Triathlon Medicine

Sergio Migliorini
Editor

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In memory of Elisa Geranio, Springer Clinical Medicine Book Editor, who gave me the idea, the suggestions and the enthusiasm to produce this book and who supported me throughout this project.

To Marisol Casado, ITU president and IOC member, for the full support that I have had as chair of the ITU Medical Committee and in particular for having improved sports science research for triathlon, including the Science+Triathlon World Conference.

To my wife, Francesca, and my daughter, Lisa, who share my passion for this fantastic sport and for understanding this another commitment of mine.

To all the ITU Medical Committee members and the ITU Sport Department, for all the efforts made over the years to improve race safety and protect triathlete health.

I would like to sincerely acknowledge all the contributing authors for their work and dedication. It was a pleasure and a privilege to work with such an exceptional group of experts.

Sergio Migliorini

Foreword

I am delighted to have been asked to write the foreword for Dr. Sergio Migliorini's comprehensive book *Triathlon Medicine*, an indispensable reference that reaches right across our sport and into all aspects of this highly technical and demanding world.

Sergio's knowledge of physical and rehabilitation medicine is second to none. It is what prompted ITU to choose him as the medical delegate for the London 2012, Rio 2016 and Tokyo 2020 Olympic Games and preside over the ITU Medical and Anti-Doping Committee and is what makes him such an authority in an endurance sport that will always test the athletes' bodies to their limits.

It has been my great honour to have been the International Triathlon Union president since 2008, during which time the sport has enjoyed huge growth and development. Working closely with Dr. Migliorini, the IOC and our National Federations around the world, ITU will always strive to uphold the Olympic values of fair play and equality as it expands into new regions, and it is thanks to the dedication of the experts who have contributed to this book, and the triathlon family as a whole, that we have been able to do so.

Today, the Olympic world is moving forward ever faster, innovating, reinventing and reaching larger audiences than ever before and bringing new generations to discover the sense of achievement, sportsmanship and belonging to a global movement in the way that only sport can do. ITU is proud to be one of the driving forces behind that momentum but is acutely aware of its responsibility towards athletes to keep them safe and healthy and understand the demands of our sport.

For the athletes, coaches, national federations, technical officials and all those who take their tri life seriously, *Triathlon Medicin*e offers precious insights from specialists in every field, with advice on injury prevention and recuperation, nutrition strategies, race day best practice and much more from those who know the sport inside out.

For everyone who thought they knew everything about this great sport, I would urge you to rethink and read on.

Marisol Casado
ITU President, IOC Member
Madrid, Spain

Preface

I am honoured with the opportunity to serve as guest editor for the book *Triathlon Medicine*.

Triathlon Medicine is the culmination of almost 30 years of experience on the field with injuries and conditions of triathletes, working with many sports medicine doctors, orthopaedics, exercise physiologists and researchers.

The cooperation with the ITU Sport Department was very useful to improve the medical assistance of the ITU races during these years, including the Olympic Games, and to continuously update the event rules to have safe races. Triathlon is the most modern of all the endurance sports, and the triathletes experience a range of environmental conditions and physiological demands, depending on the race, that must be taken into consideration when preparing for the race medical assistance.

Combining research perspective with many years of experience practicing in the field, this book offers to sport medicine physicians, orthopaedics, physical therapists, athletic trainers, coaches and officials a comprehensive guide for the evaluation, treatment and prevention of all the overuse and medical conditions and to improve athletes' performance protecting also their health. The book contains 26 chapters in which scientific and practical aspects of medicine applied to the triathlon are analysed in depth, including the ITU medical and competition rules. The book addresses in detail the topics of cardiovascular adaptations, overuse injuries, overtraining syndrome, endurance anaemia, nutrition and the physiological aspects associated with the discipline. It provides information on the training and technical aspects on the different triathlon' distances. Dedicated chapters also cover issues related to female, young, master and para-triathletes.

It was a pleasure and a privilege to work with this exceptional group of authors, many of whom are considered leaders in endurance sports medicine; they have shared with me the passion for triathlon, transmitting their invaluable practical expertise in this matter. Very special thanks also to the ITU Sport Department and ITU Medical Committee members for their work, dedication and love for this amazing sport.

Lausanne, Switzerland — Sergio Migliorini

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ITU Triathlon History

1

Gergely Markus and Antonio Arimany

Triathlon was first created in the early 1970s by the San Diego Track Club, where members were looking for an alternative workout to the rigours of track training. The club's first event consisted of a 10 km run, an 8 km cycle and a 500 m swim, but over the next decade, triathlon grew in leaps and bounds and soon gained recognition throughout the world. The first Ironman competition was held in Hawaii in 1978 and gained worldwide recognition in 1982, when Julie Moss bravely struggled to the finish line.

In the early 1980s, national governing bodies started to be formed and the sport started its dynamic spread all around the world. In 1984, the European Triathlon Union was formed by a dozen federations in the old continent, while in the same year triathlon caught the attentions of the leaders of the Olympic movement during the Games in Los Angeles.

In the years that followed, an active dialogue started among some of the early leaders of the sport such as Les McDonald and the former President of the IOC, Mr. Juan Antonio Samaranch, in order to find the best possible way to get the sport even more internationally established.

The first attempts were made to join the International Modern Pentathlon and Biathlon Federation, but because of their reluctance, in early April 1989 the International Triathlon Union (ITU) was founded at the first Congress in Avignon, France. That same city hosted the first official World Championships on August 6 later that year. ITU established its headquarters in Vancouver, Canada, before later moving to Lausanne in 2013 under the leadership of President Marisol Casado.

Triathlon was awarded full Olympic medal status in 1994 and debuted at the Sydney 2000 Olympic Games. The official distance for Olympic racing was set at a 1.5 km swim, a 40 km bike and a 10 km run—taken from existing events in each

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discipline already on the Olympic programme. There were further official distances established based on double and half of the Olympic distance, as well as on the classic Ironman distance.

Being an endurance sport, the medical aspects of triathlon have always played a significant role in its development. Various committees were quickly established in the early 1990s to advise the leadership of the organisation and to safeguard the sport on different matters, among those the medical considerations.

Triathlon has always been an inclusive sport, in which the rules were not just focused on the top elite participants, but also on the millions of amateurs—known in triathlon as Age-Groupers—for whom events are usually held together for all categories.

Since 2004, paratriathletes have also joined the World Championship program and, thanks to its rapid development since 2010, paratriathlon made its debut on the Paralympic program in 2016.

The latest achievement in this story was the inclusion of the Mixed Relay for the first time in the Tokyo 2020 program, giving many of the athletes the opportunity to perform twice during the Olympic Games.

Triathlon still has great potential to grow even further in the future, thanks to the collaborative work of all of the stakeholders within the ITU Family, as well as those connected with the sport by many other means.

Part I

Physiological and Epidemiological Aspects

Physiological Requirements of the Different Distances of Triathlon

2

Avish P. Sharma and Julien D. Périard

2.1 Introduction

Triathlon is a multisport event comprised of swim, cycle and run disciplines performed consecutively and completed over a variety of distances. Each discipline is connected by a brief transition period during which an athlete changes between disciplines (swim to cycle, cycle to run) within a specific zone, before continuing the race. Modern triathlon is thought to have commenced in San Diego, USA, in the mid-1970s and has grown in popularity ever since, with the sport making its debut in the Olympics in 2000, as well as expanding into a variety of different events. Standard triathlon distances governed by the International Triathlon Union (ITU) and regularly raced over during international competition are presented in Table 2.1.

Of these six events, the first three are "draft legal" at the elite level, indicating that athletes can shelter or "draft" in the slipstream of preceding athletes during any of the three disciplines, with the benefit most noticeable in the cycle and swim. In the "long-distance" events a set distance of at least 10–20 m must be maintained between athletes during the cycle discipline. The ITU also governs other forms or variations on the traditional "swim, bike, run" events. These include the aquathlon (2.5 km run, 1 km swim, 2.5 km run), duathlon (10 km run, 40 km cycle, 5 km run), cross-triathlon (an off-road triathlon discipline that typically takes place over a 1 km swim, 20–30 km mountain cycle and 6–10 km trail run) and winter triathlon (involves a run, mountain cycle and cross-country ski—all on snow, with distances set on the day to achieve a winning time of around 80–90 min). This chapter will

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Table 2.1 Standard (swim, bike, run) triathlon distances

Name	Swim, distance (m)	Bike, distance (km)	Run, distance (km)
Mixed-team relay (MTR)	300	7	2
Sprint distance (SD)	750	20	5
Olympic distance (OD)	1500	40	10
Long distance (LD)	4000	120	30
Half-Ironman distance (70.3)	1900	90	21.1
Ironman distance	3800	180	42.2

OD distance used during the individual event at the Olympic Games and most frequently during the ITU World Triathlon Series. *MTR* two female and two male athletes relay each other to complete an abbreviated individual triathlon—the first World Championships for this event were held in 2009, and it will debut at the Olympics in 2020. *70.3 and Ironman* Governed by the World Triathlon Corporation, these events are the most popular long-format triathlons completed by elite and non-elite competitors. The Ironman World Championships are held yearly in Hawaii

address the physiological requirements and responses associated with the different distances of triathlon, from sprint to Ironman distance.

2.2 Energy Systems Specific to Triathlon

Despite the many variations in distance evident within the sport of triathlon, the overall duration of these events (~20 min per athlete in MTR competition up to ~8–9 h for Ironman competition—Table 2.2) results in the main source of energy production originating from aerobic sources of metabolism (Fig. 2.1). Here, the synthesis of adenosine triphosphate (ATP) occurs in the mitochondria, involving the combustion of fuel in the presence of sufficient oxygen, and is the dominant source of energy production for intense exercise lasting longer than 75 s [1, 2]. The fuel can be obtained both within (i.e. free fatty acids and glycogen) and outside (e.g. blood free fatty acids, or blood glucose, from dietary ingestion/liver) the muscle [2]. However, due to a relatively low rate of ATP turnover, the aerobic system is incapable of meeting the energy demands at the beginning of exercise, irrespective of the exercise intensity, or during high-intensity exercise bouts [2]. Consequently, anaerobic (without oxygen) sources of energy are capable of responding immediately to the energy demands of intense exercise and can support extremely high muscle force application and power outputs [1, 2]. Two main anaerobic systems exist—the phosphagen or alactic system, which produces ATP through the breakdown of phosphocreatine, and the glycolytic or lactic system, involving the anaerobic breakdown of muscle glycogen or blood glucose, with lactate being produced as a by-product [2]. It is generally accepted that the phosphagen system is the dominant source of energy production for exercise lasting up to 6 s [3], though glycolysis also occurs during this period. Glycolysis reaches its maximal rate of ATP regeneration after 10–15 s of exercise, remaining at a high rate whilst aerobic metabolism increases its contribution to energy production over the next ~60 s [1, 2, 4].

Table 2.2 Fastest time split (hh:mm:ss) during each of the triathlon disciplines and overall in elite males and females from 2018 World Championship races

	Swim		Bike		Run		Total	
	Male	Female	Male	Female	Male	Female	Male	Female
Mixed relay	0:03:42	0:03:53	0:09:49	0:10:37	0:04:32	0:04:58	0:18:54	0:20:44
Sprint	0:08:54	0:09:25	0:29:10	0:31:21	0:13:59	0:15:48	0:53:24	0:58:06
Olympic	0:18:32	0:19:27	0:54:03	0:57:16	0:29:44	0:33:44	1:44:34	1:52:00
Half-Ironman	0:21:53	0:23:01	2:04:16	2:15:28	1:06:34	1:15:11	3:36:31	4:01:13
Ironman	0:46:29	0:48:14	4:09:06	4:26:07	2:41:31	2:55:22	7:52:39	8:26:18

Sprint data obtained from 2018 Hamburg World Series race as World Championship is not contested for this event. Sources: wts.triathlon.org and ironman.com

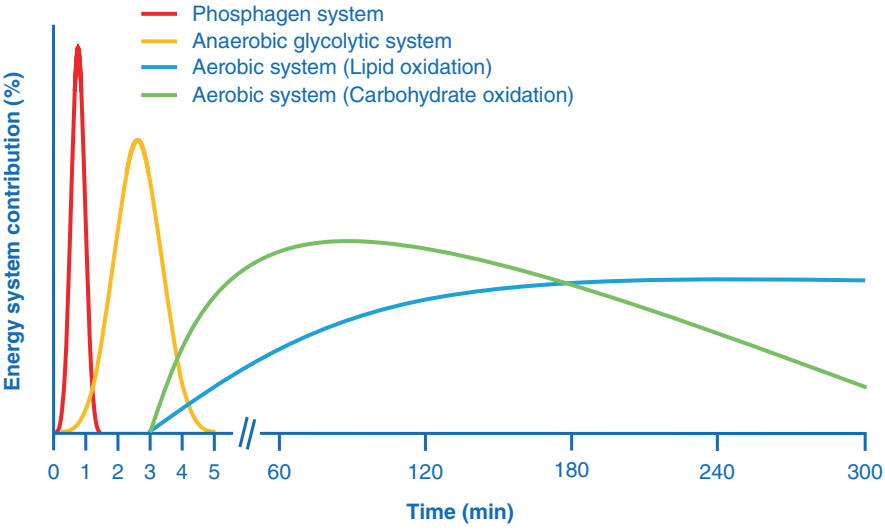

Fig. 2.1 Energy system contribution and interaction during exercise of increasing duration. The phosphagen system provides energy by hydrolysing creatine phosphate stored in the muscle, which releases a phosphate that transfers to ADP and resynthesises ATP. The anaerobic glycolytic system provides energy by breaking down carbohydrate in the form of blood glucose or muscle glycogen to resynthesise ATP. The aerobic system provides energy through mitochondrial respiration, which occurs via the oxidation of lipids (blood free fatty acids and triglycerides) and carbohydrates (blood glucose, muscle and liver glycogen)

Within a triathlon, fast starts (swim and run), intermittent short-duration spikes in power output (cycle), a stochastic intensity profile (swim, cycle and run) and resuming intense exercise after a period of cessation during transition [5–7] necessitate athletes having well-developed anaerobic qualities to meet these specific requirements. Whilst athletes focus most of their training on developing aerobic capabilities to meet the overall demands of the event, a portion of time is also dedicated to high-intensity training and speed work in the training programmes of elite athletes to develop anaerobic power and capacity [8].

2.3 Specific Aspects of Triathlon

As well as the variations in distance, it is crucial to account for event characteristics unique to triathlon that may influence the physiological demands imposed on athletes by each of the different events described above [9]. Firstly, the draft-legal nature of certain triathlon events lowers the energy cost of exercise relative to not drafting, a trait that has been reported in all three disciplines, but most prominently highlighted in the cycle portion [10]. Indeed, when cycling at a speed of 40 km h^{-1}, the trail cyclist can reduce oxygen consumption by up to 25% when following within 0.5 m of the lead cyclist [11]. When placed behind a large group (e.g. eight cyclists), the ability to draft allows for a further reduction in energy costs (~40%) [11, 12]. The lower physiological demand associated with drafting relative to cycling in isolation can manifest in a 4% improvement in subsequent running performance over 5 km in international standard male triathletes [13]. Similar to cycling, drafting when swimming can reduce the amount of frontal resistance experienced by the trail swimmer [14]. In a triathlon, drafting allows the athlete to swim at a greater speed (e.g. 3.2% over 400 m) for a given energy expenditure than swimming alone, thereby conserving energy for the subsequent cycling and running disciplines of an event [14]. In the context of a race, the lessened physiological cost of exercise associated with drafting may lead to alterations in pacing, race strategy, physiological demand and ultimately performance [5, 13–18], when compared to non-drafting events.

Secondly, the combination of swimming, cycling and running performed in sequence during a triathlon, regardless of overall duration, creates a unique physiological profile that differs from isolated exercise in any of the three disciplines. For instance, ventilation, perceived exertion and blood lactate were all elevated during 9.3 km of running preceded by a bout of Olympic distance triathlon-specific cycling (periods at low to moderate intensity interspersed with high-intensity and sprint efforts) [6], compared with no cycling [19]. Similarly, a fixed intensity bout of cycling elicited a higher physiological cost when preceded by 750 m of swimming compared to no swimming [20]. As such, it is important to account for the influence of the triathlon as a whole (e.g. fatigue development, substrate utilisation), when considering the physiological profile of each individual discipline. Furthermore, strategically manipulating performance (i.e. pace, effort) in one discipline may affect subsequent disciplines. As Vleck and colleagues [21] suggested, tactically reducing swimming effort can result in greater work in the early stages of the cycling section of an elite OD race, which may influence running performance.

Together, the unique aspects of triathlon including the open water nature of swimming, ability to draft and higher physiological cost of triathlon cycling and running relative to isolated cycling and running should be considered in planning and executing race tactics, strategies and laboratory testing [19, 22]. In addition to these factors, due to the large variations in distances, and thus exercise duration (Table 2.2), the metabolic demands and physiological responses during such races vary greatly. Over the remainder of this chapter, we will discuss the

competition demands of the different distances of triathlon, specifically mixed-relay, sprint-distance, Olympic-distance and long distance (half-Ironman and Ironman).

2.4 Draft-Legal Triathlons: Mixed-Team Relay, Sprint and Olympic Distances

2.4.1 Swimming

The MTR triathlon was included into international competition within the last decade. As such, studies describing the physical and physiological requirements of the event are relatively sparse in the literature. Importantly, the demands of competition are likely to differ substantially between each of the four "legs" of the relay. For example, the first and second relay athletes can draft more during the swim and cycle legs of the triathlon as the initial mass start allows for the athletes to remain together, whereas the third and fourth relay athletes are more likely to be racing alone or in smaller groups, as the race and athletes fraction over time. An analysis of the 2014 MTR World Championships in Hamburg, Germany, provides some insight regarding the demands of competition [23]. Swim velocities were reported as 1.30 ± 0.04 m s^{-1} for females and 1.44 ± 0.05 m s^{-1} for males [24]. These velocities are faster than similar data collected from ITU sprint and Olympic distance events, although unsurprising given the substantially shorter event distance (Table 2.1). The relative intensity (e.g. percentage of maximal aerobic velocity) to which these speeds correspond is unclear in the literature. Data from international-level male triathletes (average swim velocity of 1.29 m s^{-1} over 1500 m) indicate that a 1500 m trial completed immediately prior to an hour-long cycle was performed at 98% of maximal aerobic speed [25], suggesting that MTR and SD triathlon swimming are likely completed at or above maximal aerobic speed. However, given the characteristics of triathlon swimming (e.g. fast start and positive pacing, drafting), measures obtained from isolated swimming in a closed environment (i.e. swimming pool) may not accurately portray the physiological demands of swimming during shorter triathlon events.

Unpublished data from four Australian athletes competing in the MTR World Championships revealed positive pacing strategies were adopted during the swim. Such a strategy during the opening legs was likely due to a desire to be at the head of the swim group and avoid being disrupted (i.e. stroke mechanics, breathing, "fighting" for position) by swimming in a large group, as well as exit the water in a primary position, or during later legs of the race to catch up to leading athletes and gain the benefits of drafting on the cycle leg [13].

A high swimming position correlates strongly to high overall finish position in OD triathlon, and therefore positive pacing strategies are commonplace in this form of triathlon [5, 7, 21, 26]. For instance, during elite OD competition, swim velocity decreased from 1.39 to 1.27 m s^{-1} and 1.21–1.14 m s^{-1} over the course of 1500 m in males and females, respectively [7]. Moreover, swim speed over the first ~200 m of

the swim correlated strongly with final swim and overall race position in OD triathlon for both males and females [7]. Similar findings may be apparent in MTR and SD triathlon; however published evidence from these events is lacking. Importantly, positive pacing in swimming has been shown to elicit a greater physiological cost than even or negatively paced trials (i.e. higher blood lactate, perceived exertion) [27], eliciting a high anaerobic demand and necessitating the development of these qualities in triathletes. When considering the relative importance of the swim to overall triathlon performance, Peeling and Landers [5] highlighted the necessity of making the first pack in the swim leg to minimise the risk of expending further energy by chasing on the bike, but cautioned against the potential excessive energetic cost required in doing so. They therefore emphasised the importance to improve swimming ability such that an athlete may be capable of swimming in the first group whilst maintaining a lower energy cost, thereby increasing the likelihood of riding in the lead group of cyclists after the transition [5]. As a benchmark, Ofoghi and colleagues [26] noted that the average fastest time in ITU OD World Championship races between 2008 and 2012 was 17 min 19 s for men and 18 min 43 s for women, with medallists swimming within ~17 s of this time. Therefore, training to attain these speeds with a positive pacing strategy, whilst minimising physiological cost, would appear advantageous for performance in OD triathlon. Given the shorter duration of MTR and SD events, higher velocities in training and a greater level of anaerobic conditioning are likely required to adequately prepare for these events, though it would appear similar pacing profiles to OD triathlon exist here.

2.4.2 Cycling

Cycling has been reported to have a greater influence on individual performance in the MTR than in other triathlon events [21], perhaps given bike groups are smaller and the possibility for drafting is reduced [23]. In particular, male medallists (ranked 1–3) achieved significantly higher speeds than athletes in the other teams did during the second and fourth relay legs [23]. Mean power outputs of German athletes during the 2014 MTR World Championships have been reported as ~210 W for females and 325 W for males [23]. Unsurprisingly, mean cycling power outputs during OD triathlon are slightly lower at 250–270 W for elite males and ~180 W for elite females [24, 28].

The distribution of power output during cycling in elite OD triathlon competition was reported to be 51% in zone 1 (below first ventilatory threshold), 17% in zone 2 (between first and second ventilatory thresholds), 15% in zone 3 (between second ventilatory threshold and maximal aerobic power) and 17% performed at workloads higher than that eliciting maximal aerobic power (zone 4) [6]. The authors concluded that OD triathlon cycling requires higher aerobic and anaerobic contribution than constant workload cycling exercises classically analysed in laboratory settings (e.g. time trial) or Ironman triathlons [6], where the intensity profile is far less stochastic [29]. Similarly, Etxebarria and colleagues [28] observed elite male

triathletes recorded on average 34 peaks of power output above 600 W and ~18% of total time spent at or above maximal aerobic power during the cycle leg of ITU OD competition (~60 min). Naturally, these findings depend on the route and geographical profile of the cycling course, but provide a good indication of the demands of OD triathlon racing.

Observations of elite Australian athletes competing at the MTR World Championships indicate a similar level of high-intensity efforts during cycling (unpublished). The two male triathletes recorded 11 and 12 peaks of power output above 650 W during ~10.5 min of cycling, with time spent above 85% of their 4 min maximal average power output as 48% and 62% (relay positions 2 and 4 in the team, respectively). In the females (relay positions 1 and 3), 17 and 8 peaks of power output above 8 W kg^{-1} (~400 W) during ~11.5 min of cycling were recorded, respectively, with time spent above 85% of their 4 min maximal average power output recorded as 58% and 64%, respectively. The difference in the number of high-intensity efforts in females may be explained by race situation, with athlete 3 chasing a lead pack for the entirety of the ride, thus adopting a more even "time-trial" pacing approach, whereas athlete 1 chased initially, then sat within a lead pack, alternating between leading and drafting. The MTR thus highlights the divergent physiological requirements within the same events, necessitating well-rounded athletes capable of managing different racing situations that require particular physiological responses.

2.4.3 Running

The run leg is frequently viewed as critical to performance in draft-legal triathlons, with several studies reporting strong correlations between running speed and overall position (i.e. higher running speed with better overall ranking) [21, 24]. In MTR triathlon, running speeds were reported as 4.91 ± 0.17 m s^{-1} for females and 5.60 ± 0.23 m s^{-1} for males [23]. Additionally, significant differences were found between male medallists and remaining athletes on the final leg (5.98 ± 0.13 m s^{-1} vs. 5.57 ± 0.26 m s^{-1}), replicating the discriminatory nature of running in deciding overall performance, as observed during OD triathlon [21]. Interestingly, whilst these speeds are higher than those reported for OD triathlon [7, 24], they are similar to those observed during SD triathlon, despite the shorter duration [23]. Limited potential for drafting during the swim and cycling leg during MTR (given the likelihood of smaller groups forming as the race breaks up) leads to a higher energy expenditure compared to when drafting occurs [10], which could explain the similar average running speeds observed [23]. Unpublished data from elite Australian athletes indicate a fast-starting pace during the MTR, which suggests that the ability to tolerate the physiological cost of a fast start during both swimming and running, as well as resist the additional fatigue associated with not being able to draft, may be important qualities for success in MTR triathlon.

In OD competition, Vleck and colleagues [21] reported that the top 50% of finishers completed the run course significantly faster than the bottom 50% (5.24 m s^{-1}

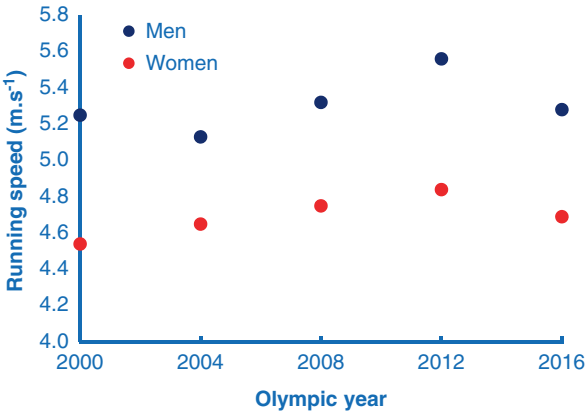

Fig. 2.2 Mean running speed of the top ten finishers for men and women in the Olympic Games where triathlon has featured. Races were conducted over the Olympic distance with athletes completing a 10 km run. Publicly available data were obtained from www.triathlon.org/results

vs. 4.98 m s^{-1} respectively). Velocities in elite men over OD triathlon have been reported to range between 5.0 and 5.3 m s^{-1} over the course of run, with corresponding values in women of 4.3–4.6 m s^{-1} [7]. Interestingly, mean running speed of the top ten finishers from each of the five Olympic Games where triathlon has featured reveals running speeds of 4.5–4.9 m s^{-1} for women and 5.1–5.6 m s^{-1} for men (Fig. 2.2). The data trend towards faster running speeds in both men and women since 2004, though this pattern was reversed during the 2016 Olympic Games, likely due to a combination of hot and humid weather in Rio de Janeiro, as well as course profile. Le Meur and colleagues [24] have reported that the run phase of a World Cup OD triathlon is completed at 93–94% of maximum heart rate for both males and females. It has also been observed that in highly trained male triathletes (maximal oxygen uptake: VO_{2max} = 69 mL kg^{-1} min^{-1}), completing a 10 km run with a fast start during a simulated OD triathlon yielded relative intensities of 96%, 80% and 80% of VO_{2max} at the start, beginning and finish of the run, respectively [30].

A positive pacing strategy is frequently observed during the run leg of elite OD triathlons in both males and females [7, 24, 31], with the progressive reduction in velocity over these distances likely due to metabolite accumulation and associated fatigue [18]. Interestingly however, studies have revealed that adopting a slower pace during the initial stages of a run (e.g. even or negative pacing strategy) of an OD triathlon leads to faster overall running performance [30, 31]. This approach may not be observed often during OD racing as athletes tactically exit the transition area quickly to establish or reduce gaps between themselves. Conversely, Taylor and Smith [32] demonstrated a positive pacing strategy improved 5 km run performance following a simulated SD swim and cycle at fixed intensity, compared to negative and even pacing. This approach, however, also yielded the highest physiological strain. The authors suggested any benefit of an initially aggressive running pace is unique to the shorter SD triathlon format, given that a 10 km run characteristic of OD triathlons provided a relatively greater remaining distance and lower increase in speed to overcome a conservative pacing strategy [32]. Additionally, a faster start has been shown to improve performance over middle-distance events by

speeding up oxygen uptake kinetics [18], perhaps explaining its suitability to SD and MTR triathlon, though it must be noted that oxygen kinetics would already be "engaged" during the run leg, due to prior swimming and cycling. However, the faster start inherent of a positive pacing strategy may also lead to suboptimal distribution of energy resources, potentially leading an athlete to earlier glycogen debt, which would impair performance over longer duration events such as OD triathlon [18]. OD triathlon is likely to induce greater glycogen depletion, neuromuscular fatigue and increases in core temperature than SD or MTR triathlon, due to the longer duration [18, 33]. At the speeds run by elite male triathletes (5.6 m s^{-1}), drafting may have some benefit on oxygen consumption and therefore performance; as such triathletes may want to adopt a faster start to keep up with leading runners. Additionally, triathletes present in the front group and thus in contention for the victory could have a psychological advantage over chasing athletes and therefore perform better [34]. However, balancing the benefits of drafting against the physiological cost of a faster start would be a key consideration, with the potential for specific athletes (i.e. those with fast oxygen uptake kinetics or higher anaerobic tolerance qualities) to target an aggressive pacing strategy.

2.5 Non-drafting Triathlons: Long Distance, Half-Ironman and Ironman Distances

Given that the energy contribution from the immediate (phosphagen) and short-term (glycolytic) energy systems is considered relatively insignificant when examining events of prolonged duration (e.g. half or full Ironman), performance in long-distance triathlon is mostly dependent on the rate and efficiency of aerobic ATP resynthesis [35]. As protein oxidation is relatively small during exercise, carbohydrate and lipid oxidation rates become a main determinant of success in ultra-endurance triathlon racing [35]. Given that humans have a greater capacity for storage of fats than carbohydrates (in the form of glycogen), there is a progressively greater use of lipids as fuel over the duration of the event (up to 80% of the total caloric expenditure) [36, 37]. Indeed, rates of maximal fat oxidation have been shown to correlate with Ironman performance, with the fastest athletes (finishing time of 9 h) displaying the highest rates [38]. Therefore, increasing this ability through training or nutritional strategies would seem appropriate to maximise performance over long-distance events. However, a recent review [39] suggested that for elite competitors (finishing time of 8 to 9 h), a hybrid approach consisting of training to increase fat oxidation combined with increasing endogenous (loading pre-race) and exogenous (during competition) carbohydrate stores be adopted, given that the energy demands of the event (17–20 kcal min^{-1} for women and men, respectively) are unlikely to be met from even the highest rates of fat oxidation alone.

Competition data from elite athletes regarding exercise intensity during long-distance triathlon events is limited. Similar to their OD counterparts, elite long-distance triathletes possess a high VO_{2max} (>70 mL kg^{-1} min^{-1} for male

competitors), as well as equivalent values for peak power output and ventilatory thresholds, despite completing a greater volume of training [40]. In well-trained triathletes, heart rate during the cycle and run phases of LD triathlons were significantly less than the second ventilatory threshold identified from incremental exercise testing, however not significantly different from the first ventilatory threshold [41, 42]. Field-based research has shown that well-trained triathletes perform the cycling leg of an Ironman triathlon at 80–83% of maximum heart rate [42] and 55% of peak power output [29]. Additionally, in highly trained triathletes (VO_{2max} = 67 mL kg^{-1} min^{-1}), swim velocity during an Ironman race equated to swim velocity at the first ventilatory threshold and was performed at a relative intensity equating to 92% of maximum heart rate and estimated VO_{2max} [43]. In contrast to draft-legal triathlons, swimming time has been reported to have a weak correlation with overall Ironman performance [42–44], likely due to its small contribution to overall race time (8–10%). Meanwhile, cycling and running exhibit much stronger correlations with overall performance (e.g. R = 0.90 in highly trained triathletes) [43].

Exercise intensity during an Ironman tends to progressively decrease over the course of the event. Heart rate, power output and speed have been observed to decline over the course of cycle and run portions of an Ironman triathlon in well-trained athletes [29, 42]. The progressive reduction in absolute exercise intensity may be the result of increased glycogen depletion resulting in altered substrate utilisation and neuromuscular fatigue [44]. Thus, the ability to resist fatigue may play a significant role in the regulation of pace during ultra-endurance events. It has been suggested that minimal variations in speed/power output (i.e. adopting an even pacing strategy) may assist in maintaining an even distribution of energetic resources and thus may be a more optimal strategy for longer triathlon events [18].

2.6 Conclusion

The physiological requirements of the different distances of triathlon are varying and largely dependent on a combination of event duration, as well as the specific aspects of competition. At the elite level, draft-legal (MTR, SD and OD) triathlon events are characterised by positive pacing strategies during the swim and run disciplines and a stochastic intensity profile during cycling whereby longer periods of low-intensity cycling are interspersed with bouts of high-intensity efforts and sprinting. Given the duration of ~18 to 120 min, these events are predominantly aerobic in nature, with elite triathletes reporting similarly high VO_{2max} values during running and cycling as their single sport counterparts [45]. However, the frequent high-intensity bursts observed across all three disciplines, as well as the positive pacing strategies often characteristic of the medal winning performers, necessitate well-developed anaerobic qualities in elite triathletes. Moreover, these high-intensity efforts are predominantly fuelled through aerobic or anaerobic glycolysis, thus necessitating sufficient carbohydrate intake [22, 46, 47]. Alternatively, the extended duration of half-Ironman (3–4 h) and Ironman (8–9 h) competition at the elite level,

along with the non-drafting nature of these events, elicits a different set of physiological requirements. Exercise intensity across disciplines is far more submaximal in nature compared to shorter-distance events and characterised by a far more even pacing profile. Therefore, aerobic metabolism is almost solely responsible for energy production during these events, and the role of fat oxidation is considerable. In summary, considerable differences emerge across the spectrum of competitive triathlon events. The variety of event-specific demands and physiological requirements of triathlon should be therefore considered when planning and executing race tactics, developing training strategies, performing laboratory testing and conducting talent identification.

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Epidemiological Aspects of Illness and Injury

3

Veronica Vleck and David Hoeden

3.1 Introduction and Definitions

This chapter extends on previous reviews of both the extent of, and the risk factors for, injury and illness in triathlon [1–4]. The reader is asked to consider [3] and our 2014 review in "Sports Medicine" [1] (the most recent and detailed of said documents) as an adjunct to this chapter. Although several papers [5–32] have been published since [1], they are mostly case reports. References [1] and [3] include the following summary tables of the literature:

(a) The timeline of immunological, oxidative and cardiovascular responses to competition [1];
(b) The proportion of athletes affected overall or by either traumatic, overuse or temperature-/fluid-related injury [1, 3];
(c) A percent comparison of anatomical location of injury [1, 3];
(d) Percent comparison of injury types (e.g. contusions/abrasions vs. fractures) [1, 3];
(e) Injuries or illnesses that have only been reported in case studies and for which assessment has not necessarily been conducted in any of the population or group-specific studies in the triathlon injury literature to date [1];
(f) The extent to which injury sustained within triathlon training and/or racing hinders activity, leads to seeking of professional help and/or recurs [1, 3];
(g) Finally, the results of studies examining the putative relationship between selected intrinsic and extrinsic risk factors and injury occurrence [3].

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We also refer the reader to Gosling, Gabbe and Forbes' excellent 2008 discussion [4] of the extent to which the research into musculoskeletal injuries in triathletes, in particular, has addressed each stage of the "Translating Research into Injury Prevention Practice" (TRIPP) framework—as most of the points they make within it still apply.

Given the continuing dearth of information on the extent of maladaptation that exists in youth and para-triathletes [3], we have drawn on both our unpublished data and the academic literature to synthesize an updated snapshot of current knowledge as regards the extent and risk factors for maladaptation in able-bodied road-based triathlon. Recommendations for research are also provided. We clearly differentiate between those (minority) data that have been collected by or in conjunction with clinicians and that which have not. Few triathlon-specific data can easily be classified according to the Orchard Sports Injury Classification System (OSICS) or similar and even less according to the Subsequent Injury Categorization (SIC) model [33, 34]. Unless otherwise stated, the non-clinical data that we report were all obtained using the *same* injury definition, i.e. that which results in either cessation or modification of training for at least 1 day and/or a visit to a clinician. In this way, we were able to consolidate the results of multiple studies that were conducted by ourselves. Four of these studies—consecutive 5-year [35, 36] retrospective pilot and 1-month retrospective proof of pilot studies (the race-based section of which was conducted at the same time as a further medical tent-based study at the same event [37]) and a 7-month prospective training diary-based study that involved predominantly short-distance specialists [38–40] – followed directly on from, and built on the results of, each other. We also present unpublished data from what we believe to be the largest retrospective study thus far conducted on injury and pain in long-distance athletes, and their associated risk factors, since they started regular triathlon training [41] (Table 3.1).

3.2 The Extent to Which Injury/Illness Occurs

Values for the incidence of catastrophic injury are not available for triathlon training. However, Harris et al. [19, 43, 44] have estimated the equivalent values for catastrophic injury whilst racing at 1.5 deaths per 100,000 race participations. This value exceeds that of marathon running but on the whole triathlon training and competition is generally considered safe for the well-prepared athlete who both performs within his or her individual limits and respects the technical guidelines that are in force. As for less severe injury, widely varying values for the extent to which it occurs have been reported in the academic literature [1, 3]. The possible reasons behind such variation have been discussed by [4]. They include the use of heterogeneous study samples and inconsistencies across studies in the definition of injury. Detailed examples of the confusion that the inconsistencies in injury definition across studies can cause are provided in [41]. The variations in prevalence thus far reported may also be a function of how far back the study participants have been asked to recall their injuries for. Egermann et al. [45] and Hoeden [41] found 74.8%

Table 3.1 Characteristics of the interlinked studies the data from which are presented in this chapter

Design	Sample	Administration	Duration/recall	n		% starters (*n*/population)	Sample subgroups
Retrospective online survey [41]	Ironman mailing list	July 2011 to February 2012	Since starting regular triathlon training	1159		3.9 (1159/30,000)	990 M
							169 F
Retrospective questionnaire [35, 36, 40]	British National Squad	1993, hard training week without taper close to their respective National OD or IR Championships	5 years	164	77	54 (7/13)	ND elite F OD
						52 (11/21)	ND SE F OD
						78 (7/9)	ND elite F IR
						75 (12/16)	ND elite M OD
						92 (22/24)	ND SE OD
						95 (18/19)	ND elite M IR
	British North East Region Clubs				87	? (87/?)	ND club M OD
Online retrospective survey of both training and racing [42]	Competitors, ITU age group Sprint and OD World Championships, Pruhealth World Triathlon Grand Final	Open 3 days post, completed within 1 month of 2013 event	1 month	660			
Prospective observational race medical tent-based study [37]		Ongoing throughout event, September 2013	N/A	182	32	8.4 (32/381)	Elite DL, Pruhealth World Triathlon Grand Final
					125	3.1 (125/3995)	ND ITU World age group Sprint and OD Championships
					25	1 (25/2465)	ND open race
Prospective longitudinal training diary-based study [1, 38–40]	British and Scottish National Squad OD athletes	February–July 1994	Filled in daily	51		72 (51/71)	British Senior OD National Squad

AG age groupers, *Club* club-level athletes, *Comp* competitive age groupers, *DL* draft legal, *F* female, *IR* Ironman, *M* male, *NA* not applicable, *ND* non-drafting, *OD* Olympic distance, *SE* sub-elite

and 71.6% of Ironman athletes, respectively, to have incurred at least one injury "during their active time in triathlon" and "since starting regular triathlon training". Shaw et al. [46] reported 62% to be affected over the course of one season and [47] noted that 37% of their group were injured within a two-month period. Overall, 15.3% and 26.2% of our 2013 group, respectively, reported themselves as injured "within the last month" and "right now" (post a key race).

A consistent finding across the studies to date is that more triathletes sustain so-called overuse injuries (without a specific, identifiable cause) than are affected by traumatic injuries, i.e. those injuries that are caused by a hazard encounter such as falling and/or either being hit by or hitting an obstacle. Only one (retrospective) study has thus far compared the actual prevalence of overuse and traumatic injuries in male vs. female (1993 level), elite vs. national squad development-level vs. club-level athletes and short- vs. long-distance specialists, using the same methodology and injury definitions throughout [35, 36, 40]. Overuse injury and traumatic injury were reported by 56.3–75% of athletes and 29–50% of athletes, respectively. The difference in the extent to which the athletes in each group reported themselves to have sustained overuse and traumatic injury was 7–42%. The results implied that neither the relative proportion of athletes affected, the relative proportion of all injuries, nor the number of injuries accounted for by overuse and traumatic injury is significantly affected by ability-level, gender or the race distance towards which the athletes' training is focused, but the study sample sizes were small. Our unpublished data from our one-month online questionnaire-based retrospective study, conducted immediately post the 2013 ITU World elite and age group (short-distance) championships, also suggest that the proportion of athletes who have (self-reported, mainly training-related) injury does not markedly differ across the majority of triathlete age groups. In all of the age groups from 20–24 to 75–79 years of age, injury was self-reported by below 50% of those athletes who took part in the research. The extent to which illness leading to cessation or modification of training or racing was experienced during the month preceding completion of the survey, which was 11.7% of those 197 athletes who responded to this question overall, may, however, change with age. As the response rate to the illness section of said retrospective questionnaire was less than that for the injury section, however, said finding needs to be confirmed.

As for competition-related injury and/or illness, it is logical that both the extent to which it occurs and its distribution should possess similarities but also differ in some ways from training-related injury. The cycle and run sections of competition take place on closed roads, which is uncommon in the case of training, so decreasing the risk of vehicle-related hazard(s). The level of risk of crowd-related injury(ies) both within the swim and the cycle disciplines of the triathlon, however, is likely higher, even within those longer-distance races that have recently introduced rolling swim starts. Our prospective observational medical tent-based data, collected within the (same) ITU 2013 World (world-ranked) elite and age group world short-distance championships [37] at which the one-month retrospective online survey was administered, indicate that the extent of event-based presentation for medical assistance can vary with sex. The overall casualty rate was higher in females than it was in

males (at 3.9% of 2095 vs. 2.5% of 4008 starters). This same finding was maintained across different (team relay, sprint and Olympic distance) race formats—except in the case of an "open" race, held at the same location and during the same weekend at the ITU World Championships, for which no ability-related prequalification was necessary. Race-based requirement for medical assistance also varied with athlete ability level. Over the same weekend, 8.4% of (381) elite (draft legal race) starters, 3.1% of (3995) age group (non-drafting race) starters and 1.0% (of 2465) "open (non-drafting) race" starters were seen to by the on-site medical staff.

Illness was not classified as such within the medical report forms. However, in all the age, sex, race format and (wholly short-distance) race distance groups that were assessed, far more athletes presented with topical problems (which included issues such as exercise-induced asthma and heat-related illness such as hypothermia)—than required medical assistance for constitutional issues (such as abrasions, blisters and chafes), or for musculoskeletal issues (such as joint pain/strain and fractures). The proportion of athletes who presented with topical issues was higher for Sprint than for Olympic distance competition (at 76.4% of 72 vs. 47.2% of 110 presentations, respectively). It was also higher for age group than it was for elite competitors. Our 1994 prospective training data study [40]—which a decreasing number of the original (71 top national-level competitive) triathletes of both genders completed per consecutive month—also assessed both injury and illness. The illness reports were in this case confined to viral-, gastric- or infection-related episodes (i.e. the kinds of problems which are more expected to occur within training and are amenable to self-report) and cannot therefore be directly compared to our clinical race data. The data so collected, nonetheless, suggest that in triathletes, (mainly training-related) self-reported illness incidence may also generally exceed self-reported injury incidence. The athletes reported themselves to be affected by illness without concomitant injury but coinciding with a performance decrease and by injury occurring without illness but with a coincident performance decrease 0.21–0.31 vs. 0.00–0.06 times per 10,000 calculated [40] training units, respectively.

3.3 Where Does Injury/Illness Occur?

Table 3.2 provides rankings, in terms of the proportions of athletes affected, for those training-related retrospective studies that used the same or similar definition of injury, of the anatomical sites that have been reported to affect the largest proportion of athletes. This way of showing the data may to some extent circumvent the problem that the actual values from each study might partly be different because the studies report the proportions of athletes who are affected over different periods of time. The sites that are ranked 1 are those that affected most athletes. It is clear from Table 3.2 that, overall, more athletes are affected by (mostly training-related) injury to the lower back, knee, calf, Achilles tendon and shoulder than to other anatomical locations.

Table 3.2 Ranking of anatomical sites affected by injury, expressed as the percentage of athletes affected, by study

Recall	Since starting regular triathlon training [41]		5 years [35, 36, 40] (OU injuries only)					1 month (all)	
Group	IM M	IM F	IR M	IR F	(*E* + SE) OD M	(*E* + SE) OD F	Club M	OD M	OD F
1	Knee (32.1)	Knee (34.5)	Knee (44)	Ant th (50)	Knee (44.9)	Knee (31.3)	Knee (40.7)	Knee, calf (15.1 each)	Knee (14.9)
2	Ach (21.8)	Foot (23.2)	Calf, ham, lower back (20 each)	Knee, ham, lower back, ach, neck, ank (16.7 each)	Lower back (34.5)	Ach, other (25 each)	Lower back (30.2)		Sh (11.9)
3	Sh (17.6)	Hip (19.6)			Ach (31)	Calf (18.8)	Calf (22.1)	Ach (9.4)	Hip (9.0)
4	Foot (13)	Sh (16.7)			Calf, sh, ham (17.2)		Ach (19.8)	Lower back (6.6)	Ribs, ant th (6.0)
5	Lower leg (12.9)	Ach (16.0)	Ach (8.9)			Lower back, sh (12.5 each)	Sh (17.4)	Hip, sh (5.7 each)	
6	Ank (11.3)	Ank (14.2)	Ant th, other, sh (8 each)				Ham (14.0)		Sole, ach, butt, other (4.5 each)
7	Lumbar (10.1)	Lumbar (12.5)			Ank, other (7 each)	Ank, ant th, ham, upper back (6.3 each)	Neck, comb	Ankle, other (4.7 each)	
8	Hip (10)	Toe (10.2)							
9	Thigh (8.3)	Lower leg (10.1)	Neck, upper back (9 each)		Neck (6.9)		Other	Upper arm, multiple sites (3.8 each)	
10	Cervical sp, toe (6.4)	Other					Ant th (4.8)		Upper arm (3.0)

Ach Achilles tendon, *Ank* ankle, *Ant th* anterior thigh, *butt* buttocks, *comb* combination of sites, *E* elite, *F* female, *ham* hamstrings, *IM* Ironman, *M* male, *OD* Olympic distance, *OU* overuse, *Sh* shoulder, *sole* sole of foot, *sp* spine

Table 3.3 Incidence of self-assessed injury to specific anatomical sites, per 1000 training hours, in athletes training mainly for Olympic distance competition [40]

Site	Incidence (each)	Site	Incidence (each)
Lower back, posterior thigh	2.33	Collar bone	0.85
Posterior thigh	2.33	Neck, buttocks, abdomen, shoulder	0.85
Knee	1.9	Hip	0.63
Inside of knee	1.69	Groin, ankle	0.42
Calf	1.48	Achilles tendon	–
Shin	1.27	Head, ear, elbow, fingers, inner thigh, bridge of foot, wrist, arch of foot	0.21
Upper arm	1.05	Sole of foot, eyes, trunk, ribs, forearm, heel	Less than 0.01

Our 7-month prospective training diary-based survey [40], which involved a mixed gender group of athletes who were training mainly for Olympic distance competition, generated the following values for new injury to specific anatomical sites per 1000 combined training and racing hours: knee 3.59 (of which 1.9 was for knee and 1.69 for "inside of knee" injury), lower back 2.33, posterior thigh 2.33, calf 1.69 and shoulder 0.85 (Table 3.3).

Said data are the most accurate of those which relate to the incidence of (mostly training-related) injury to different anatomical sites in triathletes of which we are aware. They do not allow for comparative analysis of where injury occurs as a function of ability-level, sex or distance specialization. The (1994 National Squad) athletes in question were of a relatively homogenous ability level and were all focusing their training on preparation for their (at that time, non-drafting) National and then European Olympic distance Championships. No gender-based analysis of their data has been carried out to date.

Thus, the premise that the relative proportion of (predominantly training-related) injuries that occur to each one of said and other anatomical sites may differ between males and females [40], between elites and age groupers and between short- and long-distance specialists [35] is, to date, based purely on the results of the retrospective studies that have implemented the same or similar injury definitions. The data supporting the assertion that the anatomical distribution of injury varies with ability level are few as far as short-distance athletes are concerned. It may be that this finding only holds as regards elite- vs. age group-level triathletes, as opposed to those athletes of differing ability levels who are within the same age group(s). It has been reported that Achilles tendon injury may be more prevalent in National Squad-level athletes [36, 46, 48] than it is in non-elite short-distance athletes, for example—a finding that is based (and necessarily so, because it relates to top-level athletes) on small sample sizes. We found the anatomical distribution of injury, over the same five-year recall period, to differ between [12] Olympic distance and [18] long-distance triathletes, with fewer Ironman distance than Olympic distance athletes incurring Achilles tendon injury (12.0% vs. 23.9%, $p < 0.01$) [35]. However, both groups were predominantly racing non-drafting events, the sample sizes and the

recall period were (again) small [35], and the data were collected in 1993. No direct comparison of injury occurrence as a function of the event distance towards which the athletes' training is geared, using the same recall period in each case, has since taken place. In Hoeden's 2011–2012 survey of the injury incurred by 1159 Ironman distance athletes over their triathlon career [41], 10.9% of survey participants recorded themselves to have incurred Achilles tendon injury once, and 10.1% reported recurrent Achilles tendon injury. Of 660 short-distance triathletes, asked within the competition period of 2013 to recall their injury occurrence over the preceding month, 7.7% (9.4% of males and 4.5% of females) reported themselves to have sustained Achilles tendon injury. The true extent to which the anatomical distribution of injury may differ between athletes focusing on short- vs. long-distance events, the training intensity distribution of which athletes likely significantly differ [3, 35], is not yet, therefore, clear.

3.4 When Does Injury Occur?

Our 1-month retrospective study (n = 660) indicated that training is when most of the injury to the lower back, knee, calf, Achilles tendon and shoulder, i.e. likely the most commonly affected anatomical locations overall, occurs. Only 51 athletes reported race-related injury in our 1-month retrospective survey, for which the anatomical locations that affected most athletes were the knee (11.8% plus 25% for "inside of the knee"), calf (11.8%), Achilles tendon (15.7%) and "multiple sites" at the same time (11.8%). Shoulder injury was only reported by 2%. True comparative injury incidence data for the various anatomical sites—couched in terms of incidence rate differences per 1000 training hours vs. per 1000 racing hours [49]—are not, however, yet available. Most mainly training-related injury, with the exception of lower back injury, is usually attributed to either overuse or (less commonly) to overexertion. Both our 1-month and our 5-year retrospective studies found that most athletes blamed their shoulder injury on swimming—a finding that was not echoed by the results of our prospective study. There, shoulder injury was most often attributed to weight training. In our 1-month retrospective study, lower back injury was not overwhelmingly attributed to one discipline in particular. Knee injury, calf injury and Achilles tendon injury were consistently mostly attributed to running—a consistent finding across our 5-year and 1-month retrospective studies. Indeed, running was, as reported by most of the literature [1], the exercise mode that to which most (33% in this case) injuries, including those that were recurring injuries, were attributed by our prospective survey.

As for racing injuries, according to our 1-month retrospective survey, most of these were traumatic in origin (i.e. due to collisions, falls, slips or trips). Thirty two percent, 20% and 8%, respectively, of race injuries were apportioned, by the athletes who participated in our 1-month retrospective survey to "other issues", to overuse and to overexertion. The results of the one-month retrospective study revealed that the majority of race-related traumatic injuries occurred whilst athletes were cycling, followed by when they were running. The oft most cited contributing

factors to said injury (out of a choice of overuse, overexertion, collisions, falls, slips or trips, being hit by other athlete(s) or other issues), for each segment of the event, were as follows: for swimming, other issues (66.7% of cases); for the swim-to-bike transition (T1), slips or trips (50%); for cycling, collisions, falls or slips or trips (72.5% of cases, to which weather and surface were considered major contributing factors in 45.5% and 40.9% of cases, respectively); for the bike-run transition (T2), collisions, slips or trips or other issues in equal measure (33.3% each); and for running, overuse (in 46.7% of cases). These data broadly agree with prospective observational medical tent-based data that were collected at the same event [37]. The majority of injuries were attributed to constitutional issues in the case of swimming (70% of ten cases, of which 40% and 30% were due to chest problems and hypothermia), to topical issues (82.1% of 117 cases, of which 81.2% were abrasions) in the case of cycling, to topical and musculoskeletal issues in the case of the run section (50 and 45.5% of 22 cases, respectively—of which 45.5% were strain related and 22.7% each were blisters or abrasions) and to constitutional issues (53.6% of 69 presentations, of which 21.7% were chest related) as regards presentations at the race finish, respectively.

According to our 1-month retrospective study of injury at the same event, those injuries that, within the bike section of competition, were attributed to slipping or tripping (50% of cases), collision with a fixed object (40%), falling or being hit by another athlete (5% each) accounted for the majority (here 80%) of the traumatic injuries that were sustained within the event as a whole, respectively. Injury to multiple anatomical sites, to the knee, the elbow and to the hip was usually the result, accounting for in 25%, 20%, 10% and 10% of such cases. Seventy percent of the overuse injuries that were sustained within the race (which were to the knee, calf, foot, Achilles tendon and shoulder) occurred within the run segment. The Achilles tendon was the most affected site (at 30% of self-reported overuse injuries) whilst running. As for self-reported illness, only 14 self-reports were obtained from the athletes, and these were not the same athletes who entered the medical tent. Exhaustion, cold/shivering and breathlessness accounted for 7.1% each, wheezing for 21.4%, gastrointestinal cramps for 14.1% and "other issues" for 35.7% of said athlete self-reports. Of the 48 medical tent attendances for constitutional issues (representing 0.78% of the 6103 event starters), 20.8%, 41.7% and 18.8% of instances, respectively, were ascribed to exhaustion, to chest-related issues and to hypothermia. In the event in question, temperatures dropped by 15 °C over the course of 1 day—with the age group swim duly being shortened—and scattered heavy showers (coinciding with large ingresses into the medical tent of athletes with cycle fall-related injuries) also occurred. Very few athletes needed medical attention for cycle fall-related injuries in the subsequent year of the event—which took place at the same locale and over the same course but experienced fine, dry weather. Neither our unpublished data nor that which is contained within the literature [50] are sufficient to support the conjecture, but we suggest that the influence of the prevailing weather conditions—and particularly as regards whether it is raining or not—on race injury incidence (and especially on that sustained whilst cycling) may be far greater than has as yet been quantified.

3.5 What Are the Injury Outcomes?

According to [41], the percentage of long-distance athletes who were affected by swimming injury since they started regular triathlon training was 18.7% of the 990 males and 21.4% of the 169 females who took part in said study. Overall, 84.9% of males' and 76.5% of females' shoulder-related injuries that were incurred within swimming were overuse injuries. The most common such overuse injury was reported to be shoulder injury. Less than 2.5% each of athletes incurred cervical spine, elbow (joint) or upper arm injury. Of those overuse injuries that were sustained to the shoulder area, most were reported as being at the tendon (47.9% in males vs. 54.2% in females) or muscle level (at 38.7% vs. 50.0%). Injury to the joint capsule accounted for 14.8% and 12.5% and to the tendon sheath for 14.1% and 12.5% of male and female swimming-related overuse injuries to the shoulder, respectively. The proportions of swimming-related overuse injuries to the shoulder that were reported by the athletes to be sustained to the sub-acromial bursa, ligament or cartilage were 9.9%, 9.9% and 2.8%, respectively, in males, and 12.5%, 4.2% and 4.2%, respectively, in females.

In cycling, within the same study, there were no significant sex differences as regards the most common traumatic injuries that were sustained. The traumatic injuries that affected the largest proportions of (male and female) athletes were to the shoulder area (19.5–30.1% of which were to the clavicle) and to the hands (13.1–36.8% of which were to the wrist). As for atraumatic cycling-related injuries, these were largely located in the area of the lumbar spine (36.4% overall), the cervical spine (29.3%), the genitals (18.5%) and the knee(s) (6.9% of the total number of athletes). They affected similar proportions of males and females—except in the case of cycling-related genital area injury, which more women reported than men (31.5% vs. 16.3%). Similar proportions of males and females had first reported themselves to have incurred such injury—without specifying whether it was whilst swimming, cycling, running or weight training, over the course of their triathlon career, however.

As for atraumatic running-related injuries, the largest proportions of athletes were affected, in decreasing order: achillodynia (34.3% overall of athletes), "runner's knee" (28.5%), iliotibial band friction syndrome (ITBFS, 28.3%), shin splints (17%), plantar fasciitis (14.0%), lumbar spine injury (12.4%) and stress fractures (3.5%).

According to our 1-month retrospective study, most of the injuries that were sustained to the most commonly affected anatomical sites in triathletes, i.e. to the knee, Achilles tendon, calf, shoulder and lower back, were non-severe in nature. The most common initial treatment for such injuries was usually rest, ice, compression and elevation (RICE), or stretching—this respectively being the case for 57.7% and 34.6% of knee injuries, 30.8% and 46.2% of Achilles tendon injuries and 44.4% and 50% of calf injuries. The injury was classed as minor (necessitating less than 1–4 days of training cessation or modification) in 61.4% of cases of knee injuries, with running training subsequently being stopped in 57.7% of cases. Running was the most affected of the triathlon disciplines in the case of Achilles tendon and calf

injury. In most cases swim training (in five intensity zones of which zone 5 was the highest intensity level [38, 40]) was relatively unaffected, and in some cases cycle training in the lower intensity zones was increased. When the athletes sustained shoulder injuries and lower back injuries, weight training was most affected. We would consider insufficient data have as yet been collected to allow for a valid comparative assessment of the severity of injury to different anatomical sites in triathletes, however. According to our first retrospective study, injury to the Achilles tendon, shoulder, calf, knee, lower back and hamstrings accounted for most days off training, by a total of 164 competitive athletes, over the 5-year period in question. Calf, knee, lower back and posterior thigh injury accounted for the most number of injury days within our 7-month prospective study. Nonetheless, although athletes who sustained groin and inner thigh injury were not affected by injury to these sites for as many days, more of such days coincided with self-reported performance drop(s) than was the case for other sites. No index as yet exists of the extent to which the severity of the initial instance of injury to a specific anatomical location—couched in terms of the extent to which the athlete subsequently modifies his or her training within the process of rehabilitation process from said injury—may be counterbalanced by its tendency to recur.

As for the extent to which athletes seek professional help for their injuries or illness, we mainly obtained such information from our prospective observational race medical tent-based study [37], with less detail on this topic being available from our training-related datasets. In the former investigation, 100%, 73.6% and 95.8% of the athletes seeking medical attention for topical, musculoskeletal and constitutional issues, respectively, were discharged within 2 h. The proportions of musculoskeletal injuries for which the athletes were hospitalized immediately, within 2 h and after 2 h were 13.2%, 11.3% and 1.9%, respectively. Overall, 2.1% of constitutional presentations were immediately hospitalized. The same proportion of athletes with constitutional issues was discharged after 2 h in the medical tent. For issues related to the swim, cycle and run sections of the race, in particular, as well as at the finish line, again the majority of presentations were discharged within 2 h. The proportion of cases requiring hospitalization was higher for swimming than it was for cycling and running, however, (at 10%, 8.3% and 0% of overall presentations). At the finish line, 1.4% each of athletes required hospitalization within, and outside, 2 h of presentation for medical aid.

Although both the literature and our unpublished data indicate that the injury or illness that is sustained by triathletes is usually minor in terms of its impact on training or racing, our prospective training diary-based study suggested that, within any given month, few athletes *neither* sustain injury nor illness. Up to 42% and 21% of the same athletes over the same periods also reported illness, with and without an accompanying drop in performance, respectively, in each of the months that were surveyed. The proportion of athletes who did not get injured within a given month of the prospective study ranged from 39.5% to 83.3% of participants, but those who reported themselves as neither sustaining injury, nor illness, nor a drop in performance accounted for less than 18%. This finding also raises the possibility that some of both our data and that in the literature that relates to the severity of a given

injury, in terms of the effect it has on training or racing, may in some cases be skewed by the athlete having a concurrent illness. Our prospective data suggested that injury recurrence rates in triathletes may also be high. Within any given month, the proportion of athletes who reinjured themselves was up to 20.8%. Those who sustained both a new injury and a re-injury accounted for up to 23.7% of our respondents. The case rate per 100 athletes, where every injury occurring more than once in a given month was counted once, was 0.74–0.75. The equivalent case rate per 1000 training hours was between 10.0 and 27.6 depending on the month in question. These values jumped to 10.0–58.3 and to 17.5–58.0, again depending on the month, respectively, when every injury occurring more than once was counted every time it occurred. The number of recurrences of a particular injury to a particular anatomical site in a particular athlete—when it was expressed as a percentage of the total number of reported injuries for the month in question—was also always over 40% for the first recurrence and up to nearly 30% for the second recurrence.

3.6 Risk Factors

3.6.1 General

Our prospective study [40] showed some evidence of cycle run transference—with some injuries being attributed to running on their first occurrence and to cycling on subsequent occurrences, as well as vice versa. To our knowledge, ours are the only actual data [39] relating to how this unique aspect of the sport [51] might impact on injury epidemiology that have been thus far collected, although the idea that it might do so has been raised by several authors [52, 53]. Most of the triathlon-specific injury literature up to now relies on the use of correlational analyses and is based around a linear, reductionist model of causation [54]. Our summary tables of said literature [1, 3] (including supplements) can only be used as a general guide as to what may influence the probability of an injury occurring, its severity and the likelihood that it will recur. Those studies that have used more advanced statistical techniques such as big data analytics of prospective data (Vleck et al., unpublished), to explore the links between putative risk factors and the occurrence of injury in triathletes, are very much in the minority.

Within our prospective study [40], we found, using binary logistic regression analysis, that the occurrence of new overuse injury was associated with an increase in the log of (combined weighted) run and cycle training in training intensity zones 3–5 (of 5, of which zone 5 was the highest), 2 weeks beforehand ($p < 0.05$). Such combined weighted data were obtained by multiplying training duration in zones 1, 2, 3, 4 and 5 by 1, 2, 3, 5 and 8 [55], respectively, and then summating the values so obtained for the disciplines that were involved. No weighting factors for exercise mode were used because the literature does not provide guidance as to what they could be. New overuse injury was also associated with the log of (composite weighted) swim training 4 and 2 weeks prior ($p < 0.05$). A significant relationship also existed between the occurrence of new injury and the percentage difference in cycle zone 1 training between 4 and 5 weeks prior. Similar findings were made concerning the relationship between percentage differences in swim zone 1 training

duration between 1 week prior to and the actual week in which the injury occurred ($p < 0.05$). In a subset of eight athletes who provided 26 weeks of data, some (but not all) new injuries coincided with an increase in a weekly composite swim, cycle and run training stress recovery to stress ratio (unique to the study) over that normally evidenced by each athlete in each week. A greater increase in the composite value for swim, cycle and run training stress than was normally evidenced, also within the week that the injury occurred, might also have contributed to the onset of injury. We note that when the data for the first 6 weeks of endurance base, pre-competition and competition training macrocycles for each of the athletes who took part in the study were synchronized, the weekly rate of change in training stress was found to be higher within the competition than within other macrocycles of the athletes' training year [40]. The extent to which these changes were mirrored by changes in, e.g. acute to chronic workload ratio [9, 56] has not yet been examined in triathletes only.

3.6.2 Risk Factors for Injury to Specific Anatomical Locations

The following section of this chapter presents the latest aetiological evidence [41] as regards the risk factors that are significantly related to overuse injury occurrence to the *specific* anatomical sites that were previously found, within each of the triathlon disciplines, to affect the most triathletes. The data in the study presented here, as previously described, were obtained from a total of 1159 long-distance triathletes. Although the accuracy of their recall was necessarily limited by the fact that it covered "since they started regular triathlon training", this method maximized the number of injury occurrences to the athletes in question that could then be considered by the analysis. The overuse injuries or pain in question, as previously discussed, were sustained to the shoulder (18.7% of males M and 21.4% of females F) in swimming and to the lumbar spine (36.6% of M and 35.2% of F), cervical spine (28.2% of M, 35.9% of F), knee (6.8% of M, 7.7% of F) and genital area (16.3% of M, 31.5% of F) in cycling. For running overuse injuries or pain, the risk factors that were considered in depth were to the Achilles tendon (35.8% of M, 24.8% of F), the lateral knee/ITBFS (27.7% of M and 21.1% of F), anterior knee/runner's knee (28.1% of M and 31.1% of F), anterior tibia/shin splints (16.3% of M, 21.1% of F), the sole of the foot/plantar fasciitis (13.0% of M, 19.5% of F) and lumbar spine (11.6% of M, 17.0% of F).

Bivariate analyses were carried out first. For this the dependent variables were each dichotomized into pain or injury vs. no reported pain or injury. Independent variables were dichotomized or split two or three times, depending on the distribution of the responses. Only independent variables that had a p value less than 0.25 were included in the subsequent multivariate binary logistic regression analysis. In the multivariate model, independent variables such as age (age groups) and anatomical abnormalities that were relevant for the respective disciplines were included in the model because they (almost) cannot be changed by training. The remaining independent variables—which fell under the headers of training, technique, recovery, ability level and injury prophylaxis—were added to the model using a backward selection model to check their impact on the respective dependent variable. The final models were all independent variables with a p value of less than 0.05. Table 3.4

provides an overview of the respective discipline-specific dependent and independent variables that were carried forward to the binary logistic regression analysis.

Table 3.4 Risk factors for injury or pain that was sustained since the commencement of regular triathlon training assessed in [41]

		Swim	Bike	Run
			Lumbar pain	Achillodynia
			Cervical pain	Knee pain
			Genital pain	Shin splints
			Knee pain*	Plantar fasciitis
Dependent variables		Shoulder pain		Lumbar pain
Intrinsic risk factors	Age	Age (groups)		
	Anatomical abnormalities	Postural deformities		
			Axial abnormalities	
			Leg-length inequality	
			Pronation (foot)	
			Supination (foot)	
Extrinsic risk factors	Training-related risk factors	Total training volume (h)		
		Kilometres		
		Hours		
		Intensive sessions (>85% of max. performance)		
		Upper body-strength sessions		
		Core-strength sessions		
		Lower body-strength sessions		
		Discipline-specific stretching/flexibility sessions		
		Discipline-specific stabilization sessions		
		% Swim training with paddles	% Training on aerobars	% Hill work
		% Resistance training		Belt training sessions
		% Pull buoy training		
		% Training using swim paddles with pull buoy		
	Technique training	Discipline-specific technical training		
			Adjusted racing bike	Foot plant type
				Gait-specific running shoes
	Recovery	Regeneration—sessions per week		
		Break time—individually adjusted		
		Break time—subjectively sufficient		
		Training attitude		
	Performance	Long-distance participations—total		
		Swim level	Bike level	Run level
	Prevention	General stretching sessions per week		
		Amount of preventative consultations per year		

*The cycle-related lumbar pain, cervical pain and genital pain data; and the run-related achillodynia, shin splints, plantar fasciitis, knee and lumbar pain data were obtained from questions that were each specific to that area in particular. The cycle-specific knee injury data were obtained from the responses to a question that also asked about injury in other locations

3.6.3 Swimming-Related Shoulder Pain

According to the results of the analysis, shoulder pain in swimming was less likely to have occurred in the male triathletes who did not undertake regular technique training (odds ratio OR 1 vs. OR 0.69, 95% confidence interval CI 0.47–1.00, $p = 0.048$) and, increasingly less likely, the less core training they did (for > once, once and no weekly sessions, the OR were 1.0, 0.91 (95% CI 0.60–1.37, $p = 0.656$) and 0.55 (95%CI 0.33–0.91, $p = 0.021$), respectively). Swimming-related shoulder injury or pain became more probable, the higher the proportion of the athlete's training time that was spent doing resistance (e.g. dynamic band, sponge) training (OR of 1.0 for 0% and of 1.68, 95% CI 1.04–2.69, $p = 0.033$, for above 0% of training time). In females, shoulder pain became more likely as weekly swimming mileage increased (OR for <6 km, 6–10 km and >10 km were 1.0, 4.00 (95% CI 1.45–10.99, $p = 0.007$) and 4.84 (95% CI 1.34–17.40, $p = 0.016$), respectively).

3.6.4 Cycling-Related Traumatic Injury (Including Shoulder Pain)

Overall, the athletes in Hoeden's study sustained 1376 traumatic injuries (1165 in the males and 211 in the females)—of which, in the males and in the females, respectively, 16.8% and 14.2% were to the shoulder, 10.8% and 14.4% to the area of the hand, 8.8% and 11.8% to the knee and 8.4% and 7.6% to the thigh. A total of 299 and 52 fractures were recorded, of which 23.4% and 19.2% were in the shoulder area and 20.4% and 19.2% were around or to the hand(s). The equivalent values for the ribcage and elbow (joint), in males and females, were 9.0% and 11.5% and 6.0% and 13.5%, respectively. That is, those regions that support or catch the body during a fall—such as the wrist and collarbone, which made up the majority of fractures to the hand and shoulder areas—were those that were most often affected by traumatic injury.

3.6.5 Cycling-Related Cervical Spine, Lumbar and Knee Pain

As for the most common areas to which cycling-related atraumatic injuries and pain occurred, i.e. the lumbar spine/lower back, cervical spine area, genital area and knee, some gender differences were found in their associated risk factors. Males with postural abnormalities exhibited more lumbar spine pain (OR = 1.58, 95% CI 1.05–2.38, $p = 0.029$) than those who did not, whilst no significant relationships between this and the extrinsic risk factors that were evaluated were seen in the females. As for cervical spine pain, this was reported more by those males who did not have a race bike that had been fitted to their proportions (OR = 1.74, 95% CI 1.06–2.85, $p = 0.028$) than by those that did. The females who did not do cycle technique training also experienced more cervical spine-related pain than those who

did (OR = 3.98, 95% CI 1.19–13.37, $p = 0.025$). Less data were available as regards the risk factors for cycle-related knee pain than for the other most commonly affected sites, as the survey did not feature a question specifically directed at it. Those male triathletes who performed two to three stretching units per week, or less than or up to one unit a week, exhibited over twice the risk for knee pain (OR = 2.51, 95% CI 1.29–4.90, $p = 0.007$, and OR 1.9, 95% CI 0.93–3.43, $p = 0.082$, respectively), of those who stretched more than three times per week (OR = 1). No such association was found in the females.

3.6.6 Genital Pain

The male triathletes who exhibited postural dysfunction (i.e. scoliosis, "hollow back" or hyperkyphosis) had double the risk of groin injury or pain than those who did not (OR = 1.66, 95% CI 1.05–2.62, $p = 0.030$). Those who did not perform lower body strengthening exercises, or only did one such session per week, also felt significantly more pain (OR = 1.90, 95% CI 1.05–3.45, $p = 0.035$ and 1.09, 95% CI 0.57–2.09, $p = 0.786$) than those who did more than one unit of such training per week. The males who felt less genital area-related pain did not do regenerative training (i.e. sauna, contrast hydrotherapy, ice baths, massage, electrostimulation, etc.) at least once per week (OR = 0.57, 95% CI 0.37–0.88, $p = 0.010$). Of the females, those who did more than one intensive cycle training session per week reported significantly more such pain (OR = 3.45, 95% CI 1.32–9.05, $p = 0.012$) than those who did not. Moreover, the females who did not have a cycle technique training background reported more genital area pain (OR = 3.59, 95% CI 1.0–12.89, $p = 0.050$) than those who did.

3.6.7 Achillodynia

As for running-related injuries, the males in the study sample who were 41–50 years (OR = 2.13, 95% CI 1.28–3.54 $p = 0.003$), or over 50 years (OR = 2.82, 95% CI 1.52–5.21, $p = 0.001$) of age, presented significantly more frequent Achilles tendon complaints than those who were up to and including the age of 30 (OR = 1). Males with leg-length discrepancies also had greater risk of achillodynia (OR = 1.44, 95% CI 1.01–2.05, $p = 0.042$) than those who did not. Moreover, those athletes who performed only one regenerative unit per week exhibited more pain (OR = 1.58, 95% CI 1.03–2.42, $p = 0.036$) than those athletes who did so more than once a week (OR = 1). Interestingly, the males who performed at least one coupled (bike-run or "brick") session experienced significantly less Achilles tendon-related pain (OR = 0.62, 95% CI 0.42–0.93, $p = 0.021$) than the athletes who did not do so. Additionally, the athletes who experienced pain were more likely to visit a sports physician more than once a year, on a preventative basis (OR = 1), than those who never visited one (and for whom the OR was 0.67, 95% CI 0.45–1.00, $p = 0.047$), or only visited one once a year (OR 0.63, 95% CI 0.41–0.98, $p = 0.038$).

Female triathletes who were between the ages of 31 and 40 exhibited less Achilles tendon-related problems (OR = 0.11, 95% CI 0.02–0.53, $p = 0.006$) than did younger athletes. Those females who ran more than 50 km (OR = 9.50, 95% CI 2.00–45.05, $p = 0.005$) a week were also significantly more in pain, for this reason, than those whose mileage was up to and including 30 km a week. The females who perform more than one upper body effort per week also experienced significantly more Achilles tendon-related pain (OR = 6.70, 95% CI 1.40–32.06, $p = 0.017$) than those who did not. Finally, those female triathletes who did not use a running shoe that had been chosen according to whether they had a pronated or supinated gait were less likely to present with achillodynia (OR = 0.23, 95% CI 0.07–0.74, $p = 0.014$) than those athletes who had done so.

3.6.8 Anterior Knee Pain/"Runner's Knee"

These male triathletes who were between the ages of 41 and 50 (OR = 0.47, 95% CI 0.28–0.78, $p = 0.003$), and over 50 years of age (OR = 0.34, 95% CI 0.16–0.70, $p = 0.004$), were less likely to exhibit runner's knee-related pain than those who were 30 years old or less. Athletes with a toe-first foot strike evidenced less pain (OR = 0.52, 95% CI 0.30–0.93, $p = 0.026$) in the area of the kneecap than those with a heel strike. The males who did not stretch at least once weekly (OR = 2.17, 95% CI 1.33–3.54, $p = 0.002$) were more affected by runner's knee than those who stretched up to at least thrice weekly. Interestingly, those males who subjectively perceived their extent of intersession recovery to be insufficient had a lower risk (OR = 0.56, 95% CI 0.31–1.00, $p = 0.049$) of knee pain in the knee than the men who considered it to be adequate. Of the female triathletes in the study, those who performed two units of core strength training a week (OR = 0.23, 95% CI 0.06–0.84, $p = 0.026$) complained less about knee pain than those who completed more than two such units. The females who did two to three stretching sessions per week exhibited lower odds ratios for runner's knee (OR = 0.26, 95% CI 0.08–0.82, $p = 0.022$) than those who scheduled more than three such units. The women who rated their level of rest between pauses as inadequate were more likely (OR = 6.14, 95% CI 1.67–22.57, $p = 0.006$) to have knee pain than those who did not. Mid-ability-level long-distance females (i.e. those who were completing the run section of a 3.8/180/42.2 km event in 03:40–04:43 hh:mm) had a lower odds ratio for knee pain (OR = 0.33, 95% CI 0.12–0.91, $p = 0.033$) than the group of low ability females who took 04:44 hh:mm or more to do so (OR = 1).

3.6.9 Lateral Knee Pain/Iliotibial Band Friction Syndrome (ITBFS)

Males within the 41–50-year age group (OR = 0.55, 95% CI 0.31–0.95, $p = 0.031$) were statistically less likely to present with ITBFS injury or pain as compared to 30-year-olds and younger athletes. Male triathletes who ran over 5 h per week

were also less likely to experience pain (OR = 0.54, 95% CI 0.36–0.81, $p = 0.003$) than those who ran up to 5 h. Athletes who do not use a foot-specific running shoe were less likely to complain about problems (OR = 0.64, 95% CI 0.43–0.94, $p = 0.022$) than those who wore a recommended shoe. The males who did not stretch at least once a week had a lower chance (OR = 0.56, 95% CI 0.33–0.94, $p = 0.029$) of lateral knee pain than those who did so at least three times a week. Female athletes with postural injuries were less likely to experience pain (OR = 0.36, 95% CI 0.13–1.00, $p = 0.051$) than those without postural damage. The female triathletes who never stretched or stretched once a week (OR = 0.32, 95% CI 0.13–0.82, $p = 0.017$) reported fewer injuries or pain than did the control group (OR = 1)—who stretched three or more times a week. The incidence of pain was also less frequent for mid- and high-level female athletes who were completing the (42.2 km) run section of a 3.8/180/42.2 km event in or less than 04:43 hh:mm (OR = 0.27, 95% CI 0.12–0.60, $p = 0.001$) than it was for those who took more than 04:43 hh:mm to do so.

3.6.10 Anterior Tibial Pain/"Shin Splints"

The males in the sample who were over 40 years old exhibited lower risk of pain from shin splints (OR = 0.33, 95% CI 0.18–0.60, $p = 0.000$) than those who were 30 years old or younger. The athletes whose knees pointed inwards or outwards had nearly double the risk (OR = 1.86, 95% CI 1.03–3.36, $p = 0.039$) of developing shin pain than those who did not. Male triathletes who ran between 31–50 km (OR = 2.08, 95% CI 1.18–3.69, $p = 0.012$) and more than 50 km (OR = 3.54, 95% CI 1.84–6.80, $p = 0.000$) a week were also significantly more likely to suffer from tibial pain than athletes who ran 30 km or less a week (OR = 1). Those men who did a brick training session each week (OR = 0.42, 95% CI 0.23–0.76, $p = 0.004$) noted less pain in the area of the tibia than those who did not do so. As for the females, those who were over the age of 40 (OR = 0.12, 95% CI 0.03–0.56, $p = 0.007$) were less likely to complain of pain than those who were 30 years or younger. Female athletes with a supinated foot position also reported significantly more pain (OR = 6.83, 95% CI 1.39–33.56, $p = 0.018$) than those who did not. The athletes who refrained from special stabilization/compensation work had a significantly higher incidence (OR = 3.31, 95% CI 1.78–9.32, $p = 0.023$) of shin splints than the comparison group—the athletes within which performed a minimum of one such unit per week.

3.6.11 Pain to the Sole of the Foot/Plantar Fasciitis

Male triathletes who scheduled one (OR = 0.58, 95% CI 0.35–0.96, $p = 0.033$) or more (OR = 0.41, 95% CI 0.22–0.75, $p = 0.004$) brick training units per week into their training were less likely to complain of pain than those who did not do a double workout per week. Men who did not wear a foot-specific running shoe were less likely to experience pain (OR = 0.40, 95% CI 0.26–0.61, $p = 0.000$) than triathletes

who did. Furthermore, higher-level male triathletes (OR = 2.44, 95% CI 1.29–4.59, $p = 0.006$) had a significantly higher risk of pain in the sole of the foot when they ran than did male athletes of a lower performance level. The run performance ranges for such higher- and lower-level males, obtained within a 3.8/180/42.2 km event, were faster than 03:30 hh:mm and equivalent to or exceeding 04:30 hh:mm, respectively. Among the women who had a total exercise volume of over 15 h a week, there was a nonsignificant trend for the onset of pain (OR = 2.31, 95% CI 0.99–5.39, $p = 0.052$) to be higher than in those who did up to 15 running hours per week. Those females who ran mainly on hard ground (i.e. asphalt/track) also exhibited a higher chance (OR = 3.01, 95% CI 1.04–8.68, $p = 0.041$) of pain in the sole of the foot than did the females who mainly trained on soft running surfaces.

3.6.12 Running-Related Lumbar Spine Area Pain

There were no significant associations between "pain in the lumbar region" and the independent variables that the study assessed, in males. Female triathletes who exhibited postural defects displayed a fourfold risk (OR = 4.04, 95% CI 1.42–11.49, $p = 0.009$) of pain in the area of the lumbar spine. Female athletes who either never stretched or only stretched once a week (OR = 0.26, 95% CI 0.08–0.86, $p = 0.027$), and those who stretched two to three times (OR = 0.14, 95% CI 0.04–0.53, $p = 0.004$) per week, reported significantly fewer pain symptoms than did the control group, who stretched more than three times per week.

3.7 Context, Conclusions and Recommendations

As Sands et al. so aptly put it in their 2017 paper [57], "although analytics hold considerable promise", "one cannot postdict without rich, thorough, relevant, and accessible data on the events that transpired". The consensus statement on the definition and recording of training and racing load, injury and illness in triathlon that could allow for (relatively) seamless integration of data from more than the two research groups whose data were presented here, such that, like the sport of triathlon itself, they become "more than the sum of its parts", does not yet exist. This current lack of comparability between other studies than ours makes it impossible to build up an accurate view of how the various risk factors may be interacting with each other, as well as how their relative influence may vary in different situations and athlete subgroups. Moreover, if "sparse data bias" is to be avoided, and optimal progress is to be made in this field, considerably larger sample sizes are required than have been obtained by most of the studies thus far [58].

Progress has been made, however. Technical guidelines are being updated in the light of advances in research knowledge [7, 59]. A better understanding of the problems that may face different athlete groups, and their associated risk factors, has been arrived at. How the relative influence of these risk factors changes at different stages of the training year, and how it may also vary between males and females and

between athletes within different ability-level and event distance specialization groups and age groups, and then the extent to which such differences may translate into differences in the injury profiles of such groups, needs to be examined more closely, on a prospective longitudinal basis. This can inform future research that uses both a group and an "individualized to the athlete", multifactorial approach to health and safety-related risk [60]. When such future studies are, in the first instance, based around self-reported injury or illness in an effort to maximize sample size, clinical follow-up within the data collection period should be implemented whenever possible. As most athletes lack medical knowledge, this clinical support is crucial to obtaining the specific diagnoses for injuries and profound onset mechanisms [61] that, further down the line, could be used to develop injury prevention programmes for the most common triathlon injuries.

We finish with a quote from the triathlon chapter of the IOC handbook on "Epidemiology of Injury in Olympic Sports" [3]: "We strongly urge that a collaborative research team of race organizers, technical officials, coaches, athletes, medical support staff and researchers working at both the 'grass-roots' and the 'top end' of the sport be established, for an adequate database of injury" (and illness)-related "data to be compiled and used to drive continuous improvement [62] in triathlon training and competition practice, as well as athlete, coach, and both technical and medical staff education" [63].

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Part II

Environmental Conditions

Heat-Related Illness

4

David Gerrard

4.1 Introductory Comments

History vividly records the inaugural women's Olympic marathon in Los Angeles 1984, an event celebrated for the victory of the hometown favourite Joan Benoit. But for many, the enduring memory of that hot July day will be of Swiss athlete, Gabriela Andersen-Schiess, dragging her pathetic contorted frame around the final 400-metre lap of the Los Angeles Coliseum [1]. Hyperthermic, dehydrated and hemi-paretic, her extreme distress drew disgust and dismay from helpless onlookers. And in the cauldron of the Coliseum, this hapless, ataxic athlete crossed the finish line to collapse into the arms of waiting medics, a vision indelibly etched in the memories of spectators and television viewers alike. Fast-forward 13 years to 1997 when, at the World Ironman Championships, the punishing Hawaiian heat took its toll on two accomplished triathletes, Sian Welch and Wendy Ingraham. Again, TV viewers joined onlookers, to witness the bizarre end of an epic event. For the final 200 m, two incapacitated, heat-stressed athletes, incapable of remaining erect were humiliated into an infantile crawl across the finish line to be gobbled up by concerned medical staff [2]. And at the 2016 International Triathlon final event in the heat of Cozumel, Mexico, a further arresting incident took place, this time the collapse of Olympian Jonny Brownlee with the World title and the finish literally in his sight. Neurologically impaired to a point of obvious disorientation, Brownlee was presumably hyperthermic, as well as energy and electrolyte depleted. His limp frame was dragged bodily across the line by fellow competitor Alistair, coincidentally his older sibling and the reigning Olympic champion [2]. Although his recovery was uncomplicated he was clearly affected by the combination of prolonged physical exertion and environmental stressors.

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The common thread connecting these examples is the phenomenon of exercise-associated collapse. However, in each case the high ambient temperature and the obvious signs of neurological impairment highlight the potential for far more sinister clinical outcomes. Those responsible for the provision of on-site medical care to endurance athletes must remain vigilant to the possibility of exertional heat stress and its consequential challenge to thermoregulatory mechanisms. Further, such well-supported, elite events represent only a fraction of the cases of exertional collapse in endurance athletes including many unheralded, unreported, mass participation events involving a cohort of social, club-level athletes, the so-called "weekend warriors". Collectively however, these instances underscore morbidity in contemporary endurance sport that cannot be ignored, placing heat-related illness in alarming perspective. Throughout this chapter, the terms heat "illness" and "injury" are interchangeable, denoting the consequences of prolonged exertional heat stress.

4.2 Exertional Heat Stress: What Have We Learned?

The past three decades have witnessed an explosion of interest in endurance sport, precipitating advanced coaching and a parallel scientific focus on the stresses of prolonged physical exertion [3–9]. Yet the cases referred to previously beg the fundamental question of heat injury prevention, an approach that appears to challenge the prevailing default stance of active resuscitation. Given the timing, duration and energy requirements of contemporary endurance events, heat-related injury with attendant serious health implications will always be a major concern. Traditionally, events such as triathlons or multi-sport variations are held in spring and summer, exceed one-hour duration and engage athletes in a combination of land-based and aquatic disciplines. They invariably challenge the regulation of body temperature in circumstances where too frequently, the sum of energy production and environmental stressors exceeds the body's innate capacity to dissipate heat. Several international sporting federations including Triathlon (ITU), Swimming (FINA), volleyball (FIVB) and Athletics (IAAF) have made significant research investments leading to a greater understanding of the pathophysiology of exertional heat illness [10–12]. In turn, this has spawned an extensive body of literature on thermal regulation in athletes, enhancing knowledge of its genesis and systemic implications [6–8, 13–16]. Informed by this research, sports physicians are becoming more adept at recognising signs of impending heat illness, and coaches are implementing strategies to adapt athletes to the impact of heat stress while event organisers seek scientific and clinical input into planning major races. FINA has applied research-informed evidence to regulate temperature limits for the 10 km marathon swim, popularised since its addition to the Olympic programme in Beijing 2008. Similarly, water temperature guidelines in triathlon competition with implications for wetsuit use have also been adopted by the ITU and FINA [10, 11]. Both the ITU and the IAAF have introduced temperature restrictions on land-based endurance events, governed by wet globe bulb temperature (WGBT) monitoring. The WGBT device measures ambient temperature, relative humidity, wind, and solar radiation to determine safe

conditions for prolonged exercise [12]. The FIVB medical rules have been influenced by recent research into the prevailing centre court temperatures at World Tour and World Beach Volleyball Championships (2009–2011) where WGBT recordings were matched against instances of medical forfeiture by athletes. These data have informed subsequent guidelines for minimising heat stress injury in the professional beach volleyball community [14]. In preparation for the 2020 Olympics and Paralympic Games in Tokyo, organisers are already expressing concern over the expected temperatures that will prevail during the period of the Games [17]. WGBT indices are being applied to the schedule of Games training and competition programme in an effort to minimise the potential for heat stress injuries. There has also been a call by the American College of Sports Medicine (ACSM) for the Games Organising Committee to limit competition times to early morning or evening in an attempt to minimise the effects of solar radiation on potentially affected outdoor disciplines [18]. While proactivity by responsible governing bodies is acknowledged, the literature and public media reports would suggest that some experienced, well-trained endurance athletes still remain susceptible to severe heat-related injury.

Research has shown that for a number of reasons including individual variability, acclimatisation, training and nutrition, not every athlete with a rectal temperature of 40 °C will advance to catastrophic end-stage exertional heat stroke with multi-organ failure [3, 6–9]. Athletes including marathoners, soccer players, open water swimmers and American football players have been reported with sustained rectal temperatures above a 40 °C threshold without any serious medical consequence [7]. In a similar vein, the interplay between the concepts of hyperthermia, exhaustion and dehydration is important to recognise. While the concept of exhaustion as the inability to sustain physical output is a well-reasoned physiological response, the time to complete exhaustion is also shown to diminish with rising ambient temperatures above 20 °C [6, 7, 16].

This chapter addresses the basic understanding of thermal regulation, considers the spectrum of heat-related injuries and suggests strategies to prevent heat illness, informed by recent research and clinical experience.

4.3 Thermoregulation in Humans

Thermal regulation is the ability of the body to maintain a core temperature of 37 °C to within a relatively narrow range (33.2–38.2 °C), a fundamental homeostatic mechanism acknowledged by observational studies and reliant upon several physiological factors [4, 5, 7]. Normal core body temperature rises rapidly in response to thermal challenge and in the context of endurance sport this is the product of the energy generated from sustained physical activity plus recognised environmental influences. These factors, including ambient temperature, humidity and solar radiation are beyond the control of the athlete and remain the responsibility of event organisers. As already referenced, these external influences have been recognised by the medical advisory committees of the ITU, FINA, FIVB and the IAAF [10, 11, 14]. Their interest in preserving the safety of athletes is embedded in sport-specific rules and event regulations. In addition, the International Olympic Committee has

published a position statement acknowledging a number of environmental influences including temperature, that challenge the contemporary elite athlete in a variety of sporting disciplines [15].

As mammals we possess an innate propensity for homeostasis, with four recognised pathways for the transfer of body heat. These are via the primary mechanisms of convection, conduction, radiation and evaporation. Collectively, these are essential to the regulation of core body temperature and critical to homeostasis, with fluctuations beyond a couple of degrees posing a significant challenge to thermoregulation [19]. Heat loss mechanisms, triggered in the anterior hypothalamus, reflect the temperature differential between skin surface and the environment. As body temperature increases, active sympathetic cutaneous vasodilation increases blood flow in the skin and this initiates sweating through eccrine gland activity. If skin temperature exceeds that of the surroundings, heat loss occurs through radiation and conduction. But, if the ambient temperature exceeds that of the skin, the body gains heat that is dissipated through the evaporation of sweat. Consequently, physical activity in high ambient temperatures depends upon adequate evaporation and where this is impeded, the consequential rise in core body temperature may precipitate severe heat injury [6, 7, 9]. A well-hydrated athlete will have the capacity for adequate sweat production. And adequate fluid intake is quite obviously an influence over which the athlete has control, enhanced by the provision of sufficient food and drink stations around any given course. Ambient factors such as temperature and relative humidity remain an issue for race organisers charged with venue selection and seasonal timing. As already described, when the surrounding temperature is higher than that of the skin, any impedance to evaporation, the primary means of heat dissipation, will cause the internal body temperature to rise. Prolonged physical exertion in excessive humidity impedes sweat evaporation, a mechanism essential for the maintenance of thermal neutrality [3, 6, 19].

In summary therefore, under normal circumstances, body heat generated from physical activity is quickly dissipated by the adaptive "cooling" mechanisms of radiation and convective loss through the skin, evaporation of sweat and via small amounts of evaporation through respiratory loss. These mechanisms diminish the potential hazards of exertional heat injuries, the most serious of which is heat stroke, commonly associated with a body core temperature exceeding 40 °C. While some authorities quibble over the nomenclature, there is an agreed "spectrum" of exertional heat injuries reported in the sports medicine literature, notably in events of high intensity or long duration [20]. Clinicians agree that heat illness, unrecognised and untreated, may present quite innocently with initial signs of oedema, heat rash, cramps and syncope, and progress to heat exhaustion or heat stroke representing the most severe, life-threatening end of this continuum [6, 8, 9, 14–16].

4.4 Pathophysiological Factors in Heat Injury

Our initial understanding of heat injury and its serious clinical sequelae has its genesis in the general population during times of excessively high ambient temperatures. Heat stroke with attendant life-threatening consequences is considered a

potential outcome from exposure to a high environmental temperature or prolonged strenuous exercise. A universally accepted definition includes a core body temperature above 40 °C, the absence of sweating and accompanying signs of central nervous system dysfunction [21]. These signs may include confusion, disorientation, ataxia, delirium, convulsions or coma. From a pathophysiological perspective, it is also generally agreed that unremitting heat stroke is essentially a form of hyperthermia that progresses to multi-organ failure as the result of a systemic inflammatory response. Cases of non-exertional heat stroke, involving individuals with existing comorbidities such as aging, obesity, diabetes, renal impairment and heart disease, demand acute medical intervention and have been associated with high rates of morbidity and mortality, widely reported in North American and European data [22, 23]. However, the extrapolation of the pathophysiology of non-exertional heat stroke has informed the contemporary understanding of exertional heat stress in sport, with sustained strenuous physical activity the obvious additional stressor to thermoregulation [21].

As already discussed, there is general acceptance of a spectrum of heat-related injury. At points along this continuum an athlete may display signs of poor adaptation to the escalating consequences of increasing body temperature. However, where there is little or no challenge to the homeostatic mechanism of thermoregulation, the adapted athlete will simply experience "normal" energy depletion and ultimate fatigue [6]. This may or may not result in what is commonly seen as exertional collapse, a relatively benign condition that responds to conservative management. However, sustained, increasing body temperature will stimulate cutaneous vasodilatation, thereby diverting blood from central viscera to the skin where sweating is initiated as a primary means of heat dissipation. This shunt of blood from the central circulation to the periphery challenges major organs and precipitates a relative reduction in intravascular volume that may lead to hypotension, associated with the phenomenon of "heat syncope". Meanwhile, sweating-induced losses exacerbate dehydration and sodium depletion, linked with heat exhaustion and generally considered as contributory to heat-related muscle cramping. Any further loss of salt and water further impairs normal thermoregulatory mechanisms and together with the reduction in visceral perfusion may precipitate cellular damage and ultimate organ failure if the challenge to core body temperature continues unchecked [7, 17, 19, 21].

4.5 Pathogenesis of Severe Heat Injury

The underlying, complex cascade of events in exertional heat stroke (EHS) commences at a cellular level where the membranes of heat-stressed cells become damaged, their energy systems disrupted and cell membrane permeability becomes altered. These changes precipitate the leakage of intracellular endotoxins into the systemic circulation that ultimately give rise to a well-documented syndrome of multi-organ damage and failure [24, 25].

A process labelled the "systemic inflammatory response syndrome" is also described, as the consequence of endotoxin leakage through the intestinal mucosa

accompanied by interleukins (IL)-1 and (IL)-6 from skeletal muscle. These proteins enter the systemic circulation to activate leukocyte production causing diffuse inflammatory effects that damage vascular endothelium with a consequential risk for microthrombotic events [25]. Platelet production is suppressed by heat, there is enhanced fibrin formation and ultimately a diffuse coagulopathy manifests as disseminated intravascular coagulation (DIC). Further complicating gut cell membrane damage is the leakage of intestinal gram-negative bacterial fragments into the systemic circulation that are responsible for the ominous consequence of endotoxic shock. These effects are enhanced in the presence of dehydration with overwhelming clinical consequences for any vulnerable athlete [26–28].

In addition to these sequelae, it is also well established that the integrity of skeletal muscle is seriously challenged by temperatures exceeding 40 °C [29]. Sustained heat insult gives rise to structural changes at a cellular level resulting in diffuse muscle fibre destruction. This pathophysiological process causes a clinical condition known as rhabdomyolysis [28, 29]. Muscle enzymes, in particular creatine kinase (CK), leak into the circulation from damaged cells, with CK levels more than a thousand times normal deemed to be an accurate marker of rhabdomyolysis. Free myoglobin fragments, another product of muscle damage, give rise to dark-stained urine, the typical discolouration of frank myoglobinuria. Exercise-induced rhabdomyolysis may advance to produce severe renal insult through tubular toxicity and obstruction. Intracellular potassium, also released into the systemic circulation from damaged cells gives rise to hyperkalemia, a recognised precipitant of fatal cardiac arrhythmia [28]. Collectively, these potential life-threatening consequences underscore the seriousness of prolonged physical exertion in the heat [30].

4.6 Exertional Heat Illness: A Continuum of Disorder

There is an acknowledged potential for heat illness in any situation involving sustained, intense physical activity, particularly in hot-humid conditions. Exertional heat illness, affecting athletes during high-intensity or long-duration exercise may result in withdrawal from an event or collapse during or soon after its conclusion. Although a wide variation in individual athlete response to heat stress is acknowledged [6], these illnesses include exercise-associated muscle cramping, heat exhaustion or exertional heatstroke. Athletes who are dehydrated, not well acclimatised, who use certain medications, or have recently been unwell, are at greater risk and more vulnerable to thermal challenge. A constellation of signs and symptoms associated with exertional heat illness exist along a recognised continuum of disorder. Early recognition and rapid cooling can reduce the morbidity and mortality associated with these clinical challenges that in many cases are subtle and easy to miss. Therefore, coaches, medical personnel, and race officials must be vigilant and monitor at-risk athletes closely. Heat exhaustion and exercise-related muscle cramps are not typically linked with excessive hyperthermia, but result from fatigue, dehydration and/or electrolyte depletion. But where central mechanisms of heat regulation fail and normal heat dissipation

processes fail, unidentified athletes may succumb to serious outcomes. The following is a brief summary of common exertional heat injuries.

1. Heat cramps:

   Painful involuntary episodes of muscle spam associated with prolonged exercise in hot conditions have given rise to the term "heat cramps". There is also a significant body of research interest in the same condition for which the term Exercise-Associated Muscle Cramps (EAMC) has been coined. This form of cramping in athletes occurs in the muscles of the calves, but may also affect the arms and abdomen. There is scientific debate between two possible aetiologies for EAMC. The first implicates prolonged sweating with attendant electrolyte loss, particularly of sodium, as a precipitant of heat cramps, while the alternative hypothesis is that of "altered neuromuscular control". Contemporary debate would favour the latter as the most valid pathophysiological mechanism [31]. Combined eccentric-concentric, repetitive muscle contraction through a short range of motion during the foot plant and "toe-off" phases of running would suggest the propensity for common cramping of the gastrocnemii (calf muscles). Similarly, a prolonged erect running posture engaging static contraction of the anterior abdominal wall, with rigid, adducted arms and flexed elbows provides additional logic for cramps of these regions. It is also proposed that the spontaneous discharge of hyper-excitable axon terminals results in muscle fasciculation that may progress to debilitating muscle cramps [32]. In light of the informed debate over the pathophysiological basis of heat cramps it remains generally agreed that rest, passive stretching and oral fluid replacement to replenish sodium losses are accepted measures in the management of heat cramps. Maintaining regular fluid intake during the event is still sound advice to all endurance athletes. Dehydration in itself represents an issue for all athletes but particularly those who are sweating profusely during events in hot climates. Setting aside the electrolytes lost in sweat, once fluid depleted, an athlete will no longer have the capacity to initiate sweating and thereby fails to engage a key thermoregulatory mechanism. While heat cramps represent the mild end of the spectrum of heat injuries, this phenomenon, unchecked, may be the precursor to more sinister injury associated with prolonged physical activity.
2. Heat exhaustion:

   Heat exhaustion from prolonged physical exertion in high temperatures frequently co-exists with high humidity. Athletes suffering from full-blown heat exhaustion invariably look distressed but as with all forms of heat injury, signs and symptoms appear on a spectrum from mild to moderate to severe. The early signs of impending heat exhaustion may be those of severe heat cramping associated with profuse sweating and tachycardia [3]. An unidentified athlete may progress to becoming confused and disorientated, representing early ominous central nervous system consequences of raising body core temperature. Uncharacteristic behaviours, worsening confusion, unsteady gait and inappropriate response to instructions may also be subtle signs of progressive heat stress implicating the brain [6]. Heat exhaustion is frequently accompanied by symp-

toms of dizziness, nausea, and fatigue and signs such as vomiting and mild temperature elevation. Athletes may have complained earlier of muscle cramping and on examination are found to have a rapid thready pulse and cold clammy skin in the presence of excessive sweating and dizziness. There is often an associated feeling of fatigue and impending collapse suggesting that these athletes deserve close monitoring by race officials. Unheeded, and untreated, the gain in body heat may completely overwhelm thermoregulatory mechanisms and progress to severe heat stroke. That said however, heat exhaustion, detected early, generally resolves with symptomatic care and oral hydration [3, 6, 8, 11].

3. Exertional heat stroke:

As already emphasised, heat exhaustion and heat stroke may occur along a recognised continuum of heat-related injury, but by definition, heat stroke always implies a core body temperature above 40 °C. Exertional heat stroke (EHS) is the most severe form of heat injury, whereby sustained, excessive body heat results in a syndrome of central nervous system dysfunction and a systemic inflammatory response [21, 33]. The mechanisms by which an athlete with exertional hyperthermia proceeds to a state of collapse is characterised by a cascade of disturbances of the nervous, cardiovascular, haematological and renal systems. It represents a life-threatening injury resulting from the progression of non-specific symptoms including headache, nausea and general malaise to severe systemic effects typified by the absence of sweating (anhidrosis), hepatic failure, disseminated intravascular coagulation and rhabdomyolysis. The progressive neurological signs of confusion, delirium and coma are frequently associated with arrhythmia and a potentially fatal outcome [3, 6, 8]. The effective management of heat stroke demands prompt recognition, immediate withdrawal from the event, protection from the prevailing elements, aggressive whole body cooling and appropriate fluid and electrolyte repletion [6, 7].

4.7 Susceptibility to Exertional Heat Injury

A significant body of experience derived from military reports and associated clinical data has informed the understanding of EHI and individual susceptibility. These reports are rich in details of clinical presentation, and corroborate the pathophysiological basis for heat injury. Factors relating to the individual susceptibility to heat injury may be classified as either extrinsic or intrinsic. The former includes environmental factors such as ambient temperature, humidity and solar radiation [34–37]. And in multisport events this extends to water temperature. As already alluded to, the control of these factors resides with international federations and race organisers who bear the responsibility for venue selection and the seasonal timing of major events [32, 38].

However, it is also understood that individual athletes preparing for endurance events should be obviously well trained, habituated (acclimatised) to exercising in heat, pre-hydrated, free from the effects of recent systemic illnesses and not taking medications known to increase susceptibility to heat stress. Drugs predisposing to

heat stroke include stimulants, antihistamines, anticholinergics and phenothiazines. The use of sunblock and the choice of appropriate clothing (texture and colour) are additional intrinsic factors that remain the responsibility of athlete. The education of coaches and athletes in areas of hydration, acclimatisation and freedom from intercurrent illness are important measures to minimise the risk of exertional heat stress. Heat illness and debilitating consequences are most commonly recognised by race officials whose responsibility it is to alert appropriate medical support. This acknowledges pre-race planning to include the provision of adequate medical staff with tents, awnings or suitable shelters for affected athletes.

4.8 Concluding Comments

Illnesses associated with sustained physical activity in hot humid conditions are widely reported. These have become a contemporary challenge for all endurance athletes. Well-adapted, pre-hydrated athletes are less likely to succumb to heat stress, however there are documented instances of heat-related injury at the highest competitive levels. When the normal mechanisms of heat regulation cannot cope with the imbalance of heat production and heat dissipation, there are recognisable clinical signs of distress that must be acknowledged. It behoves all organisers of endurance events to take into consideration prevailing environmental conditions, using instruments such as WGBT indices. They must also make adequate provision for the medical care of athletes and through vigilance, intervene appropriately where clinical signs indicate.

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Environmental Conditions: Cold

5

Michael Tipton

5.1 Introduction

Global warming will increase average air temperature, it will also bring larger variations in weather and more severe conditions. In 2018 the weather played a major role in changing triathlon events, with swims cancelled and extra runs added. This culminated with the organisers of the 2018 Ironman 70.3 Zell am See-Kaprum in Austria, being forced to change the event to a swim-run race due to severe weather and snow blocking the bike course. The air temperature was as low as 2 °C, a year earlier it was 34 °C. In general, the main cold-related threats to performance and safety for a triathlete in less extreme circumstances are: water temperature during the swim; transition from water to bike (T1) due to body cooling experienced during the swim; and evaporative cooling when wet on a bike after the swim. Cold conditions impact on energy output, neuromuscular function and psychological factors; each of these potentially reduces performance and safety [1]. As a consequence, the cold-related threats in triathlon range in nature from drowning, through injuryv caused by cooling, to significant decrements in performance.

In this chapter, we examine the impact of cold on triathlon performance and safety. We focus on the Olympic distance; although cold can clearly be an even larger problem in longer distance events, the nature of the problems remains the same, they are simply more likely to occur, or can be more severe, with longer duration events.

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5.2 Reponses to Immersion

Thermoneutral water temperature for a resting swim-suited adult human is about 35 °C. There is no strict definition of 'cold water', but temperatures below 15 °C can, somewhat arbitrarily, be regarded as 'cold'. The first cold-related risks to a triathlete are the responses evoked by immersion in cold water. These include: the initial responses to immersion; the impact of local cooling on neuromuscular function and therefore performance, and hypothermia.

5.2.1 Cold Shock Response (CSR)

The initial cardio-respiratory responses to immersion in cold water, which peak in swim suited individuals in water temperatures between 10 and 15 °C, represent the initial threat to triathletes. The responses, given the generic name 'Cold Shock' (Fig. 5.1), are initiated by the dynamic response of cold receptors in the superficial subepidermal layer of the skin, and include a decrease in peripheral blood flow (cutaneous vasoconstriction) an increase in cardiac output and blood pressure, a 'gasp' response, an inspiratory shift in end-expiratory lung volume and uncontrollable hyperventilation [3].

The inspiratory gasp and uncontrollable hyperventilation prevent conscious control of breathing and can result in the aspiration of water. Certainly, the inability to control respiration makes the co-ordination of swim stroke and breathing difficult, and can lead to early swim failure [5]. The hypocapnia [6] that results from the hyperventilation probably accounts for the tetany, reduced cerebral blood flow, disorientation and clouding of consciousness observed on cold water immersion [7, 8]. The inspiratory shift in lung volume results in a sensation of dyspnoea.

The combination of the cold shock response and the confusion of a mass start can make this period of a triathlon very challenging, and potentially dangerous.

Saycell et al. [9] recently reported an investigation aimed at providing a scientific rationale for the low water temperature limits for elite, high-level age group triathletes. Twelve (ten male) triathletes undertook 20 min race pace swims in a swimming flume in a range of water temperatures (Tw) wearing race suits (Tw 14 and 16 °C) or triathlon-specific, well-fitting, full-length wetsuits (2–4 mm thick neoprene: Tw 10, 12 and 14 °C). Each swim commenced with a 3 min static upright immersion to allow breathing and heart rate to recover before swimming. After each swim the triathletes attempted a simulated transition and up to a 40 km cycle. For the cycling phase, they wore their race suits, their own triathlon-specific bike shoes and used their own bike. Ambient temperature was maintained at 12 °C and wind speed (fans) for the cycle was 15–20 km h^{-1}, adequate to remove the boundary layer of air surrounding the skin. All volunteers were highly trained, experienced triathletes who either compete in elite youth/junior racing at a national level, or compete internationally in Age-Group World Championships. They were physically fit, healthy and lean (body fat 12% or less for men, 16% or less for women as calculated from the sum of seven skinfolds).

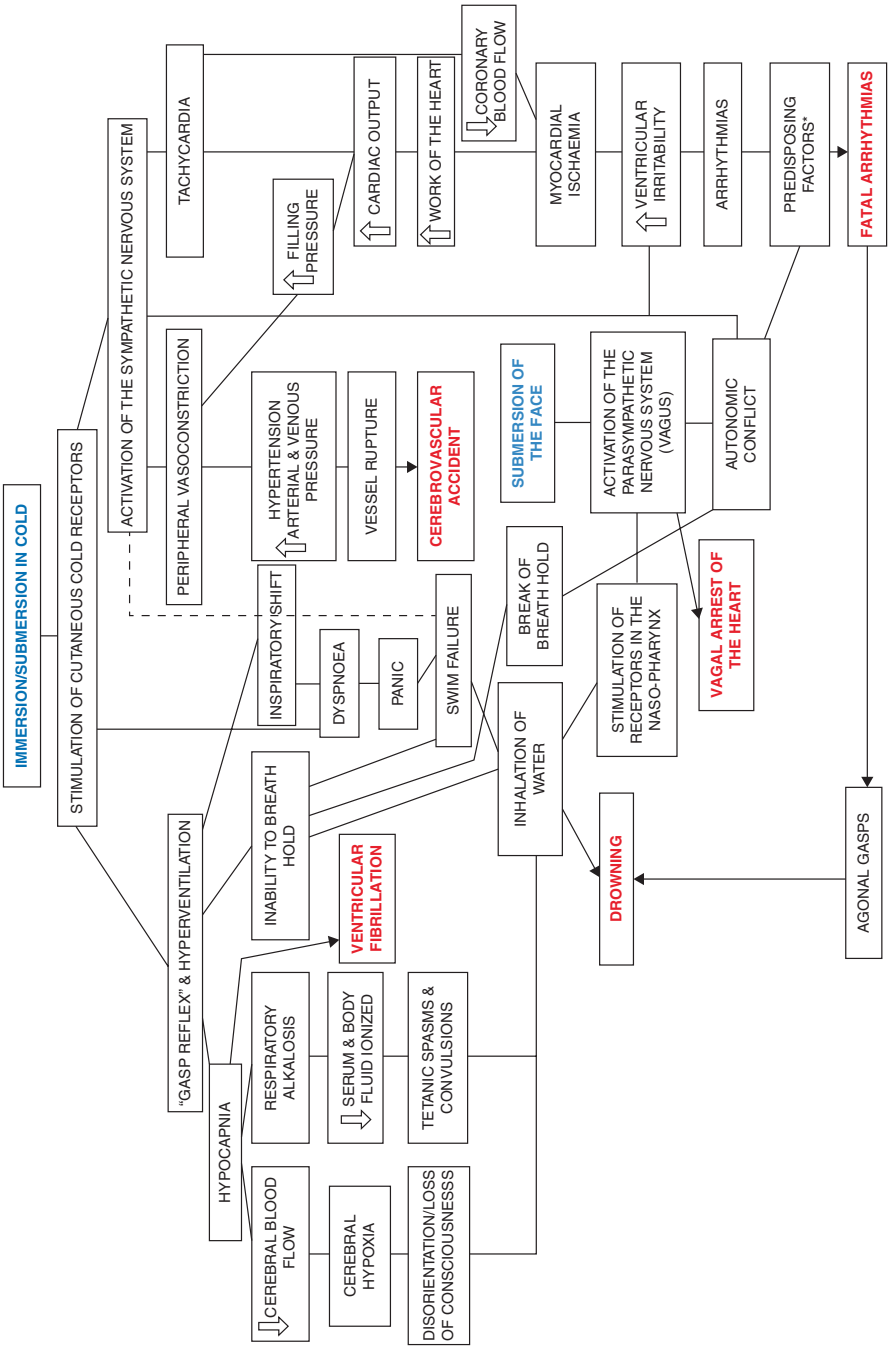

Fig. 5.1 The cold shock response: a contemporary view (Tipton (1989), Shattock and Tipton (2012), Tipton (2016) [2–4] with permission). (Asterisk) Predisposing factors include: Channelopathies; Atherosclerosis; LQTS; Myocardial hypertrophy; Ischaemic heart disease

With regard to cold shock, the results showed a clear and strong heart rate response to entering the water, at all water temperatures, in all conditions. There was some individual variation in the size of the response, but there were no significant differences in the heart rate response between the water temperatures and wetsuit conditions. Despite having an initial static period to allow cold shock to attenuate, four of the seven triathletes who attempted the 10 °C wetsuit condition failed to complete the swim due to cold shock; complaining of breathing difficulties and headache. It is known that for swim suited individuals at least, the cold shock response peaks somewhere between 15 and 10 °C [10], it appears to become problematic for high-level triathletes wearing their own well-fitting wet suits at about 10 °C.

One problem caused by cold shock is the inability to co-ordinate swim stroke breathing pattern with the uncontrollable gasping and hyperventilation on entry into the water and during the first minutes of immersion [5]. However, the respiratory responses are not the only potential hazard. A USA Triathlon Fatality Incidents Study [11] reported that 79% of deaths in triathlons in the USA between 2003 and 2011 occurred during the swim, with unexplained sudden cardiac death being the most likely cause of death in most cases. All of the deaths occurred in open water, and it is a reasonable assumption that many of those that died during the swim will have trained in open water with similar temperatures and conditions. This seems to exclude environmental factors and underlying pathology, in isolation, as a contributory cause, and raises the intriguing question of what it is about a mass participation competitive event that can result in a sudden cardiac death? In 2012 Shattock and Tipton introduced the concept of 'Autonomic Conflict' (AC) as a mechanism for the production of arrhythmias on immersion in cold water. This is the coincidental stimulation of both branches of the autonomic nervous system: the sympathetic arm via cold shock as well as exercise (promoting tachycardia), and the parasympathetic arm by immersion or wetting of the face and the activation of the diving response (promoting bradycardia). When combined with breath holding, AC can produce arrhythmias in over 80% of otherwise young, fit and healthy individuals [12]. It is thought that predisposing factors such as ischaemic heart disease, long Q-T syndrome (chanelopathies or drug-induced) and myocardial hypertrophy may result in more serious, life-threatening, arrhythmias as a result of AC (Fig. 5.2 [2]). But why should this mechanism be more likely in competition than training? In addition to cold shock and exercise, sympathetic stimulation can also be increased by anxiety and competitiveness. In addition, of all the emotions, anger is the one most associated with ventricular fibrillation; it increases the sympathetic tone while maintaining a parasympathetic tone [13, 14]. Anger, anxiety and competitiveness are more likely in a competitive event than training. Also, in a competitive event, parasympathetic activation can occur simultaneously as a result of facial wetting (experienced in training), water entering the nasopharynx (a powerful parasympathetic stimulus) and extended breath holding (both more likely in the confused water of a competitive mass swim than in training (Fig. 5.3); [15]).

Being an electric disturbance of the heart, it is not possible to detect AC postmortem, and other pathology is usually sought (e.g. 'dry drowning') or erroneously

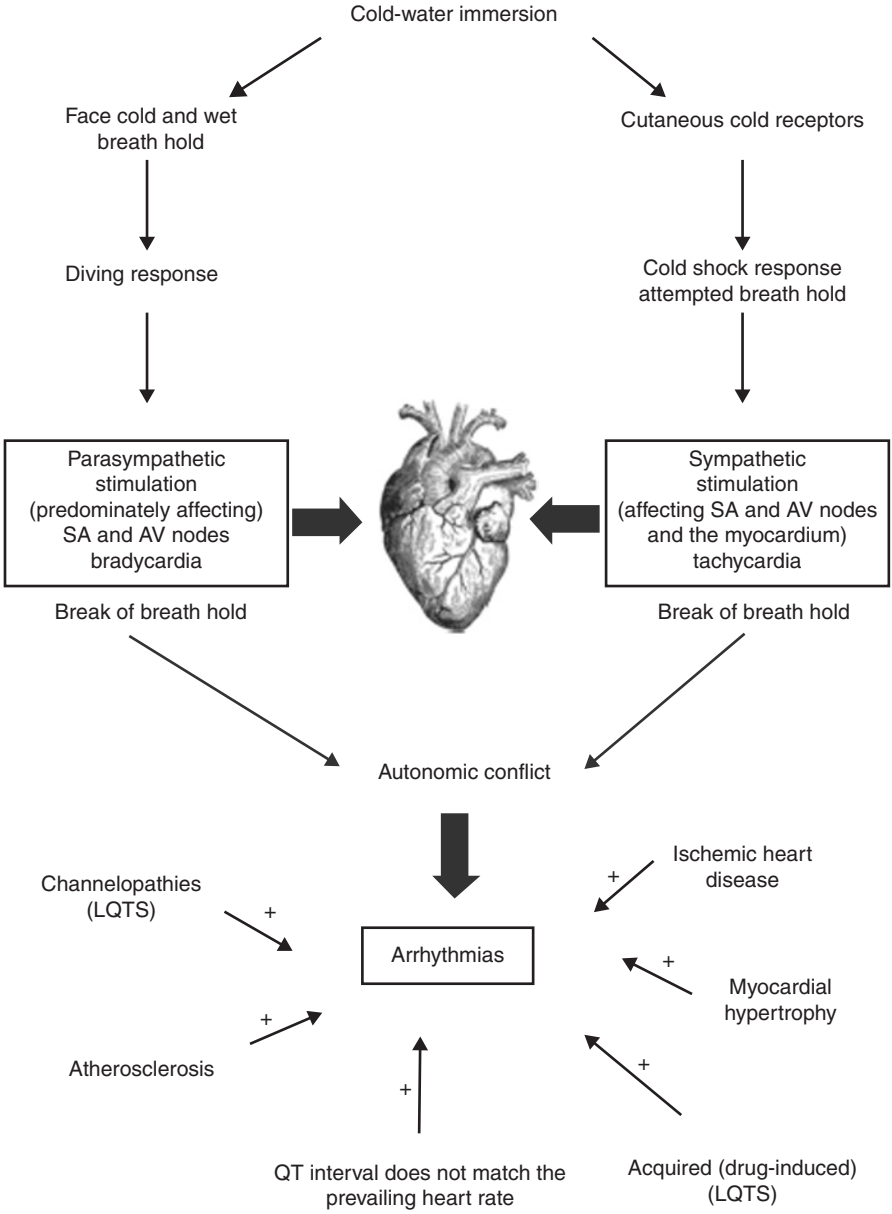

Fig. 5.2 Autonomic conflict. Coincidental stimulation of a bradycardia and tachycardia by the diving (vagal) and cold shock (sympathetic) autonomic responses. This places the heart in conflict leading to arrhythmias, usually when breath holding ceases. Progression to more serious arrhythmias are more likely in individuals with predisposing factors (Shattock and Tipton (2012) [2], with permission)

Fig. 5.3 A triathlon mass start. Note: in addition to intense exercise, the possibility of the need for extended breath-holding, water entering the naso-pharynx, ocular pressure, anger—all precursors to Autonomic Conflict

identified (e.g. drowning). This is especially the case when undetectable cardiac problems result in the agonal aspiration of water [2]. It seems clear that AC is a cause of cardiac arrhythmias during triathlon, but AC may not be unique to immersion; it is entirely plausible that AC may trigger sudden cardiac death in a wide range of sporting and non-sporting scenarios [2].

The vasoconstriction seen on whole-body immersion in water below 33–30 °C for lean individuals [16, 17] not only contributes to the cooling of the extremities (see next section) but, along with the hydrostatic squeeze on immersion, results in an increase in central venous and mean arterial pressures. This increase in pressure initiates *cold-induced/immersion-induced diuresis* via homeostatic mechanisms such as the inhibition of the release of vasopressin. As a result, soon after immersion, urine production increases at the expense of circulating blood volume; 1 h of immersion in 14 °C water increases diuresis by 163% [18], and cold-induced diuresis can reduce circulating plasma volume by 24% [19]. We have seen (Tipton, unpublished) urine production approaching 1 l after 1 h of static immersion in cold water. However, there is evidence that cold-induced diuresis is prevented or reduced by moderate exercise in the cold [20].

For the triathlete it is clearly disadvantageous to remove a significant volume of the circulating blood volume at the start of an endurance event (e.g. by "pre-cooling"). Furthermore, any attempt to pre-hyperhydrate using approaches such as sodium loading can be negated by immersion (Tipton, unpublished). That the magnitude of any cold-induced diuresis may be linked to the time spent in the water not exercising has implications for what triathletes do before their event and the method of pre-adapting to cold water to help attenuate the cold shock response (see below).

5.2.2 Neuromuscular Cooling

The next tissues to cool are the superficial nerves and muscles. Low temperatures affect chemical and physical processes at the cellular level. These decrease, impair

or reduce numerous functions including: metabolic rate (Q_{10} values of 1.6–3); enzymatic activity (e.g. decreased myosin ATPase activity); cross-bridge formation; calcium and acetylcholine release and diffusion rate; calcium sensitivity; nerve membrane excitability; as well as the series elastic components of connective tissues; and increased synovial fluid viscosity [21–23]. The time taken for these changes to occur depends on water temperature, clothing worn and heat production.

The physiological consequences of the changes described above include slowing of action potential propagation (15 m s^{-1} per 10 °C fall in local temperature) with a reduction in their amplitude at nerve temperatures below 20 °C and a loss of synchrony among motor units [24–27]. If nerve temperatures decline from 36 °C down to 20 °C, nerve conduction velocity falls from 49.6 to 7.2 m s^{-1} or at a linear decrease of 1.5–1.8 m s^{-1} per °C [28]. No nerve conduction occurs below local temperatures of 10 °C [29]. Below a muscle temperature of 27 °C the contractile force and rate of force application is reduced, with maximum power output falling by 3% per °C fall in muscle temperature. EMG amplitude increases on cold exposure [21, 30] as more muscle fibres must be recruited to perform a given work output, the power frequency distribution shifts to the left, similar to that observed with fatigue [21]. There is a slowing of twitch and tetanic contractions [25, 30], and an increased level of co-activation of agonist and antagonistic muscle pairs.

As a result of these temperature-related impairments there is a consequent reduction in the speed of movement, dexterity, strength and mechanical efficiency. For example, maximum dynamic strength, power output, jumping and sprinting performance are related to muscle temperature with reductions ranging from 4–6% per °C fall in muscle temperature down to 30 °C [31, 32]. Davies and Young [33] reported a decline in peak (31%) and average (44%) power output on a force bicycle as muscle temperature was reduced by 8.8 °C. These authors also observed a shift to the left in the force-velocity curve in cold muscles compared to control conditions. Numerous studies have demonstrated that cold exposure reduces manual dexterity [34, 35], with skin temperature being the primary correlate of manual dexterity, and with manual dexterity declining more rapidly as finger skin temperature decreases below 15 °C [34, 35].

The arms are particularly vulnerable to cooling due to their cylindrical shape, short-conductive pathway from the centre of the limb to the surface, large surface area to mass ratio and the superficial pathway of the ulnar and median nerves and musculature. With cooling, hand function is affected first and then whole body activity, such as swimming or self-rescue, that is dependent on arm function. Eventually, physical incapacitation and swim failure occur. Giesbrecht et al. [36] demonstrated that local muscle cooling, as opposed to whole body hypothermia, accounts for most of the impact on hand and arm performance, and the data from a few studies have suggested that the temperature of the triceps in particular can have an influence on swimming performance. Tipton et al. [37] reported that the reduction in swimming performance in cold water (as low as 10 °C), as defined by the change in swimming efficiency, was most closely correlated with the sum of the skinfold sites of the upper limbs (triceps, outer forearm, and subscapular, $R^2 = 0.68$).

Wallingford et al. [38] examined the influence of arm skinfold thickness during clothed mock survival swimming in 14 °C water. Triceps skinfold thickness showed a significant correlation ($r = 0.7$) with swim distance. The authors concluded that, for a healthy individual, triceps skinfold thickness is a stronger predictor of swimming distance in cold water than body adiposity, aerobic fitness or the fall in deep body temperature.

Lounsbury and Ducharme [39] examined whether providing arm insulation reduced the incidence of swim failure and improved swimming performance in a swimming flume. Deep body cooling rates during swimming were slower with arm insulation, but arm insulation did not greatly improve physical performances. However, video analysis showed that swimming technique with arm protection was maintained 10–15% better than without it between minutes 30 and 50. The authors conclude that equipping volunteers with neoprene armbands appears to have partially preserved muscle function, but with unimpressive effects on overall performance. They further conclude that swim failure is a complex entity that is related to both triceps skinfold and arm girth.

The changes in physiological function as a result of neuromuscular cooling can result in early swim failure across the spectrum of novice to elite triathletes, with novices suffering the biggest decrements, probably due to having a less entrained motor programme for swimming and therefore a more vulnerable technique. As we shall discuss below, for triathletes, the impact of the neuromuscular cooling incurred during a swim may not be limited to that swim, and may have consequences for the transition from swimming to cycling (T1) and the early stages of the cycle.

5.2.3 Deep Body Cooling

It is unlikely that any adult will become hypothermic (deep body temperature below 35 °C) in any water temperature in less than 30 min, even when swimming in just a swimming costume. However, deep body temperature may fall during both shorter and longer triathlons with implications for performance in the shorter swims and performance and safety in longer events.

The large body mass and consequent thermal inertia of an adult human mean that thermal changes in the deep tissues of the body take time to occur [40]. They also take time to be reversed when the thermal circumstances of the body change. The deep body (rectal) temperature response to a 20 min triathlon swim in a range of cold water temperatures is shown in Figs. 5.4 and 5.5, these data are from the paper of Saycell et al. [9] described above. There are several typical patterns in these data that are worth noting:

1. In both the wet suit and race suit swims it usually takes a little time for deep body temperature to start to fall. This is related to the conductive pathway between the skin and the deep body tissues, and the time taken for that pathway to be estab-

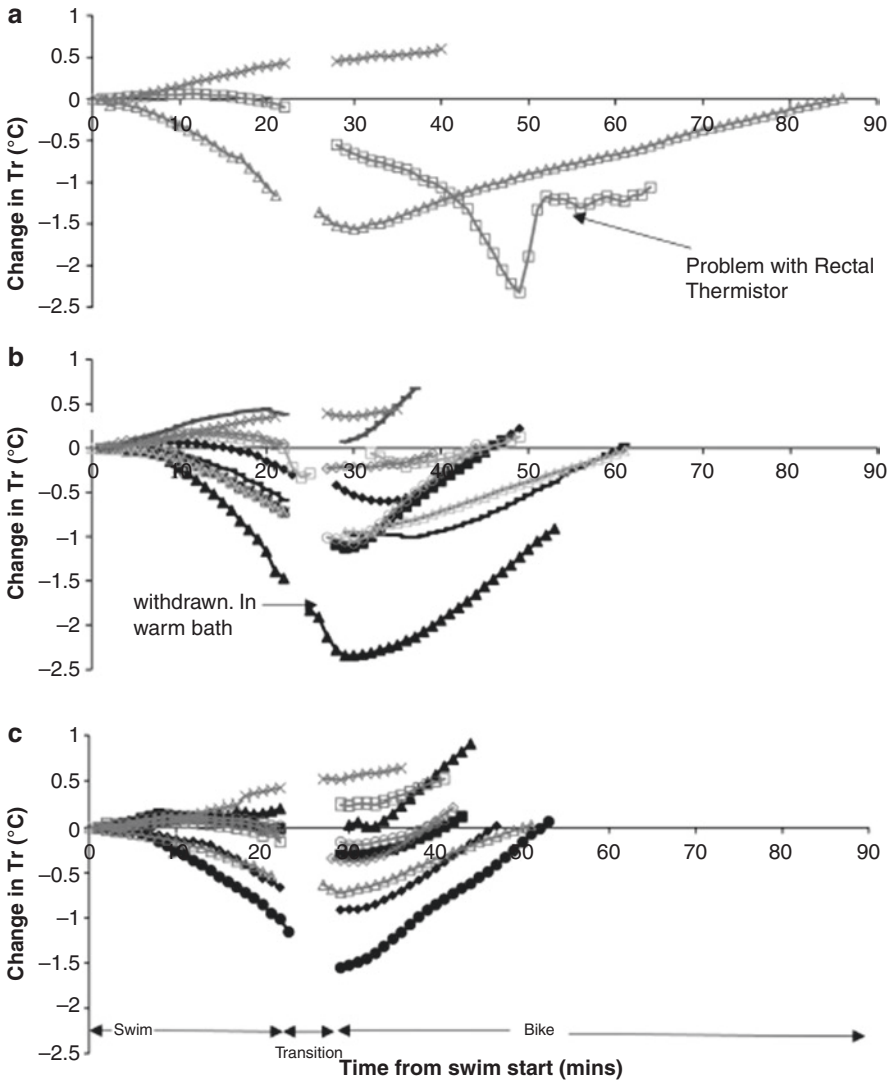

Fig. 5.4 Deep body (rectal) temperature changes for triathletes during a 20 min wet suit swim, transition and up to 40 km cycle. (**a**) Tw 10 °C, $n = 3$ (**b**) Tw 12 °C, $n = 10$ (**c**) Tw 14 °C, $n = 9$. Air temperature 12 °C, 15–20 km h^{-1} wind speed (From Saycell et al. (2018) [9], with permission)

lished and heat to start flowing from the body. During this time the skin and superficial tissues of the body are cooling (see above) [40].

2. Following a swim the deep body temperature continues to fall during transition and, in many cases, the early part of the cycle. In the situation of a triathlon where the arms do most of the work during the swim and the legs are relatively

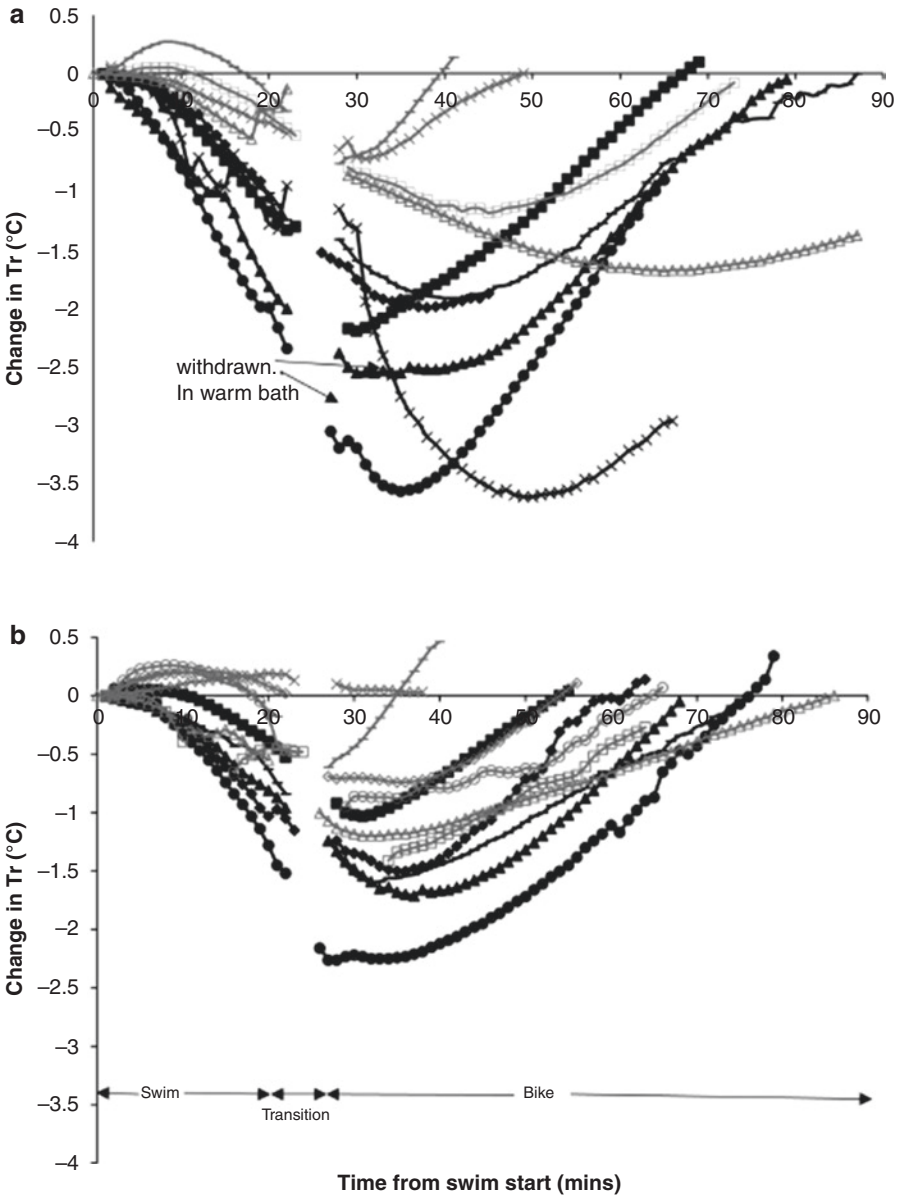

Fig. 5.5 Deep body (rectal) temperature changes for triathletes during a 20 min race suit swim, transition and up to 40 km cycle (**a**) Tw 14 °C, $n = 10$ (**b**) Tw 16, $n = 11$. Air temperature 12 °C, 15–20 km h^{-1} wind speed (From Saycell et al. (2018) [9], with permission)

passive, the continued fall in deep body temperature following the swim is due to a combination of: continued conductive heat loss within the body down the physical thermal gradient established during the swim—this will take time to reverse [41]; the introduction of evaporative cooling due to leaving the water wet; the re-perfusion, due to running in transition and then cycling, of leg muscles cooled during the swim and consequent cooling of blood that returns to the deep tissues [42, 43].

3. As might be expected, the changes in deep body temperature are smaller when a wet suit is worn compared to a race suit. On average, the fall in the deep body (rectal) temperature of elite triathletes was five times greater over 20 min swims in 14 °C water when wearing a race suit compared to a wet suit [9].
4. There is wide variation in the changes seen between individuals in deep body temperature. This is due to numerous thermal and non-thermal factors that include: water temperature, fitness, body morphology, exercise intensity, fatigue, sex, age, clothing quality and fit [44] (Fig. 5.5).

With longer swims and continued cooling, hypothermia is possible. The classic signs, symptoms and clinical features of hypothermia include:

- *Mild* (deep body temperatures 35–32 °C): mild incoordination, cold extremities, tachycardia, shivering.
- *Moderate* (deep body temperatures 31–28 °C): apathy, poor judgement, slurred speech, amnesia, clumsiness, weakness and fatigue, dehydration, reduced level of consciousness.
- *Severe* (deep body temperatures <28 °C): reduced level of consciousness/unconsciousness, cardiac arrhythmia, hypotension and bradycardia, muscle rigidity, pulmonary oedema, lack of tendon reflexes, fixed dilated pupils.

In triathlon the clinical picture is somewhat confused by the swimming that is being undertaken during the cold exposure; swimming hides some of the signs and symptoms of hypothermia (e.g. shivering, tachycardia, tachypnoea). Normally, swim impairment/failure, due to peripheral neuromuscular cooling, will occur before moderate or severe hypothermia, and a deterioration in swim performance is therefore a sign of impending significant deep body cooling [37]. However, anecdotal accounts (Phil Rush] [3-way English Channel swimmer] personal communication to author) suggest that if an individual is highly motivated and swimming hard they may be able to swim to the point of unconsciousness, which then intervenes suddenly. In theory this is possible if the muscle blood flow associated with intense exercise mixes deep and more superficial tissue temperatures so the gradient between them is small, muscle function may then continue down to a muscle temperature of 27 °C while unconsciousness intervenes at a brain temperature of 30–33 °C. However, as noted, normally

a large temperature gradient between the deep body tissues and superficial tissues results in neuromuscular impairment before unconsciousness.

In cold water, a fall in deep body temperature intensifies shivering, which raises oxygen consumption during submaximal exercise (9% in water at 25 °C; 25.3% in water at 18 °C). The increase is greater in leaner individuals. Thus, the energy cost of submaximal exercise is increased in water cooler than 26 °C; this can result in a more rapid depletion of carbohydrate and lipid energy sources and earlier onset of fatigue.

In terms of the actual falls in deep body temperature recorded during swimming in cool and cold water, Tipton et al. [37] studied ten competent swimmers up to elite level attempting to swim breaststroke in a flume for 90 min wearing just a swimming costume in water at 10, 18 and 25 °C. All ten swimmers completed 90 min swims at 25 °C, eight completed the swims at 18 °C, and five at 10 °C. In 10 °C water, one swimmer reached swim failure after 61 min and four were withdrawn before 90 min with rectal temperatures of 35 °C and close to swim failure. Swimming efficiency and length of stroke decreased more, and rate of stroke and swim angle increased more, in 10 °C water than in warmer water. These variables seemed to characterise impending swim failure (Fig. 5.6). More recently, Saycell et al. [9]

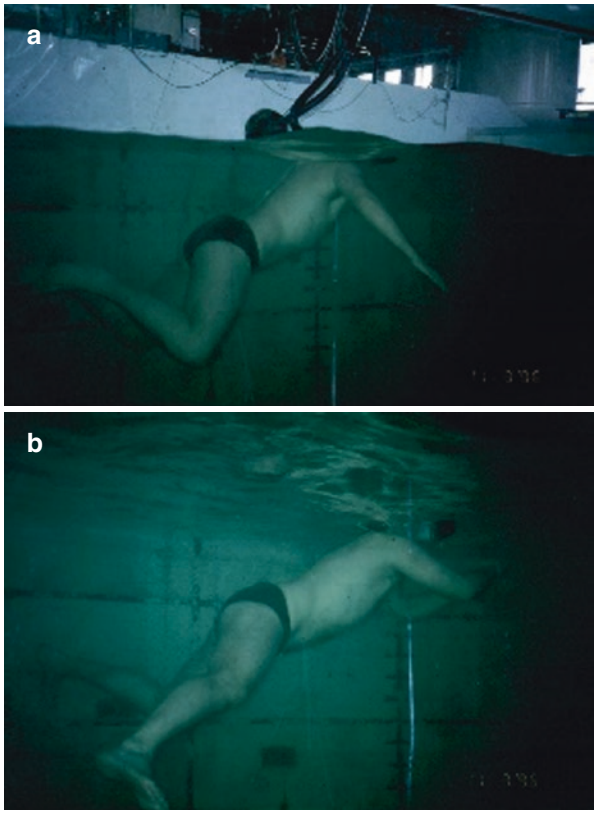

Fig. 5.6 Example of impending swim failure. (**a**) International class breaststroke swimmer after 5 min swimming in a flume in 10 °C water. (**b**) Same swimmer after 60 min, note: uncoordinated, inefficient stroke, more upright posture in the water increasing drag and sinking force and decreasing efficiency (Photos: M Tipton. For details of experiment see Tipton et al. (1999) [37]) (From Golden, F. St.C & Tipton, M. J. (2002) Essentials of sea survival. Human kinetics, Illinois. ISBN 0-7360-0215-4)

failed to observe the reduction in functional swimming capability or swim speed noted by Tipton et al. [37]. The main reason for this will have been the shorter swim times (20 min) in the study of Saycell et al. [9]. A part of the reason may also have been that the triathletes were highly trained and were therefore able to work at high levels of heat production, thereby better maintaining muscle temperature and function. The penalty for doing this is a loss of overall tissue insulation and a more rapid fall in deep body temperature than might normally be expected (Fig. 5.5). This combination of hard work (high heat production), well-perfused muscles and thermal mixing of deep and more superficial tissues in lean, fit individuals are the prerequisites needed to swim to unconsciousness, described above.

In terms of performance, with generalised muscle and deep body cooling, there is a decrease in limb blood flow; this has occurred by the time deep body temperature reaches 36 °C [45]. The exercise hyperaemia normally observed when swimming in warm water is probably attenuated in cooled individuals by a sympathetically mediated vasoconstriction of muscle resistance vessels [45, 46]. Consequently, oxygen delivery to, and utilisation by, cooled working muscle, and the removal of the end products of metabolism, may be reduced in cold water. This, plus a left-shift in the oxygen dissociation curve with cooling means that cooled muscle is required to use anaerobic metabolism at lower sub-maximal workloads, resulting in an earlier appearance of blood lactate [47], more rapid depletion of carbohydrate stores and an earlier onset of fatigue [48]. Higher oxygen consumptions, particularly in lean individuals, have been noted in cold compared with neutral environments during exercise requiring oxygen consumptions of up to 2.0 L min^{-1} [47, 49], but not at 3.0 L min^{-1} [50]; at the higher workload the superimposition of shivering on exercise is prevented by the muscles being fully recruited to support the exercise; the graded inhibition of shivering during exercise is thought to be of central origin [43].

Increased muscle tone during submaximal exercise in cold water may further reduce mechanical efficiency by increasing the activity of antagonistic muscles [43, 45]. Maximum aerobic capacity ($\dot{V}O_{2max}$) falls in relation to muscle and deep body temperature, with a 0.5 °C fall in deep body temperature resulting in a 10–30% fall in $\dot{V}O_{2max}$ and maximum cardiac output [31, 32, 45, 47].

5.3 Transition

Following a swim in water that results in body cooling, that cooling will normally continue through T1 (Figs. 5.4 and 5.5). The run from the swim, change of clothes, run out with a bike, bike mount and cycling amongst other competitors requires spatial awareness, co-ordination, balance, fine motor skills and dexterity. There is a paucity of work examining the impact of ambient temperature or body temperature on transition performance. However, we can conclude from more general work undertaken on the impact of cold, that most of the skills and capabilities required in transition are negatively impacted by cold. We have already described how even short-term superficial cooling can impact a wide

range of neuromuscular functions. Even moderate body cooling can impair cognitive function and psychomotor skills: cold exposure and body cooling have been shown to affect working memory, choice reaction time and executive function [51–53]. Cold exposure can also degrade both static and dynamic balance [54, 55].

The ability to handle a transition and cycle a technical course soon after swimming is an event-specific skill, related to the risk of accidents in T1 and at the start of the bike element of a triathlon. The recent study of Saycell et al. [9] looked specifically at the impact of cooling during a swim on T1. The psychomotor (four-choice reaction test), grip strength and bike control skills on and off the bike were tested before and immediately after a 20 min swim in a race suit and wet suit in water ranging from 10 to 16 °C. The test of bike control involved a short course (about 10 m) with a restriction and corner, with tram lines 20 cm apart (wider at the corner) marked out with tape on a non-slip floor. The elite triathletes ran through the course with their bike in one direction and cycled back through it in the opposite direction, they were asked to complete the course as quickly as possible without making any 'errors', defined as a front wheel over a tram line or putting a foot down. The problems and decrements observed occurred following the coldest swims. One triathlete did not feel able to complete transition following the 12 °C wetsuit swim. Following the 14 °C race suit swim most of the participants struggled, two were withdrawn after the swim, half of the remaining athletes (four out of eight) were not considered able to complete the T1 course safely. Of those triathletes that did complete T1, almost all had an increase in the number of bike handling errors in the colder compared to warmer conditions (e.g. race suit 14 °C vs. 16 °C; wet suit 12 °C vs. 14 °C). Four-choice reaction time was slowed following the swims in race suits. Across conditions the time taken to complete the bike course was not affected by the swim, but error rate was, with almost all triathletes showing, as expected, a greater increase in error after the swims resulting in the coldest body temperatures. Of course, these results are specific to the conditions undertaken, as swim time increases, water temperature falls or insulation worn reduces, the impact of a swim on T1 will be increased.

It is concluded that after a swim in cool water, deep body temperature continues to fall through T1 and can have an impact on performance in T1. It appears that when even moderately impaired by cooling from the preceding swim, triathletes sacrifice accuracy for speed during T1.

5.4 Cycling and Running

At the start of a triathlon cycle the body of a triathlete is in a complex thermal state; it can still be cooling from the swim, it is now in air rather than water but the surface of the body is wet so there is enhanced evaporative cooling at the start of the cycle and forced convection increases with bike speed, at the same time heat production is increasing rapidly due to exercise intensity. Because of the differences in the thermal properties of air and water, and the level of heat production of triathletes

when cycling and running, it is highly probable that deep body temperature will increase during these phases. In support of this, the thermal responses of a 25-year-old male (70 kg body mass, 1.8 m height, 8% body fat and 1.83 m^2 surface area) undertaking one hour of cycling (power outputs of 250 and 350 W, metabolic rates of 546 and 765 W m^{-2} respectively) in clothing equivalent to a race suit, after 20 min swims in 14 °C with wetsuits and 16 °C without wetsuits, has been mathematically modelled [56] for air temperatures ranging from 5 and 15 °C. The output from the model predicted a rectal temperature of greater than 37 °C. Even starting with a lowered post-swim rectal temperature of 36 °C, deep body temperature was predicted to exceed 37 °C after 1 h of cycling. The only scenario where rectal temperature was predicted to decrease was when clothing insulation was completely removed from the model, and then only for 5 °C air at the lower cycling power output (250 W). It was concluded that in the circumstance of an elite triathlon, in air temperatures of between 5 and 15 °C, the heat produced by these athletes should result in positive thermal balance and, therefore, deep body temperature should increase. This conclusion should be taken with some caution and further research and modelling in this area is recommended. Non-elite athletes, with a lower cycling power output, may not be able to achieve a positive thermal balance at low (circa. <5 °C) air temperatures.

In some special and generally rare circumstances cooling can occur during cycling and running. These circumstances always include a combination of very cold air temperatures, rain or snow and a reduction in heat production due to weather conditions, road conditions, exhaustion or injury [57–59]. If deep body cooling does occur the responses observed will be the same as those described above (neuromuscular cooling, hypothermia) for the swim.

Much more probable than hypothermia during the cycle or run is that the extremities of a triathlete will remain cold for some time into the cycle; at least until such time as the heat debt accumulated in the swim is reversed and deep body temperature begins to rise, resulting in a peripheral vasodilatation and increased blood flow to the extremities—the major source of heat for these areas. This may have consequences for bike handling in the early part of a cycle. Saycell et al. [9] report that following a 20 min swim in 14 °C wearing a race suit, four out of eight elite triathletes were unable to grip the handlebars of their bike due to cold hands. It can be seen from Figs. 5.4 and 5.5 that the lowest deep body (rectal) temperatures recorded in this study were during the initial part of the cycle, the major reason being, as discussed, the ongoing impact of the swim in cold water. Despite the fact that the elite triathletes were cycling at ‘race pace’ for a 40 km bike leg, it took an average of between 1.4 and 10.6 min to reverse the fall in deep body temperature established in the swim. For those that finished, the deep body temperature of all but one of the triathletes returned to pre-swim, resting levels but this took an average of between 10.6 and 54.6 min. The static cycle was conducted in air at 12 °C with a wind speed of (fans) of 15–20 km h^{-1}. The time taken to reverse the fall in deep body temperature during the bike ride did not differ between race suit and wet suit conditions, confirming that this continuation of cooling is primarily the result of a physical process set up during the swim [41]. However, the use of a

wet suit in the swim did reduce the time taken to return to pre-swim deep body temperatures.

Clearly, the data presented above are specific to the individuals and conditions tested. However it does seem reasonable to conclude that following a cold water swim, the thermal response seen during a triathlon cycle are influenced by the preceding swim and, as a consequence, during the cycle body temperatures may be falling initially and lower than would be the case if just a cycle was undertaken. The implications of this unusual thermal profile for training, performance and nutrition/hydration are yet to be fully elucidated.

5.5 Prevention, Mitigation and Treatment

In this final section, we examine how the problems associated with the cold can be prevented, mitigated or treated. The primary interventions are technical, physiological, technological and organisational. The starting assumption is that the person who enters the triathlon is reasonably fit and healthy.

5.5.1 Rules

The International Triathlon Union (ITU) has rules for water temperature and wet suit use. Until recently elite racing was permitted in water above 13 °C, with wetsuits mandatory below 14 °C, optional between 14 and 20 °C and forbidden above this. Recent research on elite triathletes [9] demonstrated that without wetsuits, a water temperature of 14 °C was too cold for racing due to the rapid fall in deep body temperature and inability to safely control a bike. A water temperature of 10 °C even with a good fitting, full length wet suit was too cold due to the cold shock response. As a result of this research, the ITU rules for racing in lower water temperatures were amended to allow racing from 12 °C with wetsuits mandatory up to 16 °C (ITU [60]).

5.5.2 Physiology

The cold shock response is of less magnitude in fitter individuals but still occurs. It can be habituated [61] with as few as 5 × 3 min immersions in a representative water temperature. A 40% reduction in the cold shock response has been reported, with up to 31% of the habituation remaining 7 months later, even without any intervening cold water exposures [62]. The water temperature used to habituate can be warmer than that expected in completion and still be of benefit in terms of attenuating the cold shock response [63]. Cold showers [64] and psychological skills training [65] can also reduce the cold shock response.

Unfortunately, the impairments suffered as a result of neuromuscular cooling do not respond to habituation or acclimation. In terms of swimming the evidence is that maintaining the temperature of the triceps may be important [37].

With longer-term swimming immersions in cold water, subcutaneous fat provides the majority of the resistance to heat loss from the body [66].

5.5.3 Technology

For a triathlete, a correctly (tightly) fitting wet suit is the best protection again the cold-related impairments of cold water. Other clothing that may be necessary in some more adverse climatic conditions includes cycling gloves and wind and waterproof outer layers. It is particularly useful to have the ability to vary the level of insulation and wind and waterproofing depending on alterations in exercise intensity and environmental conditions with, for example, altitude. In the 2014 Nice Ironman triathlon the conditions in the city for the swim and run were warm (water temperature 23 °C; air temperature 20 °C with high radiant heat load); however, the cycle climbs 1800 m into the surrounding mountains where it was raining (sleet), windy and the air temperature was <10 °C. The road conditions meant that the triathletes had to reduce speed (power output/heat production), this combination of factors moved the heat balance equation in favour of heat loss and some competitors began to cool. Thankfully the organisers of the event had the forethought to obtain and hand out disposable wind and waterproof ponchos in the mountains.

5.5.4 Prevention and Treatment

Those organising and supervising an event should be aware of the dangers associated with cooling, in particular cold shock, autonomic conflict, swim failure and the possibility of poor bike handling capability in T1 and early in the cycle. These are briefly addressed in Table 5.1, it should be noted that the level of evidence underpinning many of these recommendations is weak/hypothetical; this area requires further investigation.

Table 5.1 Potential cold-related problems during triathlon and some suggested organisational and supervisory mitigations

Potential problem	Mitigation
Cold shock response on initial immersion/drowning	1. Allow an initial period of immersion for attenuation of the cold shock response 2. Set triathletes off in a controlled manner in small groups (‘waves’) 3. Start the event from the water 4. Provide sufficient cover in first 400 m of course; time to rescue and medical intervention is critical

(continued)

Table 5.1 (continued)

Potential problem	Mitigation
Autonomic Conflict/Sudden cardiac death These are aimed at minimising the need for breath holding, the chance of aspirating water into the nasopharynx, and the potential for crowding, conflicts and anger [67]	1. Limit wave/group sizes 2. Have a wide start line/course width, with the caveat that it can be properly surveyed 3. Have reasonable time gaps between wave starts 4. Have a good number of easily visible (from water level) buoys to prevent sharp turns and poor navigation 5. At the start, have as long a straight line distance before requiring swimmers to make a turn, allowing the swimmers to spread out and find their own pace 6. Ask swimmers to 'self-select' into waves of appropriate ability or ask weaker/novice swimmers to start at the back of a wave 7. Advocate: acclimatisation, reduced anxiety and anger management 8. Given that Autonomic Conflict is most likely when swimming in large groups and that the first part of a swim (up to 400 m) is where the greatest number of incidents occur, increase the amount of safety cover in the first 400-m section and at turns. Time to rescue and intervention is critical 9. Brief swimmers to take their time at the start (particularly if a slower, less fit, or a novice swimmer)
Swim failure	1. Recognise the early signs of swim failure (Fig. 5.6) *i.e.* shortening of stroke, increased stroke frequency, more upright posture in the water 2. Appreciate the possibility that in some circumstances (cold water, longer duration events, triathlete working hard) people may swim until unconscious 3. Identify those at particular risk of getting cold: (a) Slow swimmers with poorly fitting wet suits (b) Very lean individuals: the most vulnerable athletes are those with a sum of seven skinfold measurements of 40 mm or less [9] (c) Encourage the use of wetsuits
Bike handling in T1 and early in bike stage	1. Provide sufficient space for T1 2. Avoid technical sections early in the bike ride
Hypothermia	1. Be aware of the signs, symptoms and clinical features of hypothermia 2. Remember, athletes may have their lowest deep body temperatures during the early part of the cycle 3. Be aware of conditions likely to result in body cooling: combination of very cold air temperatures, rain or snow and a reduction in heat production due to weather conditions, road conditions, exhaustion or injury

5.6 Out of Hospital Treatment

The primary cold-related injuries that are most likely to require treatment are drowning, cardiac arrest and hypothermia.

Persons suspected of aspirating water (abnormal lung sounds, severe cough, wheezing, blue lips and nailbeds, rapid shallow breathing, rapid pulse, frothy sputum/foamy material in the airway, depressed mental state or hypotension) should be evacuated to advanced medical care. Both sea and fresh water drowning represent a form of suffocation and therefore the speedy relief of hypoxia is the primary treatment objective and the one that has the largest impact on outcome (best result when treatment received in under 10 min). Present recommendations are for a high fraction of inspired oxygen, delivered by any means possible. Other objectives are restoration of cardiovascular stability, prevention of further heat loss (wind proof/waterproof out layer and insulation, e.g. heavy duty plastic bag and blankets) and speedy evacuation to hospital.

Because cardiac arrest in drowning is a consequence of anoxia, Basic Life Support should include ventilations and chest compression [68]. Ventricular fibrillation is rare in drowning (<10%) so incorporation of AED in the initial minutes of drowning treatment should not interfere with oxygenation and ventilation. Otherwise AED use should be considered and is not contraindicated in a wet environment [69].

Immersion hypothermia is usually 'acute'; occurring rapidly due to the cooling power of the environment. Those with mild hypothermia can be given a warm sweet drink for comfort, fluid and energy, and external heat applied to speed the return of thermal comfort. Thus, external active rewarming is permissible in cases of mild hypothermia but should be avoided in severe hypothermia. Fully conscious casualties can be removed and rewarmed in a hot bath (39–40 °C) or seated (not standing) in a hot shower. Rewarming should cease before the casualty becomes too hot (i.e. the appearance of sweat); over-warming can result in rewarming collapse.

Those suspected of suffering from severe hypothermia should be regarded as critically ill and handled as little and as gently as possible; the risk of ventricular fibrillation is increased in a cold myocardium and can be precipitated by movement, cardio-pulmonary resuscitation (CPR), insertion of a supraglottic airway or tracheal intubation. Unconscious casualties, whose airways are not under threat, should be carefully removed from the water horizontal and kept in this position during evacuation to hospital. Out-of-hospital, moderate and severe hypothermic casualties can be treated with passive rewarming (insulate and allow the casualty's metabolism to rewarm then slowly ~0.75–1.0 °C h^{-1}); this is the safest way to rewarm [70, 71]. Patients in cardiac arrest should receive continuous cardiopulmonary resuscitation during transfer to advanced care; preferably a centre with expertise and experience in extracorporeal life support. If continuous CPR is not possible, then either delay starting CPR, institute mechanical CPR or administer CPR intermittently during evacuation [72, 73].

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Part III

Acute and Overuse Injuries

Iliotibial Band Syndrome (ITBS)

6

Sergio Migliorini, Marco Merlo, and Lisa Migliorini

6.1 Introduction

Iliotibial band syndrome (ITBS) is an overuse injury caused by repetitive friction of the iliotibial band (ITB) and underlying bursa across the lateral femoral epicondyle. ITBS is the most common causes of lateral knee pain in runners, with an incidence estimated to be between 5% and 14% of all the overuse injuries related to running [1–3]. Renne, in 1975, first described the ITBS in marines, who underwent heavy endurance training [4]. Considering the success of road endurance races in these years, the ITBS diagnosis is now really frequent and other sports disciplines, such as triathlon and cycling, are involved in that pathology.

6.2 Etiopathogenesis

The ITB is considered a continuation of the tendinous portion of the tensor fascia lata muscle and is indirectly attached to parts of the gluteus medius, glutes maximus and the vastus lateralis muscles. The intermuscular septum connects the ITB to the line aspera femuris until just proximal to the femoral lateral epicondyle. Distally, the ITB spans out and inserts on the lateral border of the patella, the lateral patellar retinaculum, and Gerdy tibial tubercle. The ITB is only free from bony attachment between the superior aspect on the lateral femoral epicondyle and Gerdy tubercle

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[5, 6]. A bursa, like synovial tissue, insinuates under the ITB and acts as an interface between ITB and lateral femoral epicondyle [7].

The ITB assists the tensor fascia lata as it abducts the thigh and controls and decelerates adduction of the thigh.

With the knee in full extension and until 20°–30° flexion, the ITB lies anteriorly to the femoral lateral epicondyle and acts as an active knee extensor. From 20° to 30° of knee flexion, the ITB is posterior to the femoral lateral epicondyle and acts like an active knee flexor [8]. During walking and running, the ITB in an important stabilizer of the lower limb.

A biomechanical study of runners found that the posterior edge of the band impinges against the lateral epicondyle just after foot strike in the gait cycle, and the friction occurs at or slightly less than 30° of knee flexion. Orchard [9] suggests that the impingement, in runners who had ITBS, occurs in the foot contact phase at an average 21.4° ± 4.3° angle knee flexion, at or slightly below the 30° of flexion traditionally described in the literature (Fig. 6.1).

As in the other overuse injuries, the ITBS has intrinsic and extrinsic causes [10–12] (Table 6.1).

The most important extrinsic factors are the training errors (sudden increases in mileage or intensity, excessive running in the same direction on the track, hill running and time trial training, lack of warm-up, insufficient muscle stretching execution), and bad shoes, inadequate to the running biomechanics. In triathlon, the transition cycling to running, with the change from concentric muscular contractions of cycling to eccentric contraction of running, and from unloaded cycling phase to the load state of running, is an extremely delicate phase in which some kilometers are required to regain neuromuscular efficiency and elasticity

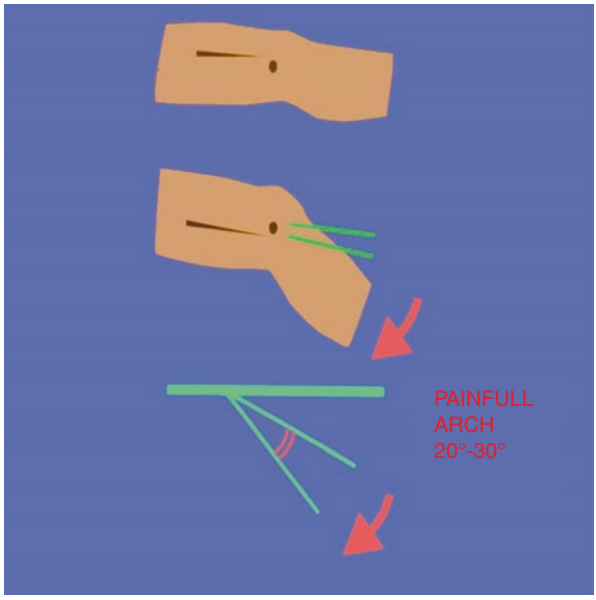

Fig. 6.1 During running the friction between ITB and lateral femoral epicondyle occurs at 20°–30° of knee flexion

Table 6.1 ITBS intrinsic and extrinsic factors

Intrinsic	Extrinsic
Varus knee	High impact running styles
Internal tibial rotation	Training errors
Cavus-varus foot	Hill running
Femoral antetorsion	Insufficient muscle stretching
Knee lateral laxity	Bad or inadequate running shoes
Excessive foot pronation	Incorrect cleat pedal and saddle alignment
Hip abductors weakness	Cycling to running transition in triathlon
Lower leg discrepancies	

indispensable for proper running style [12–14]. In this phase, the inability to dissipate the load forces of the locomotor apparatus by the lower limb can favor the transmission of stress to the knee [15]. The runner's training at the lower pace and the high impact running style induce the friction of ITB with less than 30° knee flexion at the footstrike.

In cycling, the abnormal lateral knee stress is most often the result of incorrect cleat pedal alignments, particularly with varus knee or external tibial rotation greater than 20°, when riding with internally rotated cleats. Generally, this injury has increased since the introduction of rigid clipless pedal systems in 1985. Also a too high saddle results in knee extension behind 150° causing the distally ITB to abrade across the lateral femoral epicondyle.

The surfaces of activity can also contribute to the development of ITBS in runners: running on surfaces with excessive camber can put excess strain on the lateral aspect of the knee. Downhill running tends to be worse because of the decrease in knee flexion that is present at the time of footstrike, thus increasing the force experienced by the knee within the Orchard impingement zone [9].

Anatomic factors that contribute to ITBS include excessive varus knee, excessive internal tibial torsion, foot pronation, hip abductor weakness, lower leg length discrepancies, cavus foot, femoral antetorsion. Fredericson [16–18] found that runners with ITBS had significant weakness in the hip abductors of their affected limb; when these muscles do not act properly throughout the support phase of the running cycle, there is a decreased ability to stabilize the pelvis and eccentrically control the femoral abduction. As a result, other muscles must compensate, often leading to excessive soft tissue tightness and myofascial restrictions. The fatigued runner, and the fatigued triathlete after the cycling, or those who have a weak gluteus medius are prone to increase thigh adduction and internal rotation at midstance, leading to an increased valgus vector at the knee. These condition increases tension on the ITB, making it more prone to impingement to the lateral femoral epicondyle, especially during the foot contact, when maximal deceleration absorbs ground reaction forces. In leg length discrepancy, the ITBS is more frequent in the lower leg due to the increasing of varus forefoot and increased knee Q angle [19].

Sixty-five percent of athletes with ITBS are male, due to greater training volume and more number of practitioners [1]. In women, the incidence of ITBS is less than men because of anatomical factors: valgus knee, medial knee laxity, femoral lateral epicondyle less prominent. Considering the gender differences in gait mechanics Phinyomark

[20] suggests that female ITBS runners exhibited significantly greater hip external rotation compared with female healthy runners. On the contrary, male ITBS runners exhibited significantly greater ankle internal rotation compared with healthy males.

Also the lack of muscular elasticity of the tensor fascia lata, knee and hip flexors, is an injury risk factor, particularly in the triathletes that compete in triathlon after years of cycling competitions [12].

Other authors sustain a different pathogenesis of ITBS. Fairclough [21] suggests that ITB overuse injuries may be more likely to be associated with fat compression beneath the ITB, rather than with repetitive friction as the knee flexes and extends. Ekman [22] and Hariri [23] consider that the inflammation of the bursa underlying the ITB is the only cause of ITBS.

6.3 Patient Evaluation

The main symptom of the ITBS is sharp pain or burning on the lateral aspect of the knee. The patient typically localizes the pain to the region of the distal ITB between the lateral femoral condyle and its insertion on the Gerdy tubercle. Runners often note that they start running pain-free but develop symptoms after a reproducible time or distance. If ITBS progresses pain can persist even during walking, particularly when the patients ascent or descent stairs. In some cases, the triathlete with ITBS can perform painless other sports activities, like swimming, skiing, basketball and football.

The knee examination is typically negative except for local tenderness and, occasionally, swelling over the distal ITB where the band moves over the lateral femoral epicondyle.

Some provocative tests are commonly used in the assessment of ITBS and ITB function. Application of the direct pressure to the distal fibers across the lateral femoral epicondyle as the athletes flexed and extended knee from 90° to 180° usually elicited a very positive response (Holmes test). The Noble test is performed with the patient lying supine; beginning with the affected knee flexed at 90°, the leg is extended with direct pressure over the lateral femoral epicondyle, with reproducible pain near 30° of knee flexion. The Thomas test is used to determine the tightness of the iliopsoas muscle, rectus femoris muscle and ITB. The patient is instructed to lie supine at the edge of the examination table with both knees held to the chest. The patient holds the unaffected leg to the chest and the affected leg is extended and lowered. A positive test results if the patient cannot completely extend and lower the affected leg to horizontal. Useful is also the Migliorini-Merlo sign: with the patient lying supine and the completely extended knee, it can observe in case of ITBS an evident sulcus between the ITB and the lateral femoral epicondyle (Fig. 6.2).

In particular cases, it is important to perform an evaluation of biomechanics of running, evaluation of triathletes and cyclists on their bicycles, bikes, running shoes, foot orthosis and shock-absorbers review.

The differential diagnosis includes lateral meniscal pathology, popliteal or biceps femoris tendinopathy, patellar syndrome, common peroneal nerve injury, lateral myofascial thigh pain, osteochondral lesion of the lateral femoral condyle.

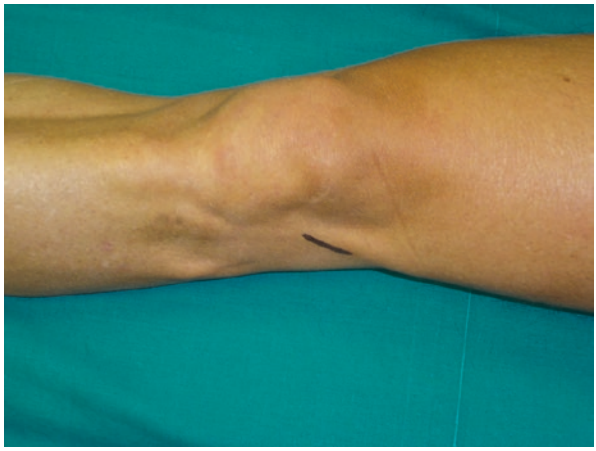

Fig. 6.2 Migliorini-Merlo sign. With the fully extended knee, it can observe an evident sulcus between the ITB and the lateral femoral epicondyle

6.4 Imaging

Although not routinely required, radiography imaging can be used to supplement the physical examination. Routine radiographs of the knee, including AP, lateral and sunrise view can be used as a diagnostic adjunct to rule out other possible causes of the lateral knee pain, such as lateral joint space narrowing from degenerative disease, patellar maltracking and stress fractures.

Ultrasonography of the knee lateral compartment is the first radiological evaluation considering low cost and availability of this modality compared with MRI. Usually, ultrasonography performs a study to measure ITB thickness and the underlying bursa.

All athletes were examined with MRI to confirm the diagnosis because ITBS may be confused with other derangement of the knee, such as lateral meniscal tear, popliteal tendinopathy, lateral collateral ligament strain, hamstring strain, lateral femoral condyle osteochondral lesion or transchondral fracture. Some authors [22, 24] reported the presence of high-intensity signal, representing a fluid-filled collection, over the lateral epicondyle deep to the ITB as well as a marked thickening of the distal ITB. Eckman [22] demonstrated a significantly thicker ITB over the lateral femoral epicondyle. Thickness of the ITB in the disease group was 5.49 ± 2.12 mm, as opposed to 2.52 ± 1.56 mm in the control group.

6.5 Conservative Treatment and Rehabilitation Programme

Nonsurgical management is the mainstay of treatment of symptomatic ITBS.

In the acute phase, the therapy must reduce the pain and we suggest to rest from the inciting activities, such as running or cycling, for 2–3 weeks, until pain has resolved. Swimming with the pool-buoy, without using lower extremities, normally

is the only sport activity allowed in the acute phase. Daily cryotherapy is useful in this phase and should be incorporated into the physical therapy program in an effort to reduce the inflammatory component of the ITBS.

Oral nonsteroidal anti-inflammatory drugs (NSAIDs) can be used to reduce the acute inflammatory response, alone have not been found to be effective in providing symptom relief. On the contrary, local corticosteroid injections can relieve pain as well as aid in the diagnosis of ITBS, after 3 days of pain and, particularly infiltrating the underlying bursa. Local corticosteroid infiltrations are effective and safe in the early (first 14 days) treatment of recent onset ITBS [25].

Therapy with PRP (platelet-rich plasma) should be an opportunity, but actually it needs more studies to determine the real efficacy.

Rehabilitation program is one of the most important parts of ITBS conservative treatment. Stretching of tensor fascia latae, hamstrings, gluteus medius, ileopsoas, rectus femoris, gastrocnemius, and soleus muscles should be started after acute inflammation subsides. Each stretch position should be maintained for 20 s and repeated at least 5 times (Figs. 6.3a–c, 6.4, and 6.5). Manual therapy that consists of soft-tissue and medial patella mobilization techniques may also contribute to lengthening the ITB. Additionally, the patient can use a foam roller as a myofascial release tool to break up soft-tissue adhesions in the ITB (Fig. 6.6). In subacute phase, it is important the treatment of fascia lata and vastus lateralis trigger points with deep stripping massage, compression, fibrolysis, dry needling and mesotherapy. Shock waves should show good results if applied to the ITB and to the trigger points in the musculotendinous junction of the distal vastus lateralis. A randomized controlled trial showed that shockwave therapy and manual therapy are equally effective in reducing pain in subjects with ITBS [26].

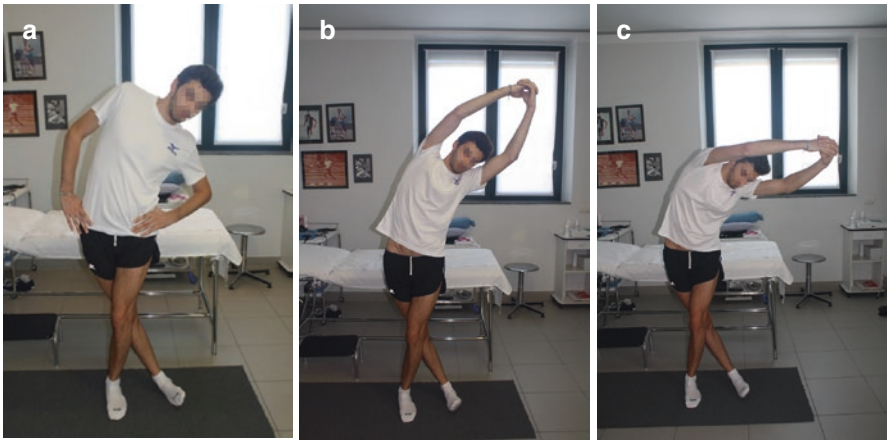

Fig. 6.3 Iliotibial band-specific stretching regimen (**a**–**c**). To perform the standing stretch (**a**) the patient stands upright, using a wall for balance if needed. The symptomatic leg is extended and adducted across the noninvolved leg. The patient slowly flexes the trunk laterally the opposite side until a stretch is felt on the side of the hip. The arm-overhead standing stretch (**b**) accentuates the stretch by increasing lateral trunk flexion. Further progress is made by teaching the patient to bend downward and diagonally (**c**)

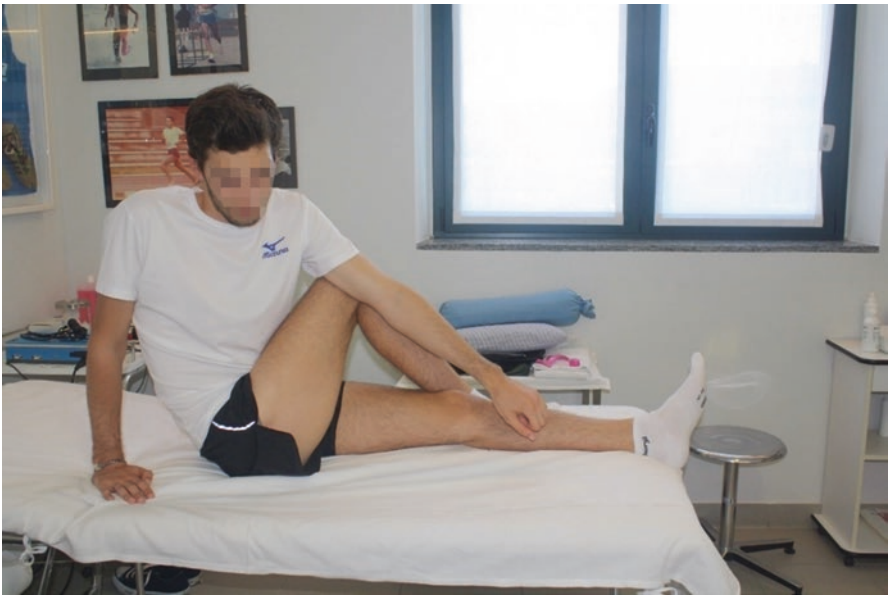

Fig. 6.4 The patient is seated with the symptomatic leg crossed over the noninvolved one. Rotating the trunk and helping with the elbow, the patient pushes the knee medially until a stretch is felt on the side of the hip

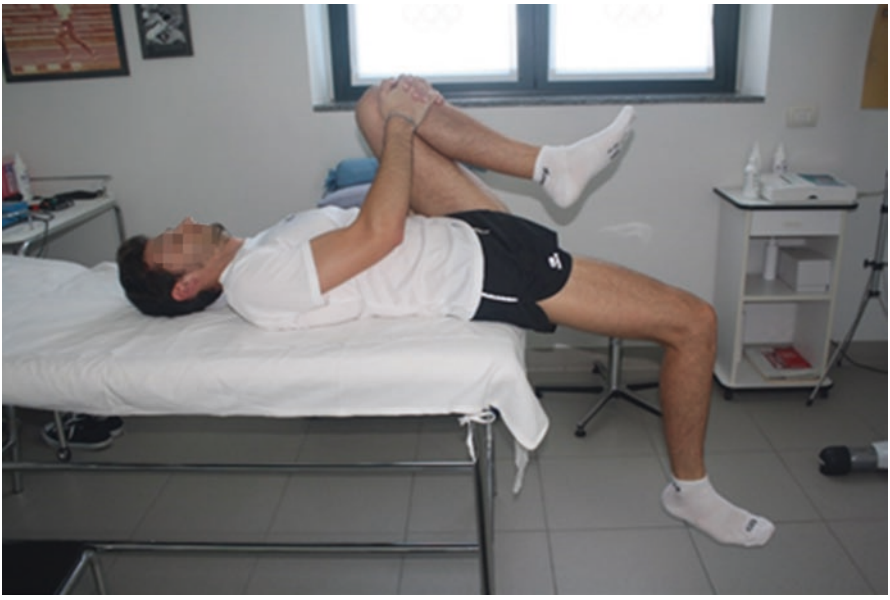

Fig. 6.5 The patient is seated on the end of the examining table in a supine position. Then he/she holds the hip on the asymptomatic side close to the chest but without inducing excessive posterior tilt or bringing the hips off the table. Keeping this position, the patient adduces the affected leg until a stretch is felt on the side of the hip

Fig. 6.6 ITB foam roller position. The patient supports the upper body with the hands on the floor and the foam roller is placed under the side of the involved leg, which is held straight. The patient crosses the uninvolved leg over involved one and rolls over the roller from hip to knee, emphasizing tight area

Once the patient is capable of performing the stretching regimen without pain, strengthening is added to the rehabilitation program. Attention is paid to the proximal strengthening of the hip abductors (gluteus medius) and the core muscles to stabilize the pelvis to prevent excessive adduction of the hip [8]. The initial exercise is side-lying leg lifts to help the patient learn to isolate the gluteus medius muscle (Fig. 6.7) introducing in the programme also the single leg bridge and the lateral bridge exercises (Figs. 6.8 and 6.9). Because training is specific to limb position, it is essential that patient progresses to weight-bearing exercises. Initially patient was asked to perform a step-down exercise (Fig. 6.10); once this is mustered patient is taught to perform the pelvic drop exercises (Fig. 6.11) [16].

Great attention should be focused on sport equipment, considering running shoes and biomechanics of the foot contact on the ground, the bike saddle position (avoiding knee extension beyond 150°), the incorrect cleat pedal alignment (varus knee or external tibial rotation more than 20% cause a significant stress on distal ITB when riding with internally rotate cleats). The hyperpronated runner should use stable running shoes and change the shoes every 500–600 miles considering that shoes loose almost 60% of their capacity to absorb ground reaction forces [27].

Length discrepancies of the lower legs should be corrected with orthosis for the running shoes or with thickness between cycling shoes and cleat pedals. In some cases, it is useful putting inside the shoes the shock absorbers, in order to increase the knee flexion at the foot strike [14]. During the subacute phase, the athlete can

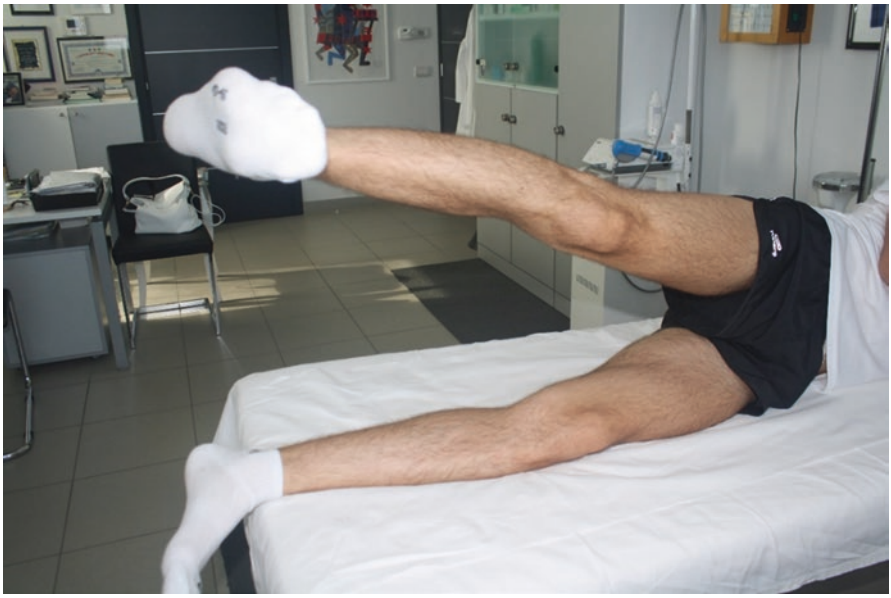

Fig. 6.7 Side-lying leg lifts are the first exercise performed to strengthen the gluteus medius muscles. The patient is instructed to keep the lower leg flexed for balance. With the leg slightly extended and internally rotated, the patient abducts the affected leg 30°, holding the position for 1 s. The leg is slowly lowered into maximal adduction. Start with 3 sets of 10 repetitions

Fig. 6.8 Single leg bridge. The patient lies on the back. Then he/she lifts pelvis and shift weight slowly onto one foot, extending the other leg, not allowing the pelvis on that side to drop. Hold for 5 s, lower pelvis back down and repeat on the other side. Start with 3 sets of 10 repetitions

Fig. 6.9 Lateral bridge. Legs are extended. The patient supports him/herself on one elbow and the feet while lifting his/her hips off the floor to create a straight line over his/her body length. This position should be maintained as long as possible (1 min it is enough) and repeated 3 times each side

return to cycling, easy pedaling (80 rpm or more) with a little resistance at a pain-free cadence on flats terrains. Return to running depends on the severity of the condition and patient's premorbid function. We recommend running every 2 days, starting with easy sprints on the level ground over slow jogging; biomechanical studies have shown that faster-paced running is less likely to aggravate the ITBS [9]. Another way to maintain aerobic conditioning during rehabilitation is deep water running, and it could be started even in the acute phase, especially in elite athletes. When performing water running, the athlete runs in the deep end of the swimming pool with the aid of a commercial flotation belt that helps support the head above water [28, 29]. A study showed that during 4/8 weeks of deep water running, the athlete maintains cardio-respiratory performances (VO2max ventilatory threshold, running economy). We suggest a gradual increase in distance running and frequency, avoiding hill training and intensive training (too much, too soon). Most of the patients have symptomatic relief without surgery within 6–8 weeks and are capable of returning to their athletic activities with no long-term sequeale.

6.6 Surgical Treatment

Surgical treatment is indicated only after extensive nonoperative measures have failed to relieve symptoms (3–6 months).

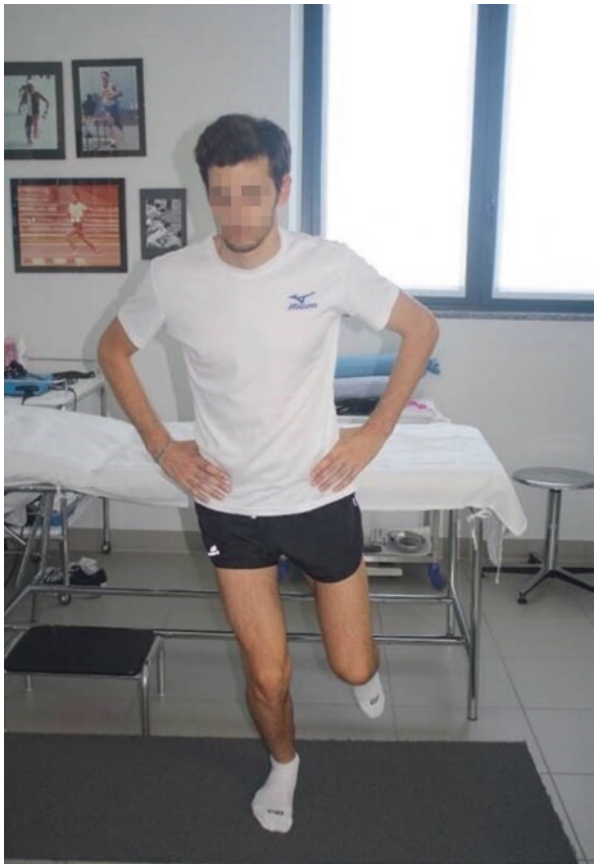

Fig. 6.10 Step-down exercises are learned in front of a full-length mirror so that the patient can monitor proper form. The goal is to maintain pure sagittal plane motion, avoiding excessive hip adduction or internal rotation

Holmes [30] suggests two procedures: percutaneous release and the open release excision. The percutaneous incision is performed under local anesthetic with the knee held at 90° so that the posterior fibers are free from the lateral femoral epicondyle. The open release surgery consists in the excision of an ellipse tissue measuring 4 cm at the base and 2 cm at the apex.

Martens [31] resected from the posterior part of the ITB a triangular portion 2 cm wide at the posterior base and 1.5 cm height. The ITB Z-lengthening is also an option described by Richards [32]. Hariri [23] suggests an open ITB bursectomy. Michels [33] described an arthroscopic technique to treat refractory ITBS using a synovial shaver to resect the lateral synovial recess accessed through an accessory superolateral portal.

Our personal surgical procedure [34], usually in local anesthesia and in day-surgery hospital, is performed by a small access (less than 2 cm) and consists in a subtotal vertical tenotomy of the ITB with the knee held at 70° of flexion (Fig. 6.12). We do not remove the bursa underlying the ITB, because of the high risk of hematoma and persistent synovial reaction. The ITB release is sufficient to remove the ITB impingement with the lateral femoral epicondyle and consequent reduction of

Fig. 6.11 Pelvic drops. To begin, the patient stands on a step with the involved leg, holding the wall for support if needed. With both knees locked, the patient lowers the uninvolved pelvis toward the floor by shifting body weight to the inside foot of the involved leg. The patient returns to the starting position by contracting the gluteus medius on the involved side. Exercising in front of a mirror will help the patient learn proper technique

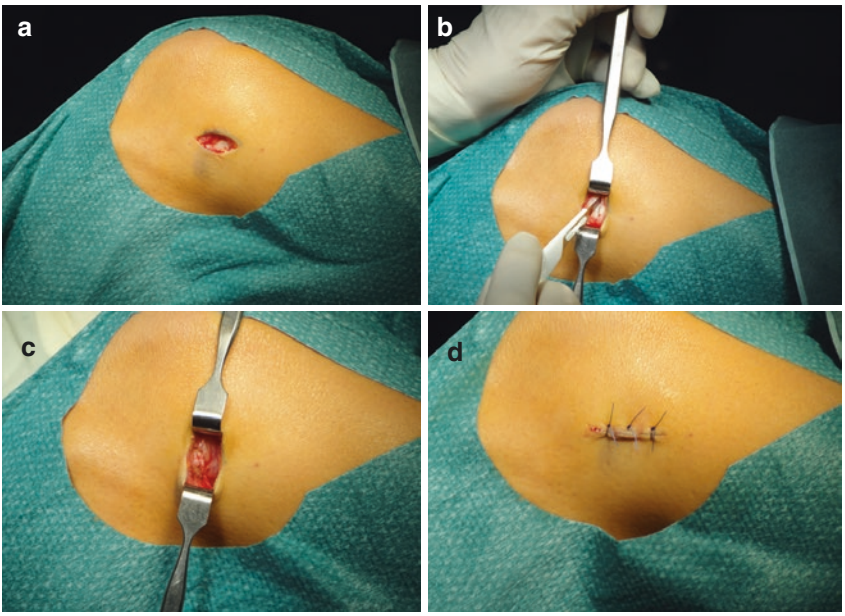

Fig. 6.12 Our personal procedure. (**a**) approach to ITB; (**b**) vertical incision of ITB; (**c**) complete release of ITB; (**d**) skin suture

the bursitis. Compression bandage and cryotherapy is mandatory at the end of the procedure. In some cases, we perform an arthroscopic evaluation of the knee joint to rule out associated other articular lesions, particularly involving the lateral meniscus.

Postoperative protocol began immediately after surgery. Patients are allowed to be weight-bearing as tolerated with bilateral crutches until their gait normalized. Typically they are able to walk without crutches between 3 and 5 days postoperatively. Some athletes develop small seromas or hematomas near the incision during the second postoperatively week. These resolved spontaneously in 3–5 days with intermittent icing, local compresses or aspiration. Passive range of motion for the hip and knee began on postoperative day 1 with full knee extension achieved by day 3 and full hip and knee flexion by the end of week 2. Patellofemoral joint mobilizations are also performed with specific emphasis on medial glides of the patella. Gentle massage and range of motion for the ITB are initiated in week 1 and progressed to gradual stretching in week 2.

Isometric exercises for the hip abductors are performed during week 1 and progressed to active range of motion and progressive resisted isotonic during week 2–4. The initial exercise is side-lying leg lifts to help the patient learn to isolate the gluteus medius muscle. For all strength exercises patients start with one set of 20 repetitions and built up to 3 sets of 20 repetitions daily. Because training is specific to limb position, it is essential that patients progress to weight-bearing exercises. Initially, athletes perform step-down exercises. Once this is mastered, patients are taught to perform the pelvic drop exercises. Neuromuscular electrical stimulation is applied to the quadriceps to prevent the effects of postoperative inhibition. The patients are evaluated by the operating surgeon at week 4 and cleared to begin a gradual progression of swimming and cycling as tolerated with a return to running between 6 and 8 weeks postoperatively. As a general rule, athletes can return to running once they can perform all strength exercises without pain. We recommend running every other day for the first 4 weeks, starting with easy sprints on level ground. Biomechanical studies [9, 16] have shown that faster-paced running is less likely to aggravate ITBS because at foot strike the knee is flexed beyond the angles that cause friction, and faster strides are initially recommended over a slower jogging pace. In our experience, according to Evans [5], the ITB release does not affect the biomechanics of the hip and knee.

6.7 Conclusions

ITBS is a common cause of lateral knee pain in the athletic patients' population, particularly in endurance sports, like running and triathlon. We focus the importance of the exact etiology underlying the condition that it is different for every triathlete considering the intrinsic and extrinsic factors. Most patients with ITBS improve with aggressive protocol rehabilitation and activity modification. Surgery release of the ITB is indicated only after extensive nonoperative measures have failed to relieve symptoms. Between a variety of surgical management options, our personal surgical procedure reported good results and return athletes to pain-free athletic activity.

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Anterior Knee Pain

7

Thilo Hotfiel, Martin Engelhardt, and Casper Grim

7.1 Introduction

In triathlon knee injuries are frequently observed in both recreational and elite athletes and are some of the most common injuries. A prospective study has demonstrated a 56% prevalence of knee pain in long-distance triathletes, who were preparing for an Ironman-distance (26 weeks follow-up, n = 174) representing almost one quarter of all assessed injuries [1]. Knee-related disorders can generally occur as a cause of overuse or as an acute traumatic injury, whereas traumatic injuries may happen in context of cycling accidents and display a great variety of different injuries. These range from soft tissue injuries like contusions or lacerations up to harmful sequelae covering complex ligament, tendon or meniscal injuries, or even fractures and dislocations [2].

Overuse injuries mainly occur as a consequence of repetitive overload in conjunction with extrinsic or intrinsic risk factors. In regard to the different disciplines in triathlon, running displays the highest prevalence of overuse injuries of the knee [3, 4] (Fig. 7.1). A study evaluating chronic knee pain in triathletes (n = 58) could demonstrate that 72% of all knee-related injuries occurred during running events and affected predominantly the anterior and lateral aspect of the knee [5]. Although triathlon has been estimated to be a varied sport, the incidence of knee-related disorders is not less than in running itself, which has been indicated to be between 7% and 50% [6]. Cycling is seen as a further discipline in which anterior knee pain including patellofemoral pain and patellar tendinopathies are commonly observed [4, 7, 8]. Overuse injuries have to be evaluated carefully as a great number of

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Fig. 7.1 Anterior knee pain is commonly observed in triathletes. Overuse injuries predominantly occur during running and affect mostly the anterior and lateral aspect of the knee

athletes will develop recurrence, a leading cause of longer rehabilitation time and time loss in sports [9]. Diagnostics as well as a successful treatment remain challenging to ensure both a successful treatment, a small loss of training or competition time as well as avoidance of reinjuries. In the following chapter we will differentiate among patellofemoral pain, patellar tendinopathies, plica injuries, and disorders of the infrapatellar fat pad with special attention on pathomechanism, diagnostics, and treatment.

7.2 Patellofemoral Pain

7.2.1 Etiology and Pathomechanism

Patellofemoral pain syndrome (PFPS) is a broad and rather unspecific term used to describe pain in the front of the knee and around the patella and is one of the most common musculoskeletal disorders. It is of high relevance since PFPS is a frequent cause for presentation at physiotherapy, general practice, orthopedic, and sports medicine clinics [10, 11]. PFPS is common in young adolescents with a prevalence of 7–28% and an incidence of 9.2% [12–14]. Specialization in a single sport seems associated with a relative risk (1.5: 95% CI: 1.0 to 2.2) of PFPS incidence compared to multisport athletes [12]. Increasing evidence suggests that it is a recalcitrant condition that can persist for many years [15, 16]. Despite its high incidence and plenty of available research, the pathogenesis of PFPS is still unclear ("The

Black Hole of Orthopaedics"). Anterior knee pain is a commonly used synonym. The numerous treatment regimens that exist for PFPS highlight the lack of knowledge regarding the etiology of pain. Crossley et al. summarize the current theories on pathophysiology of anterior knee pain and patellofemoral pain in their latest consensus paper [17].

Different mechanisms seem to be involved in the pathophysiology of pain sensation in PFPS and this can be grades in four general types. Nociceptive pain is referring to a response to a noxious stimulus. Homeostatic pain is considered to arise from healing of injured tissue as a cascade to reestablish the tissue homeostasis. Spontaneous pain and hypersensitivity in association with damage of the somatosensory nervous system is considered to be a neuropathic pain. Functional pain in PFPS may result from abnormal central stimulus processing of normal input. PFPS may include symptoms such as allodynia and hyperalgesia known as increased perception to a stimulus that is normally not painful. The phenomenon of pain in rest or while sitting ("movie goers syndrome or movie sign") in absence of osteoarthritis might act an example of allodynia. The pain arising from a non-nociceptive activity may reinforce an abnormal processing [17].

The pathophysiology also includes considerations about anatomical abnormalities and extensor mechanism disorder, resulting in patellar malalignment. Furthermore it is assumed that anterior knee pain arises from overuse, especially in adolescents [18], which seems to play an important role in athletes.

Patients experience anterior knee pain mostly nonspecific and localized diffuse around the patella. Other patients complain about a feeling of patellar instability. The first group usually has normal patellar mobility, no instability risk factors at the clinical and radiological evaluation and they mainly report the symptoms to occur after physical activity. Those with patellar instability often present with a patellar hypermobility with perceptible tracking problems and they rather complain of knee pain during physical activity [19, 20]. Patients with anterior knee pain should be divided into two groups: Pain versus patellar instability problems.

Patients representing any history of dislocations, or who report perceptions of subluxation and instability should not be a subgroup of patients with patellofemoral pain. Since they have a distinct disorder, biomechanical risk factors and regularly require different treatment approaches, e.g., realignment surgery. From a clinical point of view, patellar instability should be discussed separately and will not be discussed in this chapter.

7.2.2 Symptoms

The most common symptom of PFPS is a dull, aching pain in the front of the knee. This pain usually begins subtle and is frequently related to activity. The pain may be present in one or both knees. In triathletes the pain is frequently related to running and cycling with high resistance, while knee complaints during swimming a rare.

Other common symptoms include:

- Pain during exercise and activities that repeatedly bend the knee, such as climbing stairs, running, jumping, cycling with high resistance, or squatting.
- Pain after/while sitting for a longer period with flexed knees (e.g., movie theater, airplane). Pain related to changes in activity level, intensity, training volume, or equipment (e.g., running shoes, plate position on cycling shoes).
- Clicking or snapping sounds while climbing stairs or when standing up after prolonged sitting.

7.2.3 Diagnostics

7.2.3.1 Clinical Examination

The clinical examination is the cornerstone to diagnose a PFPS, but a definitive clinical test to diagnose PFPS does not exist. The best available test is anterior knee pain seems to be a squatting maneuver, since PFPS is evident in 80% of people who are positive on this test [21]. Additional tests have limited evidence. Tenderness on palpation of the patellar margins can be found in 71–75% of people with PFPS. Tests with limited diagnostic value are patellar grind or compression tests (e.g., Zohlen sign, Clarke's test, patellar grind test). They have low sensitivity and limited diagnostic accuracy for PFPS [21]. Clinical examination should particularly aim to exclude patellar instability.

Weakness of the quadriceps muscle, especially during eccentric contractions, is usually present in the majority of anterior knee pain patients. A relative hypotrophy and reduced activity of the vastus medialis is often observed, which result in muscular imbalances. The athletes should also be checked for segmental dysfunctions of the lower back and the SI-joint. A gait analysis with the focus on heel-strike pattern can be helpful to identify functional problem and possible patellofemoral overloading. Forefoot strike-pattern leads to increased Achilles tendon loading, while a heel strike pattern (hard impact) increases patellofemoral loading [22]. The athlete should be checked for proper muscle flexibility. Soft tissue and muscle flexibility is essential in musculoskeletal evaluation and has specific implications in patients with anterior knee pain. A poor flexibility of the knee extensors and knee flexors seems to be correlated with anterior knee pain [23]. In addition, tightness of the tensor fascia lata and iliotibial band is associated with anterior knee pain [24]. Patients with PFPS sometimes also show tightness of the lateral retinaculum. This might also lead to an altered patella tracking.

7.2.3.2 Imaging

There is no specific investigation or imaging that can proof PFPS. Radiological evaluation of PFPS should in first instance aim for exclusion of patellar instability, impending instability, and other differential diagnosis, e.g., patellofemoral osteoarthritis, patellar tendinopathy. With imaging the risk factors for patellar instability

should be evaluated. Information such as patella height, patella tilt, trochlea morphology, TTTG/TTPCL, and leg malalignment have to be obtained. Imaging in PFPS aims to exclude other diagnosis.

7.2.3.3 Therapy

The expert panel from the 4th International Patellofemoral Pain Research Retreat [25] gave six evidence-based recommendations for treating PFPS:

1. Exercise-therapy is recommended to reduce pain in the short, medium, and long term, and improve function in the medium and long term.
2. Combining hip and knee exercises is recommended to reduce pain and improve function in the short, medium, and long term, and this combination should be used in preference to knee exercises alone.
3. Combined interventions are recommended to reduce pain in adults with patellofemoral pain in the short and medium term.
4. Foot orthoses are recommended to reduce pain in the short term.
5. Patellofemoral, knee and lumbar mobilizations are not recommended.
6. Electrophysical agents are not recommended.

These recommendations differ from those described in the "Best Practice Guide (BPG) to Conservative Management of Patellofemoral Pain" [26]. The BPG recommended consideration of acupuncture and combined interventions (using a combination of exercise therapy, targeting knee and hip musculature, patellofemoral taping, mobilization and foot orthoses). It appears that clinicians have a strong preference for combined interventions. However, exercise therapy has been evaluated accurately within the scientific literature and the recommendation as a stand-alone treatment is based on the strength of supporting evidence [25]. In contrast, the combined interventions have very limited supporting evidence, although they play an important role in daily sports medical practice. The lack of supporting evidence does not mean the absence of positive treatment effects. Since triathletes are highly active, a load-vs-capacity problem should be considered. Like in any other overload-problem the load and activity has to be adapted to current tissue capacity and training level. Athlete and coach education is necessary for gradually building up the capacity to handle training loads without overloading. If the athletes keep fully participating in their sports (with the same training errors) during a patellofemoral pain episode, the capacity to handle that training load may be compromised. Their goal should be to balance their training loads with current capacity (Fig. 7.2a, b).

An activity ladder (Fig. 7.3) can be a useful tool to raise the motivation and to adhere to the treatment protocol by meeting the athlete's desires (e.g., participating in sports requires running). By using the ladder and pain-scale, which has to be transferred to a training schedule, the focus gradually shifts from pain avoidance toward pain-management and functional goals [14].

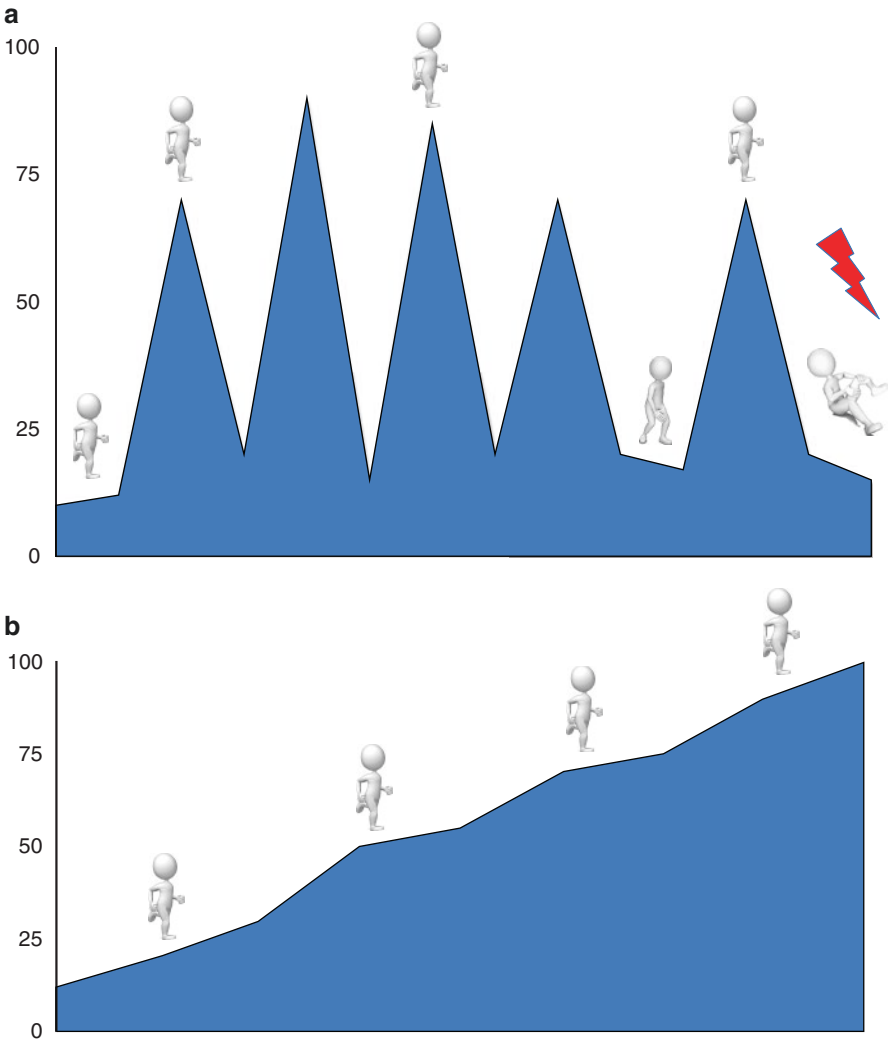

Fig. 7.2 (**a**, **b**) If the athletes keep fully participating in their sports (with the same training errors) during a patellofemoral pain episode, the capacity to handle that training load may be compromised. Their goal should be to balance their training loads with current capacity

7.3 Patellar Tendinopathy

7.3.1 Etiology and Pathomechanism

Patellar tendinopathy is a common overuse injury associated with pain and dysfunction of the patellar tendon [27, 28]. There are several factors that have been

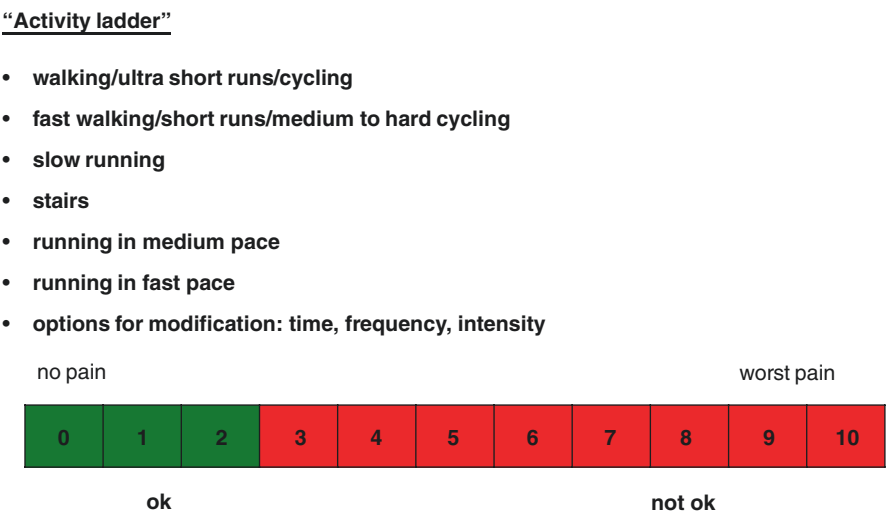

Fig. 7.3 An activity ladder can be a useful tool to raise the motivation and to adhere to the treatment protocol by meeting the athlete's desires

estimated to be responsible for the development of patellar tendinopathy. However, clearly defined risk factors for both the pathophysiological process of acute PT and the development of chronic PT are not uniformly accepted [29]. One theory to explain the pathophysiological process includes the continuum model placing the underlying cause of the pathophysiological process in three groups: (1) collagen disruption/tearing, (2) inflammation, and (3) tendon cell response. Thereby interchangeable stages of reactive tendinopathy, tendon disrepair, and degenerative tendinopathy have been postulated [29]. Mechanical overload has been proposed to be the most relevant pathway usually associated with several extrinsic and/or intrinsic risk factors including leg length discrepancy, arch height, quadriceps flexibility and strength, hamstring flexibility, ankle dorsiflexion, BMI, changes in surface density, or shock absorption [27–30]. Tendon overload has been defined as an activity level which exceeds the load of the tendon is adapted to and symptoms can occur by a sudden or substantial increase in the volume of loading to the tendon. However, repetitive low loading as swimming or running rarely aggravate the PT and additional risk factors have to be considered cautiously in these athletes [27].

7.3.2 Symptoms

PT is characterized by anterior knee pain and tenderness to the patellar tendon mainly localized at the proximal tendon attachment during high-level patellar loading or following repetitive running or cycling loads [27, 31]. More widely and diffuse distributed pain offers different diagnosis. Clinical signs include reduced force capacities and increased painful restriction of flexion, stiffness, swelling, and also altered

biomechanics to the adjacent joints. Sometimes sensations of "weakness" or "giving way" phenomena can be observed. PT usually represents a gradual onset of pain.

7.3.3 Diagnostics

In general, a detailed history (i.e., assessing changes in training volume and characteristics as well as risk factors) and clinical examination with inspection, palpation, general functional testing of the knee, and an assessment of the related kinetic chain can provide important information about the extent and severity of injury—or if different structures may be the source of pain. PT is often accompanied by tenderness and swelling upon manual testing and the clinician may be able to palpate a local or even global area of pain and swelling [32–34]. The single-leg decline squat has been established as a key test, leading to pain isolated to the tendon at a critical angle of knee flexion [27]. PT mostly leads to impaired strength delivery to the anterior thigh and calf muscles.

7.3.3.1 Imaging

Imaging of the knee is essential for providing a correct assessment of the severity of tendon damage [27]. In diagnostic imaging of PT, conventional ultrasound and magnet resonance imaging (MRI) have been reported as the preferred modality providing detailed image analysis and characterization of this kind of tendon injury. However, it has to be noted that pathologies observed in imaging do not correlate to clinical signs of painful PT [31]. In *MRI* the normal patellar tendon appears in homogenous low signal intensity. It should not exceed 7 mm in anteroposterior (AP) diameter, may demonstrate a convex anterior border, and should consist of a distinct posterior border [35]. PT can be detected on MRI as an intratendineous hyperintense signal, mainly at the proximal one-third of the tendon with patchy high signal changes (Fig. 7.4). Partial or complete tears to tendon are detectable mostly in T1-weighted sequences.

Ultrasound is able to demonstrate the presence of tendon pathology with high spatial resolution. It is recommended to use a linear probe with high frequency scanning the tendon in longitudinal and transversal axis. Abnormality criteria include heterogeneous tendon structures (anechogenicity), swelling, and a neovascularization observed in PW-Doppler [36, 37]. Both in vitro and in vivo studies have shown that neovascularization can contribute to pain and chronic conditions [36]. Beside conventional gray-scale ultrasound, elastography provides a noninvasive tissue characterization.

7.3.4 Therapy

7.3.4.1 Nonoperative Treatment

PT can mostly be addressed conservatively. Treatment considerations should focus on different aspects, including strategies to target symptoms as pain and swelling, interventions targeting function, load capacity and structure, and finally interventions

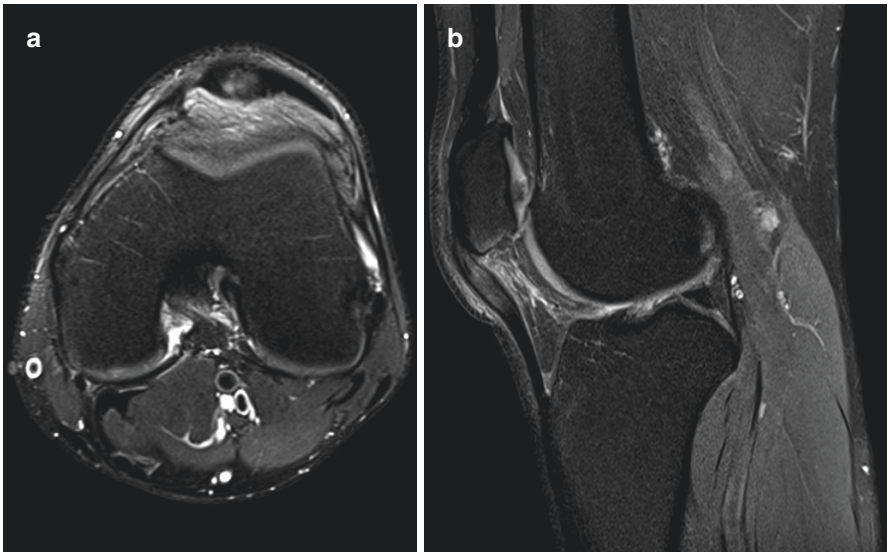

Fig. 7.4 MR images [PD tse fs tra (**a**) and sag (**b**)] in case of refractory patellar tendinopathy showing increased diameter at the proximal origin of the patellar tendon with hyperintense signal in the tendon as well as in the infrapatellar fat pad and adjacent distal patellar bone

to address underlying risk factors [27, 29–31, 38]. Meanwhile, for all mentioned aspects, there are several available methods to accelerate the rehabilitation process and thereby restoring tendon function and capacities. However, management of PT in elite athletes, particularly during in season remains challenging because of the required time for recovery [38]. Due to the growth of diverse published interventional strategies, not all treatments concerning this topic can be addressed to its full amount. The most frequently applied nonoperative interventions in clinical practice are listed below.

7.3.4.2 Acute Phase: Symptomatic Therapy and Pain Management

During the acute phase after the onset of tendinopathy, load management including restrictions in training volume and intensity (high-load drills) should be assigned preferably [27, 38]. However, a complete cessation of tendon loading should be avoided [30]. Further local anti-inflammatory strategies like intermittent cooling and the temporary use of topic or oral use of NSAIDs can be considered, whereas a longstanding use of NSAIDs should be regarded critically in degenerative tendinopathy [28]. Even during the acute phase, isometric training forms can be appropriate as they have demonstrated significant pain relief [39, 40]. Potential obtained associated risk-factors should be addressed promptly.

7.3.4.3 Active Interventions

Loading exercises even applied to the painful tendon are one of the most important steps to target symptoms as well as to restore tendon capacities [28, 31]. Resistive

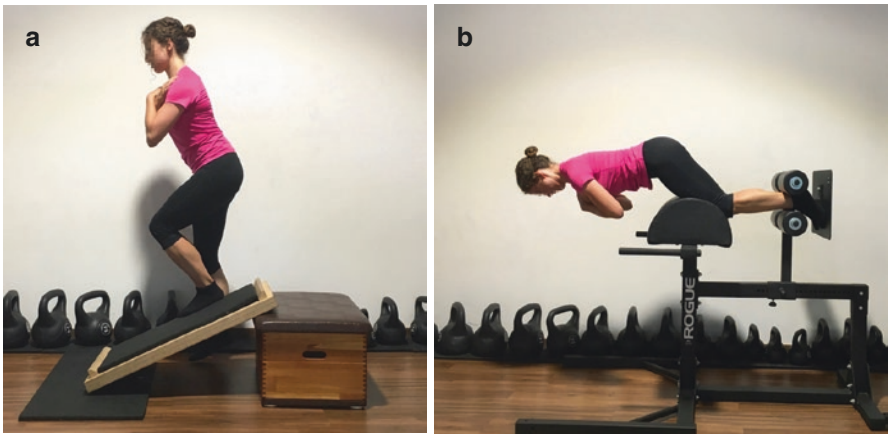

Fig. 7.5 (**a**) Loading exercises applied to the tendon are one of the most important steps to target symptoms as well as to restore tendon capacities: Single-leg squad using an inclined surface. (**b**) Functional strengthening including the trunk and kinetic chain

exercises should be provided gradually and thereby increased in its volume and intensity according to the individuals pain level [27]. Thereby eccentric, heavy slow resistance, isotonic, and isometric exercises have been described to be effective in PT. Eccentric exercises have generally been established by demonstrating good short- and long-term outcomes [27, 31]. The single-leg-squat with a 25° inclined surface has been integrated in most of the existing resistance drills (Fig. 7.5a). Functional strengthening, particularly including the trunk and kinetic chain should be performed as well (Fig. 7.5b) [31]; however there are no high-quality studies that have addressed these functional aspects to its full extent. Once an improvement of clinical symptoms is obtained, sports-specific training can be started gradually as easy running drills like skipping, jumping, or hopping. Additionally, kinetic chain exercises and education remain an integral part of a well-rounded rehabilitation program [27].

7.3.4.4 Physical Therapy

Extracorporeal shock wave therapy is often used in the management of common lower limb conditions [28, 41]. A systematic review concluded that low-level evidence suggests that focused ESTW is superior to conservative treatment including the ingestion of NSAIDS, physiotherapy, knee strap, modification of activity in long-term follow up (2–3 years) respecting functional, and pain-related outcomes [41]. However, no protocols could be recommended due to substantial heterogeneity in PT studies [41]. From the clinical perspective, ESWT should not be used as monotherapy. Instead it has to be seen as an additional modality in context of an individualized rehabilitation protocol.

7.3.4.5 Injection Therapy

Injections with various substances are frequently used in the clinical setting. Although some studies demonstrated good short-term results in PT [28], the local

and systemic application of corticosteroids cannot be recommended in the treatment of degenerative tendinopathy. The intratendinous presence of corticosteroids is associated with several adverse effects in regard to the healing response, and cases of patellar tendon ruptures have been reported [28]. A recent review concluded that corticosteroid injections should generally be used cautiously in athletes [42]. The injection of platelet rich plasma (PRP) has gained increasing attention during the last years. There are many studies focusing on the effectiveness in terms of patellar tendinopathy. A recent meta-analysis of randomized trials concluded PRP to be superior over other established nonsurgical treatments (dry needling and ESTW) for refractory cases [43]. Beside corticoids and PRP there are many other agents in use, involving hyaluronic acid, aprotinin, high-volume injections, or sclerotic agents [28, 30, 44–46]. However, there is no clear-cut evidence to support their use to date. Generally, injections are just one component of a comprehensive rehabilitation plan and the indication of its use should be evaluated seriously.

7.3.4.6 Operative Treatment

Operative treatment has to be considered in case of failure of conservative treatment or refractory cases. Depending on the underlying pathology, a surgical approach can be performed either in arthroscopic or open technique as debridement, resection, or drilling of the affected tissue and adjacent hypertrophic synovium and infrapatellar fat pad [28].

7.4 The Infrapatellar Fat Pad

7.4.1 Etiology and Pathomechanism

The diamond-shaped infrapatellar fat pad (IFP; Hoffa fat pad) is the largest soft tissue structure of the knee consisting of white adipose tissue delimited by connective septa [47]. As a flexible, deformable, and displaceable structure, it is covered by synovial membrane posteriorly and is closely related to articular cartilage, being intracapsular and extrasynovial [47]. A cadaveric study could demonstrate the IFP to be closely attached to various structures including the inferior patellar pole, femoral intercondylar notch, proximal patellar tendon, intermeniscal ligament, both menisci and the anterior tibia via the meniscotibial ligaments [48]. The IFP has demonstrated an extensive anastomotic network of vessels [49], an high-grade innervation and represents a tendency to express scar tissue [47]. Studies have found that the IFP is extremely sensitive to pain, and a potent source of stem cells [48]. Hence, the IFP has generally been attributed to have an important role for the knee joint biomechanics and function. This includes mechanical cushioning and an interaction with joint kinematics that could be demonstrated by altered knee joint kinematics and patellar contact pressures as a consequence of fat pat resection [48]. From a functional point of view, IFP facilitates the distribution of synovial fluid and acts to absorb forces through the knee joint [47]. The high-grade mobility and intraarticular interaction makes it vulnerable to interference from trauma or

repetitive overload, which may lead to inflammatory and hemorrhagic reactions resulting in swelling and loss of plasticity of the IFP [48, 50]. Chronic conditions are associated with a variety of inflammatory and mechanical changes to the peripatellar fat pad, including synovial proliferation, fibrosis, metaplastic nodules of cartilage or bone friction, and impingement as well as inflammatory reactions [50]. Alterations of the infrapatellar fat pad also have demonstrated high association with patellar maltracking or malalignment including patella alta and lateral subluxation of the patella [35, 51].

7.4.2 Symptoms

The "Hoffa's disease" was first described by Hoffa in 1904 [52], who reported an inflammatory fibrous hyperplasia of the infrapatellar adipose tissue. Up to now diagnosis including it signs and symptoms are not usually accepted [53]. IFP disorders include a wide variety of symptoms and most of them have to be seen as unspecific and do not indicate abnormalities to the IFP per se [50]. Symptoms range from diffuse infrapatellar anterior knee pain, swelling and warming up to mechanical friction, and patellofemoral impingement. Restrictions in range of motion, particularly in extension can be observed as well. Pain sensation may develop slowly during or even after exercises and repetitive loading of the knee. As pathological changes in the IFP mostly develop secondary as a consequence of malalignment, patellofemoral maltracking, or intraarticular pathologies further symptoms may be present.

7.4.3 Diagnostics

As usual in diagnostics of anterior knee pain, a detailed history and general clinical examination of the knee and adjacent joints and functional chain should be performed [53]. Focusing on the IFP, a test primary described by Hoffa can be conducted: Applying pressure directly or beside the patellar tendon without tearing the tendon (extension) may lead to tenderness and pain sensation. Imaging of the knee is essential for further diagnostics. So far, MRI has been developed as the preferred modality providing detailed image analysis and tissue characterization. Further intraarticular pathologies that may be associated with IFP can be detected as well in one setting. MRI imaging mostly offers a focal increase in signal intensity on fluid sensitive MR sequences between the posterior superolateral patellar tendon and the lateral femoral condyle [54] (Fig. 7.6). Increased signal intensity of the IFP particularly to the posterior border is commonly described as indirect sign of edema. Beside IFP hyperintensity further imaging findings include fibrosis or synovial thickening, or ganglion cysts [54, 55]. Patellofemoral maltracking or malalignment has been posturized to have a key role for the development of IFP pathologies [55]. A prospective cohort study in cross-sectional design (1134 knees)

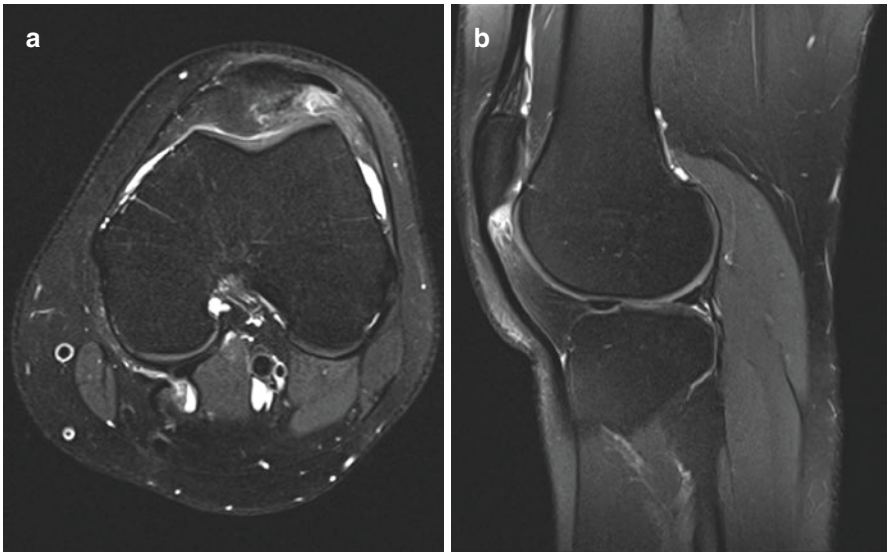

Fig. 7.6 MR images (PD tse fs) in transversal (**a**) and saggital (**b**) axis demonstrating areas of infrapatellar fat pad hyperintensity at the superolateral aspect and accompanying hoffa synovitis

could demonstrate an association between SHFP hyperintensity and measures of patellofemoral maltracking including patella alta, lateralization of the tibial tuberosity, and anterior position of the lateral femoral condyle [56]. In regard to associated cartilage damages the Multicenter Osteoarthritis (MOST) study including 1094 knees observed a cross-sectionally and longitudinally association between Hoffa synovitis and structural damages of the patellofemoral joint, regardless of definition, in all compartments. The authors concluded IFP hyperintensity to be a potential local marker of PFJ structural damage [54]. Ultrasound as further soft tissue imaging tool may reveal anechogenicity, swelling, and increased perfusion as sings of inflammatory reaction. As differential diagnosis a variety of pathologic conditions including lipoma, hemangioma, synovial chondromatosis, focal villonodular synovitis, ganglion cysts, osteochondroma, and chondrosarcoma have to be considered [57]. Histological diagnosis should be reserved in case of suspect findings in MRI.

7.4.4 Therapy

The management of IFP disorders remains challenging as anatomy, function, and pathophysiological responses of the IFP are not fully understood. This makes the treatment one of the most complex among the various pathologies affecting the knee. Based on the pathomechanism, treatment strategies should focus both to target inflammatory responses of the fat pad itself, as well as on accompanying

intraarticular pathologies and underlying patellofemoral maltracking, joint instability, or functional disorders. Treatment strategies and interventions for these athletes are currently uninformed accepted and, therefore, often unsuccessful. One of the most important step is the assessment of associated risk factors and pathologies that may reinforce inflammatory response and structural abnormalities.

7.5 Plica Injuries

7.5.1 Etiology and Pathomechanism

Plicae are normal structures frequently observed within the joint capsule presenting a thin, soft, flexible, and vascularized synovial structure [58, 59]. The most encountered plicae in regard to the localization are mediopatellar, suprapatellar, infrapatellar, and much less frequently, the lateral plica [35, 59]. There is still an ongoing discussion about its embryological origin. Attempts to explain include the theory, placing a plica as remnants of embryological membranes separating the knee into three different compartments as medial, lateral, and suprapatellar during uterine life [58]. As a consequence of an incomplete synovial membranes resorb while forming a single cavity, plicae are thought to exist [58]. Other studies suggest plicae are more likely to be true anatomical features not embryological remnants. It is proposed that incomplete resorption of mesenchymal tissue leads to failures of forming one single cavity and therefore is responsible for plica formation [58]. Thereby plicae are declared to be a remnant of mesenchymal tissue, but not of a septum. Particularly the mediopatellar plica has been implicated to be responsible for anteromedial knee pain when it becomes inflamed, thickened, and fibrotic [35, 60]. Plicae usually appear silent and asymptomatic, without signs of clinical significance. Hence, there is still little knowledge regarding the clinical significance of a plica, as its appearance and size do not necessarily confirm its pathological nature, its correlation with associated symptoms, or the necessity for treatment [59]. The prevalence of plica syndrome varies between the different types; however as an overall prevalence a range between 20% and 25% has been reported [58]. As a consequence of trauma, repetitive knee movements, or any form of transient synovitis with chronic inflammation may result to a hypertrophic thickened fibrotic plica with subsequent loss of elasticity [35, 60]. An altered plica in this way may cause abrasion and impingement against intra-articular structures and may lead to further synovitis and loss of articular cartilage. In adolescence, symptoms can occur during a period of growth spurt [61].

7.5.2 Symptoms

Due to the proposed mechanical nature of a developing plica syndrome, an increase in training volume and intensity may reinforce the degree of plica irritation and allows explaining its increased prevalence in sporting individual. Patients commonly report intermittent nonspecific anterior or anteromedial knee pain. Signs and symptoms can

appear diffuse and often nonspecific, resembling those of a meniscal tear or patellofemoral malalignment [60]. Sensations of snapping, clicking, grinding, "giving way," or popping closely attached along the medial side of the knee during flexion and extension have been reported [61]. Some patients report a palpable tender band discerning as cord-like structure in the medial peripatellar area [35]. In advanced stage, the knee may be painful to palpation, swollen, or may present restrictions in range of motion.

7.5.3 Diagnostics

As well as with other pathologies, one of the most important steps in diagnostics of a plica syndrome is to assess an appropriate history from the patient [61]. A symptomatic plica syndrome has been attributed to be a clinical diagnosis that may be supported by specific tests and imaging. During the palpation of the knee, the plica may be perceptible by rolling one finger over the plica fold [61]. A palpable mediopatellar plica typically present as a ribbonlike fold of tissue, which can be rolled directly against the underlying medial femoral condyle. While some patients may have a sensation of mild pain when palpating the synovial plica, it is important to ascertain while performing this test, if this reproduces their symptoms. It is also very important to compare the sensation to the opposite knee to see if there is a difference in the amount of the produced pain [61].

Dynamic ultrasound has been described as highly effective in detecting abnormalities of medial plicae in the knee, representing a good sensitivity and specificity [58]. During MR imaging, a physiological plica demonstrates a low signal on both T1- and T2-weighted sequences and is easy to identify with some degree of joint distention [35] (Fig. 7.7). 3T MRI may improve the detection of plica structures due

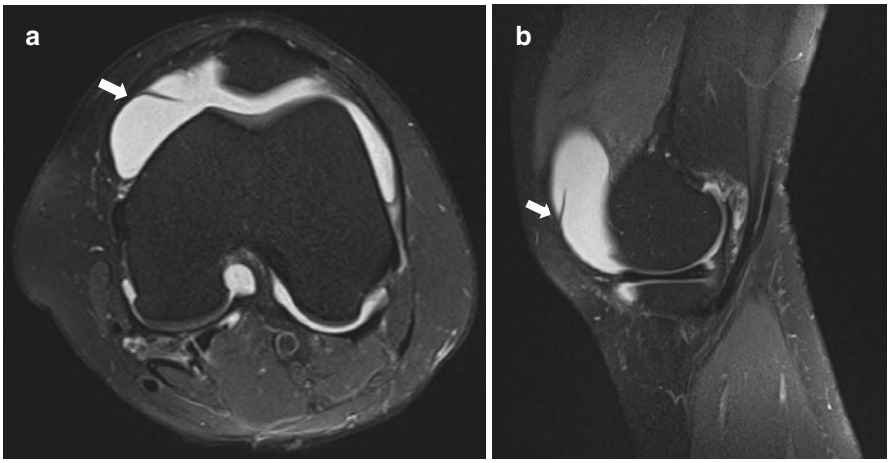

Fig. 7.7 MR sequences [PD tse fs tra (**a**) and sag(**b**)] demonstrating the presence of mediopatellar plica (white arrow). The intraarticular fluid improves the visualization of the plica due to increased tissue contrast and joint distension

to a high signal-to-noise ratio and spatial resolution [59]. Increased T2 signal intensity may be observed in symptomatic plica [35]. In unclear cases, particularly MR arthrography (MRA) may display an additional tool for detecting plicae [61]. As generally recommended (in particular in acute injuries), it is important to evaluate comprehensively, if there are different signs of pathology that are closely related to the synovial plica including infrapatellar fat pad, meniscal, patellofemoral, cruciate, and collateral ligament injuries [61].

7.5.4 Therapy

Therapeutic approaches to target a plica syndrome include both nonoperative and operative treatment. Initial nonoperative treatment focuses on symptomatic pain relief involving anti-inflammatory medication, combined with limitation of the reinforcing activities (patella-femoral loading) [58]. Stretching of the quadriceps, hamstrings, and gastrocnemius in combination with topical anti-inflammatory agents as cryotherapy and elevation has further been described to relief symptoms [58, 62]. Emphasize should be paid on patellofemoral mechanism and any imbalance in patellofemoral tracking should be addressed comprehensively [60, 61]. Quadriceps and particular vastus medialis strengthening exercises have been estimated to improve patellofemoral abnormalities [60, 61]. A recent systematic review focusing on anterior knee pain demonstrated a volume of low quality yet consistent evidence supporting the use of exercise therapy as treatment option. It was suggested that the use of exercise therapy alone resulted in a clinically relevant reduction in symptoms, increase in function, and improved overall recovery [61]. Although commonly applied in clinical practice, intraplical steroid injection should be considered critically as success may be transient and harmful sequelae (risk of infection, necrosis, chondrotoxicity) have to be taken into account [30]. Some studies declared success rates of conservative management to be low as they depend on a number of variables. As generally proposed, the infrapatellar and lateral plica are rarely amenable to conservative management and require surgical excision when symptoms are sufficiently severe [60]. However, it is advisable to consider a course of nonsurgical treatment for any form of synovial plica syndrome [60].

In case of failure of conservative treatment, the only remaining treatment is surgery by arthroscopic removal of the symptomatic plica (usually the medial plica). This is performed through the superolateral or direct medial portal and mainly successful [58].

7.5.5 Prevention of Anterior Knee Pain

Preventive interventions have received increased attention from elite as well as recreational triathletes. According to the concept published by van Mechelen in 1992 [63] the first and second step to reduce the risk of injury is to monitor the incidence of injuries and to identify underlying risk factors, respectively ("injury surveillance"). The next step is to introduce intervention programs that are likely to reduce the risk and/or severity of injury. Finally, the effect of the interventional programs must be evaluated by repeating to assess the first step. This approach requires clear-cut definitions of injury and well-designed prospective studies during all steps. However, triathlon specific studies mostly include small cohorts and are predominantly of retrospective design. Therefore, there are currently no evidence-based preventive programs or guideline that can be given in terms of the above mentioned "ideal concept" of prevention.

However, recommendations can be made on the basis of generally accepted mechanisms of injury. An overuse injury is a type of injury that is caused by repetitive impact/trauma, thereby exceeding the level to which the structure is adapted to. Hence both changes in trainings volume or intensity (training errors) as well as individual alterations in physical resilience (technique errors, individual risk factors) may prone to this type of injury. Training errors are the most common cause of overuse injuries and involve rapid acceleration of the intensity, duration, or frequency of training.

Focusing on the knee joint, imbalances between strength and flexibility, lower limb alignment or the technique may impact overuse injuries (Please refer to pathomechanism). Hence, individual deviations from physiological movement patterns in all disciplines as well as muscular imbalances and functional deficits should be cautiously and routinely assessed in injured as well as in healthy adult and adolescent athletes and have be addressed promptly [64–66]. However, even in healthy athletes, a general and individual athletic program focusing on core stability, static, and dynamic neuromuscular control should be implemented in the training process (Fig. 7.8). Deviations from an optimal movement pattern are estimated to be contributing factor to tissue stress with specific concern for excessive frontal plane knee motion [67]. A systematic review examining the evidence for the foot-shoe-pedal-interface (FSPI) intervention in competitive cyclists concluded wedges and orthoses to alter kinematics of the lower limb while cycling [67]. Hence, technical equipment and its adjustment as the bike fitting, the cycling and running shoes or orthoses, and wedges should be assessed cautiously in every athlete.

Fig. 7.8 Selected sequences of an individual developed athletic program with weight lifting exercises focusing on core and lower limb stability (**a**–**c**); (**d**) demonstrating nordic hamstring exercises (With kind permisssion: German Triathlon Union, DTU)

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Stress Fractures

8

Sergio Migliorini and Marco Merlo

8.1 Introduction

In the triathlon, the combined practice of swimming, cycling, and running permits the reduction of overuse injuries compared to the practice of running alone. Redistribution of stress over several parts of the body and the correction of muscle imbalance are cited as reasons for the reduction in injuries occurrence. Nonetheless, like other endurance sports, 80–85% of injuries can be ascribed to overuse [1]. Injury risk is greatest in elite athletes, in the youngest age categories, and in those competing in the greater distances, especially while running [2]. Running has been the most commonly reported action associated with injuries in triathletes, followed by cycling and then swimming [3, 4]. The amount of training, and therefore potential for associated injuries, varies by the distance of the triathlon races; the run may be the portion most predictive for overall success in the race [5]. Stress fractures usually appear in the lower leg due to repetitive loading and high impact forces during running, and they are commonly seen in triathletes.

8.2 Pathophysiology of Stress Fractures

Stress fractures represent a form of bone response to repetitive and excessive stress. The bone is an active substance that adapts to the loads it is placed under. There is a spectrum of bone response to repetitive stress: normal response, stress reaction, and stress fracture. Stress reactions occur when repeated abnormal bone stress without appropriate rest causes osteoclastic activity to outstrip osteoblastic activity [6]. The

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eventual adaptive response is periosteal new bone formation to provide reinforcement; however, if physical stress continues, the osteoclastic activity predominates resulting initially in microfractures (commonly seen as bone marrow edema on MRI, consistent with a stress reaction) and eventually a true cortical break (stress fracture) may result. Cortical bone turnover is much slower and most stress fractures occur in the cortical bone. Cancellous (trabecular) bone is located in the metaphysis and epiphysis of long and square bones. More active remodeling occurs in cancellous bone. Thus, there is a continuum of normal bone strain leading to appropriate remodeling, but if strain becomes excessive or adequate rest is not taken, stress reaction and eventually stress fracture can result [7].

8.3 Risk Factors

The main risk factors that may lead to running injuries in the triathlon can be subdivided into extrinsic and intrinsic factors. Identifying and modifying risk factors is the key to management of stress fractures.

8.3.1 Extrinsic Factors

From an etiopathogenetic point of view, all overuse injuries must be studied in relation to the technical characteristics of the triathlon, to the transition phases between the disciplines and particularly to the cycle–run transition, to the different load of training that the preparation for the Olympics requires compared to the long distance triathlon. Evaluation of triathletes has revealed that 68% of the preseason and 78% of the competitive season injuries were related to overuse and that 71% of the preseason and 78% of the competitive season overuse injuries were attributed to running [8]. In one study, 78% of injured triathletes identified running as the triathlon component in which they were unable to participate because of their injuries [9]. The run leg of the triathlon is more physiologically demanding than an isolated equidistant run and the cycle–run transition presents unique challenges related to the change in muscle activation [10, 11]. Athletes training for longer distance triathlons reportedly average 10.5 km swimming, 304.3 km cycling, and 71.9 km running weekly [10, 12]. Although most of the time, training for a triathlon is spent in cycling, running injuries are the most common [8]. The transition from cycling to running is usually the phase of the race in which greater performance is seen among the elite athletes compared to the middle-level athletes; this is when athletes suffer stress reactions due to minor muscle fatigue, minor changes in energy expenditure, and reorganizing of the correct running mechanics [13]. With longer biking, more time is required to regain the neuromuscular and elastic efficiency indispensable for a proper running style [4, 14]. After cycling, a time lag exists before the running technique can reach its optimal level. In this phase of the training and of the race, the inability to dissipate the load forces of the locomotor apparatus by the lower limbs can favor the transmission of stress to the lumbar–sacral region [15] and to

the knee [4, 14, 16]. The change from concentric muscular contractions to eccentric ones and from the unloaded cycling phase to the load state of running is in fact an extremely delicate phase in the triathlon [14]. Higher amounts of running are associated in general with increased risk for stress fractures [17] and a common aetiological feature is the change in either the quality or the quantity of training [18]. The rapid increase of the training program (frequency, duration, intensity) and the failure to schedule rest days, with insufficient rest and time for bone recovery, is a big risk for the development of stress fracture. Running is different from most other energetic sports due to the neuromuscular pattern and generation of loads up to 8 times the body weight. The fatigued muscle is less effective at dispersing forces, which tend to concentrate in the bone [19]. Moreover, poor running technique with high impact running style is an important extrinsic risk factor. Calf tightness with premature lifting of the heel while running transfers a significant amount of forces to the forefoot and is involved in metatarsal stress fracture. The running terrain affects the risk of stress fractures. Runners with stress fractures run particularly on irregular or angled surfaces or on hilly terrain. Moreover, a switch from marching on flat terrain to marching on hilly, rocky terrain has been shown to cause a higher incidence of stress fractures [20]. Training in shoes older than 6 months is a risk factor for stress which is likely related to the decrement in shock absorption as the shoe ages. Most doctors and trainers suggest changing running shoes every 300–500 miles and it is recognized that after 500 miles, shoes often retain less than 60% of their initial shock absorption capacity [21]. Nutritional deficiencies in calcium and vitamin D increase the risk of stress fractures [22, 23] and the supplementation of these nutrients may be protective against stress fractures [24]. Dietary insufficiencies increase the risk of stress fractures in both sexes [25, 26].

8.3.2 Intrinsic Factors

The female sex has been most frequently associated with an increased incidence of stress fractures [27]; in particular, female long-distance runners appear to have the highest risk for sustaining stress fracture [28]. Although long-distance running and triathlons can predispose the athletes (male and female) to stress fractures, it appears that the associated nutritional and menstrual irregularities seen with increased frequency in long-distance endurance running women are the major contributors to the increased risk. Abnormal levels of hormones, LH (luteinizing hormone) pulsatility, inadequate body fat stores, low energy availability (EA), and exercise stress may be aetiological factors in menstrual disorders in athletes [29]. The relative energy deficiency in sports (RED-S) has adverse consequences on the bone. Even a slight estrogen/progesterone imbalance, as seen in subclinical disturbances with low EA may produce negative changes in the bone[30] and the changes in bone structure lead to an increased risk of stress fractures [31]. Although low bone mineral density (BMD) was first attributed to hypoestrogenism caused by menstrual dysfunction, low EA is now recognized as an independent factor of poor bone health at all levels of energy deficiency due to decreased IgF-1 and bone formation markers levels

[32]. High-risk stress fractures (i.e., femoral neck) occur in adolescent athletes with RED-S and can have serious long-term consequences [33, 34]. Biomechanical factors can also contribute to stress fractures. The smaller calf girth and less mass in the lower limb of female runners were associated with stress fracture [35]. This finding may be because there is less muscle mass to absorb energy, which subsequently transmits to the bone. Poor muscular endurance is also considered a risk factor when muscle fatigue transmits more force to the adjacent bone. Static alignment and anatomic issues may contribute to stress fractures, including leg length discrepancy [35–37] and cavus [36, 38] or planus type foot [39]. Bone geometric properties may predispose an individual to stress fractures, including a thinner cortex of tibia in triathletes [40] and smaller tibial cross-sectional area in runners [41]. Dynamic biomechanical loading patterns experienced during running may also contribute to injury [42], particularly higher peak hip adduction, internal knee rotation, knee abduction, internal tibial rotation, and rear foot eversion [43, 44]. Running with excessive hip adduction and rear foot eversion are predictors of tibial stress fracture [43]. Moreover, the average vertical loading [43, 44] and higher peak acceleration [45] contribute to injury.

8.4 Evaluation

Initially, the pain of insidious onset is not present at the start of running but occurs toward the end of a run. A classic physical examination presents focal point tenderness with direct and/or indirect percussion, covered by a single finger; a swelling, a warmth or an erythema can also be present at the area of tenderness. If the triathlete continues to run, the pain typically occurs earlier in successive runs and affects the routine ambulation and the rest. Less sensitive tests for stress fractures of long bones include the fulcrum test and the hop test. It is always important to elicit history regarding predisposing intrinsic and extrinsic factors to stress fractures, considering that runners often do not recollect a specific inciting event or injury to the area.

8.4.1 Imaging

Imaging is utilized to supplement the history and physical examination and confirm the diagnosis of stress fractures. Radiographs have low sensitivity to stress fractures, and it can take up to 3 weeks for the symptoms to become evident. Often they are negative; however, an early radiographic sign can reveal periosteal reactions or cortical irregularities. Other findings include a poorly defined cortical margin, and a discrete fracture line in severe cases (Fig. 8.1). A visible callus is the most common finding of a healing fracture [7]. Bone scans are a highly sensitive modality for diagnosing stress fractures but they are nonspecific and a poor choice to monitor recovery. CT (computed tomography) can be particularly helpful in differentiating a stress reaction from a true fracture by readily visualizing the fracture line; it can also help to differentiate a stress fracture from osteoid osteoma,

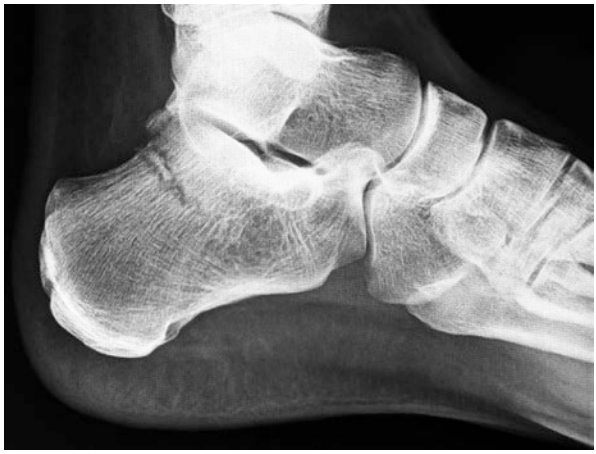

Fig. 8.1 Radiographs of a lateral calcaneus show a calcaneus stress fracture in a 35 year old woman triathlete

osteomyelitis, and malignancy [46]. CT scan appearances of healing stress fractures can be deceptive in some cases as the fracture is still visible well after clinical healing has occurred [47]. MRI (magnetic resonance imaging) is being increasingly advocated as the gold standard investigation for the evaluation of bony stress injuries. Even though MRI do not image fractures as clearly as CT scans do, the advantages of MRI include no ionizing radiation, the ability to visualize soft tissue, and its utility in visualizing a consistent bony edema with a stress reaction. Its drawback is that not all stress reactions seen on MRI relate to clinical symptoms. The MRI assists in planning treatment and counseling runners, but does demand an experienced diagnostician to decrease false-positive injuries [48]. Fredericson et al. have developed an MRI grading system [49] that has been most recently updated by Nattiv et al. [50].

8.5 High and Low Risk Stress Fractures

One common adopted general convention is to classify stress fractures as high risk or low risk, based on the anatomic location of the fracture, the natural history of fractures occurring at this anatomic site, and considering the time taken to heal and the risk for nonunion. High risk stress fractures are classified by their tendency for incomplete healing or fracture nonunion. Low risk stress fractures tend to heal with conservative measures and restriction of activity only.

8.5.1 High Risk Stress Fractures

8.5.1.1 Femoral Neck

The insidious nature of femoral neck stress fracture presentation combined with a low clinical suspicion, commonly leads to serious complications with higher

incidence of complete fracture, malunion with resultant impingement, nonunion particularly on the tension side of the bone, avascular necrosis, and arthritic changes [27]. Johansson et al. have reported a 30% complication rate in a series of athletes with femoral neck stress fracture [51]. In the young active population, progression to avascular necrosis has been reported in 20–86% of cases by various authors [52]. Runners have a history of anterior hip or groin pain which worsens with activity; if the index of suspicion is high and plain radiographs are negative, it is recommended to proceed to MRI. The femoral neck is subjected to tensile forces at the superior aspect of the femoral neck and to compressive forces at the inferior aspect. Because tension side femoral neck stress fractures are at higher risk for nonunion and displacement [53, 54], it is generally recommended that these fractures be managed with internal fixation [55]. However, there are reports of successful conservative management for non-displaced tension side fractures; tension side fractures may be managed with bed rest, as long as widening of the cortical fracture is not observed on serial imaging [56]. For compression side fractures and lesser trochanteric fractures, we recommend the use of crutches to maintain non-weight bearing status, with clinical evaluation and repeat imaging to ensure bone healing. Triathletes can advance to cycling, swimming, and cross training exercises when pain free on examination and cortical bridging is present in radiographs [42]. Resumption of running can be considered when full weight bearing is asymptomatic, there is no tenderness to palpation on physical examination, and imaging studies are consistent with healed fracture [7]. Completed healing is expected by 2–3 months [57].

8.5.1.2 Anterior Tibial Cortex

Anterior tibial stress fractures are tension side fractures and are at risk for delayed healing and nonunion. Normally, the runner has an anterior leg pain and a localized discomfort; the physical examination may reveal erythema and swelling at the site of the fracture. “Dreaded black-line” radiographs, visualized as horizontal radiolucency localized at the middle-distal junction of the anterior tibia is pathognomonic for this fracture. Both surgical and non surgical methods have been used effectively to treat anterior tibial cortex stress fractures [58]. The conservative treatment with rest and restricted weight bearing with gradual return to running has been described as unsatisfying [59]; surgical management of anterior tibial shaft fractures can lead to fracture union and a return to sports up to 5 months earlier than conservative managed fractures [7].

8.5.1.3 Medial Malleolus

Stress fractures of the medial malleolus are infrequent and are significantly less common than fractures at the distal fibula and lateral malleolus. The pathogenesis of medial malleolus stress fractures is related to abnormal weight transmission and torsional forces without a significant role for muscular forces [60]. Plain radiographs are often initially negative; the MRI is a sensitive and useful tool for the diagnosis of this fracture. Considering the contributions of the medial malleolus to the ankle mortise, we recommend an initial immobilization for 6 weeks

unless there is a significant displacement of the fracture or involvement of the talocrural joint. A surgical treatment with open reduction and internal fixation with screws, with or without plate, has a good outcome [61].

8.5.1.4 Tarsal Navicular

The navicular bone is highly vulnerable to stress fractures because of the relative avascularity complicated by a physical compression between the talus and the cuneiforms with load-bearing in plantar flexion [62]. The tenderness at physical examination is over the navicular tuberosity or the navicular–cuneiform joint and the pain during running is over the medial dorsum of the foot. There is no advantage in surgical treatment compared with non-weight-bearing immobilization. Maintaining strict non-weight bearing status initially to promote healing may result in the best clinical outcome [63]. Stress fracture of the navicular bone has a particularly high rate of failure when treated with decreased activity, but continued weight bearing [47].Those that do not respond to conservative measures, including non-weight bearing periods and immobilization, are surgical candidates [64]. Surgical treatment of navicular stress fractures is generally preferred in elite athletes and patients with high functional demands [65]. Malee et al. demonstrated an earlier return to sport at 16.4 weeks in patients treated operatively compared to 21.7 weeks when treated non-operatively in a cast for 6 weeks [66].

8.5.1.5 Fifth Metatarsal

Fifth metatarsal stress fractures are found in the proximal 1.5 cm of the diaphysis; this is in contrast to avulsion fractures that typically occur with acute inversion of the foot and peroneus brevis tendon traction. Also known as Jones fracture, it is an important fracture in runners given its high rate of delayed healing and nonunion because of the relative avascularity of the bone distal to the tuberosity. For nondisplaced fifth metatarsal fractures, 6–8 weeks of non-weight bearing immobilization is generally recommended. Use of a CAM (controlled ankle motion) walker boot may off load this site most effectively to promote initial healing [67] but there is a high rate of nonunion in partial weight-bearing programs. Surgery should be considered early in triathletes who do not respond to conservative measures or have a displaced fracture. Surgery is also an option for the elite athlete desiring an expedited return to competition by as early as 12 weeks [68].

8.5.1.6 Base of the Second Metatarsal

This fracture is considered high risk, especially if the fracture extends to the Lisfranc joint (metatarsal cuneiform joint). Immobilization for a minimum of 4 weeks is recommended. Morton toe could be an associated biomechanical risk factor for this injury because of the increased force transmitted through the second metatarsal of the foot [69].

8.5.1.7 Hallux Sesamoids

Sesamoid stress fractures are important injuries to consider in runners with forefoot pain and they represent approximately 1–3% of stress fractures in athletes [70]. The

sesamoid bones function as a pulley for the flexor hallucis longus and brevis tendons and provide stabilization at the metatarsophalangeal (MPT) joint. Sesamoid stress fractures are at high risk for delayed union, nonunion, osteonecrosis, and sesamoiditis. Stress fracture may be difficult to diagnosis considering that bipartite sesamoid with sesamoiditis may radiographically appear as a split sesamoid. The radiographs of the contralateral asymptomatic foot can show this anatomic variant. A prolonged period of non-weight bearing or casting (6 weeks) is recommended and in some cases, the use of cushioned orthosis with accommodative insole to offload the sesamoids is helpful to reduce biomechanical stress and promote healing [42]. If conservative treatment fails to relieve tenderness and pain, sesamoidectomy should be considered [71].

8.5.2 Low-Risk Stress Fractures

8.5.2.1 Sacrum

Sacral stress fracture often mimics other causes of gluteal and back pain. MRI may reveal linear abnormal signal intensity paralleling the sacroiliac joint, whereas CT may reveal linear sclerosis with cortical disruption [7]. The conservative treatment consists of rest and cross training: the triathlete usually returns to running activity around 12 weeks [72].

8.5.2.2 Pubic Ramus

Stress fractures most commonly occur at the inferior pubic ramus [57, 73]. The cause of this stress fracture is repetitive tensile stresses of the adductor magnus at its origin on the inferior pubic ramus [74]. The athlete has groin pain that is initially often mistaken for an adductor strain. Weight bearing and the return to sport should be guided by pain. Usually, it will take 2 or 3 months for the stress fracture of the pubic ramus to heal.

8.5.2.3 Femoral Shaft

This injury most commonly occurs in the mid-medial and posteromedial cortex [75] and is caused by compressive forces. Athletes generally present with insidious onset of pain that is nonspecific and localized in the groin, thigh, or knee. Injuries without evidence of displacement or cortical break tend to heal and allow return to running within 8–12 weeks [57].

8.5.2.4 Tibia

The majority of these stress fractures occur at the posteromedial aspect of the middle third of the tibial shaft; they account for nearly 50% of all stress fractures in athletes [76]. MRI may be required to reveal stress fractures and early stress reactions. A rest period of 4–6 weeks from running is generally recommended, with swimming and cycling permitted. It may take several months to return to the previous level of function [77].

8.5.2.5 Fibula

Fibular stress fractures most commonly occur at the distal stress fractures in athletes and are referred to as "runner's fractures" occurring at the narrow cortical bone; middle-aged women are more likely to have stress fractures 3–4 cm from the lateral malleolus in the cancellous bone. Distal fractures occurring at the lateral malleolus may be related to osteoporosis and intrinsic bone quality given the tendency for these fractures to occur in middle-aged women [78]. Muscular forces have been implicated in the development of fibular stress fractures, considering the fibula has less of a role in weight bearing. Easy to examine given the surface bony anatomy, these fractures tend to heal in a similar time as of other low risk stress fractures.

8.5.2.6 Metatarsal

Stress fractures of the metatarsals are common and with the exception of the base of the second metatarsal and fractures involving the metaphysis of the fifth metatarsal, fractures involving the shaft of metatarsals 2–4 are considered low risk and have a good healing response. Without the presence of a fracture line and in case of non-displaced metatarsal shaft stress fractures, the treatment is nonoperative and the use of a metatarsal pad and a firm-sole shoe may allow the athlete to ambulate without pain [57]. With the presence of a fracture line, prefabricated walker boots, or a short-leg cast with progressive weight bearing for 4 weeks may be useful. A second radiograph at 4 weeks is recommended to document evidence of bony bridging and cortical hypertrophy over the fracture site, before starting cross-training and return to running. Most injuries heal within 6–8 weeks to allow return to ground running [42].

8.6 General Treatment and Prevention Strategy

The best form of treatment of stress fracture is prevention. Poor bone mineral density (BMD) is a very common cause of stress fracture and a cancellous bone stress fracture should immediately alert the physician to investigate BMD. In athletes with low EA, eating disorders (ED), and or amenorrhea of over 6 months, BMD should be measured by DXA [79]. In the athlete population, low BMD is defined as a *Z*-score between−1.0 and 2.0 SD, together with a history of nutritional deficiencies, hypoestrogenism, stress fracture or other secondary clinical risk factors for fracture. The recommended interval to reassess BMD via DXA scan for athletes at risk is 12 months in adults and a minimum of 6 months in adolescents. Female athletes with lower BMD and hormonal irregularities have a markedly increased incidence of cancellous fracture when compared with men or other women without the aforementioned attributes. The right levels of calcium (1500 mg/day) and vitamin D (1500–2000 UI/day) supplementation appear to be protective against development of stress fractures [80]. Given the prevalence of vitamin D deficiency, it is important to measure the 25-OH vitamin D level and provide supplemental vitamin D if needed. Biphosphonates which inhibit the resorption of bone and may delay fracture maturation, are not recommended for women of reproductive age, as they are

stored in the bone for prolonged amounts of time and have been shown to be teratogenic [81, 82]. To avoid the RED-S it is important to ensure adequate energy availability and allow for ovulatory menstrual cycles and maintenance of bone mass [83].The performance improves in female athletes who maintain ovulatory function with adequate nutrition [84]; women taking oral contraceptives had a decreased risk of stress fracture [85]. In order to get back to running as soon as possible, athletes with stress fracture should avoid using over-the-counter non steroidal medications, since these drugs have been shown to interfere with bone remodeling [86]. Frequent running shoes change (every 6 months or 300–500 miles) is essential for the prevention of stress fractures; certain shock-absorbent materials (e.g., in-soles) can independently decrease the risk of stress fractures [87]. After 500 miles, the shoes often retain less than 60% of their initial shock absorption capacity [88]. It is important to note that a switch to minimalist footwear has been correlated with the development of foot injuries, most notably metatarsal stress fractures [88]. Capacitative coupling electric fields can be used safely in the treatment of stress fractures; more severe stress fractures healed more quickly with this therapy [89, 90]. Extracorporeal shock wave therapy (ESWT) has been used successfully on fracture complications, such as delayed union and nonunion; it is also a successful and noninvasive therapy for the management of resistant stress fractures [91–93]. The time to full recovery is significantly faster in the ESWT group compared with patients who only performed a graded running program [94]. Maintaining fitness during treatment of lower limb stress fracture is of significant concern to the athlete. Normally, the triathlete can continue to swim and cycle; deep floatation device is an effective form of cross training for injured athletes. During a 4–8 week deep-water run training program, runners maintained VO_2 max, anaerobic threshold, leg strength, and 2 mile and 5 km run performance [95, 96]. Gravity eliminated running (antigravity treadmills) are gaining popularity in some universities and elite running programs to keep athletes training while injured or to decrease risk in high-volume runners. Strengthening the piriformis, the gluteus maximus, the tensor fascia lata, and the digital toe flexors, may play a role in the prevention of the stress fractures of the femoral neck, femoral shaft, and the metatarsals. Runners heal at different rates and conventional formulas used to predict recovery for specific stress fractures are extremely inaccurate. In general, the severity of the stress fracture and pain dictate how quickly a runner can progress back to regular running. A runner with a low-risk stress fracture should have adequate stress fracture healing within 4–8 weeks of modified activity [97, 98]. The athlete should be pain free in all activities of daily living and there should be no tenderness for approximately 2 weeks before return to run training. The athlete must run on alternate days and return to running with a walk/run combination. It is important to increase weekly mileage on a case by case basis with significant increases in weekly distance only after 3 weeks. Athletes returning to activity after relative rest should escalate their activity by no more than 10% each week. Unfortunately, athletes minimize the prescribed rest period and often try to return to full training too early. The surgical management of certain high risk stress fractures has an increased role in improving return times and rates to sport [99, 100].

8.7 Conclusions

Stress fractures are a common injury in endurance athletes, in particular runners and triathletes. They may occur virtually in any bone in the body, but the most common affected bones are the tibia, fibula, metatarsals, tarsal navicular, femur, and pelvis. Accurate and timely diagnosis and treatment are essential in the care of the athlete with stress fracture. Possible predisposing factors in the development of stress fractures in triathletes are extrinsic and intrinsic factors [98]. The best form of treatment of stress fractures is prevention; low risk stress fractures tend to heal with conservative treatment and restriction of activity. In the high risk stress fractures, surgical intervention can be considered. Triathletes should be pain free in all activities of daily living before returning to running.

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Operative Treatment of Tendinopathies of Achilles Tendon

9

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9.1 Introduction

Achilles tendon disorders are common in physically active individuals, but can also be seen in normal non-athletic people. There are several different entities of these pains, usually called "tendinopathies" of Achilles tendon [1, 2]. This general term is diffuse and can be used clinically, when we do not have an exact diagnosis of the disorder. With history, clinical examination, and radiographic examinations, a more exact diagnosis usually can be done better. In operation and with histological specimen, more accurate diagnosis is finally done. Acute, recurrent and chronic peritendinitis tendinopathy, tendinosis of the mid-portion area or distal Achilles tendon, partial degenerative tear, retro calcaneal bursitis with or without prominent Haglund's heel, distal insertional or tendon body calcification, and some anomalies of the tendon, are the most common non-traumatic Achilles tendon overuse disorders or tendinopathies in endurance athletes [3]. In Table 9.1, Achilles tendinopathies, treated surgically, are shown in two patient series.

9.2 Indications for Surgery

If Achilles tendinopathy has been causing annoying symptoms for more than half a year, despite proper conservative treatments and rest periods, surgery becomes the last treatment option. Often, pain comes gradually and is tolerated quite well. If the tendinopathy makes endurance training impossible or symptoms reoccur after rest and conservative treatment, decision for surgery needs to be considered

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Table 9.1 Distribution of diagnoses of surgically treated chronic Achilles tendon tendinopathies in athletes

Diagnosis	1991 (330 operations)	2005 (283 operations)
Year	1991	2005
Number of operations	330	283
	%	%
Peritendinitis	54	36
Tendinosis	15	24
– Mid-tendon	10.6	
– Distal tendon	3.7	
Partial tear	15.5	11
– Mid-tendon	11.5	
– Proximal tendo	2.1	
– Distal tendon	1.9	
Retrocalcaneal bursitis (of which real Haglund's heel 3%)	17	26
Anomalous soleus	0.6	0.6
Calcification	3.0	3.6
– Tendon	0.9	
– Insertion	2.3	

Two materials from Finland

[4, 5]. There are no absolute indications for surgical treatment. In all cases, the decision has to be done individually. Surgery should be done at a time that suits best to the athlete [6]. After surgery, individual rehabilitation and recovery plan needs to be done [7]. Special attention is given to the gradual onset of training after surgery, physiotherapy, and muscle care before returning to high—load and maximal training.

9.3 Effects and Aims of Surgery

With Achilles tendon tendinopathy operations, we can alter and correct several faulty mechanical, frictional, inflammatoric and tissue compression, or breakdown problems. These surgical procedures include, for example, division of pathological pressure of the thickened crural fascia and adhesions around Achilles tendon disturbing its gliding, removing bony calcaneal prominence and inflamed retrocalcaneal bursa, repair or suturation of partial tears and degenerations, as well as making tenotomies to tendinotic tendon [7, 8]. By these local corrections, it is possible to diminish extra pressure, help circulation, release compression and friction, and thus allow the tendon tissue regenerate better. The operations also affect pain and general condition positively. Achilles tendinopathy operations are relatively small surgical procedures. However, Achilles tendon is a sensitive structure, experience and knowledge is needed of an orthopaedic surgeon to choose right timing, operation technique, and approach in each case individually [3].

9.4 Operation for a Chronic Peritendinitis Tendinopathy

In chronic peritendinitis of the Achilles tendon, there are inflammatoric changes around the tendon. Organic adhesions are seen on both sides of the tendon and in Kager's triangle. There is excessive amount of unmature proteins around (e.g. bibronectin). The crural fascia is thickened and fibrotic [5, 9]. Ultrasound and MRI examinations are recommended before surgery. They usually support the clinical diagnosis and exclude and show other possible reasons to Achilles tendon pain. Chronic peritendinitis tendinopathy can be localized around the mid-tendon area, near to myotendinous junction or is in the distal part of the tendon, too [9–13]. Mostly, peritendinitis is diffuse but can also be localized. One form is "stenosing" chronic peritendinitis, which is usually localized around the proximal tendon. In stenosing form, a fibrotic sheath is formed to cover the tendon [6]. This sheath is hard and needs to be removed radically. This type is often seen and treated in reoperations following a previous, often inadequate Achilles tendon operation.

Our approach is usually open surgery. With tenoscopy, it is sometimes possible to make medial crural fascial release and cut or excise the plantaris tendon, if the chronic disorder is also related to it [14, 15]. The open wound can be done medially or laterally. The crural fascia covering the tendon is opened longitudinally. We suggest removing part of it posteriorly to Achilles tendon. Kager's triangle is freed on both sides and adhesions cut or removed. The paratenon layer attached to fascia becomes excised, but the layer over the tendon needs to be left untouched. If there are defects in paratenon, they should be tried to suture with thin resorbible suture material. Fascial incision is continued proximally [3, 5, 7]. If the visibility is poor, endoscopic division of the crural fascia is possible to avoid too long wound. Good hemostasis is important. Post-operative hematoma can cause recurrent peritendinitis. It is also important not to pull the skin too much, because skin edge necrosis may follow it. Therefore, too small wound can cause more complications than a few centimeters longer incision. We do not make or recommend longitudinal tendon incisions (scarification, tenotomies) in chronic paratenonitis tendinopathy operations. Crural fascia is newer sutured again. Subcutis is closed with interrupted 4-0 Vicryl knots. Skin closure can be done with interrupted or continuous 4-0 or 3-0 Nylon, on with intracutaneous thin resorbible sutures.

9.5 Operative Treatment of Tendinosis Tendinopathy

In tendinosis tendinopathy, there is no inflammation in the tendon. Unmature connective tissue grows to the tendon between normal tendon fibers. A fusiform, tender swelling usually develops in the mid-tendon area. Achilles tendon tendinosis with long lasting symptoms despite rest and all conservative treatments has long been treated surgically by longitudinal tenotomies. This open surgery included longitudinal opening of the diseased tendon area, removal of soft degenerative tissue (if found). One or more tenotomies could be done to the posterior tendon or from inside to the tendon. Loose suturation of tenotomies is done. Another method is to make

subcutaneous longitudinal tenotomy via a short incision, put blade of the knife inside the nodulus, and move the ankle from full dorsiflexion to plantarflexion resulting longitudinal tenotomy of the tendon nodulus [16]. In open surgery, excessive pathological blood neovessels ventral to the Achilles tendon can be cut, sutured, or cauterized. Longitudinal tenotomy is still possible today for a chronic painful fusiform tendinosis bulging. Our approach is to make "microtenotomy" with Topaz radiofrequency method. It makes 1 mm holes to the tendon, and then tissue is transformed to H_2O and CO_2. The method is not burning or causing tendon necrosis. The holes are made in two three rows, 5–6 mm distance between the foration holes. They are made to different depths. Physiological NaCl drop is needed during the procedure, because the radiofrequency device is not functioning in dry surroundings [17]. After the procedure, good flushing with NaCl removes "free radicals" and also makes the hemostasis better. Crural fascia and possible adhesions are treated as in peritendinitis operation. The radiofrequency holes take away the internal "pressure" from the tendon and trigger healing mechanisms. Since the start of using radiofrequency mictotenotomy, no longitudinal open tenotomies have been done in our clinic. The "tendinosis tissue" seen in MRI examination is not necessary to remove. Only in cases with associated partial tendon tear that more radical operation and suturation is added to the surgery.

9.6 Surgery for Partial Achilles Tendon Tear

In top level athletes, a small partial tear of Achilles tendon may appear after direct or stretch trauma, recurrent hard performances, or from pure overuse. Symptoms can be very similar to peritendinitis or tendinosis tendinopathy. In young athletes (under 30 years), tendinosis is rare and painful swelling of Achilles tendon must be considered as a partial tear. The rupture can be very small, consisting only a tiny band of tendon fibers, or bigger, usually flap like lesion. It is difficult to find with clinical examination, and ultrasound examination only shows oedema changes around tendon. In MRI, there is better diagnosis, but the findings of superficial tear are sometimes minimal and pre-operatively right diagnosis cannot always be set. In operation, the lesion is seen. The superficial tear can be at the degenerated area, it can be partially healed and glued to the main tendon. If degeneration or bad tissue is seen, it is excised and the site is sutured with slowly absorbable or thin non-absorbable suture material. If the tear is bigger, consisting one fourth of tendon, it should be repaired with stronger suture material, and fascial or plantaris tendon augmentation [3]. In these cases, post-operative non-weight bearing and plaster, or walker boot, can be used for 2-4 weeks. Recovery takes usually 3-4 months. In athletes, partial Achilles tendon tears need to be treated operatively, to avoid total tears.

9.7 Surgery for Distal Enthesitis and Retrocalcaneal Bursitis

Distal Achilles tendon enthesitis tendinopathy is usually associated with prominent superior calcaneal corner or Haglund's deformity. Retrocalcaneal bursitis is the entity that causes most of the posterior heel pain. Friction and compression of the

Achilles tendon to the prominent bone corner also causes local pain, and in rest after exercise tendon swelling makes the heel stiff, swollen, and painful next morning. Bone may cause local friction damage to the tendon to its inner surface, sometimes partial tear. Risk for a total tear is imminent in severe cases. Tendinosis thickening usually occurs, and due to thick distal tendon, friction is increased and bursitis gets worse. Operation can be done using straight incision at the lateral distal tendon margin and insertion or straight posterior incision to the tendon insertion and heel [8, 18]. The lateral wound is less invasive. Bursectomy is done and bony prominence excised. We rasped the bone surface even and curved anteroposteriorly and from side to side. Especially important is to remove lateral, superior, and also medial bone margin with periosteum, which can also be cauterized with diathermy. New bone may grow from periosteum, not from the middle of the resected bone. In cases with bigger partial tear, bone anchor plasty to the distal tendon may be needed. In very severe cases with Haglund's heel, calcaneal osteotomy can be done. Sometimes, reinsertion of the Achilles tendon is required too. Endoscopic technique is increasingly used today for excision of the posterosuperior calcaneal corner [19].

9.8 Surgery for Achilles Tendon and Heel Enthesitis Calcifications

Calcified spurs at the Achilles insertion are rare, but can be seen in over 20 year old athletes, as well as calcifications in the body of the tendon, too. If a calcification is seen developing, it is treated long conservatively. At least 1 year has to pass before operative treatment is considered. In some cases, the outlines of the calcification may remain obscure, and the bone mass is growing for a long time. It is better to wait until the mass is solid and "mature," before surgery is possible. Operations are done with bloodless field tourniquet. The calcified mass is carefully removed from the tendon. In distal calcification spurs, the tendon may remain thinner and weaker after removal of triangular bone spike. Bone anchor repair can then be used, too [20]. We routinely use indomethacin 50 mg daily after surgery for 4-5 weeks to prevent early recurrences of calcifications.

9.9 Achilles Tendon Anomalies

Achilles tendon can be long or short. In cases with short tendon, the soleus muscle is often hypertrophied. It can cause chronic peritendinitis tendinopathy symptoms. By surgery, not only liberation of the tendon is done. Resection of the oversized muscle below and on both sides of the tendon is likewise done. Fasciotomy on both sides of the tendon and removal of fascial flaps is recommended. There may also be an anomalous soleus muscle on the medial side [21]. This excessive muscle comes from medial soleus and a fusiform muscle is attached with an own tendon to the medial calcaneus. The treatment is done to remove this muscle-tendon anomaly. Sometimes, the tendon has septum-like fold from the inner tendon to calcaneal

bone. The two main Achilles tendon bands can be partially separated from each other. The tendon can also be too tight, even without any neurological reason. The medial side can be thick and tight. The treatment in these cases is individually selected.

9.10 Results of Operative Treatment

The end results of Achilles tendon tendinopathy operations are usually excellent or good in 60–80% of surgically treated cases [8, 22–25]. The results depend on the experience of orthopaedic surgeons performing these operations. One needs to see and examine lot of athletes and tendon problems to be able to estimate which disorders require surgery. There are different surgical methods and techniques, which may affect the post-operative healing time, complications, and end result [3]. In addition to the earlier mentioned surgical techniques, there are several additional procedures used in special cases [26]. In Table 9.2, post-operative results in two patient series are shown. In the tendinosis groups, longitudinal tenotomy was used in the earlier series and radiofrequency coblation technique in the later series. Results from that were significantly better.

9.11 Post-operative Care and Rehabilitation Guidelines

After surgery, the operated leg is kept in elastic compressive bandage, if cast is not required. Supporting bandage is recommended at least for 2 weeks. Elevation of the extremity in bed is recommended as much as possible during the first 2 or 3 days and after that, several times daily for 1 week. Anti-inflammatoric medicines are prescribed to be taken by mouth for a few days and local anti-inflammatory gels after removal of sutures. Walking crutches are used for a few days and partial weight bearing is allowed next day after surgery. Ankle motion is kept limited (only 20–30°) until the removal of sutures, but toes can be flexed and extended freely. Cold packs are used several times a day for 3 days. Removal of sutures can usually be done 10–14 days after surgery. After that, when the wound has been seen to be healed well, full mobility of the ankle is allowed. Then swimming,

Table 9.2 Excellent or good results of Achilles tendinopathy operations in two patient series, both comprising approximately 300 operations

	Excellent or good result (%)	
Diagnosis of tendinopathy	1990	2005
Chronic paratenonitis	81	84
Retrocalcaneal bursitis	75	76
Partial tear	69	72
Tendinosis	47	70

water running, and other water exercises can be started. Bicycling and light gym training are allowed 3 weeks after surgery. Walking is done increasingly weekly, but only for a few kilometers 1 month after surgery. Later, light jogging, more gym and weight training, and cycling are allowed. Returning to "normal" running takes usually 2 months [6, 8, 13]. If there is excessive stiffness, swelling, or pain, physiotherapy may help and one needs to proceed with training more slowly. Cold treatment after exercises is good during the first few weeks. If the symptoms have lasted long before surgery, calf muscle care and strengthening is necessary. After tendinosis tendinopathy operations, eccentric training is started 3 – 4 weeks after surgery and continued for 2 months. In other Achilles operations, individual plan is done for stretching, strength training, physiotherapy, and footwear. In cases with partial tear or bone anchor plasty, the post-operative recovery takes longer time.

9.12 Complications of Achilles Tendon Overuse Injury Operations

The rate of complications following Achilles tendon tendinopathy operations has generally been counted to be from 10% to 20% [7, 8, 23–25]. Most of them are minor and tolerable and only shortly delay final healing. With good operation technique and considering all possible factors on the background of complications, their number is reduced. Severe complications may also occur as skin necrosis demanding big plastic surgery repairs to cover the skin defect with vascularized skin grafts [27]. We could decrease the number of post-operative complications from 12.5% to 8.8%, with good planning and with special care for operation technique. Orthopaedic surgeons treating athletes and Achilles tendon tendinopathies have learned to respect Achilles tendons and try to make operations as gently and smoothly as possible with atraumatic technique. In Table 9.3, complications related to Achilles tendon tendinopathy operations are shown.

Table 9.3 Complications seen after Achilles tendon tendinopathy operations in athletes

Skin necrosis (partial, moderate, severe)
Infection
Hematoma
Seroma
Pseudocyst
Fibrosis, contraction, sheath formation
Calcification, spur
Nerve injury (n. suralis)
Scar keloid formation

With good planning and careful operation technique the number of complications could be decreased from 12.5% to 8.8% (Paavola, Orava et al. Am J Sports Med 2002)

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Part IV

Medical Conditions

Airway Dysfunction in Elite Athletes

10

James O'Donovan, Michael Koehle, and Don McKenzie

10.1 Introduction

The mental and physiological demands of competition as an elite triathlete are extreme and challenge the limits of human performance. Most exercise scientists agree that oxygen transport is the major limiting factor to performance in events such as these and with years of training there are significant adaptations that occur in the musculoskeletal, cardiovascular and hematological systems that lead to improved performance. Larger lung volumes and an increased pulmonary diffusion capacity have been observed in swimmers and there are measureable changes in the strength and endurance of the respiratory muscles with training. However, there are no structural changes in the lung parenchyma, airways or chest wall that accompany a training program [1]. There is general consensus that the respiratory system is overbuilt for the demands of ventilation and gas exchange required by the elite athlete [2]. Minute ventilation can increase 20-fold from resting values, implying an adequate buffer exists to deal with the demands of heavy work. Nevertheless, there are unique physiological conditions that expose the respiratory system as a weak-link that could lead to decreases in endurance performance.

Exercise-induced arterial hypoxemia (EIAH) is seen much more frequently in elite athletes. This condition occurs in healthy athletes with no evidence of respiratory disease. It is observed in more than 50% of highly trained athletes. EIAH is more prevalent in female athletes and occurs earlier in high intensity work than with males [3]. The mechanisms remain unclear and complex: diffusion limitations, relative hypoventilation, V/Q mismatch, rapid transit time, transient pulmonary edema and intra-pulmonary shunts all have been implicated in the development of EIAH [4]. Of concern to the athlete, EIAH has been associated

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with reduced performance, with a linear relationship between the decrease in hemoglobin saturation and work capacity on a cycle ergometer [5].

Expiratory flow limitation (EFL) occurs in many endurance athletes during heavy exercise. This leads to an increased work of breathing, dyspnea, EIAH and poor performance. Again, EFL is more prevalent in females; and the masters athlete, capable of heavy work, is also compromised with EFL as the aging process results in a loss of elastic recoil leading to a reduction in the maximal flow volume loop [6].

While these physiological conditions are present in many endurance athletes, asthma remains by far the most common clinical problem in the triathlete and, in fact, is the most common medical condition seen at major games. In the elite athlete, asthma is much more prevalent in sports with a significant endurance component and triathlon ranks with cycling and swimming as the sport with the highest percentage of asthmatic athletes at the Olympic Games [7].

10.2 Clinical Presentation

Asthma is a condition defined by inflammation and bronchial hyperresponsiveness with characteristic clinical symptoms. The Global Initiative for Asthma (GINA) defines asthma "as a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness, and cough that vary over time and in intensity, together with variable expiratory airflow limitation" [8]. The clinical presentation features much overlap with other common respiratory conditions, causing difficulty in athletes when distinguishing between normal symptoms associated with vigorous exercise. This is particularly true for elite sporting activities where breathlessness, wheeze, cough, chest tightness, and phlegm are often present in relation to extreme exercise [9]. This creates a diagnostic challenge for the physician when diagnoses are based on symptoms alone, which with asthma, is commonplace in the general population.

Exercise-induced bronchoconstriction (EIB) describes the acute onset of bronchoconstriction occurring during, or immediately after exercise, and is a particular clinical phenotype of asthma, relevant to endurance athletes. It is important to highlight the importance of exercise for asthmatic patients, as distinguished from this specific phenotype. However, there is some overlap between the conditions. In EIB, exercise acts as a trigger for bronchoconstriction through an inflammatory cascade or physiological adaptation. Symptoms usually appear within 10 min of exercise, generally peak within 10–15 min, and will have resolved by 60 min. The typical symptoms are shortness of breath, chest tightness and cough. Breathlessness is the dominant symptom, reported in 50% of winter athletes, in contrast wheezing and waking with breathlessness are less prevalent in athletes than in the general population [10]. Any associated hoarseness or stridor is uncommon and should raise the possibility of exercise-induced laryngeal obstruction (EILO).

When taking a history from a suspected EIB case, symptom presence, the rigor and duration of exercise, as well as the environmental conditions must be considered, as these have an impact on EIB [11]. One must be mindful to have a higher

suspicion in athletes participating in winter or endurance sports. Seventy percent of asthmatics are atopic and a history of allergies, hay fever, eczema etc. is important to document. Other possible triggers should be identified.

10.3 Diagnosis

The IOC Medical Committee and WADA Physician Guidelines recommend that an objective test be used to confirm the diagnosis of asthma, such as spirometry demonstrating airflow limitation with reversibility following inhaled bronchodilator or a positive bronchoprovocation test.

In athletes, respiratory symptoms are non-specific and provide no diagnostic certainty in the confirmation of EIB. It is recommended that a diagnosis of EIB in an athlete should not be based on symptoms alone. In the absence of objective tests of airway function, other diagnoses such as upper airway dysfunction, i.e. EILO, may be overlooked and also may lead to the unnecessary prescription of therapy [7]. An objective test that shows reversibility of airway obstruction is recommended and this can be achieved by evaluating the response to a bronchodilator medication with exercise or from bronchoprovocation testing (Table 10.1).

Table 10.1 Tests to assist in the diagnosis of asthma and/or airway hyperresponsiveness

Type of test	Test and description	Criteria for positive
Bronchodilator	Inhale a rapid acting beta 2 agonist	≥12% or greater increase in baseline FEV_1
Provocation tests		
Indirect tests		
Exercise	Exercise challenge in the laboratory or sport specific in the field of 6–8 min duration achieving 80–90% of maximum heart rate	≥10% reduction in baseline FEV_1
Eucapnic voluntary hyperpnea (EVH)	Ventilate at a target of 30 × FEV_1 for 6 min breathing dry air containing 5% carbon dioxide	≥10% reduction in baseline FEV_1 in two consecutive time points
Hypertonic saline	While tidal breathing, inhale nebulized 4.5% hypertonic saline for increasing time periods	≥15% reduction in baseline FEV_1
Mannitol	Inhale increasing doses of dry powdered mannitol to a maximum cumulative dose 635 mg	≥15% reduction in baseline FEV_1
Direct tests		
Methacholine	Increasing concentrations of aerosol methacholine are delivered either via the tidal breathing (preferred) or dosimeter methods	≥20% reduction in FEV_1 to a PC_{20} ≥ 4 mg/mL or if glucocorticosteroids have been inhaled ≥ 1 month, a PC_{20} ≥ 16 mg/mL

Bronchial provocation tests (BPT) are used to identify airway hyperresponsiveness and to aid the diagnosis of asthma in athletes. BPTs are either indirect or direct. The majority of athletes with respiratory symptoms will have resting FEV_1 values within normal range (80% of their predictive value). In these individuals, an indirect airway challenge is the most appropriate means of establishing a diagnosis. The term "indirect" challenge is used because a positive response is dependent on the release of mediators (e.g. prostaglandins, leukotrienes and histamine) produced by inflammatory cells in the airway, which then cause an airway smooth muscle contraction, with subsequent airway narrowing. The indirect tests include the exercise challenge, the eucapnic voluntary hyperpnoea (EVH) test and the administration of hyperosmolar aerosols such as 4.5% saline and mannitol. To make the diagnosis, using exercise or EVH, a reduction in FEV_1 of 10% is consistent with EIB. A fall in FEV_1 greater than 15% in response to 4.5% saline or mannitol is consistent with the clinical diagnosis of asthma.

An exercise challenge test is often preferred in a sport-specific setting, as it allows the test to be undertaken in an environment where the athlete is usually symptomatic. For example, a swimmer would undertake their exercise challenge in the pool, while a skater would be tested in an ice rink. An exercise challenge test typically incorporates a 2-min warm-up directly followed by approximately 6–10 min of sport-specific exercise sufficient to raise the heart rate to 80–90% of the predicted maximum. A test is generally considered positive if the FEV1 decreases by 10% or more from rest to consecutive time points during the post-exercise spirometry measures which are recorded at 3, 5, 7, 10, and 15 min post-exercise. While any exercise test of sufficient intensity and duration, under dry-air ambient conditions, can be used to elicit a bronchoconstricting response for EIB screening, it is best to employ an exercise challenge that mimics the individual's actual athletic event [12]. Some studies have demonstrated that an individual may present EIB symptoms during their particular sporting event, yet be symptom-free during a laboratory exercise challenge. Conversely, those who react to a non-exercise laboratory test (e.g. methacholine or EVH challenge) may be asymptomatic during their actual event [13]. To ensure that an athlete is appropriately treated, a sport-specific challenge following medical intervention should be employed, if possible. This may provide useful information on the degree of asthma control and permit an adjustment in the treatment plan if required. These tests can be done frequently if there is a question about adequate control.

The EVH test is considered to be the most sensitive indirect airway challenge to diagnose EIB. It is also useful to identify patients with EIB who have had a false negative exercise test [14]. However, in some cases, it is considered too sensitive and has been postulated that it may result in overdiagnosis due to the implementation of screening in squads/athletes who may have been asymptomatic and not had any detriment in performance prior to diagnosis. On the other hand, one could argue that the screening process revealed many athletes who did not recognise and/or report exercise respiratory symptoms and therefore trained and competed with uncontrolled EIB [15]. During the EVH test, the athlete is required to breathe at 85% of their maximal voluntary ventilation (30 times FEV1) for 6 min while

breathing dry air containing 5% carbon dioxide [16]. The added carbon dioxide from a compressed gas cylinder prevents the athlete from becoming hypocapnic from the hyperventilation. The test duration, temperature, and ventilation level can be varied to simulate the conditions of the sport. As with the exercise challenge test, a positive test is regarded as a falling FEV_1 of 10% or greater from two consecutive time points post-EVH challenge. Hyperosmolar aerosols such as the mannitol challenge test have a similar sensitivity and specificity to EVH challenge in summer athletes, but reduced sensitivity in winter athletes. The mannitol challenge test is not available in all countries.

10.4 Management

The goal of treating an athlete with asthma is to control symptoms of asthma and prevent progression, with minimal effects on sporting performance. The management of athletes with asthma should follow current national or international guidelines (Fig. 10.1). There is no evidence supporting different treatment for asthma or exercise induced bronchoconstriction (EIB) in athletes vs. non-athletes. However, some specific issues need to be considered for the elite athletic population.

Non pharmacologic treatments must be maximised in athletes. Education is critical as athletes are frequently training or competing away from their source of medical support and they must have an action plan for exacerbations of their asthma. Fundamentally, reduction in associated environmental exposures is very important. Athletes are continually exposed to differing air quality, allergens, and temperatures often during times of high minute ventilation associated with intense exercise. Potentially controllable factors are the magnitude of minute ventilation and the temperature and humidity of the inspired air. Improving an athlete's cardiovascular fitness reduces the minute ventilation required for a given exercise. However, for elite athletes this is often at a maximal regardless. Bronchoconstriction is lessened when inspired gas is warmer and more humid. It has been advised to breathe through a loosely fitting scarf or a mask when exercising in cold dry conditions, although this has limited use when high minute ventilation is required. The importance of temperature is reflected in the high prevalence of 30–50% airway hyper responsiveness in cross country skiers, who have high minute ventilations sustained in cold/dry ambient conditions [17]. In addition, other environmental factors must be considered such as outdoor air pollution, known allergens, exhaust fumes from ice resurfacing equipment, and chlorine gas from swimming pools. For example, a swimmer who is sensitive to chlorine may be less symptomatic if using a pool with lower chlorine levels (e.g. one filtered by UV or ozone). These triggers must be identified, and exposure reduced wherever possible, especially during training.

Warm-up is important to the asthmatic athlete as exercise can influence the respiratory response during competition or training. Interval and continuous exercise as a warm-up both induce an effect [18]. A continuous 15-min warm-up at ~60% maximal aerobic capacity will reduce post-exercise bronchoconstriction in moderately trained athletes [19].

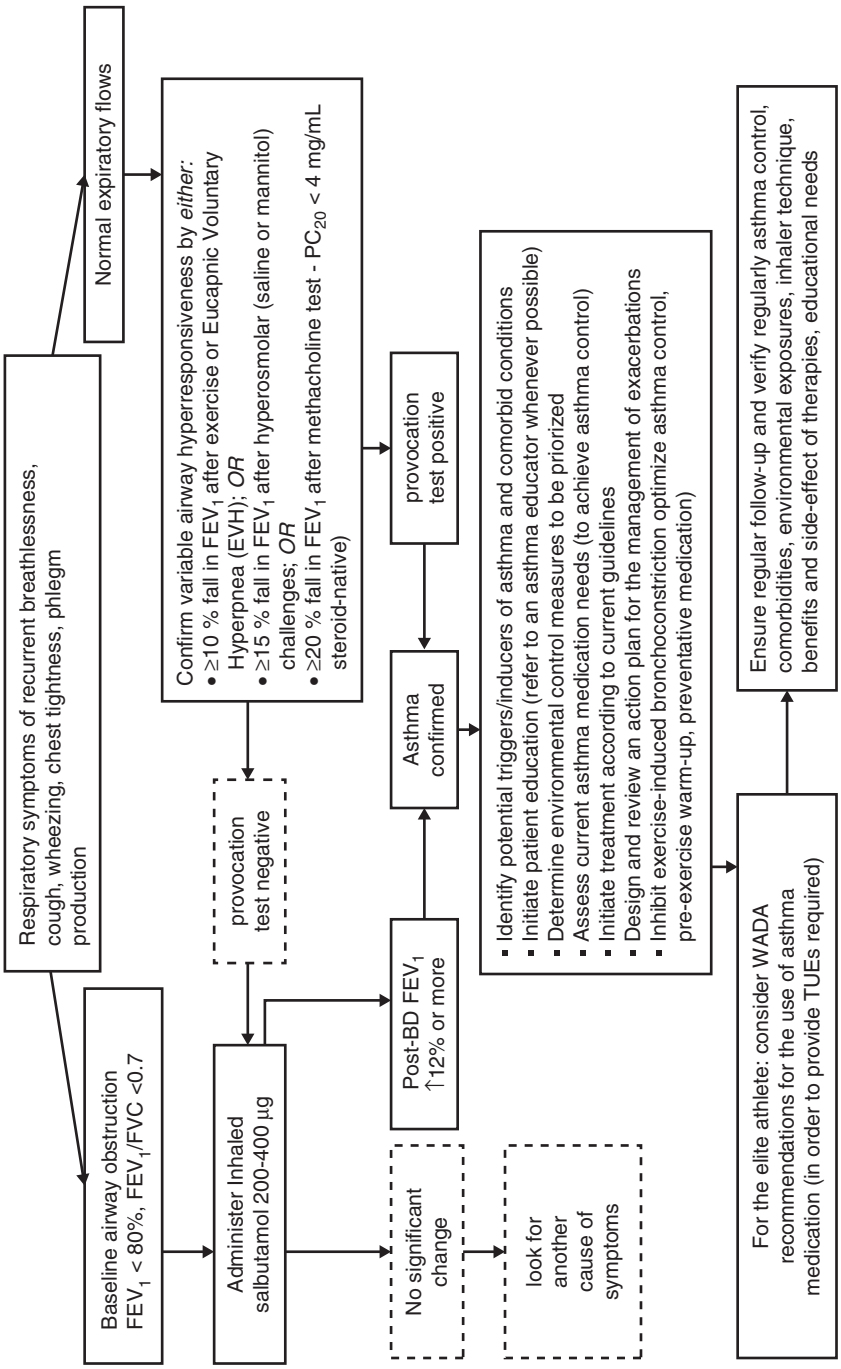

Fig. 10.1 Asthma management for the athlete (From [7]). Reprinted from J Allergy Clin Immunol. 2008 Aug;122(2):254–60, 260.e1–7. Fitch KD, Sue-Chu M, Anderson SD, Boulet LP, Hancox RJ, McKenzie DC, Backer V, Rundell KW, Alonso JM, Kippelen P, Cummiskey JM, Garnier A, Ljungqvist A. Asthma and the elite athlete: summary of the International Olympic Committee's consensus conference, Lausanne, Switzerland, January 22–24, 2008. with permission from Elsevier

Approximately 40–50% of individuals who have an initial episode of EIB experience a refractory period of diminished responsiveness that can last 1–4 h after the initial warm-up exercise. The cause of this refractory period is not fully understood but may be caused by the depletion of catecholamines, increased circulation of prostaglandin, or degranulation of mast cell mediators [11].

Pharmacological treatment of asthma in elite athletes should follow standard guidelines for treatment individualised in a step-up and step-down approach to achieve asthma control with the effects being constantly monitored (see Fig. 10.1). Inhaled corticosteroids (ICS) represent the gold standard for long term control of asthma and the prevention of EIB [7]. Inhaled short acting B_2 agonists (SABA) form the mainstay of immediate treatment of EIB and for the athlete with intermittent asthma. Further pharmacological intervention is guided by frequency of use of B_2 agonist and presence of other symptoms.

Athletes with well-controlled asthma, but who frequently have symptoms with exercise, are often instructed to use prophylactic treatment approximately 5–15 min before exercise, usually with two puffs of SABA. However, tolerance (tachyphylaxis) occurs rapidly after a few days of regular use. Thus, to maintain bronchoprotection, limiting the use of SABA to competition or workouts where EIB might develop is another strategy to consider in the triathlete. One might imagine that an inhaled long acting B_2 agonist (LABA) would be useful in this situation. However, current guidelines do not recommend regular use of LABAs as monotherapy [7]. Tolerance is neither prevented by inhaled corticosteroid treatment nor overcome by using a higher dose of B_2 agonist [20]. However, anti-inflammatory treatment may help to reduce the severity of EIB and therefore reduce the need for B_2 agonists.

For athletes not optimally controlled with ICS and SABA, oral anti-leukotriene antagonists (LTRAs) represent another line of treatment. They must be taken at least 2 h before exercise to have a maximal protective effect, but the effect lasts 12 (zafirlukast) to 24 (montelukast) hours [21]. While not effective in all patients, LTRAs appear superior to LABAs when treating asthmatics with EIB.

For patients who are intolerant of SABAs, pre-treatment with ipratropium will provide partial protection against EIB. This inhaled anticholinergic agent reduces the decrease in FEV_1 relative to placebo, but is less effective than SABAs or mast cell stabilizing agents [22].

It is important that a correct inhaler technique is learned and monitored. Use of a spacer, (also known as aerosol-holding chambers, add-on devices and spacing devices), may make it easier to inhale the medicine delivered by a pressurized MDI, improve the lung deposition and decrease the deposition in the mouth and throat. Advising an athlete to maintain a peak flow diary is also useful.

10.5 WADA

When working with athletes in the elite sporting environment, it is imperative that the physician has a sound knowledge of the current World Anti-Doping Association (WADA) Prohibited List [23]. It is very important to be familiar with the medication

prohibited lists that apply to in-competition and out-of-competition use. Oral and injected B_2-agonists are prohibited at all times. All B_2-agonists (e.g. fenoterol, higenamine, indacaterol, olodaterol, procaterol, reproterol, terbutaline, vilanterol) are prohibited and require a TUE (therapeutic use exemption), with the exception of salbutamol, salmeterol and formoterol at defined doses.

Inhaled salbutamol is currently permitted at doses of 1600 μg over 24 h, in divided doses not to exceed 800 μg over 12 h. However, the presence of salbutamol in the urine in excess of 1000 ng/mL is presumed not to be a therapeutic use of the substance and is considered an adverse analytical finding.

If systemic GCSs are required for the treatment of an exacerbation of asthma before competition, a retroactive/emergency TUE should be submitted as soon as possible to the International Triathlon Medical TUE committee. Supporting medical documentation must accompany the TUE Application.

10.6 Other Conditions

Although most respiratory symptoms in endurance athletes can be attributed to EIB as a cause, two other conditions not infrequently cause dyspnoea and other respiratory symptoms in the competitive triathlete: immersion pulmonary edema, and exercise-induced laryngeal dysfunction.

10.6.1 Immersion Pulmonary Edema (IPE)

This condition was first described in SCUBA divers, initially attributed to deep diving in cold water. Subsequently, a number of case series reported a similar condition in individuals swimming on the surface of the water [24–26]. In fact, unilateral pulmonary edema has been described in Special Forces swimmers that swim for long periods on one side [27] with the edema occurring only on the dependent side. IPE is believed to be caused by increased blood flow to the pulmonary vasculature resulting from a combination of immersion, cold and intense exercise. This overpressure in the pulmonary vessels leads to leakage into the airspaces and symptoms. One study has indicated a decrease in the number of lymphatic vessels in susceptible individuals, but this result has not yet been confirmed [28]. Key risk factors for IPE seem to be cold water, strenuous exercise and fluid loading prior to immersion [29]. Diving depth does not seem to be a risk factor [25].

IPE presents with a rapid onset of dyspnoea during immersion. IPE can be accompanied by cough, fatigue, frothy sputum and hemoptysis. Without treatment, it can be fatal in severe cases. On auscultation, crackles can be heard. Radiography can confirm the pulmonary edema, when available. Treatment is supportive, including rapid removal from the water, oxygen, and in severe cases, ventilation. These treatments are curative if administered quickly enough. Symptoms typically resolve within a few hours of supportive care [24], less in very mild cases. IPE can be differentiated from EIB due to its episodic occurrence only during immersion and (in

severe cases) the hemoptysis and severe respiratory distress. In triathlon, event organisers need to ensure that lifeguards and on-water safety personnel are aware and adequately trained in recognition and management of the condition. Anecdotally, salmeterol has been used in elite triathletes who are particularly susceptible to the condition, based on its ability to increase activity of the Na/K pump in the alveolar membrane to remove fluid. Likewise, nifedipine has been tried (but not tested) for this condition, based on its ability to reduce pulmonary artery pressure [25].

10.6.2 Exercise-Induced Laryngeal Obstruction (EILO)

Respiratory symptoms are often attributed entirely to the lung. However, one of the key conditions that cause dyspnoea in endurance athletes (including triathletes) has an extra-thoracic etiology. This condition has a variety of names, but is most commonly referred to as Exercise-induced Laryngeal Obstruction (EILO), Paradoxical Vocal Fold Motion (PVFM), or Vocal Cord Dysfunction (VCD). The aetiology of the condition is airflow obstruction occurring at the level of the larynx, specifically at the vocal cords. With a normal inspiration, the vocal cords abduct, allowing increased airflow. In contrast, with EILO the abduction does not occur, and airflow is restricted through the cords. This restriction most often occurs due to paradoxical adduction, but it can also occur as a result of dystrophy and softening of the arytenoid cartilages that support the vocal cords.

This airway obstruction is manifested as dyspnoea, stridor, cough, choking, difficulty getting air in [30] and impaired performance, typically at very high ventilatory rates [31]. The prevalence can range from 3% to 12% in different populations [32] and can occur during swimming, cycling or running, but in general, swimming seems to be the triathlon sport that is the most aggravating. EILO is more common in female athletes, and more common in athletes with a higher level of fitness. Other factors linked to EILO include anxiety, and gastroesophageal reflux disease (GERD). EILO is a challenging diagnosis and is often misdiagnosed as EIB. However, both EIB and EILO can often coexist in elite athletes.

Diagnosis is suspected based on history of mostly inspiratory symptoms, coming on during high intensity endurance exercise, with poor to no response to bronchodilators. Spirometry can be normal when asymptomatic at rest, but when symptomatic there can be flattening of the inspiratory phase of the flow-volume loop. When possible, direct laryngoscopy during an episode is diagnostic. Due to the relative rarity of the condition in the general public, it can often require a long time with multiple visits to a series of clinicians before the proper diagnosis is made [1].

The cornerstone of management is to make the correct diagnosis and to educate the patient on the aetiology and nature of their condition. Athletes often benefit from some breathing training, under the guidance of a clinician with expertise in the condition, such as a speech-language pathologist. The athlete learns awareness of the larynx's role during exercise hyperpnea, and learns strategies to properly open the upper airway to prevent or abort an attack. This can include breathing and phonation training, postural exercises, and even respiratory muscle training with inspiratory

resistive devices [33]. In difficult cases, with a significant anxiety component, counselling and therapies directed at alleviating the anxiety may also be appropriate [32]. Likewise, the treatment of other associated conditions such as EIB, and GERD (if present) is also a key to a successful outcome [31].

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Cardiovascular Adaptations in Triathlon

11

Sanjay Sharma and Maximiliano Moreira Accame

11.1 Introduction

The impact of regular physical activity on health has long been well established. Several studies have demonstrated positive cardiovascular benefits, either by reducing the risk profile for atherosclerotic coronary artery disease or through primary and secondary prevention of many chronic diseases. The volume and intensity of exercise required to achieve these health benefits are relatively modest in the general population [1, 2] in comparison with the amount of exercise performed by endurance athletes.

Over the past few decades, the number of individuals engaging in endurance sports has significantly increased, particularly in long-distance running (marathon and ultramarathons) and long-distance triathlons (half and full distance and above), and up to 45% are aged above 40 years old [3–12]. Triathlon participation is a growing sport worldwide, with more than 500,000 participants per year in the United States.

A triathlon comprises three different sports of swimming, cycling and running, across an extensive range of distances and race venues [6–8]. The intensity and workload of the training of competitive triathletes are 10- to 20-fold greater than the current exercise recommendations for the general population and may be associated with profound structural and electrical manifestations occurring as a physiological response to a chronic increase in cardiac preload and afterload [1, 13–19]. Athletes are exposed to several environmental stressors such as water or ambient temperature, which can dramatically vary along the course and pose different physiological

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demands in each part of the competition. These factors can lead to various medical problems and may trigger acute cardiovascular disturbances in athletes with underlying cardiac conditions, thereby representing a challenge for the emergency response plan during mass triathlon events [7, 20].

This chapter will focus on the cardiovascular adaptation to endurance exercise, exercise-related sudden cardiac death (SCD) during exercise and methods for preventing SCD. Precise definition of a competitive athlete is difficult to ascertain. Competitive athletic status is usually conferred to an individual who engages in regular physical training (>6 h/week) and participates in official sports competitions with an emphasis on excellence and achievement [2].

11.2 Endurance Sport and the Heart

Endurance sports are defined as those typically resulting in >70% of the maximal oxygen uptake, such as cycling, rowing, swimming or triathlon, and are associated with a five- to sixfold increase in cardiac output. Many endurance sports such as the triathlon and rowing have a high degree of both dynamic and static components of exercise [21–23]. The dynamic component is associated with a high cardiac preload, and the static component is associated with a high cardiac afterload. Chronic increases in preload and afterload for prolonged periods necessitate the development of a plethora of structural and functional adaptations within the heart [23–25]. These changes also impact on the resting ECG and are collectively termed the "athlete's heart". Sinus bradycardia and modest increases in cardiac dimensions are common; however the precise magnitude of these adaptation is governed by the intensity and cumulative duration of training protocols and also by several demographic factors including age, sex, body size and ethnicity [23–33].

11.3 Electrical Changes in the Endurance Athlete

The electrical manifestations of the athlete's heart can be broadly attributed to increased vagal tone and increased cardiac mass [23, 34]. Common electrocardiographic patterns in endurance athletes include sinus bradycardia, sinus arrhythmia, first-degree atrioventricular (AV) block, large-amplitude QRS complexes and early repolarisation pattern which are observed in over 50% of endurance athletes [34, 35] (Fig. 11.1). Such changes are most profound in large, adult male athletes with the highest training volume. Endurance athletes may also reveal junctional bradycardia or Mobitz type I second-degree AV block on the resting ECG. Sinus pauses >3 s during sleep are common. Anterior T wave inversion is generally considered abnormal in white adult athletes when it is present beyond V2 [17]; however up to 4% of endurance athletes may show T wave inversion in V3. In such cases the preceding ST segment has J-point elevation and a convex ST segment; the T wave itself is often biphasic [36]. T wave inversion in the inferior and/or lateral leads is not a feature of athletic training and warrants further investigation.

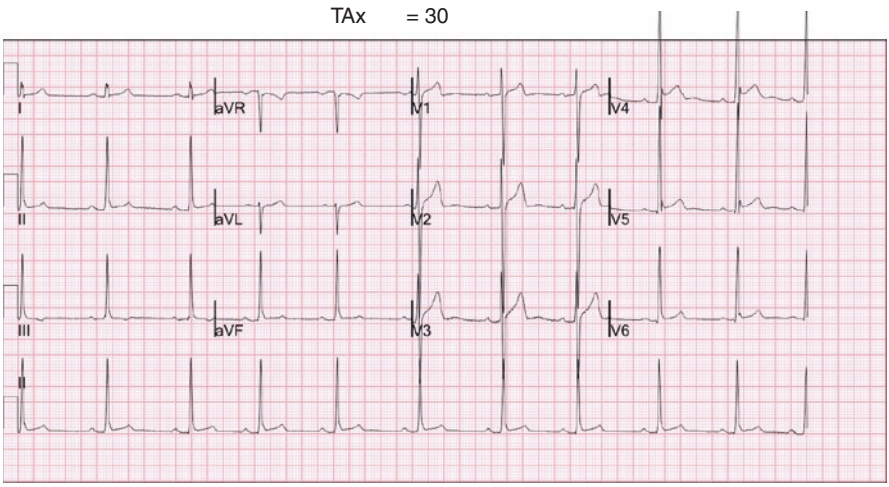

Fig. 11.1 ECG changes in endurance athletes

11.4 Structural and Functional Changes in the Endurance Athlete

The acute cardiovascular response to endurance exercise includes a substantial increase in heart rate, stroke volume, cardiac output and systolic blood pressure and a marked decrease in total peripheral resistance [37–39]. Long-term adaptations to repeated bouts of dynamic exercise include enhanced cardiac filling in diastole, increased cardiac chamber size and mass and augmentation of stroke volume even at maximal heart rates.

In the skeletal muscle, there is increased oxidative capacity due to an increase in the cellular concentration of mitochondria and enzymes responsible for oxidative phosphorylation. Furthermore, there is an increase in the density of capillaries surrounding skeletal muscle fibres. In combination, these adaptations facilitate an increased arteriovenous oxygen difference [40]. The combination of an increased cardiac output and arteriovenous oxygen difference results in a high peak oxygen consumption which is the most commonly used marker of fitness.

Endurance athletes commonly show a symmetrical increase in all four cardiac chambers and cardiac mass [31–33, 37–39, 41–43]. Endurance athletes may also reveal a slightly increased aortic root diameter but rarely >40 mml [32]. Echocardiography is usually the first-line imaging study, and assessment of athletes participating in a variety of sporting disciplines reveals a 10–20% increase in left ventricular wall thickness and 10–15% increase in left and right ventricular cavity size compared with the upper limits of the general population [30–32, 44–47] (Fig. 11.2). In absolute terms around 2% of male athletes may show a left ventricular wall thickness >12 mm; however, our own experience suggests that a left

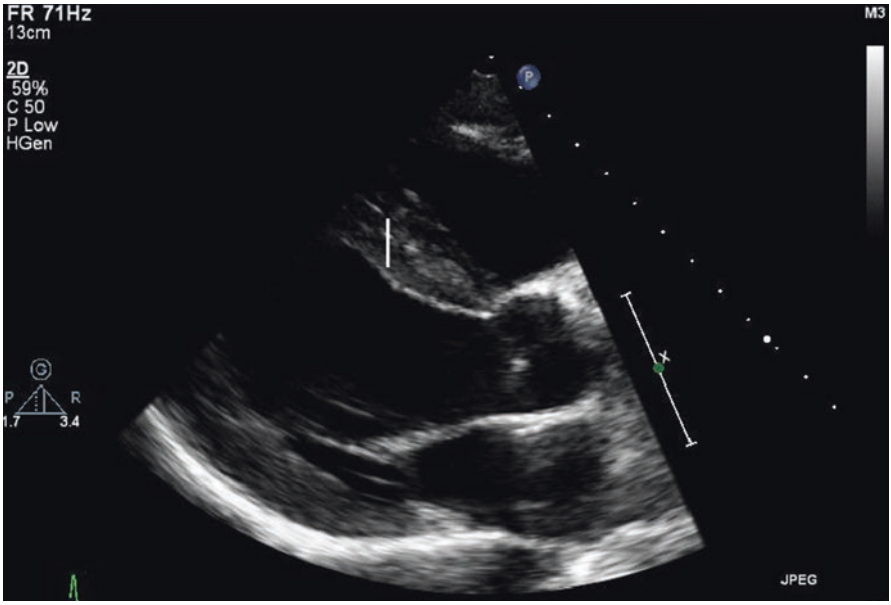

Fig. 11.2 Echocardiography and structural changes in endurance athletes

ventricular wall thickness >14 mm is extremely uncommon in triathletes. In contrast a significant proportion of endurance athletes may reveal a left or right ventricular cavity size that overlaps with dilated cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy, respectively.

Around 10% of such athletes may have a borderline low ventricular ejection fraction which further compounds the diagnostic dilemma. In contrast with patients with cardiomyopathy, athletes do not reveal regional wall motion abnormalities of the ventricle and show a rapid improvement in ejection fraction with submaximal exercise. Additionally, indices of diastolic function are normal.

Cardiovascular magnetic resonance (CMR) is increasingly used in clinical practice to assess cardiac morphology and has a pivotal role in evaluating cardiac structural changes in competitive athletes who often present with challenging diagnostic dilemmas. CMR allows a more accurate assessment of myocardial structure and tissue characteristics including fat content and myocyte mass. Gadolinium-based contrast agents enable the detection areas, inflammation, infarction or focal fibrosis, and techniques as extracellular volume imaging by T1 mapping can permit the detection of diffuse myocardial fibrosis [47–54].

Recent evidence has shown that 17% of middle-aged triathletes have focal myocardial fibrosis in the basal and mid-inferolateral LV wall which follows a non-ischaemic pattern. The precise cause or mechanisms of myocardial fibrosis remains unknown; however, a long competition history, increased cycling mileage, increased systolic blood pressure during exercise and subclinical healed myocarditis are postulated explanations for these findings [55].

11.5 Sudden Cardiac Death (SCD) in Athletes

Although endurance athletes are considered to represent the fittest and healthiest segment of society, triathlon races may be associated with sudden death. Such deaths can be attributed to race-related issues such as drowning, heatstroke, exercise-associated hyponatraemia and drowning, but a significant proportion are due to quiescent cardiac abnormalities [56–61].

The prevalence of SCD in athletes is low but varies widely depending on the population studied and methods by which the data were collected [62–65]. Data which rely on web-based search engines and insurance claims suggest that the prevalence of SCD is between 1–2/100,000, whereas data based on systematic registries such as the Football Association in the UK reveal a prevalence of almost 7/100,000 [65]. The absence of standardised post-mortem evaluation and a national registry of SCD in most of the countries means that there is also a high degree of variability on data relating to the most common causes of SCD worldwide [56, 60, 61, 66–70]. Sudden cardiac death in young athletes is usually due to inherited congenital abnormalities of the heart. Several studies from North America have shown a higher incidence of cardiomyopathies, such as hypertrophic cardiomyopathy, as a leading cause of SCD in young athletes followed by anomalous coronary artery origins [60, 61, 68]. Data from the Veneto region of Italy, where there is a mandatory pre-participation cardiovascular evaluation of athletes and maintenance of systematic registry for SCD, suggests that arrhythmogenic cardiomyopathy is the most common cause of death in young athletes [57].

Recent data from the UK, New Zealand and Australia have demonstrated that the most common finding at autopsy, in up to 40% of the cases under the age of 35 years was a structurally normal heart and was probably due to an electrical cardiac disorder [64, 66, 69, 71–73]. There is overwhelming evidence that coronary artery disease is the most common cause of death in athletes aged >35 years old [69].

The overall risk of SCD in athletes affected by the aforementioned conditions is increased by 2.5- to 6-fold depending on the condition in question indicating that exercise is a trigger for fatal arrhythmias in vulnerable individuals. The vast majority of fatalities occur in male athletes and occur during or immediately after exertion. Most (80%) of affected athletes do not exhibit any warning symptoms [74, 75].

11.6 Sudden Cardiac Death in Endurance Events

Most data on the incidence of SCD in endurance sport is derived from marathon running. However, limitations exist due to varying definitions of SCD, methodology of subject recruitment, differences in sample size and the detail of the athletic and past medical history of the deceased [3–5]. Despite such methodological inconsistencies, the current evidence has shown that the incidence of SCD in marathons ranges from 0.6 to 1.9 per 100,000 runners and is more frequent in males. The most

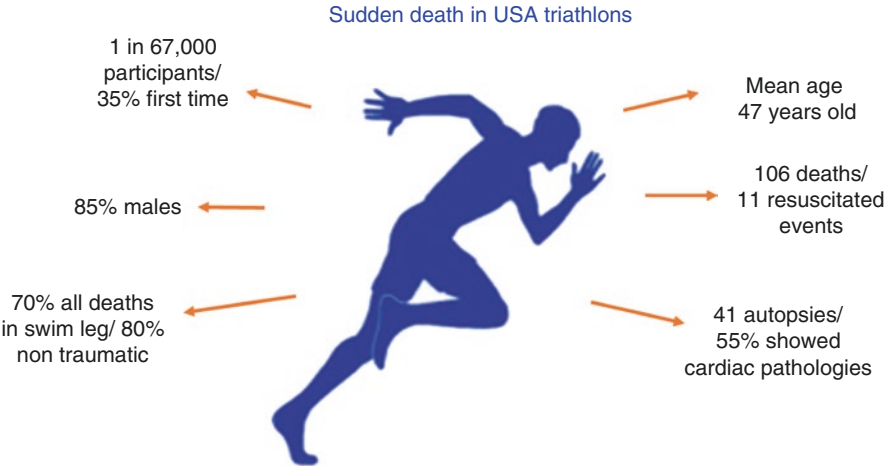

Fig. 11.3 Evidence of sudden death in US triathlons (based on data from Harris K., et al. ACC 2016)

frequent causes of cardiac arrest according to documented autopsies are hypertrophic cardiomyopathy and coronary artery disease, and the mean age of death ranges from 37 to 48 years [3–5, 9, 76].

The most accurate evidence regarding the incidence of SCD in triathlons is derived from an extensive report of case series, using the United States of America Triathlon (USAT) records and the United States registry of sudden death in triathletes over a 30-year period with more than nine million participants [8] (Fig. 11.3). Of the 135 related deaths and cardiac arrests reported, the vast majority occurred during the swimming stage of the triathlon and mostly in the open ocean.

Such deaths may be secondary to drowning, swim-induced pulmonary oedema, heatstroke, a structural cardiac abnormality or an ion channel disorder such as long QT syndrome, where the adrenergic surges from the diving reflex may precipitate fatal arrhythmias. Less than a quarter of the total of deaths were in women, and the risk appeared to be threefold higher in males aged 40 years and compared with younger males [8].

Nearly 50% of deaths occurred in short-distance triathlons, and half of the arrests were amongst first-time triathlon participants, which could indicate that less-trained or less-experienced athletes have higher risk. The mean age of SCD was 44 years old. Amongst athletes that underwent autopsy, a cardiac abnormality was detected in over 50% of cases, and the most common finding was coronary artery disease and probable hypertrophic cardiomyopathy, which is very similar to the data reported for the marathon runners [3, 6, 8].

The crude incidence of SCD reported in that study was 1.7 per 100,000 participants which seems to be higher than previous reports in long-distance running races. Survival from was only 11% during triathlons, which is much lower than the 29–43% survival after cardiac arrests reported in marathon runners [4]. Several

factors can contribute to the low survival rate amongst triathletes, and these may include the logistical challenges of a water rescue, both the recognition of an emergency situation with a swimmer and the extraction and transport to a setting suitable for advanced resuscitation and the more varied physiological environment [4, 6–9, 20].

11.7 Cardiac Screening

Awareness of SCD in young athletes has increased over the last few decades, and cardiac screening has become a crucial element to identify those who may be at increased risk of SCD with view to performing medical and surgical interventions, if appropriate and providing specific exercise recommendations [62–67].

The American Heart Association (AHA), the European Society of Cardiology (ESC) and many sports governing bodies, such as the International Olympic Committee (IOC), have developed or endorsed consensus documents providing guidance on pre-participation cardiovascular screening programmes for young athletes. However, a standardised pre-participation screening strategy for all remains elusive, given the challenges of resource limitation, logistical problems and the risk of false-positive and false-negative results [65, 67–71].

The addition of the ECG to the health questionnaire and physical examination is associated with a dramatic improvement in the sensitivity of cardiac screening in athletes considering that most athletes harbouring serious cardiac disease are asymptomatic prior to death [32, 68–73]. The ECG is effective for detecting the Wolff-Parkinson-White syndrome, long QT syndrome and marked repolarisation changes that may lead to a subsequent diagnosis of cardiomyopathy (Fig. 11.4).

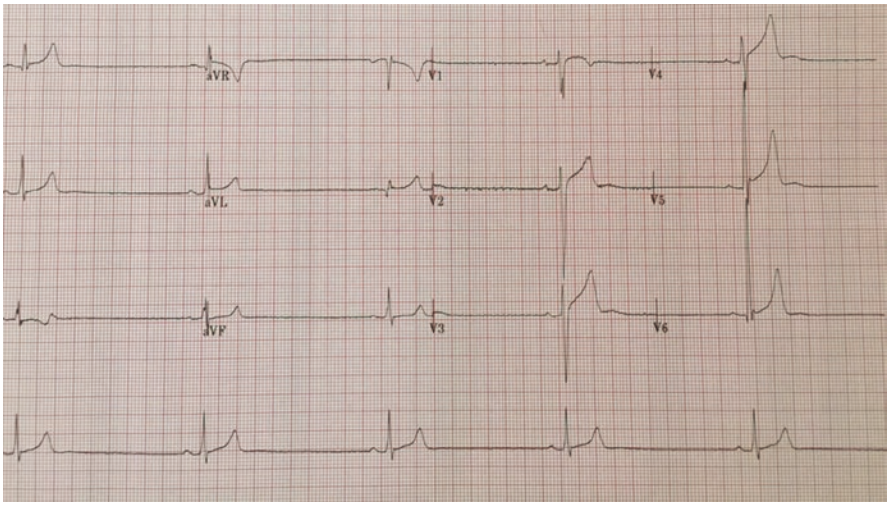

Fig. 11.4 ECG in endurance athlete and interpretation

Although there is occasional overlap between electrical manifestations of physiological cardiac remodelling, recent evidence-based studies in large cohorts of athletes and patients with cardiomyopathy have refined our approach for interpreting the athlete's ECG [4–7, 77, 78].

The new international recommendations for ECG interpretation in athletes account for age and ethnicity and non-specific ECG changes with a low diagnostic yield such as axis deviation or voltage criteria for atrial enlargement. These recommendations have improved the specificity of ECG screening without compromising sensitivity, increased the positive predictive value of an abnormal ECG and reduced the costs of subsequent investigation of an abnormal ECG by 21% compared with the former 2010 ESC recommendations [4–7, 77–79].

The cardiac screening in athletes over 35 years remains a challenge because most die from coronary artery disease where the resting ECG is ineffective for disease detection. Exercise stress tests will only detect athletes with severe (>70% stenosis) coronary artery disease. CT coronary angiography can identify silent coronary plaques but is expensive, involves radiation and only provides additional prognostic information in individuals with a moderate atherosclerotic risk profile. Furthermore, some endurance athletes show increased coronary artery calcification compared to sedentary counterparts, and the significance of such changes is uncertain.

According to the current guidelines, only athletes with symptoms and risk factors should be subjected to investigations beyond a health questionnaire, physical examination and 12-lead ECG [10–12, 80–83].

11.8 Conclusions

Endurance athletes including triathletes show several physiological electrical and structural alterations in response intensive exercise. Such changes are most marked in large adult male athlete and may occasionally overlap with the phenotype observed in cardiomyopathy. Sudden cardiac death is a rare complication and is usually secondary to an underlying cardiac abnormality. Most deaths occur during the swim phase and largely affect middle-aged males. Pre-participation screening with the ECG is most effective for identifying young athletes with inherited electrical disease and cardiomyopathy. The most effective strategy for preventing deaths involves an emergency response plan capable of delivering cardiopulmonary resuscitation and application of an automated electrical defibrillator.

Appendix: The ITU PPE

ITU Medical Documents

Pre-competition Health Screening

The ITU Medical Committee strongly recommends a periodic health evaluation (PHE) for all the triathletes, performed by a sports physician, starting with a Pre

Participation Examination (PPE) prior to engaging in competitive sport. (ITU Competition Rule 2.4 Health)

More than 90% of sudden death in competitive athletes are related to pre-existing cardiovascular problems (SCD). The SCD of an athlete on the field remains the most devastating medical event in sports.

The purpose of this screening is to identify, as accurately as possible, athletes at risk in order to advise them accordingly. According to the European Society of Cardiology (ESC), epidemiology studies on population of thousands of competitive athletes showed a decrease of up to 89% in SCDs with a PPE including a 12-lead rest ECG.

The screening takes place in three steps:

1. Answer the **Medical Questionnaire**: this questionnaire is strictly confidential and must be given to the responsible team doctor before the medical examination.
2. **Physical Examination**: by the doctor following the IOC recommendations published in the "Lausanne Recommendations" about the Sudden Cardiovascular Death in Sport Consensus.
   (a) Cardiac auscultation:
      - Rate/rhythm
      - Murmur: systolic/diastolic
      - Systolic click
   (b) Blood pressure
   (c) Radial and femoral pulses
   (d) Marfan stigmata
3. A **12-lead rest electrocardiogram (ECG)**. Doctor to look for anomalies in rhythm, conduction or repolarisation.

Select cases with a positive personal history, family history of potentially inherited cardiac disease or positive physical or ECG result will require further evaluation by an age-appropriate cardiac specialist.

All athletes competing in the ITU competitions in junior, U 23 and elite categories must have completed a pre-competition health screening which includes a questionnaire, a physical examination and an ECG following the IOC's recommended procedure.

Thereafter, all athletes junior, U 23 and elite, competing in the ITU competitions, must complete the medical questionnaire and undergo a medical examination each year and, in addition, must undergo a resting ECG every 2 years.

It is the responsibility of the National Federations to ensure that these Pre-Competition Health Screening procedures have been performed and the NFs are required to confirm to ITU with the PPE Certification that all entered athletes have completed the screening.

For all other triathletes age-group, this screening is strongly recommended.

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Endurance Anemia, Relevance to Triathlon

12

Gaetano Cairo

12.1 Introduction

Anemia is a frequent pathologic condition and a public health problem, as some years ago Global Disease Burden estimated that 2.36 billion people were affected by anemia, more than half due to iron deficiency [1]. Anemia can be defined as the reduction below reference values of the total number of erythrocytes (red blood cells, RBCs) accompanied by low concentration of hemoglobin (Hb) in the circulating blood (<5 × 10^6/mm^3 and 13 g/dL, respectively, in young males). The wide range of "normal" values (4.7–6 × 10^6/mm^3 and 14–18 g/dL) and the fact that normal levels are lower in females than in males, justified by an average smaller muscle mass in the former, indicate that a more appropriate definition of anemia could be a condition in which the Hb-mediated oxygen (O_2) transport capacity of the blood is not adequate to match tissue demand, thereby resulting in tissue hypoxia [2]. This definition is particularly suitable to sports medicine. Anemias commonly result from (1) impaired erythrocyte production, (2) blood loss (acute or chronic), (3) increased erythrocyte destruction, or (4) a combination of these three factors. Physiological conditions involving higher requirements and thereby increasing the risk for anemia are present in children under 2 years of age, adolescent girls, pregnant women, and regular blood donors. However, athletes, in particular those performing demanding activities like triathlon, are also at risk. In this context, as maximal O_2 consumption, which strongly depends on the O_2 carrying capacity of the blood, is a primary factor in endurance exercise [3], it is evident that even a mild anemia, which does not create particular problems to a sedentary individual, may instead severely affect athletic performance.

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Anemias are classified by their causes (e.g., anemia of chronic disease) or by changes that affect the size, shape, or Hb content of the erythrocyte. The most common classification is based on the erythrocyte's size (i.e., normocytic, macrocytic, and microcytic anemias).

12.2 Sports Anemia

Since an efficient O_2 transport capacity of blood exerts a highly positive effect on aerobic exercise, anemia is associated with impaired athletic performance [3]. However, a preliminary question that needs to be considered, and possibly answered, is whether sports anemia actually occurs or instead athletes present the so-called pseudo-anemia, caused by plasma volume (PV) expansion with consequent decrease of Hb concentration [4]. Of course, whether anemia should be corrected in athletes depends on the answer to this preliminary question. Measuring red cell volume (RCV) and PV through the carbon-monoxide (CO) rebreathing technique, which accurately measures total Hb mass (tHb-mass) and from which RCV and PV are derived [5], would provide an answer, but this method cannot be practically used in large epidemiological studies. In the clinical practice, anemia is generally considered as the result of impaired Hb synthesis or increased RBCs loss or destruction, whereas an increase in PV is rarely taken into account as a cause. However, this issue may be relevant in athletes. In fact, changes in PV caused by ionic disturbances in blood and muscle which are due, in part, to a redistribution of water and electrolytes between body fluid compartments are associated to exercise. A decrease in PV occurs as an acute response to moderate and intense exercise and is the result of several factors, such as (a) increased capillary perfusion, which causes water movements from the plasma compartment into both the interstitial and intracellular fluid compartments of contracting muscle, (b) changes in osmotic gradients, (c) blood redistribution from the splanchnic and renal circulations, and (d) fluid losses due to increased perspiration during exercise. However, it has been shown that adaptation to training, by stimulating renin, aldosterone, and vasopressin production and increasing plasma protein content, reduces this PV contraction and may actually lead to PV expansion, disproportionately to RCV. This adaptive process is dependent on training intensity and may lead up to 20% expansion of PV in elite distance runner, resulting in a consistent decrease of Hb concentration [4]. However, rather than being detrimental to the athlete, such a PV expansion, by rising blood volume, may increase heart stroke volume, improve thermoregulation, and decrease blood viscosity, thus contributing to better athletic performance. Together with the enhancement of RCV, which is another well-recognized effect of endurance training, PV expansion (i.e., pseudo-anemia) facilitates O_2 delivery to tissues and thereby ameliorates aerobic exercise capacity.

Having recognized that sports pseudo-anemia may occur, though often without adverse effects on performance, it should be stated that "real" sports anemia is a frequent phenomenon in athletes, particularly females, and is most commonly associated to endurance exercise. Detecting the existence of the most common

causes of anemia may help in distinguishing between the two possibilities, thus favoring therapeutic interventions in case of "real anemia."

12.3 Clinical Manifestations of Anemia

As described above, the fundamental physiologic manifestation of anemia is a reduced O_2 carrying capacity. Symptoms of anemia diverge, depending on the body's capacity to compensate for reduced O_2 availability in tissues [6]. Anemia that is mild and develops progressively is usually easier to tolerate, but may cause problems during physical exertion, and thus is particularly relevant for the great and protracted energy demand occurring in endurance sports like triathlon. As the reduction in RBCs continues, symptoms become more pronounced, and alterations of specific organs and compensatory effects become more apparent. Among the most common consequences of anemia in the general population are cognitive impairment in children, increased morbidity/mortality of mothers, worse outcome of concomitant disorders, decreased physical performance in workers, and reduced quality of life. Compensation for the reduction in the number of RBCs generally involves the cardiovascular, respiratory, and hematologic systems and may lead to severe consequences if anemia is chronic. Compensatory actions on the pulmonary system include an increased rate and depth of breathing in an attempt to increase O_2 availability. At the cardiovascular and hematologic levels, the PV expansion, in order to maintain the volume of blood unchanged, and the dilation of small vessels, in order to decrease vascular resistance and increase blood flow, contribute to a higher venous return, which leads to increased heart rate and stroke volume in a continuous effort to meet normal O_2 demand and prevent cardiopulmonary congestion. If the underlying anemic condition is not corrected, these compensatory mechanisms may eventually lead to cardiac dilation, valve insufficiency, and heart failure [2].

12.4 Anemia and Exercise

Complications like dyspnea, syncope, angina, compensatory tachycardia, and heart dysfunction are present in severe anemia but do not usually affect athletes. However, even in mild, but chronic, conditions, decreased O_2 delivery to tissues causes symptoms like increased heartbeat and fatigue when O_2 demand is increased (e.g., during physical exertion). Of course, given the well-established link between anemia-dependent impairment of O_2 transport and work capacity [7], this would affect exercise performance, in particular in endurance sports like triathlon [8]. In fact, high maximal O_2 uptake (VO_{2max}) values of elite endurance athletes are related to O_2 transport capacity and it has been demonstrated that higher Hb concentrations improve VO_{2max} and enhance endurance performance [9]. Interestingly, it has been shown that both tHb-mass and PV are similarly expanded by some 35% in elite endurance athletes [10], leading to Hb concentrations in athletes matching

those of sedentary individuals, but resulting in a substantial increase of RBCs oxygen transport in athletes that supports better performance. More recently, the relationship between hematocrit and endurance performance was analyzed in mice with different RBCs concentrations [11]. The study showed that optimal RBCs concentrations maximizing aerobic performance are the result of a balance between O_2 transport and blood flow. In fact, increased Hb values are associated with a rise in blood viscosity, higher peripheral vascular resistance, and reduced cardiac output that, in addition to increasing the risk of adverse events like thrombosis, may diminish VO_{2max} and exercise capacity. This issue impinges on the blood doping problem.

12.5 The Prevalence of Anemia Among Athletes

Maintaining an adequate Hb level is essential for athletes to avoid the effects of anemia and to maintain or improve performance. Athletes are generally affected by microcytic anemia, often determined by iron deficiency (see below). Most studies investigated anemia in women, as young, menstruating females, particularly those with heavy menstrual bleeding, are at an elevated risk of anemia. Examination of laboratory results obtained from more than 1500 cross-country female athletes showed that one in 20 was anemic [12]. Additionally, a great percentage of exercising women are not clearly anemic but show iron deficiency, a condition that may negatively affect performance or even athlete's health by leading to subsequent development of anemia [13] (see below). The elevated exercise energy expenditure of elite female athletes practicing endurance sports like triathlon partly explains the high prevalence of anemia, which is greater than in exercising women of other sports.

12.6 Etiology of Anemia in Athletes

The causes leading to anemia in endurance athletes are multifarious, and several components, such as dietary, hemorrhagic, hemolytic, and inflammatory factors, may coexist.

Chronic blood loss due to minimal gastrointestinal hemorrhages caused by injury to gut linings is a source of anemia. For example, intestinal cells may be damaged by the ischemia occurring during exercise, when blood is diverted to muscles, and by post-ischemic reperfusion after the exercise, or by the side effects of anti-inflammatory drugs often used by athletes. Furthermore, in athletes (including tri-athletes), hemolytic anemia is frequently secondary to erythrocyte damage caused by repeated mechanical stresses. Though increased hemolysis caused by circulatory trauma, oxidative stress, and osmotic changes may occur also during not weight-bearing activities like swimming, the primary cause appears to be footstrike hemolysis: the impact with the ground during running sessions damages RBCs in capillaries of the bottom of the foot [14].

Nutritional imbalance may lead to anemia when the intake of micronutrients necessary for RBCs formation, such as folates, vitamin B_{12}, and iron, is inadequate to the high requirement of a challenging activity like triathlon. Of these micronutrients, iron is probably the most important, as this essential metal plays a key role in several processes related to physical exercise beyond Hb synthesis and RBCs multiplication. In fact, iron-containing enzymes and proteins are required for many cellular pathways involved in exercise performance, such as the mitochondrial electron transport system [15] and myoglobin synthesis. Moreover, with the exception of vegetarians, triathletes are more prone to iron deficiency than at risk of incurring in B_{12} and folates deficiency.

12.7 The Importance of Iron

Iron is an essential micronutrient that is necessary for physiological processes essential for athletic performance, such as O_2 transport, energy production, and cell division [16, 17]. However, an excess of highly reactive "free" iron, by interacting with the reactive oxygen species (ROS, superoxide and hydrogen peroxide) or lipid peroxides that are inevitably and continuously generated during the normal activity of cells living under aerobic conditions, may be toxic. In fact, these reactions produce hydroxyl or lipid radicals that damage lipids, proteins, and nucleic acids, thereby leading to cell and tissue injury. Therefore, iron homeostasis is strictly regulated, and in recent years there have been important advancements in our knowledge of the underlying processes [18]. Here, I will briefly summarize how body iron balance is controlled, but it should be noted that every cell has the possibility to regulate its own iron content [19], mainly through the action of iron regulatory proteins (IRP1 and IRP2), which, in response to intracellular iron levels, modulate either the stability or the translation of the mRNAs coding for proteins involved in iron uptake, storage, utilization, and export [20].

The discovery and characterization of hepcidin represented an important advance in understanding systemic iron homeostasis (Fig. 12.1). Hepcidin, a peptide primarily produced and secreted by hepatocytes, controls body iron homeostasis by inducing the internalization and degradation of ferroportin, the major or sole cellular iron exporter. Inhibition of ferroportin leads to iron retention (predominantly in duodenal enterocytes, macrophages, hepatocytes, and placental cells) and decreased levels of plasma iron [21].

Hepatic hepcidin expression is upregulated mostly in response to iron excess and inflammation, in order to avoid additional flow of iron into the circulation [22]. On the contrary, hepcidin is downregulated in response to iron deficiency, anemia, and hypoxia (including high-altitude hypoxia), a response that increases iron availability [23]. Erythropoietin, produced by the kidney in response to decreased O_2 availability, stimulates the production and secretion of the hepcidin inhibitor erythroferrone (ERFE) in the bone marrow [24] (Fig. 12.1).

In addition to Hb, which is involved in O_2 transport, and myoglobin, a hemoprotein known as an O_2-storage protein and also an O_2-diffusion facilitator, which

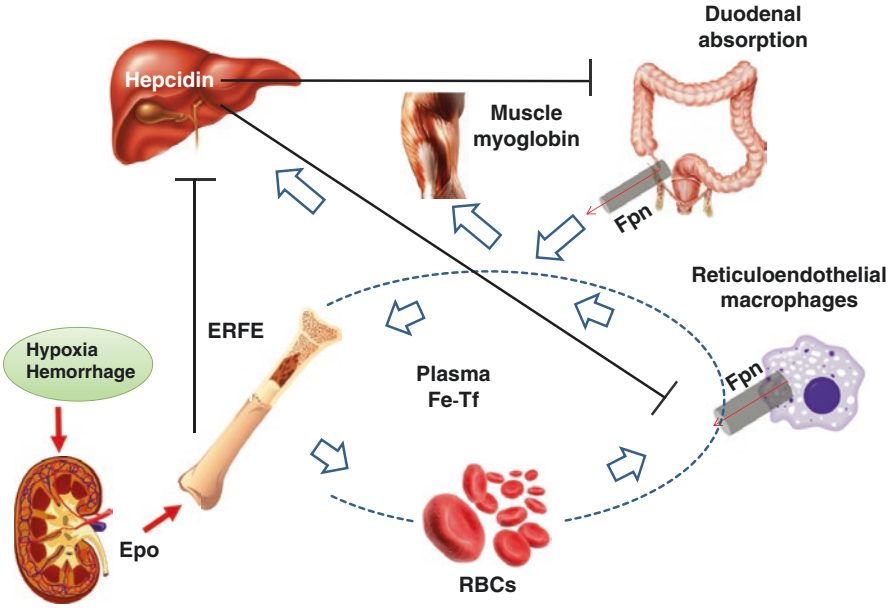

Fig. 12.1 Simplified model of hepcidin-dependent regulation of systemic iron homeostasis. Plasma iron transferrin (Fe-Tf) delivers the iron supplied by ferroportin (Fpn)-mediated duodenal absorption and the iron-recycling activity of macrophages to various organs (e.g., the skeletal muscle) and tissues including the bone marrow, which incorporates the metal into red blood cells (RBCs) hemoglobin. Liver-derived hepcidin controls systemic iron homeostasis by inhibiting Fpn-mediated iron flow into circulation. Hepcidin synthesis in the liver is regulated positively by body iron stores and inflammatory signals and negatively by erythropoietic activity and hypoxia. Under conditions of enhanced erythropoiesis, the kidney produces erythropoietin (Epo) which activates erythroferrone (ERFE) synthesis in the bone marrow. In turn, ERFE downregulates hepcidin production, thus favoring Fpn-dependent iron release into plasma and increasing iron availability

account for most body iron, the list of iron-containing proteins encompasses proteins of vital physiological significance. Among the iron-containing proteins relevant to exercise and performance, it is worth citing those involved in energy production, such as the heme-containing cytochromes and the proteins endowed with Fe-S clusters that are part of the mitochondrial electron transport chain and the tricarboxylic acid cycle [15, 25], as well as the Fe(II)- and α-ketoglutarate-dependent dioxygenase family members, which play a key role in oxygen sensing, fatty acid metabolism, etc.

Since the Hb mass is related to O_2 delivery to the working muscle, and mitochondrial energy production depends on the activity of a number of iron-dependent enzymes and proteins [15, 26, 27], it is evident that iron is key to most physiological variables of athletic performance, particularly in endurance exercise [28, 29]. The necessity of an optimal iron balance for both maximal and submaximal aerobic capacity exercises has been recently reviewed [17, 30–32].

In line with this view, a number of recent studies confirmed the well-known concept that lack of iron affects physical performance, particularly in women, who experience iron deficiency more frequently [33]. Conversely, iron supplementation improves measures of physical performance [34].

How an optimal iron balance attains this effect is still undefined. In fact, iron-deficiency anemia impairs performance by reducing blood O_2 carrying capacity, but a number of reports indicated that iron deficiency without anemia may impact physiological performance and work capacity as well [32].

The evidence that iron is a cofactor for proteins involved in a variety of functions [35] may help in explaining why recent studies showed that iron treatment also improves fatigue in subjects with iron depletion without anemia [36], confirming earlier findings in humans [37] and animals [38] and suggesting that the beneficial effect of iron may be mediated by mechanisms other than correction of anemia. Another example of the non-hematologic role of iron is provided by a recent study showing exercise intolerance in patients affected by mitochondrial myopathy caused by defective Fe-S cluster formation [25]. However, it is difficult to discriminate the relative importance of the effect of iron status on Hb synthesis vs. the consequences on non-hematologic variables. Generally, the functional outcome is believed to depend on the severity of iron deficiency: when iron stores fall below a specific threshold, Hb synthesis is impaired. Further body iron depletion leads to muscle tissue iron shortage and a decline in performance. Hence, the erythropoietic compartment appears to be a privileged iron consumer, as also suggested by our findings in subjects exposed to high-altitude hypoxia [39].

The heart may be an organ particularly sensitive to iron status. Iron deficiency is associated with mitochondrial dysfunction and cardiomyopathy (reviewed in [15, 40]), and thus under this condition, the heart may be less able to face the increased functional demands of exercise.

A recent study showed that iron deficiency and iron-deficient anemia are significant concerns for the health and performance of both males and females highly trained elite triathletes [41].

12.8 Etiology of Iron Deficiency in Athletes

Athlete's iron stores can be compromised by several exercise-related mechanisms, such as hemolysis, hematuria, sweating, etc. (see above), leading to absolute iron deficiency, but additional pathophysiological mechanisms may lead to iron-restricted erythropoiesis and anemia.

As reported above, a tight link exists between iron metabolism and erythropoiesis. Higher Hb synthesis is accompanied by ERFE-mediated inhibition of hepcidin expression, thereby increasing ferroportin activity and iron availability to the erythropoietic compartment [21–24]. Apart from rare mutations in genes coding for proteins of iron metabolism, iron-restricted erythropoiesis may develop from an imbalance between iron supply and erythropoietic needs, due to strong stimulation of erythropoiesis induced by either endogenous responses

to anemia or exogenous erythropoietin administration. However, iron sequestration is the most common mechanism leading to this condition in athletes.

In fact, a mechanism of sports-induced iron deficiency, based on the influence of a mild inflammatory status associated with physical activity on the postexercise hepcidin response, has been proposed. As reported above, inflammatory conditions upregulate hepcidin [21]; therefore, this response may represent the common mechanism behind iron deficiency, and possibly anemia, in athletes. Recent studies found elevated serum hepcidin levels after different exercise regimens, thus providing evidence of the effects of exercise on hepcidin-dependent control of iron status [42, 43]. Since a few reports did not find increased hepcidin levels, one could speculate that hepcidin is not induced by low-intensity exercises that are not sufficient to trigger inflammation [44]. However, triathlon is probably an exercise strenuous enough to expose triathletes to the risk of developing this type of iron deficiency and ensuing anemia (for detailed reviews, see [17, 45–47]).

It should be also reminded that competition for iron between different body compartments, such as the erythroid compartment and the skeletal muscle, may occur and be particularly relevant in athletes. The high heme production associated with erythropoiesis requires substantial entry of iron into erythroid precursor cells, but skeletal muscle contains a considerable amount of body iron located mainly in myoglobin. Therefore, enough iron should be available for skeletal muscle to allow for iron incorporation in the heme moiety of myoglobin, thereby preserving muscle O_2 homeostasis. However, we showed that increased iron demand due to accelerated erythropoiesis leads to muscle iron loss and decreased myoglobin in human subjects exposed to high altitude [39]. Several questions regarding the balance of iron distribution between the muscle and the erythropoietic bone marrow and its role in the capacity for physical exercise remain unanswered. For example, are skeletal muscle O_2 homeostasis and work capacity impaired more by low erythropoietic activity or by reduced myoglobin content? Moreover, based on several lines of evidence obtained both in experimental models and in humans, it is now well defined that iron is necessary for many functions beyond Hb and myoglobin synthesis that are important to exercise capacity. Many other iron-dependent enzymes might be affected, such as those involved in energy production and redox control, cell growth, etc., which are relevant for muscle function and work capacity [16, 35].

Hb concentration and serum ferritin, the secreted form of the iron storage protein ferritin which is closely related to body iron deposits, are habitually used to identify iron-deficient anemia, but the interpretation of these indicators may be more challenging in athletes, due to the fluctuations in PV cited above and the stimulatory effect of inflammation on serum ferritin. Low serum ferritin can help distinguish absolute iron deficiency from inflammatory iron sequestration, but threshold levels have not been clearly defined. Hepcidin levels could have a good diagnostic potential in this context, but the detection of this protein has not entered laboratory routine, yet. A good characterization of iron status is important for the management of the anemic triathlete and will be useful to adopt interventions avoiding iron deficiency or overload. For example, iron therapy will be of little help in case inflammation is the underlying cause of anemia, as elevated hepcidin levels would impair iron

absorption and iron release from reticuloendothelial macrophages. In case nutritional strategies to ameliorate iron intake, such as increasing the consumption of heme iron (e.g., meat) or enhancing iron absorption by means of ascorbate, or stop iron loss are not effective, iron supplementation is required [48]. In patients who do not tolerate oral iron supplementation, in severe cases, or when anemia should be quickly corrected before major competitions, intravenous iron administration may be appropriate, though the benefits of this intervention may be limited in subjects with anemia of inflammation [49].

While iron may be beneficial, it should be kept in mind that too much iron can be harmful, and therefore it is always necessary to maintain an appropriate iron balance. Indeed, it has been shown that the use of exogenous iron resulted not only in toxic iron overload in the liver and spleen [50] but also in the skeletal muscle, thereby impairing exercise performance [51]. Therefore, when not needed, iron supplementation should be avoided not only because possibly unsafe but also useless. In fact, supplemental iron did not improve performance in distance runners who were not iron deficient [52].

12.9 Conclusions

This chapter addressed the connection of anemia and physical exercise, in particular in endurance athletes like triathletes who are at increased risk for anemia for a variety of reasons. Since efficient O_2 transport is of paramount importance for performance, the increased knowledge and the more detailed understanding of the molecular mechanisms underlying anemia may provide information that ultimately allows athletes, coaches, and sport dietitians to adopt strategies for focused intervention, including prevention and treatment.

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The Youth Triathlete

13

Michael F. Bergeron

13.1 Introduction

The positive impacts of youth sports participation on health, fitness, psychosocial, and character development and numerous other traits contributing to academic and life success are widely recognized and increasingly well supported [1–7]. However, there are also many challenges that are too often related to overly intense specialization in a single sport that places unsustainable physiological, psychological, and social demands on the youth athlete [8–12]. Accordingly, various prevalent unintended consequences are frequently demonstrated by a cascade of preventable injuries, burnout, and eventual dropout from sport.

Triathlon training and participation during youth may indeed offer an attractive diverse solution, so long as the benefits of inherent variable physical activity exposure are not offset by an excessive emphasis on one or more components of the sport—that is, excessively overloading separately or concomitantly with swimming, cycling, or running. In contrast, balancing an appropriate level of alternating sport-specific training can reduce the physical and psychological burden of constant repetitive overload. However, this chapter does not focus on a specific training model or schedule for developing the youth (includes both youth and junior) triathlete. Moreover, there is not a detailed, in-depth explanation of topics (e.g., physiological demands, acute and overuse injuries, environmental-related injuries and illnesses, or various other clinical considerations) already thoroughly covered in other chapters, as these presented aspects in many ways are similarly applicable to youth, though there is an emphasis here on selected priorities and considerations specific to the developing young athlete that are integral to a positive triathlete experience for youth. The adolescent period is furthermore underscored, highlighting selected inherent changes and challenges that,

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depending on how these are managed, can reinforce or interfere with a sustainable and enjoyable sport career at all levels of triathlon participation and success.

The underlying paradigm for the successful youth triathlete is underpinned by healthy and sustainable participation with sensible progressive development that is *youth centered*. That is, the enduring benchmarks are focused on enjoyment and fun, self-motivation, interdependent sport and character development, building an athletic foundation that encourages physical and psychological resilience, and a schedule of training and competition that is balanced and complementary to healthy physical, psychological, and sociological development and other life priorities. The model must also be individualized and promptly responsive to a young athlete's changing needs, goals, and early stages of evolving problems. And, lastly, it must be appropriately implemented by knowledgeable adults (including coaches and administrators) who embrace individual assets and a wide definition of individual athletic and sport success in youth. These tenets have been well described by the International Olympic Committee (IOC) in a recent definitive consensus on youth athletic development [13]. Done right, the lasting positive effects can extend throughout the youth triathlete experience to enjoying and possibly excelling as an adult in the sport, while maintaining rewarding engagement in other physical activities that encourage a viable lifestyle of fitness and health [14].

13.2 Challenges for the Youth Triathlete Across Adolescence

Defining and implementing a developmentally appropriate and optimal individualized strategy for training, performance, and injury risk mitigation is recognizably challenging at any level. This is notably even more difficult for a youth athlete, owing to a constantly and uniquely changing base that is highly affected by normal physical growth, biological maturation, behavioral development, and their interactions. And accurately assessing the needs and progress of a youth triathlete across the multiple domains and variable stages of athletic development and sport progression and advancement further adds considerably to the practical challenges [15, 16]. Unfortunately, the competitive careers of youth athletes are too often temporarily halted or permanently derailed by overuse injuries resulting from disproportionate training and repetitive homogenous loads, further hastened and exacerbated by insufficient rest and recovery [17, 18].

The long-term, moderate-intensity, continuous physical activity characteristics of triathlon training and competition suit children well. This is attributed, in part, to their lower ratio of glycolytic (anaerobic) to oxidative enzyme activity and higher relative rates of lipid oxidation and glycogen sparing during exercise compared to adults [13, 19, 20]. As boys and girls mature through puberty, however, percent of peak VO_2 at the lactate threshold decreases. That is, across adolescence, anaerobic capacity and reliance on anaerobic energy provision during strenuous exercise progressively increase in youth triathletes. In parallel, resistance to fatigue and rate of physiological and metabolic recovery from strenuous exercise, especially with repeated intermittent bouts of high-intensity overloading during training, also

progressively lessen as an adolescent matures toward adulthood [19, 21, 22]. Yet, a frequent training error with older adolescents is when they are repeatedly pushed inappropriately harder beyond their ability to tolerate the higher workload. Moreover, the inherent risks of premature fatigue and related injury from excessive rapid overload are readily exacerbated by not allowing adequate recovery time to accommodate an increasingly lower resistance to fatigue and rate of recovery. Too often, the older athletically gifted and motivated athlete is the one expected to tolerate an undue training load and, sadly, ends up paying the costly price of a halted or ruined sport career [17].

Training and competing in the heat also present recognized challenges for boys and girls, and the risk for incurring heat-related problems is generally progressively greater as a young triathlete advances through the teen years and higher divisions and begins to participate in longer racing distances. While youth athletes do not appear to have any inherent biological maturation-related disadvantage or greater difficulty tolerating the heat [23], environmental heat stress—especially when the humidity is high—can indeed be a measurable threat to athletic performance and safety [24, 25]. Thus, it is imperative that youth athletes and those overseeing them recognize this and suitably accommodate in advance the changing heat-related challenges and threats as maturation and athletic and sport development evolve. As a youth triathlete physically and physiologically develops and matures, a greater muscle mass produces more heat during vigorous physical activity, and the more mature sweat glands respond with increased sweat production [26]. A resulting greater thermal load and total body water deficit collectively impact cardiovascular function and the ability to cope with the heat [27], as well as increase the risk for hyperthermic fatigue and exhaustion [28, 29]. Notably, substantial total body water and exchangeable sodium deficits from extensive sweating are increasingly more characteristic in mid- to late-teen athletes compared to even just a year or two earlier [30, 31]. But while adequate hydration is recognizably integral to heat safety for youth triathletes during training and competition, excessive thermal strain and exertional heat illness risk should not be overlooked simply because fluids are provided. Moreover, longer and more physically demanding workouts and competitive racing events are also increasingly expected and prevalent in those older adolescent triathletes who are more physically developed, fit, and skilled. Without sufficient recovery time, resulting greater levels of muscle damage and various other physiological carryover effects could increase thermal strain and other clinical risks during the next training session or event [32]. Adequate recovery time (especially in the heat) between training sessions and races should always be a priority. However, as young triathletes get older and train and compete more often, longer, and at a higher level, greater accommodation for recovery should be anticipated, deliberately implemented, and strictly adhered to in the short- and long-term schedules. Nonetheless, recognizing the value of sensibly adjusting for heat-related challenges and threats, with adequate preparation (including progressive heat acclimatization and ample hydration) and appropriately applying other offsetting measures, most healthy children and adolescents can safely participate in a wide range of outdoor training and competition in the heat [24, 30, 33].

The positive effects on bone health via high-impact loading in sport (e.g., gymnastics, volleyball) or odd (variable)-impact loading (e.g., soccer, basketball, tennis) are well recognized. These effects are realized through higher bone mineral composition, mineral density, and enhanced geometric properties in a sport-specific loading pattern [6], and the benefits are augmented when youth athletes regularly achieve the recommended dietary calcium and vitamin D intake [34]. While the osteogenic effect is not as effectively bolstered by non-loading activities such as cycling and swimming, youth triathletes will achieve beneficial bone health loads and stress from running and certain complementary fitness activities [35–37]. However, coaches and parents should also appreciate that sport-related overuse injuries are unduly prevalent during puberty and the adolescent growth spurt [9, 38]. During the period of peak linear growth, stress fracture risk is notably increased by the coincident dissociation between bone expansion and bone mineralization [39]. Unfortunately, young "select" athletes at this stage of physical maturation and athletic development are too often urgently encouraged by their parents and coaches to amplify intensive training, practice, and competition loads. Certain aspects of linear growth rate and maturation may also predispose some youth athletes to specific injuries involving the immature spine (e.g., spondylolysis, spondylolisthesis), joint surfaces (osteochondritis dissecans), and traction apophysitis (e.g., Osgood-Schlatter disease, Sever disease) [9, 40–43]. Therefore, because of the concomitant greater potential for overuse-related injury, it is arguably strategically advantageous to reduce (or at least maintain) training and competition intensity, frequency, and/or volume. A more pragmatic focus on foundational and holistic fitness and sound biomechanical technique would assist in mitigating overuse-related injury risk during this particularly vulnerable stage of biological development. Moreover, a timely emphasis on developing proper swim, cycling, and running biomechanics and racing tactics and skills competencies will help the young triathlete more optimally withstand the demands of training and competition as an older adolescent and young adult.

13.3 Managing and Monitoring Training and Performance Readiness

The challenges related to maturation-related biological changes across adolescence facing developing youth triathletes need to be recognized and properly accommodated. Even only periodically mismanaging training, practice, and competition load or scheduling and recovery strategies can readily lead to undue fatigue, poor performance, and a measurable increase in injury risk [17, 44]. Youth sports should not be unduly straining. Moreover, adequate rest and recovery between training, conditioning, and race events are vital in minimizing injury risk, and these elements are central to regenerative and positive adaptations to the training stimulus, especially when regularly reinforced with proper nutrition and sleep [45, 46]. This approach is also complementary and integral to achieving or maintaining optimal athletic readiness and performance. In contrast, when the young triathlete is chronically

subjected to demanding physical and psychological loading and stress without ample ongoing rest and recovery and periodic time off, the youth athlete breaks down [9, 13]. Accordingly, coaches and parents need to recognize and promptly respond to the early warning signs—specifically, complaints of pain or undue soreness, uncharacteristic fatigue, and poor performance. Disturbed or abnormal sleep patterns can also be useful indicators of underlying excessive training or sport-related psychological overload and competing demands and potentially consequent related anxiety [47–49]. Any one of these simple and typically obvious indicators could suggest evolving athletic overload, overuse, burnout, or an injury.

Rigorous and comprehensive year-round sport-specific physical training and high-frequency competition continues to be a recognized norm in youth sports. In part, this culture is encouraged by parents, coaches, and sport facilities offering a range of professional-like development programs purported to enhance ranking, recruitment/selection, national recognition, and championships opportunities. Widespread competitiveness is further fueled by the media and marketing directed to young athletes and parents. Whether a youth athlete is intensely engaged in a single-sport focus or participating in multiple sports and related activities, insufficient allocation of time for rest, regular recovery, and other non-sport activities important to developing youth comes with a price. Not surprisingly, the cost continues to be illustrated by the disturbing prevalence of preventable sport-related injuries and health problems, including overuse injury, chronic fatigue, overtraining, and burnout [9, 10, 50, 51].

For all boys and girls in sports, training and scheduling errors are arguably the greatest contributing factors to injury risk versus any specific competitive event or distance per se. Throughout the progressive development of the youth triathlete, any increase in workload (training or racing distance, volume, intensity, and/or frequency) proportionately escalates the risk of injury. However, excessive weekly training distance, a sudden change in racing distance and/or intensity (often due to a misguided sense of urgency to "age up" or rapidly improve fitness and/or competition performance), and insufficient daily rest/nutrition and weekly recovery days are the notable *red flags* to watch for. An impulsive and appreciable training and conditioning upsurge in preparation for an upcoming "important" race is also a common error. Poor biomechanics and strength and/or musculoskeletal control asymmetry and deficits will further exacerbate injury risk with increases in training distance, volume, and frequency. And, unless an ample level of proper training and conditioning is maintained over the summer months and a viable and appropriate re-entry to school strategy is planned, onset of the scholastic schedule and coincident new competing demands can also be expected to challenge and negatively impact the youth triathlete. Lastly, premature return to activity following an injury often results in one or more new or recurring physical setbacks that indeed are sometimes longer lasting than the initial problem.

Many of the traditional assumptions and "proven" practices regarding generalized training and conditioning, practice, and competition across age-related timeframes should also be carefully considered and challenged. Athlete responses and adaptation trajectories during these predefined periods are highly variable and

individual specific. That is, individual athletes respond uniquely to training and conditioning, as well as competition formats, loads, and schedules. Accordingly, a structured manipulation of program variables (e.g., training mode, intensity, frequency, and volume) cannot predictably advance athlete and sport development similarly across individuals and youth populations [52]. While this simplified traditional approach to athletic and sport periodization is convenient, widely promoted, and commonly used, the efficacy is generally not scientifically supported or empirically validated [53, 54].

The key is to strive for appropriate variation in sport development activities (training and conditioning, practice, and competition) with a balanced emphasis on other priorities that are fundamental to youth (e.g., family and school, life skills, and social development). This will reduce injury risk, help to maintain athlete focus and engagement, and more positively contribute to athletic progress and sport performance [9, 13]. For the young triathlete, the inherent variable nature of the sport—integrating swimming, cycling, and running—contributes favorably to this end. But the complementary vital component to all youth athlete development strategies and programs is ongoing sensitive and early detection of advancement opportunities and emerging threats. Close observation of and carefully listening to *each* athlete should, as needed, be promptly followed with relevant, suitable, and specific individualized redirection [54, 55].

13.4 Key Points

Closely matching individual physical, athletic, and psychosocial/emotional development to the progressive demands and expectations of the sport is essential. And while a young athlete's interest and commitment may be strong, numerous challenges and problems can still arise. Many of these, however, can be averted or minimized in the youth triathlete by ensuring a state of fundamental athletic and sport readiness that goes far beyond merely passing a pre-participation medical exam or questionnaire. And the effective strategy progressively becomes more comprehensive, holistic, and individual specific throughout development into more advanced levels of participation. This is especially crucial *and* revealing in the competitive race environment, as this is where gaps in preparedness and readiness will readily surface. Accordingly, coaches, parents, and youth triathletes all have a responsible role in ensuring proper preparation, skill competencies, and physical and psychological/emotional readiness. Moreover, it is critical to recognize and appropriately accommodate to changing personal and sport stresses, injury risks associated with workload and rest/recovery cycles, and notably periods of vulnerability during puberty and the adolescent growth spurt. Otherwise, an unsustainable overload elicited by an imbalance of physical, physiological, psychological, academic, social, and sport demands and expectations can readily result in a variety of detrimental consequences ranging from injury to burnout and withdrawal from the sport.

The recent IOC consensus defined and explained the evidence-informed tenets of healthy and sustainable youth sports participation and athlete development [13].

While the entire array of recommendations featured in this definitive document is extensive and fundamental, several notable overarching principles underscore the primary concerns and considerations outlined in this chapter for the youth triathlete. These also underpin key best practices for setting and working toward realistic and achievable goals of healthy, sustainable, meaningful, and enjoyable sport participation:

- Sustainable athletic and sport development takes time. For the youth triathlete, from entry level to advanced achievement, the sport experience and success (however defined) will always evolve on an individual trajectory along an unpredictable continuum.
- Early success or struggle as a youth triathlete is not always predictive of continued engagement or future performance, achievement, or enjoyment in the sport.
- Fitness, athleticism, skill, and physical and psychological/emotional readiness and resilience must adequately support the expectations and demands across the developmental progression of triathlon training and competition.
- Appropriate variation and diversity in progressively introduced athletic loading, with adequate regular rest and recovery between training and conditioning sessions and cycles, as well proper preparation for and recovery from competitions, are key for reducing injury risk and optimizing performance.
- Coaches and parents need to recognize and promptly respond to the early warning signs (e.g., undue soreness, fatigue, or poor performance) potentially indicating evolving athletic overload, overuse, or apparent injury, and *no youth triathlete should train or compete hurt.*
- Above all, youth triathlete development programs and priorities must be *youth athlete centered*!

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The Master Triathlete

14

Romuald Lepers

14.1 Introduction

Master triathletes are generally defined as triathletes older than 40 years who systematically train and compete in triathlon events of various distances. These athletes can still achieve extremely high levels of performance. The Swiss woman Natascha Badmann won the South Africa Ironman triathlon in 2012 at 45 years old (becoming the oldest Ironman triathlon winner) and finished sixth at the Ironman World Championship the same year. In 2018, Hiromu Inada from Japan became the oldest man to ever finish the Ironman World Championship at 85 years old in a time of 16 h 53 min. These both examples of "young" or "old" master triathletes show how it is possible to push the limits of the interaction between aging and endurance performance. This chapter addresses the specific aspects of age-related declines in triathlon performance with a special focus on the performances of the best master triathletes because they represent a unique model for studying the effects of high levels of physical training in older individuals.

14.2 Increase in Participation of Master Triathlete

Over the last decades, there has been a continual increase in the number of master athletes for short- and long-distance triathlons. For both females and males, there was an important rise in relative participation among age groups >40 years. For example, at the Hawaii Ironman triathlon, master triathletes represented 31% of the total field for the males and 23% of the total field for females in 1986, while they represented 56% of the total field for males and 47% of the total field for females in

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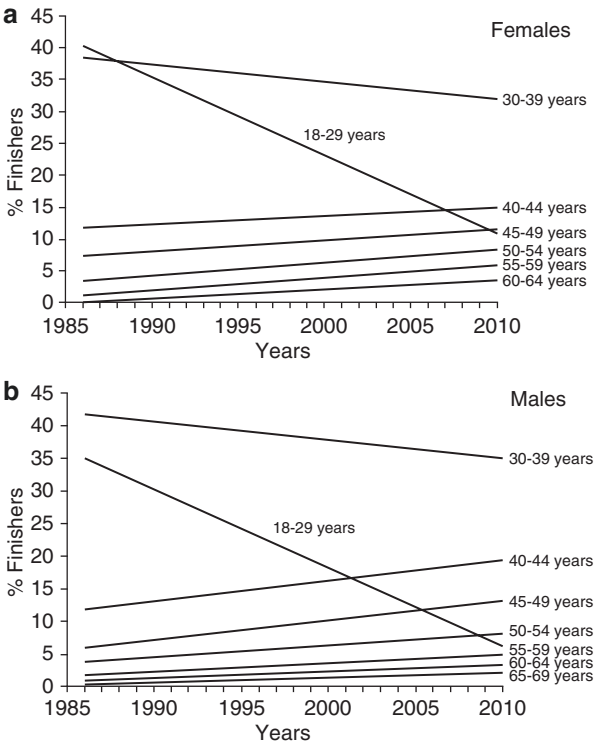

Fig. 14.1 Increase in participation of master triathletes at the Hawaii Ironman triathlon across the period of 1986 through 2010, for both females (**a**) and males (**b**). Reprinted with kind permission of the American Aging Association from Lepers et al. [1]

2010 [1] (see Fig. 14.1). In contrast, relative participation decreased among males and females <40 years, especially for the youngest age groups <30 years. It has been estimated that in all Ironman triathlons held worldwide in 2015 (≈11,000 female finishers and ≈47,000 male finishers), master triathletes represented 59% of the total field for the males and 54% of the total field for females [2]. Master triathletes are also well represented in short-distance triathlons. Sultana et al. [3] found that master athletes represented more than 50% of the total field for males and more than 40% of the total field for females at the 2006 and 2007 Olympic triathlon age groups world championships. The increase in participation of master athletes is not specific to triathlon. Indeed, increase in participation of both male and female athletes older than 40 years over the past decades has been also reported for marathon and ultra-marathon running [4–7].

Different factors could explain the increase in participation of master athletes in triathlon. The relative increase in participation of master triathletes in Ironman triathlon could result from a relative decrease in Ironman triathletes in the younger age groups (<40 years). Younger triathletes are maybe more attracted by shorter distances such as sprint or Olympic distances. Triathlon being an Olympic sport since 2000, it may have increased the popularity of the short distance especially in the young triathlete field [1]. With advancing age, triathletes naturally move towards longer distances where endurance and experience play a greater role in performance

than maximal aerobic capacity. Health, fitness benefits, enjoyment and social factors are the primary drivers sport involvement with advancing age [8]. In addition, with the increase in life expectancy and training facilities, the increased participation of master triathletes, especially for those older than 60 years old, may be a reflection that these athletes, who may in some cases be retired, have more available time and resources at their disposal to train and therefore to compete [9]. However, sporting background, social category and working profession of the master triathletes all remain unknown. Some may have the experience of many years of training and competition, while others only begin as they approach middle age and beyond.

14.3 Improvement of Master Triathletes' Performances

Age-related declines in triathlon performances have been well described in the literature for both short- and long-distance events. Triathlon performance appears to be maintained until 35–40 years of age, followed by modest decreases until 50 years of age and progressive decrease in performance thereafter. For example, the age-related declines in total Ironman triathlon performance with advancing age are about 12–13% per decade for males and 14–15% per decade for females [1]. The best male master triathletes tend to reduce the age-related decline to 10% per decade until 60 years of age (see Table 14.1).

The age of peak performance in Ironman triathlon is around 30–35 years for both females and males for elite triathletes, while the fastest race times are usually achieved between 25 and 45 years for non-elite triathletes [10, 11]. Interestingly, it has been reported that the age of the elite Ironman triathletes has increased over the past decades. For example, the age of the top ten finishers at the Hawaii Ironman triathlon increased over the last three decades from 27 to 34 years for the males and from 26 to 35 years for females [12]. It is now not uncommon for triathletes over 40 years to finish on the podium of an Ironman triathlon. For example, Cameron Brown won the 2016 New Zealand Ironman when he was 43 years old and still finished third 2 years later. Similarly, the Belgian Marino Vanhoenacker won the 2016 Austria Ironman when he was 40 years old and won the 2018 Australia Ironman when he was 42 years old. These two elite triathletes have had very long sporting careers and have remained at the highest level for about 15 years. Cameron Brown, who started triathlon when he was 20 years old, won his first Ironman when he was 28 years old, and Marino Vanhoenacker won his first Ironman when he was 27 years old.

The increase in participation of master triathletes in triathlon events over the past decades has been accompanied by an improvement in their performance at a faster rate than young triathletes, especially for the oldest categories [1, 13, 14]. For example, at the Hawaii Ironman triathlon, the best male finishers in the age group 60–64 years improved their total performance by 20% during the 1985–2010 period ([1], see Fig. 14.2).

Several reasons may explain the improved performances of master triathletes. Firstly, the increase in participation of the master triathletes increased the

Table 14.1 Total time records and corresponding split times for male and female age groups at the Hawaii Ironman triathlon

	Age groups (years)										
	PRO	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80–84	85–89
Male—time (h:min:s)											
3.8 km swim	50:37	1:01:53	1:03:18	56:07	1:01:09	58:10	1:04:42	1:47:46	1:31:39	1:49:34	1:51:26
180 km cycle	4:16:05	4:34:39	4:36:55	4:42:03	4:45:31	5:05:23	5:28:59	5:47:33	6:22:23	7:42:08	8:02:40
42 km run	2:41:32	2:59:18	3:09:02	3:21:19	3:19:35	3:36:26	4:01:18	3:52:47	5:02:30	5:41:52	6:28:18
Total	7:52:39	8:41:22	8:55:33	9:05:38	9:14:24	9:46:54	10:44:31	11:45:05	13:06:04	15:38:25	16:53:50
(Year)	(2018)[a]	(2013)[b]	(2018)[c]	(2018)	(2018)	(2017)[d]	(2013)	(2011)	(2018)	(2012)	(2018)
Female—time (h:min:s)											
3.8 km swim	57:26	1:09:26	1:03:29	1:06:50	1:06:12	1:21:11	1:11:55	1:15:22	1:45:05	–	–
180 km cycle	4:26:07	4:58:41	4:58:36	5:16:28	5:19:27	5:52:33	6:02:33	6:49:58	7:25:17	–	–
42 km run	2:57:05	3:18:45	3:15:22	3:41:36	3:57:57	4:19:50	5:03:27	5:28:42	6:19:43	–	–
Total	8:26:16	9:32:05	9:23:26	10:12:03	10:33:10	11:41:45	12:28:44	13:42:50	15:54:16	–	–
(Year)	(2018)[e]	(2011)[f]	(2018)	(2018)	(2013)	(2018)	(2018)	(2017)	(2005)	–	–

PRO professional category
[a]Patrick Lange (32 years old)
[b]Dave Scott (40 years old) did 8:24:32 (1994) in PRO category
[c]Cameron Brown (46 years old) did 8:25:30 (2018) in PRO category
[d]Rob Barel (was born in December so he was 59 years the race day)
[e]Daniela Ryf (31 years old)
[f]Mareen Hufe (40 years old) did 8:25:30 (2018) in PRO category

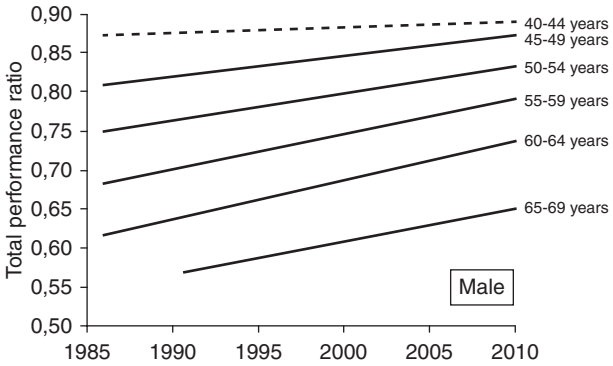

Fig. 14.2 Changes in total time performance at the Hawaii Ironman triathlon across the years for best male master triathletes. A performance ratio equal to 1 corresponds to the performance of the top ten elite triathletes (<40 years old). Solid lines represent conditions where the slopes of the linear regressions were significantly different ($P < 0.01$) from zero. Dashed lines indicate the slopes of the linear regressions were not significantly different from zero. Reprinted with kind permission of the American Aging Association from Lepers et al. [1]

probability of finding better triathletes in the older age groups. In addition, the increased facilities for older athletes, the improvement of master athletes coaching, training techniques, nutritional strategies or equipment may also explain the improved performance of master triathletes [9, 15–18]. Master triathletes that have attained good results and positive outcome have increased levels of motivation to train and compete in triathlon events and their competitive spirit [19–21].

14.4 Effects of Event Duration and Type of Triathlon

The age-related declines in triathlon performance seem to depend on event duration. It has been shown that the magnitude of the decline in cycling and running performances with advancing age was less pronounced for Olympic distance triathlon compared to Ironman distance triathlon [22]. In contrast, the age-related decline in swimming performance was not influenced by triathlon duration. For the best master triathletes of 70–74 years, total finishing time is approximately 3 h for an Olympic distance triathlon, whereas it is around 12 h for an Ironman. Factors such as greater muscle fatigue and greater sensibility to muscle damage of older triathletes could explain the greater declines in cycling and running performance when exercise duration increases.

It has also been shown that the type of discipline (road bike versus mountain bike cycling and road running versus trail running) could influence the magnitude of age-related changes in triathlon performance [23]. Indeed, the rate of decline in performance with advancing age is greater for off-road triathlon (e.g. Xterra®) than for road-based triathlon. Reduction in power to weight ratio with advancing age, lower technical bike-handling skills and reduced ability to

modify the biomechanical components of running on trails could explain the greater decline for off-road triathlon of master triathletes [23].

14.5 Effect of Discipline: The Specificity of Cycling

Triathlon involving three disciplines (swimming, cycling and running) offers the possibility of comparing age-related declines in the three locomotion modes for the same athlete. Triathlon performance decreases in a curvilinear manner with advancing age, but it has been observed a smaller age-related decline in cycling performance than in running and swimming performances for both long- and short-distance events [22–25] (see Fig. 14.3). These findings suggest that the age-related declines in endurance performance are dependent to the mode of locomotion, although the cause for such discipline specificity remains not really clear. Several hypotheses have been proposed to explain the smaller decline in cycling performance with advanced age. Lepers et al. [22] proposed that mechanical power could explain these differences. Indeed, mechanical power output (P) is dependent upon the velocity (V) for running ($P = k.V$, k: constant), whereas it is dependent upon the third power of velocity for cycling ($P = k.V^3$). As changes in aerobic capacity with age are tied to reductions in P, these reductions with age would impact cycling velocity to a lower extent than running velocity. Other mechanisms can be proposed to explain the smaller decline in cycling performance compared with the decline in the two other disciplines: a lesser reduction in lactate threshold or economy during cycling relating to a greater training volume in cycling compared to running to limit the traumatic injuries, a greater muscle fatigue during running with age and a decrease in upper body flexibility with age during swimming. Although these assumptions remain speculative and require further investigations, these data suggest that cycling is the discipline for which it is easier to maintain the highest level of performance in triathlon with advancing age.

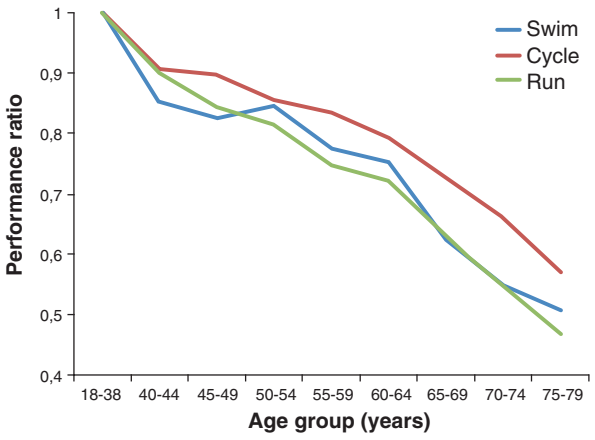

Fig. 14.3 Age-related decline in swimming, cycling and running performances at the 2017 Hawaii Ironman triathlon for the top five finishers of each age group. There was a lesser age-related decline in cycling performance compared with running and swimming after 40 years old

14.6 Sex Difference in Performance with Advancing Age

Sex difference in triathlon performance varied from 12% to 18% depending on the level of triathletes (elite versus age group) or the distance of the race [13]. Generally, time differences between sexes in swimming have been shown to be smaller than in cycling and running and could be explained in part by the biological difference in relative body fat (7–9% higher in females). Sex differences in performance are of biological origin, and the gap between elite males and females is unlikely to narrow naturally. However, several studies showed that the sex difference in triathlon performance increases with advanced age [13, 26]. For example, the sex difference in total event performance time at the Hawaii Ironman triathlon increased with advancing age from 55 years during the 2006–2008 period [26]. Data from Table 14.1 also show that sex difference in total performance at the Hawaii Ironman triathlon equals to 14% for age group 55–59 years, 16% for age group 65–69 years and 21% for age group 75–79 years. Similar findings have been observed for Olympic distance triathlon [27]. The reasons for the increased sex difference with age remain unclear and may result from physiological, sociological and psychological changes [28, 29]. The lower participation rate of female master triathletes, especially in the older categories, is likely to amplify the sex difference in performance above that due to physiological differences alone [30]. By comparison, since 2000 the sex difference in running performance at the New York marathon did not differ anymore across the age [6]. This relative stability of sex difference across the ages for running suggests that the age-related declines in physiological functions did not differ between females and males. These findings contrast with previous studies showing a greater decline in physiological determinants of endurance performance for females compared to males [29]. The increase in participation of female master triathletes associated with the appearance of well-trained females to the older age groups will inevitably reduce the gap between male and female master triathletes. In the near future, sex difference in triathlon performance should be the same for all age group categories, at least the best older triathletes.

14.7 Age-Related Changes in Physiology of the Master Triathlete

Both central (decreases in maximal heart rate, maximal stroke volume and maximal cardiac output) and peripheral (decreases in muscle mass and in maximal arteriovenous oxygen difference) factors contribute to age-related declines in triathlon performances. However, these physiological factors can be modulated by changing the volume and the intensity of master triathlete' training [7, 9, 31]. Among the three determinant factors of endurance exercise performance (i.e. maximal oxygen consumption, lactate threshold and exercise economy), the decrease in maximal oxygen consumption (i.e. VO_{2max}) seems to be the predominant contributor to the decline in endurance performance with advancing age [31]. It is generally admitted that VO_{2max} declines by ≈10% per decade after 40 years in healthy sedentary people. It is not

clear in the literature if the rate of decline is smaller or greater in master endurance athletes [32–34]. The greater rate of decline in VO_{2max} in master athletes compared to healthy sedentary adults could result from that of a greater baseline VO_{2max} with age as young adults and greater reductions in training with advancing age compared to non-trained adults. Some trained master athletes can still achieve high level of aerobic capacity even at a very advanced age. For example, it has been estimated that the VO_{2max} of the Canadian male runner (Ed Whitlock) who ran the marathon in 3 h 15 min at the age of 80 years was close to 50 ml/kg/min [35].

Reductions in the capacity to sustain a high fraction of VO_{2max}, evaluated by blood lactate threshold, and submaximal exercise economy may also contribute to the decrease in triathlon performance with advancing age. However, the decrease in running and cycling efficiency in master triathletes remains poorly understood. A higher energy cost of running in master triathletes (>40 years), compared to their young counterparts, has been observed by Sultana et al. [36]. These authors suggested that the higher energy cost of running in master triathletes could be due in part to a lower muscle power. Peiffer et al. [37] found that cycling efficiency was 11% lower and energy cost of running was 11% greater in master triathletes (60 years) compared with young triathletes (28 years). The difference was even more pronounced for the run (+22%) when values was scaled to lean body mass. Even if physiological data of well-trained master triathletes are missing in the literature, present data suggest that aging can influence exercise economy for both the run and cycle discipline. Future studies will need to focus on physiological characteristics of master triathletes and verify if they differ from other endurance master athletes. The inevitable age-related changes in physiology of master triathletes remain closely related with reductions in exercise training intensity and volume. The psychological (e.g. motivation to train hard) and physical (e.g. prevalence of injuries, see [38]) aspects of training of the master triathletes should also be considered in the age-related changes in performances.

14.8 Recovery of Master Triathlete

Master triathletes frequently said that they need more time for recovering from a hard training session or a competition. Chris McCormack, Australian triathlete and two-time winner of the Ironman World Championship, wrote in his book, “When I was twenty, I could jump out of bed after a few hours of sleep, throw some junk down my throat and run, bike and swim a ten-hour training day. If I tried to do that today (at 38 years old), I wouldn’t last six hours. You have to plan for more rest when you’re older” [39]. Unfortunately, scientific evidences of lower recovery capacities in master triathletes compared to young counterparts are missing. However, some studies suggest that while master athletes may show delays in the acute recovery of a number physiological parameters, the influence of this delayed acute recovery on subsequent physical performance seems to be minimal [40, 41]. However, despite restoration of physical performance at rates similar to younger athletes, master athletes perceive to take longer to recovery from a bout of

high-intensity exercise. These results suggest that coaches and master triathletes may have to allow for greater recovery durations between sessions to allow for psychological recovery in master triathletes. These data warrant the use of perceptual measures when monitoring the training load of master triathletes as physiological measures alone may not be sensitive enough to quantify recovery [41]. In addition, greater care may be required when training incudes exercise-related muscle damage as previous studies have suggested that this may lead to a delayed physical recovery in master athletes [42, 43].

In master athletes, the effects of dietary supplementation on recovery remain contradictory [15]. However, the role of antioxidants and micronutrients (mainly vitamins) during aging appears essential especially in master triathletes who are doubly exposed to oxidative stress. Indeed, the repetition of intensive physical exercises damaging to muscles, combined with increased oxygen consumption, and aging are factors conducive to oxidative stress. Very few studies have tested the potential beneficial effects of vitamin supplementation in master athletes. Although the ingestion of vitamin C or E alone seems to have no effect on muscle recovery, the use of complexes of antioxidants including several vitamins and minerals could be beneficial for recovery, in particular when training involves eccentric exercises well known to induce muscle damage [42].

14.9 Conclusion

Despite the age-related changes in their physiology, master triathletes can still achieve high levels of performance in the three disciplines. Different factors such as the event duration, the type of triathlon and the locomotion mode should be considered in the age-related decline in triathlon performance. The best master triathletes have improved their performances over these last decades, and the question as to whether older triathletes have reached their limits in triathlon performance maybe raised. Additional information regarding their responses to different training regimes such as concurrent strength and aerobic training or high-intensity interval training, their recovery capacities, their specific nutritional requirements or their psychological profiles could help master triathletes to extend the limits of their endurance.

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The Female Triathlete

15

Margo Mountjoy, Alex Thomas, and Jasmine Levesque

15.1 Introduction

Female athlete participation in triathlon ranges from recreational to the elite level. The Olympic triathlon event consists of three components; a 1.5 km swim, 40 km cycle, and a 10 km run. Triathlon joined the Olympic program in 2000, with 48 female and 52 male participants. The most recent summer Olympic Games in Rio de Janeiro in 2016 had 55 female and 55 male entries from 41 nations. The International Federation for Triathlon, known as the International Triathlon Union (ITU), offers a World Triathlon Series throughout the remainder of the quadrennial with an annual Grand Finale where $100,000 USD is split evenly between the top male and female athlete as calculated by cumulative points throughout the year. Other events for female triathletes include the mixed relay and the World Paratriathlon Series. The long distance triathlon is known as the Ironman series which consists of a 3.8 km swim, 180 km cycle, and a 42.2 km run. In contrast, the sprint distance triathlon has a 750 m swim, 20 km cycle and 5 km run.

Female athlete participation in triathlon is gradually increasing. Evaluation of 1,666 female athletes who participated in the Olympic distance triathlon event held in Zurich, Switzerland revealed that participation in the age range between 40 and 54 years increased significantly over the study period of 2000–2010 and that female triathletes in the 45–49 years of age category improved their total race time

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[1]. Likewise, a study of female triathletes in the long distance 'Isklar Norseman Xtreme Triathlon' between 2003 and 2015 showed that female athlete participation increased significantly over the study period, and that their performance also improved across the years in swimming, cycling and overall race time; however, not for running time in either the male or female participants. These studies demonstrate that female athletes of all ages are active in triathlon competitions of varying distances and improving their performance.

The ITU places an emphasis on gender equity in its sport. It is one of the few International Federations with a female President. It has a women's committee that showcases a series of articles to promote stories of women in triathlon titled #WomeninTri Case Studies [2]. This committee also has developed an annual Award of Excellence that "honors individuals with drive, leadership, creativity and commitment to develop, encourage and strengthen the participation of women in triathlon." The award is given in recognition of an outstanding contribution to:

- The participation of women and girls in Triathlon.
- Educating or raising awareness on gender issues in Triathlon.
- The coaching of women and girls in Triathlon.
- Supporting and coaching of women in Triathlon administrative and leadership roles.
- The promotion of women in Triathlon in the media.
- Acting as a role model for women and girls [3].

In the spirit of the ITU, this chapter serves to promote the health participation of women and girls in triathlon by reviewing the physical and mental health concerns unique to the female triathlete with the objectives of (1) increasing knowledge of relevant topics related to healthy female athlete participation in triathlon, (2) encouraging the adoption of healthy female triathlete sport practices by athletes, coaches and other members of the athlete entourage, and (3) promoting safe participation for female athletes in triathlon.

15.2 Training

There is limited published research on whether triathlon training programs should differ based on gender. The existing literature rarely controls for distance, non-elite versus elite, or male versus female. Training programs are often strategically personalized. Triathlon training typically consists of a combination of running, cycling, swimming, transitioning, and resistance training. A study of nine elite-level Olympic distance triathletes found that, on average, female triathletes swam 4.22 (±1.50) h per week, cycled 7.03 (±2.37) h per week, and ran 3.52 (±1.72) h per week [4] compared to male triathletes who swam for 5.4 h, cycled for 8.0 h, and ran for 3.13 h [5]. Male and female triathletes have a similar breakdown regarding training habits. However, one key difference is that female triathletes are more likely to train with friends or training partners whereas male triathletes are more likely to train by

themselves [6]. Compared to Ironman distance triathletes, female Olympic distance triathletes were also found to spend less time per week in low intensity long runs and long bike sessions [7]. The training variables that are most predictive of race success are experience, race strategy and weekly mileage [8].

There are several anatomic and physiologic differences between male and female triathletes that must be considered when developing training plans and strategies for the female triathlete. Anatomically, female triathletes are typically shorter, have a higher body fat percentage, and have less lean muscle mass than male triathletes. In terms of physiological differences, the most relevant difference is that VO_{2max} is typically 10% lower in female athletes than in male athletes across all sports [9]. This is due to several factors but is primarily explained by differences in body composition and oxygen transport [10]. However, when comparing highly trained male and female athletes, the difference in cardiovascular endurance capacity, relative lower body strength, and muscle fiber type are all significantly reduced [11]. Given that training and competing in triathlon is primarily an aerobic activity, these similarities are especially important and advantageous for the female triathlete.

Although more research is required to fully differentiate male and female training strategies, the subtle anatomic, physiologic, and psychosocial differences must be considered when developing female specific training plans.

15.3 Injury Prevention

According to injury prevention theoretical models, the first step in injury prevention is understanding the prevalence and nature of injuries in the athlete cohort. At the elite level, there are few injury surveillance studies published on female triathletes. However, there are data from the 2008, 2012 and 2016 Olympic Games. Female triathletes at the 2016 Olympic Games held in Rio de Janeiro (n = 55) had an overall injury incidence of new onset, acute injuries of 5.6% in contrast to male triathletes at 16.4%. The overall injury incidence for the Rio 2016 Olympic Games for all athletes in all sport disciplines and genders (n = 11,289) was 9.8%. Severe injuries, defined as resulting in >7 days of time loss from sport participation, was 1.9% for female triathletes and 7.3% for male triathletes, demonstrating that female elite triathletes have a low incidence of acute new onset injuries during the Olympic Games [12]. There are no injury surveillance studies published by the ITU.

In contrast, a larger prospective cohort study of the 5-day World Championships held in London, England in 2013 found that the female participants had a higher injury incidence than male participants. Walker et al. reported observational data of injuries in 6,103 athletes of all levels of triathlon experience from 84 countries ranging from 16 to 80 years of age. Independent variables included gender, age, event standard, event distance and event type. Elite athletes were found to have a higher injury incidence than recreational athletes. For the Olympic distance event, injuries were highest for females, the oldest and youngest athletes. This study provides a broader view of female triathlete injuries in the non-elite population [13].

As triathlon is not a contact sport, the injury burden in this population is expected to have a low incidence of new onset acute injuries during a major event. Given the training loads of female triathletes, the injury pattern experienced by these athletes is one of chronic overuse injuries. A study by Collins et al., retrospectively surveyed 60 female triathletes at a triathlon event. The response rate for the survey was 45%. Over the preceding year, 49% of the female athletes reported having suffered a training-related injury serious enough to cause them to stop training for at least 1 day, seek medical advice, or take medicine. This number was consistent with the male pattern of injury prevalence of 48%. The most commonly reported injury sites were the knee, shoulder and lower leg. Nine athletes reported more than one injury. Not surprisingly, elite female triathletes had greater injury prevalence than recreational athletes at 60%, although this finding did not reach statistical significance. Athletes with higher mileage (swimming, cycling and running) were not at a great risk for injury, nor were age, sex, or body mass index [14].

Given the paucity of literature on injuries in female triathletes, clinicians should be aware of the top 5 injuries experienced by female athletes in sport. Female athletes are more prone than male athletes to, (1) stress fractures, (2) anterior cruciate ligament ruptures, (3) concussion, (4) patellar femoral syndrome, (5) ankle sprains. Because of the biomechanical demands of swimming, cycling and running, female triathletes are therefore most likely to suffer from stress fractures, patellar femoral syndrome and ankle sprains. Concussions are more common in contact sports, however, cycling accidents may result in a concussion. Anterior cruciate ligament sprains or ruptures are more common in multi-directional sports such as tennis, soccer (football) or rugby, where the knee is placed in a 'plant and twist' position.

As outlined in the injury prevention models, injury prevention interventions are implemented based on the injury patterns in the sport. Given the overuse injury pattern in triathlon, attention to appropriately timed training loads and stroke, cycling, running biomechanics is essential.

15.4 Relative Energy Deficiency in Sport (RED-S)

Relative Energy Deficiency in Sport (RED-S) is defined by the International Olympic Committee as:

> A syndrome referring to impaired physiological function including, but not limited to, metabolic rate, menstrual function, bone health, immunity, protein synthesis, cardiovascular health caused by relative energy deficiency. [15]

Energy availability is defined as energy intake minus energy expenditure during exercise adjusted for fat free mass. (EA (kcal/kg) = EI (kcal) − EEE (kcal)/FFM (kg)) In healthy adults, a value of 45 kcal/kg/FFM/day equals energy balance. Energy balance can be challenged by either restriction of oral intake of energy, as seen in eating disorders or disordered eating, or through excessive energy expenditures, whereby the energy output exceeds the energy intake. This phenomenon

occurs in the absence of body image distortion, an eating disorder or disordered eating. Thus, athletes in endurance sports, whose training regimens include high volumes of endurance exercise, are at particular risk of low energy availability. It is evident from published prevalence data that low energy availability results in multiple health (Fig. 15.1) and performance consequences (Fig. 15.2). Although RED-S may occur in male athletes, the body of literature is more robust for the female athlete given the historical focus on low energy availability in females.

There are a few studies specifically looking at RED-S parameters in triathlon. A cross-sectional study of 15 female triathletes from the United Sates identified that 60% were found to be in caloric deficit that was defined by the authors to

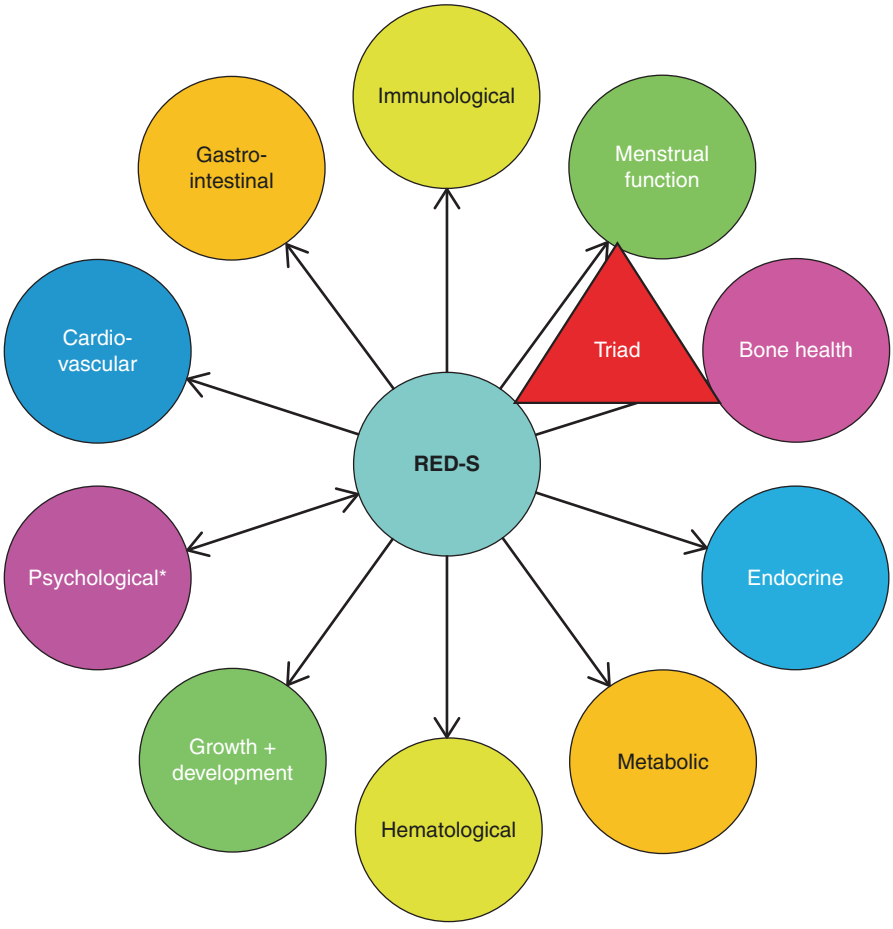

Fig. 15.1 Health consequences of Relative Energy Deficiency in Sport (RED-S) (*Psychological consequences can either precede RED-S or be the result of RED-S) [15]. (Adapted from Constantini [16])

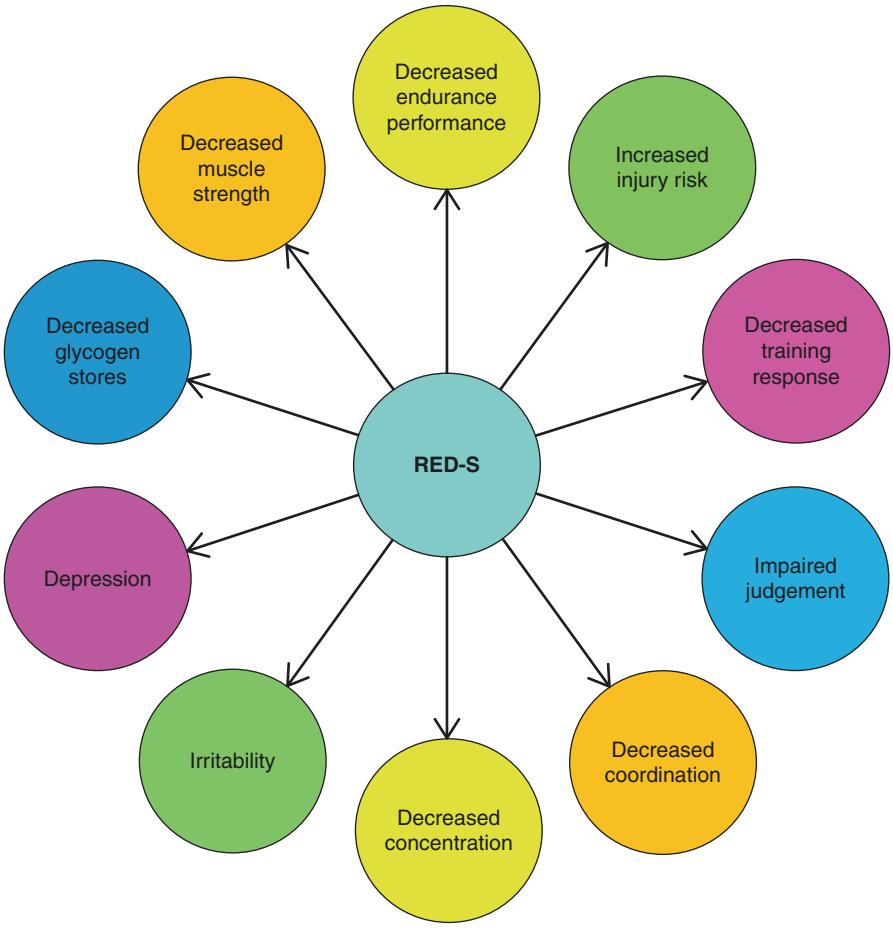

Fig. 15.2 Potential performance effects of Relative Energy Deficiency in Sport (**Aerobic and anaerobic performance) [15]. (Adapted from Constantini [16])

be consistent with a diagnosis of disordered eating. Forty percent had a history of amenorrhea. There was no evidence of osteopenia or osteoporosis. Over half of the participants were not aware that these findings were consistent with health or performance implications [17]. A study by Kimber et al. evaluated energy balance in 10 male and 8 female Ironman distance triathletes (3.8 km swim, 180 km cycle, 42.2 km run). Energy expenditures were significantly higher than total energy intakes for both male and female participants [18]. This study demonstrates the importance of proper energy fuelling during competition. Another study of Ironman triathletes by Knechtle et al. evaluated the factors associated with the outcome measure of race performance. In female triathletes, percent body fat was not associated with race time. However, average weekly training volume was. This finding is in direct contrast to the male triathlete whose total race time was correlated with

percent body fat. This study demonstrates the importance of healthy training and the differences between male and female athlete physiological function [19]. A final study by Leake and Carter assessed the body composition of 16 trained triathletes in comparison with Olympic runners and swimmers. This study also concluded that training parameters were more important than anthropometric measures in the prediction of sport performance. It was determined that female triathletes were most similar with respect to somatotype and body composition to swimmers rather than to runners [20].

Female triathletes should be screened for evidence of RED-S. The LEAF-Q (low energy availability in females questionnaire) is a validated tool to identify those female athletes at risk for RED-S [21]. The sport physician can also utilize the International Olympic Committee (IOC) RED-S Clinical Assessment Tool (RED-S CAT) to guide the diagnosis and return to play for female triathletes with RED-S [22]. Treatment should be individualised according to the specific presentation, and often in conjunction with a multi-disciplinary treatment team. The focus of treatment should be on the attainment of energy balance through proper nutritional counselling by a registered and experienced sport dietitian/nutritionist.

15.5 Nutrition

Nutritional requirements and recommendations are well studied for female athletes and for triathletes in general, although there is less research specifically addressing the nutritional requirements for female triathletes. The current guidelines and general principles for nutritional strategies among triathletes are applicable for both males and females. However, there are a few key differences that must be considered when fueling a female triathlete. On average, female triathletes typically have a smaller body size and less muscle mass in comparison with male triathletes. For this reason, it is important that nutritional requirements are scaled relative to body mass in addition to energy expenditure.

15.5.1 Energy and Fluids

As discussed in the previous section, female triathletes, like their male counterparts, are at an increased risk of low energy availability due to extensive physiological demands from long periods of endurance training, which is typical in triathlon. Intense endurance training can lead to multiple health and performance outcomes identified in the syndrome called Relative Energy Deficiency in Sport [23]. As for all endurance athletes, female triathletes should consume carbohydrates as a primary fuel source for their ability to provide adequate glycogen to muscles and the brain [24], and should include multiple ingestions of transportable carbohydrate during endurance training and racing [25]. The optimal level of carbohydrate intake should reflect daily training demands [26]. Fatigue among endurance athletes is often due to muscle glycogen depletion or dehydration due to fluid lost in sweat

[27]. Current guidelines recommend avoiding more than 2–3% of bodyweight lost in fluids [25], but to also be aware of the risk of hyponatremia secondary to excessive fluid replacement [28]. Female triathletes are at an increased risk of developing hyponatremia because of smaller body sizes and lower sweat rates [29].

15.5.2 Micronutrients

In terms of micronutrients, female triathletes are at an increased risk of being deficient in iron, calcium, and Vitamin D. Iron-deficiency anemia is more common in the female triathlete due to menstrual losses, which can lead to fatigue and consequently suboptimal performance [30]. Calcium and vitamin D deficiencies often occur in female triathletes, which can result in increased risk of bone stress injury, and over time, can lead to osteopenia/osteoporosis [31]. The current Canadian recommended dietary allowance for female adults is 8 mg of iron, 1000 mg of calcium, and 800 IU of Vitamin D [32]. However, given a high incidence of iron-deficiency anemia among female triathletes, Coates et al. recommend supplementing with 40–60 mg of iron every other day to achieve maximal fractional absorption [33].

15.5.3 Supplementation

Beyond supplemental vitamins to restore specific deficiencies, there are only a few supplements that have been shown to improve sport performance among female triathletes and these do not differ by gender. Caffeine and nitrate-rich beetroot juice are two commonly-used supplements that have been shown to have performance-enhancing effects among triathletes [24]. A different study by Potgieter et al. concludes that triathletes should ingest 6 mg of caffeine per kg of body weight 45–60 min before an Olympic distance triathlon to improve performance. However, the improved performance was only statistically significant for male triathletes [34]. Other nutritional supplements that improve sport performance and are not prohibited for athlete use by the World Anti-Doping Agency (WADA) include creatine, beta alanine and sodium bicarbonate. These have not been studied for efficacy in triathletes [35].

15.6 Bone Health

Several intrinsic and extrinsic factors have been found to impact bone health. Some intrinsic factors include genetics, sex, age, body size, race, ethnicity and family history. Extrinsic factors associated with bone health are body weight, Vitamin C, Vitamin D, exercise, sex hormones, medications, smoking and some diseases. The scientific literature with respect to athletes is mainly focused on the impact of exercise on bone health. There are various studies demonstrating that weight-bearing exercise is beneficial for long-term bone health [36]. Bone acquisition during

childhood and adolescence is a significant factor in peak bone mass and the eventual risk of osteoporosis and fragility fracture later in life. When determining the quality of bone, the most important measure is its strength, in which bone mineral density (BMD) is a large component.

The most common measurement of bone density is the dual X-ray absorptiometry (DXA). Osteoporosis is defined as a skeletal disorder where bone strength is compromised and predisposes an individual to an increased risk of fracture. In terms of diagnostic values, the World Health Organization has defined osteopenia as BMD between −1.0 and −2.5 standard deviations (SD) below normal and osteoporosis as below −2.5 SD, measured by DXA. However, for athletes with a history of nutritional deficiencies, stress fractures or hypoestrogenism, low BMD (or osteopenia) is defined by the American College of Sport Medicine as occurring when the *z*-score is between −1.0 and −2.0 SD. In the same clinical scenario, osteoporosis is a BMD (*z*-score) of less than −2.0 SD [37]. In these populations, a low BMD and secondary clinical risk factors of bone mineral loss should be present for a diagnosis of osteoporosis. Secondary clinical features include chronic malnutrition, eating disorders, hypogonadism, glucocorticoid exposure and previous fractures. Athletes in weight-bearing sports normally have higher BMD than sedentary individuals. Therefore, a BMD *z*-score below −1.0 warrants further investigation in athletes [38].

There is evidence that individuals who participate in endurance sports, such as running, and non-weight-bearing sports, such as biking and swimming, may have lower BMD than athletes participating in ball and power sports. There is also published evidence that endurance athletes can even have BMD lower than sedentary individuals [39]. Originally, the reason for these findings was postulated that endurance activities provide insufficient skeletal loading, therefore resulting in low bone mineral density [40]. More recently, with the advancement of our understanding of bone metabolism, it is clear that many endurance athletes have low energy availability, which has a direct negative influence on bone density, as well as an indirect negative effect on bone via hypoestrogenism. This scenario is referred to as Relative Energy Deficiency in Sport (RED-S) [15]. Low BMD is a concern for athletes as it increases the risk of developing a stress fracture that can have a significant impact on an athlete's performance and athletic career [39].

Triathletes are unique in that they engage in three sports that have different loading characteristics [40]. Despite the fact that triathletes spend a lot of time swimming and cycling, it is believed that the cross-training may be healthier for bone than participating in a single sport discipline [39]. A study investigating bone health in triathletes demonstrated that the BMD are often within normal range [17]. Duncan et al. also found that female adolescent triathletes, unlike swimmers and cyclists, did not have lower BMD in comparison with running athletes [41]. However, a study evaluating amenorrhoeic endurance athletes, the majority being triathletes, found that spine BMD was significantly reduced in amenorrhoeic athletes compared with both eumenorrhoeic athletes and age-matched control population, supporting the link between hypoestrogenism and low BMD as seen in RED-S [42]. A case study of an elite junior triathlete demonstrated osteopenia associated with low energy availability and menstrual dysfunction [43].

In summary, sport medicine clinicians need to maintain a high index of suspicion in female triathletes for poor bone health, especially in those with a history of amenorrhea or a history of bone stress injury. Adequate energy availability, calcium, vitamin D, and caloric intake, as well as regular high-impact cross-training, are recommended for all triathletes to maintain good bone health [39].

15.7 Menstrual Dysfunction

Menstrual dysfunction in athletes range from anovulatory cycles, luteal phase defects, oligomenorrhea and amenorrhea. Mensural dysfunction can have many detrimental effects on both health and sport performance. The prevalence of menstrual irregularities in female triathletes was reported to as high as 24% [44]. In another study consisting of 15 triathletes, 40% reported a history of primary or secondary amenorrhea of an average length of 14 months [17]. Some female athletes (between 20% and 42%) report a negative affect on their sport performance, particularly from heavy bleeding [45, 46]. In terms of health consequences of menstrual dysfunction, athletes with oligo- or amenorrhea were found to have increased risk of stress fractures compared with eumenorrhoeic individuals [47].

Many types of contraception are available for female athletes. Some of those contraceptives act through the administration of female sex hormones to alter the menstrual cycle to prevent pregnancy. Oral contraceptive pills (OCPs) deliver these hormones systemically while certain intrauterine devices (IUDs) locally deliver the hormones to the uterus. Copper IUDs prevent pregnancy through natural spermicidal action and impairment of implantation. Depo-medroxyprogesterone (DMPA) injections are a contraceptive method that require administration every 3 months and has been found to be associated with reduced bone mineral density. Thus, this method should not be first line consideration by female athletes [48]. Combined oral contraceptive pills are the most commonly prescribed form of contraception for women. OCPS can reduce premenstrual syndrome symptoms that may cause athlete discomfort during training and performance [49]. Muscular strength is unaffected by OCPs despite possible androgenic effects of progestins [49]. There is also no change in endurance performance when taking OCPs [50]. However, the use of OCPs may mask low energy availability in endurance athletes such as triathletes. A normal menstrual cycle is a sign that the athlete has adequate energy balance. The athlete with oligo- or amenorrhea is at risk for bone stress injury, and may be suffering from RED-S. Therefore, the Copper IUD is the preferred method of birth control for the endurance athletes as it does not interfere with the hypothalamic-pituitary-ovarian axis, which is interrupted in the athlete with low energy availability [15]. Although monthly withdrawal bleeds occur when taking OCPs, it is not equivalent to restoring natural cycles through improving energy availability [37]. All causes of menstrual dysfunction, including pregnancy, polycystic ovary syndrome, low energy availability, hormonal disorders should be considered in triathletes with menstrual irregularities. Treatment of menstrual dysfunction should be treated in consultation with an appropriate health professional.

15.8 Pelvic Floor Dysfunction

Pelvic floor muscles are located in the area surrounding the pelvic openings in women and provide support for the pelvic organs and prevent leakage of urine, flatus or stool. They consist of two major muscle groups: pelvic and urogenital diaphragm. Some women are not able to contract these muscles correctly, which can lead to urinary, bowel or gynecological symptoms. The most common pelvic floor disorders (PFDs) include pelvic organ prolapse, pelvic girdle pain, and urinary or anal incontinence. Urinary incontinence can be further divided into two types: stress urinary incontinence and urge urinary incontinence. Stress urinary incontinence is defined as involuntary loss of a small amount of urine as a result of strain that increases intra-abdominal pressure, such as coughing or lifting a heavy object. Urge incontinence involves larger amounts of urine leakage associated with a strong urge to void and inability to prevent voiding.

There are two opposing theories describing the effect of high endurance training in female athletes on pelvic floor muscle strength. It is hypothesised that strong pelvic floor muscles in female athletes may be due to physical activity leading to increased intra-abdominal pressure resulting in repeated contractions by these muscles. On the other hand, it is thought that female athletes may have weak pelvic floor muscles due to overload and stretching from physical activity. With such opposing theories, there is a clear need for further studies to delineate the influence of physical activity on the function of the pelvic floor muscles.

Yi et al. evaluated the prevalence of PFD in 311 female triathletes and found that 37.4% had stress urinary incontinence, 28% had anal incontinence symptoms, 16% had urge urinary incontinence, 5% had pelvic organ prolapse. Female triathletes who have had children were found to have higher rates of stress urinary incontinence and pelvic organ prolapse in comparison with nulliparous triathletes. With respect to athlete complaints of pelvic girdle pain, while 18% of female triathletes reported having such pain, it was not reported as being disabling or significant enough to interfere with training. The pelvic girdle pain was most commonly located between the sacroiliac joints or on a single sacroiliac joint and less commonly reported at the pubic symphysis. Higher levels of pain were reported in the cohort of female triathletes who also reported having a PFD. Individuals with pelvic girdle pain can have difficulty standing, walking, and sitting for longer periods [44].

As triathletes have repetitive impact of high-intensity on the pelvic floor while cycling, they are at higher risk of developing a PFD [44]. Negative effects from sitting on a bicycle saddle have been identified in female cyclists ranging from minor skin lesions to pain and peripheral nerve damage. Most of the symptoms are temporary and preventative measures such as correct adjustment of the bicycle seat can provide some relief. Other adjustments such as positioning of the bar at the level of, or higher than, the saddle, and using a short broader saddle instead of a cut-out saddle can also be helpful in decreasing symptoms [51]. An association between cycling and decreased genital sensation has also been identified in female cyclists. It is hypothesised that there may be an impact on the peripheral nervous system from

endurance sports, specifically in cycling as it involves prolonged pressure on the ischial tuberosities, pudendal nerve and its branches. Further studies investigating this relationship are needed [44].

In summary, the scientific literature indicates that female triathletes suffer from pelvic floor disorder and pelvic girdle pain, although the symptomatology usually does not interfere with training. However, the long-term impact of these pelvic issues on these athletes' performance, participation and quality of life is not well understood. For those triathletes with significant symptomatology, depending on the complexity of the athlete's presentation, a multidisciplinary approach should be considered including expertise from physical therapy, physiatry, sports medicine and if needed, a surgical specialist [52]. Functional training of the pelvic floor muscles is effective in decreasing symptoms and can be integrated into triathletes' exercise regimen [44].

15.9 Mental Health

Athlete mental health is an area of elite athlete health that has only recently begun to receive attention. According to Reardon and Factor, mental health issues can present in one of three mechanisms [53]:

1. *Athletes may obtain high levels of success in spite of coexisting primary psychiatric disorder.*
2. *Athletes may choose sport as a means of coping with mental health disorder.*
3. *Athletes may have psychiatric illness precipitated or worsened by sport participation.*

The common mental health presentations in athletes include the following diagnoses [53]:

- Mood disorders: depression and bipolar
- Anxiety disorders: generalised, obsessive-compulsive disorder, post-traumatic stress disorder
- Attention deficit disorder ± hyperactivity
- Eating disorders/disordered eating
- Addictions: alcohol, drugs, gambling
- Suicide

Female athletes are approximately two times more likely than male athletes to present with a mood disorder, anxiety disorder and eating disorders, and are less likely to present with attention deficit disorder. Athletes in individual events are more likely to present with a mental health disorder than athletes in a team sport. There are sport-specific risk periods for athlete depression including post Olympic Games, transition from sport to retirement, in conjunction with abuse or harassment and post injury. There is a direct link between concussion and depression.

There are few publications on the mental health of female triathletes. A study by Lopez-Fernandez et al. examined the relationship between sex and motivation in triathletes, and found that although there were low amotivation scores in both genders, female triathletes had a statistically significant lower score than male triathletes. This survey had a 40% response rate, and thus self-selection bias may confound these results [54].

Three studies have been published on eating disorders in triathletes. A survey of 583 triathletes from the USA by Debate et al. identified that 28% of the females and 11% of the males scored below the mid-point of the range for the Eating Attitude Test-26 (EAT-26) construct Preoccupation with Food and Weight, and respectively 39% (female) and 23% (male) scored below the mid-point of the range for the construct Calorie Control. Both male and female triathletes participated in attempts to reduce body weight by energy restriction, limitation of food groups and excessive exercise. Fifty-eight percent of female and 47% of the male triathletes demonstrated dissatisfaction with their body image, despite being at or slightly below normal weight, and exhibited an intense fear of gaining weight, becoming fat or feeling fat. Although the survey is methodologically limited by a 20% response rate, it appears that triathlon is a sport that has a risk of clinical and sub-clinical eating disorders [55]. A second study on the same cohort found that female triathletes perceived themselves to be smaller than their actual Body Mass Index, and demonstrated a desire to be smaller in size. Only 10% of female triathletes in the underweight range desired to be larger, and alarmingly, 13% desired to be even smaller. In the normal weight range, the majority of female triathletes desired to be smaller, in contrast to 19% of male triathletes [56]. A smaller study comparing 53 triathletes and 109 runners demonstrated that triathletes had a slightly healthier attitude towards eating than the running study group, and were motivated more often by the enhancement of self-esteem and fun, in contrast to the burning of calories reported most frequently by female runners [57].

In summary, it is important for the female triathlete entourage to pay attention to the prevention, screening and treatment of female triathlete mental health issues, with a particular emphasis on eating disorders/disordered eating, in addition to mood and anxiety disorders.

15.10 Non-accidental Violence

While the benefits of sport participation on athlete physical and mental health are clearly documented in the scientific literature, there are negative effects on athlete health and sport integrity from harassment and abuse experienced within sport, commonly referred to as ‘non-accidental violence’. All athletes have the right to enjoy ‘safe sport’, which is defined by the International Olympic Committee as an athletic environment that is respectful, equitable and free from all forms of non-accidental violence to athletes. Non-accidental violence can take the form of psychological abuse, physical abuse, neglect or sexual abuse. It is based in a culture of discriminations where athletes are rendered powerless. Sexual abuse in particular

is more likely to occur where there is high athlete vulnerability, high perpetrator motivation and low organizational protection through athlete safeguarding policies and procedures [58].

Prevalence data demonstrates that non-accidental violence occurs in all sports at all levels, with an increase in risk as the athlete becomes more elite. Female athletes are more often reported than male athletes to be victims of non-accidental violence, and historically male coaches were more commonly reported than female coaches to be perpetrators. Triathlete specific prevalence data is not available. Although male athletes may also be victims of non-accidental violence in sport, it is important for sport medicine and science members of the female triathlete entourage to have the clinical competence to prevent, recognize and manage allegations of non-accidental violence in sport [59].

The impacts for victims vary significantly depending on the type of abuse, the circumstances, duration and a combination of extrinsic and intrinsic factors. For many, the negative impacts can be long lasting and devastating. The impacts of non-accidental violence in sport can affect athlete physical, mental, cognitive, emotional, behavioural and relational health. These impacts may last well beyond the cessation of the abuse. Psychological harm is compounded by passive attitudes and/or non-intervention by those in positions of power in the sport; this is known as the 'bystander effect'. Sport organizations are also impacted by non-accidental violence through reputational damage, loss of players and fans, loss of sponsorship, and a reduction in public trust [58].

15.11 Anti-doping

In highly competitive sports at the elite level, athletes are prone to performance enhancement practices. While some performance enhancement substances are not prohibited (caffeine, creatine, beta alanine, nitrates, sodium bicarbonate) [35], there are many performance enhancement substances that are prohibited. The World Anti-Doping Agency (WADA) is responsible for annually determining the WADA List of Prohibited Substances and Methods [60]. The number of positive doping cases (anti-doping rule violations) reported by the ITU between 2015 and 2017 for male triathletes is seven in contrast to there being no reported anti-doping rule violations for female triathletes. This data does not include National Federation cases. Anti-doping rules and regulations are the same for male and female triathletes.

In a survey of 2,997 recreational triathletes, female triathletes were more likely than male athletes to use analgesics before a competition. The estimated prevalence of doping was significantly higher in those athletes who used analgesics (20.4%) than in those athletes who did not use analgesics (12.4%) [61]. Analgesic use in triathletes was found to be as high as 60% in the 3 months before a competition, with the most common reason being for pain control.

All practitioners in the female triathlete entourage should be cognizant of the WADA Prohibited List [60] and Therapeutic Use Exemption rules and regulations [62].

15.12 Conclusion

Triathlon is a sport with many health benefits for female athletes. While triathlon at the elite level has inherent health risks for all participants, there are some unique health concerns in particular for the female participant. This chapter has outlined the training recommendations, as well as the specific health risks for the female triathlete. Knowledge of the health risks of training and competing in elite triathlon for female athletes helps to inform and influence the development of targeted prevention strategies, surveillance programs and intervention practices. Knowledge translation of the body of science in the area of female triathlete health should target athletes, coaches and the athlete support entourage. Applying the principles outlined in this chapter will not only enhance female triathletes' health and sport performance during their elite sport career, but also improve health outcomes upon retireme from competitive sport.

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The Travelling Athlete

16

Nebojša Nikolić

16.1 Introduction

Triathlon today is one of the most challenging outdoor sports around. High-level triathlon athletes need physical abilities to fulfil demands of three different strenuous challenging disciplines, the ability to understand and solve complicated problems of multisport race tactics and different environments they are competing in, under the mental pressure to use them efficiently. One of the challenges in triathlon is in successfully manoeuvring the swim-to-cycle transition (T1) and cycle-to-run transition (T2). In such a complex sport with very narrow margin of advantage against competitors, using or alleviating different influences on the performance, including environmental ones, becomes a vital part of success.

There is no doubt that travel is an integral part of athletes' life. Triathlon season is organised as a national or international string of events that requires weekly or biweekly travel to matches. By the nature of their sport, the athletes' racing field environment can differ much from their home one and can have a decisive influence on the final success in the race. One day a racing field can be tropical one and the next week it can be in a very cold climate. Even in the same climate zone and similar outdoor temperatures, water temperature can vary significantly from one field of play to another. Different modes or transport can also influence the physical and psychological well-being of the athlete. At the new destination, they are also exposed to the new biological environment with different epidemiological characteristics. Such a major environmental change can strongly influence not only their fitness abilities but also their health. Modern top-level competing athletes travel frequently and must be able to deal with environmental problems and also with problems related to mode of transportation, major time zone changes, different

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foods, housing and different approaches and levels of medical care at the new event destination. Fatigue connected with travel can influence their fitness capabilities and unexpected events abroad can ruin months of dedicated training.

16.2 Preparing for International Travel

Sports travel medicine is developed to assist and solve the problems of the travelling athlete, including those caused by training and competing in different environments in triathlon events. Many of the travel and sport-related diseases and ailments that can affect athletes are well known, have effective treatments and are largely preventable. Using preventive measures during training, competition and travel will decrease the chance of a risk to an athlete's health and his abilities. Triathlon athletes travel widely, usually by air, and often become lax about taking precautions regarding their health. Having travelled numerous times without major health upsets and focused on the forthcoming race, they may neglect to check that they are up to date with vaccinations or take necessary precautions against minor but potentially debilitating health conditions. Such neglect could easily cost them the medal and success.

As the teams often travel without an accompanying doctor's support, it is the coach's duty to ensure that athletes follow simple precautionary measures and that nothing stops his team on the road to success. A team doctor can plan the health protection, but it is the coach who is the closest to the athlete, has the most influence and is the one that athlete will follow all the way.

In international triathlon events, organizers are obliged to ensure an adequate chain of medical help that will give prompt and effective assistance in the event of an incident [1]. This should be provided during the event and for pre-event training.

Before departing, athletes should consult with their medical team. Evacuation and travel insurance to cover health emergencies while abroad should be in place before the team departs. All that planning can be done at the level of MNA or club at the beginning of the season when plans or training and events that will be attended are made. Situation should be followed and adapted to possible changes in plans or changes in epidemiological or environmental conditions. Information given to teams and athletes should always be up to date.

However, it is possible that there will be no pre-organised local medical support, for instance, in training camps, and one will therefore have to rely on his own abilities and the efficiency of the local medical system. In that case, teams should also be provided with the basic information about the local medical system and at least with local emergency phone numbers. If an incident happens, usually the coach or physiotherapist will have to provide first aid to the casualty until adequate medical help becomes available. The aim of first aid in sports is to save and preserve life, to prevent further damage and to relieve pain, and on every team at least one person should be competent in it.

In any case, if any health disturbance, especially the fever, develops during or after the trip abroad, one should seek competent medical help immediately.

16.2.1 Pretravel Planning

The first thing to do in any prevention project is to assess the risk. In pretravel planning, a team doctor who is regularly checking and treating an athlete can easily assess the risk of the individual athlete, including the potential environmental risks that may be encountered because of their itinerary, season, the risks of the racing field, past medical history, problem list and lifestyle [2]. It is a big advantage if the team doctor is able to plan for continuity of care before, during and after competition. Once this initial assessment has been made, the doctor can schedule the pre-competition protection "package" needed by the athlete in the context of the ongoing care.

It is ideal to start the process 4–8 weeks before the trip, but in a competition program where the season is crowded with races, the decision on participation is often made at short notice [3]. Planning should start about 3 weeks before departure to countries with different environmental conditions, but even in the "last-minute visits" to the team doctor, it is possible to deliver a good pretravel and pre-competition "package" which should consist of the following:

- Pretravel advice
- Letter regarding any current illness and TUE if necessary
- Medic alert tag (allergies, blood type, medical assistance company tel. number, etc.)
- Dental care or other minor ailments
- Eye glasses if necessary (extra pair, prescription sunglasses/contact lenses)

Pretravel planning in the team doctor's office should include oral and written instructions concerning the hazards in the countries to be visited, preventive measures, vaccination and prescription of necessary medicines.

16.2.2 Immunisation

A cornerstone of *travel medicine* is the prevention of infectious diseases through proper vaccination, including up-to-date routine immunisation, specific vaccines that will be required by any of the countries visited (Table 16.1) and vaccines which will be needed based on expected exposure [4–6]. Either inactive or live vaccines or a combination of types can be given simultaneously, without loss of either efficiency or safety.

Every athlete should receive information not only about required vaccines on border crossing (yellow fever is the only one at the moment) but also about recommended vaccines for that area. National federation or team doctor should provide athlete with that information. Vaccinations are administered according to the health risk which the travelling athlete is likely to incur (Table 16.2). Short-term travellers, for instance, participating in triathlon event that last only 1 day and staying in

Table 16.1 Travel vaccines

Class	Vaccine	Booster (year)	Indication
(a) Routine	Tetanus	10 (or according to national regulations)	All travellers
	Diphtheria	10 (or according to national regulations)	All travellers
	Pertussis	Unvaccinated (or according to national regulations)	All travellers
	Poliomyelitis (oral)	Once	All travellers
	Poliomyelitis (inj.)	Once	All travellers
	Human papilloma virus[a]	None	All travellers
	MMR (measles, mumps, rubella)	None	All travellers
	Hepatitis B	None	All travellers
	Influenza[b]	Annually	All travellers
	Rotavirus[a]	None	All travellers
	Tuberculosis (BCG)[c]	None	All travellers
	Varicella[a]	None	All travellers
	Pneumococcal disease	None	All travellers
(b) Required (International borders)	Yellow fever	None (10 in some countries)	S. America and Africa
	Meningococcal disease	1 (3–5 depending on the type of the vaccine)	Hajj (S. Arabia)
	Poliomyelitis	Once	Hajj (S. Arabia)
(c) Recommended	Cholera	2	Travellers to remote high-risk areas
	Hepatitis A[d]	None	Travellers to risk areas
	Japanese encephalitis[d]	1–2 (or none depending on the type of the vaccine)	Rural Asia, S.E. Asia, long stay in transmission areas
	Meningococcal disease[d]	1 (3–5 depending on the type of the vaccine)	Sub-Saharan Africa, group accommodation on big sport events (sport camps, sport villages)
	Rabies	If VNA titre < 0.5 IU/ml	
	Typhoid fever (inj.)	3	Indian subcontinent or prolonged stay/more than a month
	Typhoid fever (oral)	3–7	
	Yellow fever[d]	None	S. America, Africa

Table 16.1 (continued)

Class	Vaccine	Booster (year)	Indication
	Tick-borne encephalitis[a]	3	Eastern and Central Europe, parts of Scandinavia
	Dengue	None	S.E. Asia, S. America, Caribbean

[a]So far, introduced into the routine immunisation programme of a limited number of countries
[b]Routine vaccination for certain age groups and for individuals potentially exposed to certain risk factors
[c]No longer routine in most industrialised countries
[d]These vaccines are also included in the routine immunisation programme in several high-risk countries

Table 16.2 Immunisations according to risk

	No
Childhood immunisation?	→ Routine vaccinations (Table 16.1)
↓ Yes	Yes
Border crossing?	→ Yellow fever (meningococcal disease, polio)
↓ No	Yes
Food/water risk?	→ Hepatitis A, typhoid
↓ No	Yes
Long-term stay?	→ Hepatitis B, rabies, TBC test
↓ No	Yes
Special risk?	→ Japanese e., hepatitis B, rabies, dengue, tick born e.
↓ No	Yes
Athlete at big events	→ Influenza (meningococcal disease)

first-class hotels in urban centres, need less protection than those staying in training camp for prolonged periods in the developing world.

16.2.3 Travel Kit

If your team doctor is not accompanying you, he/she should be able to provide you with the proper "travel kit." These kits should contain first-aid materials, such as bandages, cold preparations, sunscreen, insect repellent, iodine or other antiseptic solution. If necessary, extra prescription eyeglasses and any other routinely used medicines or supplies can be a part of its contents too. A signed prescription form should accompany all prescription drugs carried, with proper labelling on the drug container:

- Antidiarrheal (loperamide)
- Antiemetic (dimenhydrinate)
- Analgesic (nonsteroidal anti-inflammatory agents/analgesics)

- Antihistamine (hydroxyzine, terfenadine)
- Antipyrexial (paracetamol, acetylsalicylic acid/aspirin)

To avoid suspicion and misunderstanding regarding medicines that could be considered unnecessary in some countries, all the kits must have the list of original contents provided by the manufacturer or prescription and accompanying letter from your team doctor, including TUE documentation if needed.

16.2.3.1 Antibiotics

Although many medical problems that athletes incur are of non-infectious origin (e.g. injuries or environment-associated illness) in triathlon, they are often at risk for acquiring a variety of infections. Infections can not only disrupt training program and result of the race; in some instances, they can have serious health consequences if not treated. Many infectious diseases are preventable, and antibiotics play an important role in the treatment and prevention of a variety of bacterial and parasitic infections. With appropriate advice, they can be used safely for prophylaxis and self-treatment during travel. If abroad alone, athletes must be carefully educated regarding symptoms of disease and appropriate indications for empiric antibiotic use. The risks and benefits of antibiotic use during travel should be weighed carefully for each situation and, if possible, it should be done in consultation with the team medical doctor.

In the life of every athlete, there are situations when illness "cannot be afforded" and can ruin lifelong preparation. In those situations, the medical team will usually opt for antibiotic prophylaxis, while if abroad for training camp, they could make different decision. Concept of the antibiotic for all reasons is especially appealing to the travelling athletes' environment where drugs are often self-prescribed or prescribed by non-medical persons (i.e. coach). Although antibiotic resistance is a growing problem, continuously changing such an advice, for the sport environment, the obvious choice is quinolone group of antibiotics. For travel shorter than 3 weeks, triathlon athlete should carry a 3-day course of *ciprofloxacin* and *doxycycline* for primary prophylaxis or self-treatment of leptospirosis [7].

16.2.4 STDs, HIV Infection and International Travel

Advice on prevention of sexually transmitted diseases (STDs) should also be part of the pretravel planning, at least in the form of pamphlets. All athletes should be aware of the risks of STDs, taught about the dangers, constantly reminded and advised to take condoms with them. Isolation from usual social constrains back home, anonymity and experience seeking may reduce social and sexual inhibitions and precipitate higher risk behaviour [8]. Over the last decades, 5–50% of short-term travellers engaged in casual sex during foreign trips, with a substantial risk of developing STDs [9, 10]. Even those with a partner need advice about STIs, condom use, hepatitis B vaccination and the need to come for screening after return if they have engaged in casual sex during the trip [10].

Dos and don'ts of HIV	
Do not	Do
• Engage in unsafe sex	• Use latex condoms
• Pierce ears	• Associate socially
• Accept acupuncture, tattoo or injections (reused needles)	• Share food, hug etc.
• Receive transfusion	
• Use illicit injectable drugs	

16.2.4.1 HIV Screening of International Travellers

Some countries in Eastern Europe, the Middle East and Asia now have policies to screen international travellers. For the most part, screening requirements apply only to long-term travellers (e.g. foreign students and workers) [11]. It is important to note that some countries will not accept the results of HIV testing abroad and will insist on testing for HIV soon after arrival. The list of HIV testing requirements for entry into foreign countries changes frequently, and therefore your team doctor or MNA should obtain that up-to-date information from the embassy or consulate of the countries on the team's itinerary.

16.2.5 Malaria Prevention (Chemoprophylaxis) and Other Mosquito-Borne Diseases

Malaria, viral haemorrhagic fevers, Zika virus disease, chikungunya, various rickettsial diseases and African sleeping sickness are vector-borne diseases that cannot be prevented by vaccine and for which limited protection by other means is available.

Although vaccine for dengue is available in some countries, its use is currently recommended under special circumstances [12]. In most people, the infection is mild and passes in about a week without causing any lasting problems, but in rare cases it can be very serious and potentially life threatening. As there is no specific treatment or widely available vaccine for dengue, it is important to try to avoid being bitten by mosquitoes when visiting an area where the infection is found.

In pretravel counselling, athletes should be given this advice, with special emphasis on the importance of compliance to chemoprophylaxis if malaria prevention is necessary when competing or training in malaria risk zone for a prolonged time.

Although due to the short-term stays during competition events the risk for such a vector-borne infection is lower and "stand-by treatment" can be used if necessary, personal protective measures to prevent the mosquito bites should be taken throughout the whole stay. These include the use of appropriate repellents and wearing of light-coloured long sleeves/pants clothing. Repellents should contain either DEET (diethyltoluamide) 20% or more, or IR 3535, or Picaridin, and may be applied to exposed skin and to clothing including sport gear (but not under clothing). The percentage of DEET does not increase its mosquito repelling power but

rather increases its longevity of action (DEET 30%: 4–6 h protection; DEET 90%: 8–10 h protection). Repeated application is normally needed several times a day. Any sunscreen should be applied first [13].

16.3 Adaptation to New Destination

16.3.1 Exposure and Adaptation to Heat

The triathlon season can require travel and competing in very hot environments with different climatic zones. In situations like running and cycling, where unavoidable environmental factors such as heat are exceeding physiological accepted limits, its influence can become detrimental to sports results and even endanger an athlete's health. Also, conditions of the water are the essential part of open-water swimming in triathlon and inevitably influence the results of the swimming leg of the race. Heat-related illness is a serious medical concern in open-water swimming too and can happen even if water temperature is inside the limit set up by the ITU (<32 °C) [14].

Our body tends to keep its own core temperature between certain boundaries. Normally, it is maintained within 0.5 °C of 36.7 °C. To remain in thermal balance, the heat being gained by the body must equal to that being lost, such that no heat is stored in the body. To achieve that, our body uses several efficient physiological mechanisms and in normal conditions manages to reduce the core temperature. Heat is exchanged between the body and the environment by four physical processes—convection, conduction, radiation and evaporation [15]. In the cold, the body predominantly relies on radiation, convection and conduction, while with warmer ambient temperatures, the body relies predominantly on radiation and evaporative cooling [16]. Those mechanisms have several physiological consequences, some of which although physiologically necessary can influence the athlete's final capabilities to perform at a high level. Only 20–25% of the chemical energy used during muscular contraction is converted into mechanical work; the remainder is liberated as heat. During sustained vigorous exercise, heat production can reach more than 20 kcal/min [15]. In such a situation, the central body temperature can raise to a level that impairs endurance before muscle glycogen stores are depleted [17]. With muscle activity and heath production, as the core temperature rises, there is a reflex vasodilatation of the cutaneous blood vessels. Acute effects of that adaptation to heath are sweat production and an increase in skin blood flow. That results in a threefold increase in blood flow with subsequent increase in sweating and evaporation [16]. Increased sweating leads to decrease in blood volume and consequent decrease in cardiac blood flow. Because of those physiological changes, blood shift increases cardiac blood flow demand. Such a change can negatively influence athletes' aerobic capacity, cognitive ability and recovery [18].

The body temperature attained during exercise is dependent on work rate but independent of ambient conditions over a range of temperatures (5–25 °C) with relatively low humidity ("prescriptive zone"). Above the "prescriptive zone", deep body temperature rises. So, when the air temperature exceeds 25 °C or humidity rises, heat loss must occur if thermal injury is to be avoided. Luckily, wind or apparent air movement while running or cycling is a natural cooling mechanism, but certain environmental conditions can expose athletes to dangerous hyperthermia. In climates with high humidity, the body's cooling mechanism can become ineffective. In hot weather, perspiration is the only way to release heat from our body while exercising (because it lowers body temperature by evaporation). If air humidity rises over 70%, evaporation is impaired and our body increases perspiration to the level of 3 l/h in the effort to keep its temperature low. This could lead to dehydration. Problem is that even the most effective oral fluid replacement strategies will fail to prevent dehydration above this level of sweat loss [15]. The upper rate at which fluid will empty from the stomach during exercise is 1–1.5 l/h which is less than amount lost by perspiration. This may lead to thermal injury.

Thermal injury is in order of increasing severity: heat cramps, heat exhaustion, heat stroke, exertional rhabdomyolysis and death [15].

In conditions of heath, sports performance is directly influenced by the level of the physical reserve deterioration and this deterioration is individual, specific to every athlete and not linked to the skill level. Athlete must achieve balance between avoiding hyperthermia, maintaining body stores of water within safe limits while losing sweat for evaporative cooling and continuing performance at the high level [17]. Keeping thermal balance and preventing dehydration is of utmost importance to an athlete's health and sport performance.

16.3.1.1 Acclimatisation

Our body not only immediately reacts to high environmental temperature but also tries in the long term to adapt to it. By prolonged exposure to such an environment, the athlete's body adapts to these conditions and despite the necessary physiological reactions to the heat starts to function more efficiently and again reaches top level of performance.

Acclimatisation to high temperatures is achieved in a process that lasts 1–3 weeks, but the systems of the body are adapting to heat exposure at varying rates [19]. It is important to exercise during these exposures (gradually increasing exercise intensity each day until at normal pace), as resting in the heat provides only partial acclimatisation. Heat acclimation has been shown to induce several positive physiological adaptations, including reduced oxygen uptake at a given power output, muscle glycogen sparing, improved myocardial efficiency and increased ventricular compliance. In acclimatisation process, sweat glands start to work more efficiently and salt is retained better in the body by altered sodium and chloride reabsorption. Using a 10-day heat acclimation program (90 min cycling at 50% VO_{2max} in 40 °C, 30%rh) showed improvements in 1-h time trial

performance in hot (38 °C, 30%rh; +8%) and cool conditions (13 °C, 30%rh, +5%) in well-trained cyclists. Heat acclimation also increased plasma volume, power output at lactate threshold, maximal cardiac output and VO_{2max} relative to the control group that performed the same 10-day exercise protocol under cool conditions [20].

Elite athletes benefit from adaptations to training with an expanded plasma volume that allows more blood to be shunted to aid peripheral convective cooling [17]. The adjustments to the raised body temperatures associated with exercise mean that fitter individuals can acclimatise more quickly (7–10 days). However, even fit individuals need to exercise in a hot environment in order to achieve full acclimatisation [15]. The process requires minimum exposure to representative environmental temperatures for at least 2 h per day and takes a total of 10–14 days. No more than 3 days should elapse between successive exposures.

These adaptations must be in place before psychophysical tolerance levels to heath are raised. The elevated tolerance of internal temperature is about 0.2 °C [21]. In athletes early occurring adaptations that include expansion of plasma volume can be maintained 3 weeks if stimulus is appropriate. The acclimatisation acquired is specific to the climate and activity level; those acclimatised to hot dry environments will require additional time to acclimatise if they move to hot humid environments. It is important to know that during the acclimatisation period physical efficiency suffers, so if athlete wants to be at its peak while competing, acclimatisation should be completed before the race. Acclimatisation process can also have negative impact on training quality associated with lower performance in first days at the new environment.

The problem is that today athletes are travelling fast and there is no time for complete acclimatisation upon arrival. Athletes often arrive at a venue only a few days before the event begins, minimising the ability to acclimatise to the hot conditions. This can be a problem for the teams from cold climate zones, so they will be forced to acclimatise in "environmental chambers" or sauna. Although it may be difficult to find the facilities needed to create the warm environment required for this manoeuvre, the triathlete may be able to perform a modified version of the protocol used by Lorenzo et al. by completing light exercise in the heat over the final days leading up to their event [20].

16.3.1.2 Heat Acclimatisation Protocol in Saunas

Two days a week, three exposures of 6–9 min on the temperature of 90–100 °C with relative air humidity of 10–20%.

The best results are achieved by training in hot conditions as close as possible to those that athlete will be exposed during the sport event because specific combination of individual and environmental characteristics dictate the magnitude of adaptation [22].

Besides acclimatisation procedures, there are some other recommendations to consider:

- In a very hot environment, reduce physical activity to swimming during the hottest daylight hours. Use indoor gyms and swimming pools to train avoiding the heat.
- In the hot environment, the warmup should be limited in intensity and duration and adequate recovery time be provided before the start of competition to rehydrate and restore the body's core temperature.
- Before the race, avoid working or staying too long in an overheated container, even if it's the only shadow available.
- Staying in the *athlete's lounge* or restaurant may help the athletes keep their body temperature at the right level, therefore allowing them to compete better. However, entering a cooled room coming directly from outside may cause a few problems: one should not forget to put on dry and warm clothes when entering a cooled room; be careful not to enter these places with wet clothes or wetsuits.
- Athletes should wear light and light-coloured clothes with UV protection. They should always wear a cap, sunglasses and sunscreen cream. Sunscreen preparations should be applied several times a day.
- They should have enough fluids during exercise (600–800 cc/h, drink small amounts at least every 15 min), but they must remember that hydration can prevent dehydration but cannot decrease core blood temperature!
- Extra-dietary salt and adequate rest.
- Consistent daily monitoring of fluid/electrolyte balance is required.

Simple advice about proper fluid intake can save a lot of problems, especially in very humid weather, but excess dietary water and electrolytes do not speed up the process of heat acclimatisation. Also, one should not forget that the triathlon season is long and heat acclimatisation adaptation may vanish after few weeks. Not only does their performance suffer but un-acclimatised athletes in hot climates are prone to heat injury. Heat-related illness is a serious medical concern in all three disciplines in triathlon. It is important that the team doctor and medical team on the field of play are able to recognise and manage the signs of the athlete in heat distress. The team doctor should have a predetermined medical action plan in coordination with the local organising medical staff to manage hyperthermia [23].

16.3.1.3 Precooling

Precooling is the strategy to reduce body temperature and enhance the body capacity to store heat during the subsequent bout of exercise. Reducing core temperature by 0.7 °C (e.g. immersion in cool water) has been shown to increase subsequent exercise endurance in hot and humid conditions, with time to exhaustion being inversely related to initial body temperature. Cooling the skin by 5–6 °C reduces thermal strain and increases cycling performance (distance cycled in 30 min) in

warm and humid conditions. Returning body temperature to normal levels between repeated bouts of exercise in the heat helps to maintain performance, decreases physiological strain and extends work time [15].

Numerous cooling modalities currently promote both local and systemic reductions in body temperature (ice packs, cooling vests, heath sink vests, ice baths, cool air baths) but precooling in competitive situations relies upon effective strategies that cause minimal disruption to the athlete's preparation for competition. In triathlon event, precooling can be used before the swimming part in case of high outdoor temperatures and high water temperatures. The resulting thermal advantage is characterised by reductions in core body temperature, skin temperature, a decrease in cardiovascular and metabolic strain and reduced perception of effort which may be consistent with performance improvement [17].

Athletes should be encouraged to use various methods of precooling in unfavourable heath conditions, and these techniques should be discussed and approved by the team doctor.

16.3.2 Exposure and Adaptation to Different Time Zone

Every natural process within the body shows some variation in pattern between night and day. Circadian rhythms are endogenous cycles that last about 24 h (the "body clock") but may last between 20 and 28 h [24]. These cycles are synchronised (entrained) by the earth's 24-h light–dark cycle but persist in the absence of light and dark cues [24]. Many circadian rhythms have been identified, including core body temperature and behavioural rhythms, such as the sleep–wake cycle [25].

Basic components of performance also have rhythmic ups and downs that follow circadian pattern. These include leg strength, back strength, elbow flexors, sprint and anaerobic efforts and aerobic tasks or sport-specific tasks such as soccer, jumping, cycling and swimming. Significant circadian rhythmicity has also been shown in surrogates of performance such as heart rate, blood pressure and blood lactate [26]. Speed of reaction time and muscle strength peak consistently in the early evening and it is well known that world records are usually broken by athletes competing in the late afternoon/early evening hours [27, 28].

Rapid air travel across several time zones outstrips the ability of the body to re-synchronise these rhythms forcing athlete to compete at unfavourable periods of their biorhythms when their capabilities are not at their peak [29, 30].

16.3.2.1 Jet Lag (Circadian Dysrhythmia)

When the internal sleep–wake cycle is out of phase with the local light–dark cycle, it is causing drowsiness or arousal at "inappropriate" local times. The resulting physiological desynchronisation causes symptoms such as weakness, gastrointestinal disturbance, loss of appetite, tiredness during the day, disorientation, memory

impairment and reduced mental performance that every traveller recognizes very readily as *jet lag* [29, 31]. *Jet lag* can significantly influence athlete's physical and especially cognitive capabilities to perform at a high level. *Jet lag* symptoms usually persist 1 day for each time zone crossed until the body realigns its circadian clock, regardless of direction travelled [28]. Symptoms of jet lag and sleep disturbances are worse after eastward flights, when the length of the day is shortened, and the circadian system must shorten to re-establish a normal rhythm [32–35]. When travel is westward, symptoms peak in the first 3 days, while for eastward travel, the symptoms persist for as long as 7 days [35].

Often, teams are coming on racing fields of play by airplane, from another part of the world and from different time zones, immediately being engaged in competition. The team leader, who is responsible for organising the team's itinerary, should know the basics of biorhythm and desynchronisation problem so that he could organise the transport that best corresponds with athletes' biorhythm, allows proper rest and secures adequate time for adaptation on arrival. For a short-term travel (1–2 days), full adaptation to the new time zone is not always recommended, as full adaptation in short time is unlikely to be achieved. When travel across time zones is for longer than 3 days, circadian adaptation is typically recommended. The sleep–wake cycle recovers quicker (2–3 days) than do physiological cycles, which may take 8–10 days to resynchronise [24]. Luckily, higher levels of physical fitness allow people to adjust more quickly to changes in those cycles [36, 37].

Adaptation procedures to new time zone [27]:

(a) Sleep–wake time shift:	
Westbound	(Pre-departures) go to bed later and waken later
Eastbound	(Pre-departures) go to bed earlier and waken earlier
(b) Light-exposure alteration[a]:	
Eastbound:	(On arrival) ≤ 6 time zones: ↑ a.m. light
	(On arrival) 7–12 time zones: ↑ p.m. light
Westbound:	(On arrival) reverse of eastbound
(c) Melatonin: 3 mg (0.5–5)	
Eastbound:	(Pre-departures) at 2–3 a.m. "destination time" for 3 days
	(On arrival) at bedtime for 4 days
Westbound:	(On arrival) at bedtime for 4 days
(d) Caffeine: 200–1000 mg	
	(On arrival) morning, or chosen time before the match[b]

[a]Several methods of exposing the eyes to an artificial bright light that simulates sunlight for brief periods at planned times during the day are developed. Various modalities include a light box, a lamp and a light visor [38–40]

[b]While not banned by World Anti-Doping Agency, caffeine is a monitored substance [41]

16.4 Protection from the Sun and Cold

The climatic environment can have an extraordinary influence on the outcome of the triathlon competition because triathlon is a sport practised in the open, with only limited means of environmental protection, which is normally personal gear and equipment.

16.4.1 Exposure to UV Radiation from the Sun

Besides infrared rays that we feel as heat, the sun is also radiating much more damaging ultraviolet (UV) rays, particularly UVB with wavelength of 280–315 nm, that are causing serious damage to our skin (sunburn), including skin cancer. Exposure to sunlight may result in solar urticaria that can occur minutes of exposure. It can also cause damage to our eyes (acute keratitis, cataracts and macular degeneration—after long-term exposition). Exposure may supress the immune system and increase the risk of infectious disease [42].

The UV index shows us how strong the UV rays are. The values of the index range from zero upwards—the higher the index value, the greater the potential to the skin and eyes (and the less time it takes for harm to occur). The index values are grouped in exposure categories, with values greater than 10 being "extreme". Before travel, one should always obtain the UV index data at the event destination. It is as important as the weather, wind and temperature forecast!

The risk must be reduced by using the right skin protection and UV sunglasses.

To avoid some of the consequences of competing/training in countries with a high UV index (higher than 5) one should do the following:

- When training, avoid the sun between central times of the day: 11.00 h and 16.00 h.
- Use of special UV textile equipment; wraparound sunglasses with appropriate UV filters and caps with rim or flaps for a better head, neck and ear protection.
- Be sure that the remaining parts of the bodies are regularly covered with sunscreen preparations.
- Use the appropriate sunscreen:
  - *SPF 25 or greater*
- Apply appropriate quantity of sunscreen.

An adult athlete, if wearing short sleeves shirt and shorts, needs approximately 2 mg of sunscreen preparation per cm^2 of the skin. This means that most athletes will need about 15–20 ml each time. This is 1/6 of a 100 ml bottle to cover the exposed parts of the body. The reality is that the majority of people apply just 1/4 up to 1/3 of the quantity needed! This means they will have a protection of just about 1/4 or 1/3 of the indicated SPF on the sunscreen bottle: for example, using an SPF 25 sunscreen, they will just have an SPF approximately of 6–7, which will probably not be enough to protect their skin during the race that can last few hours, sometimes continuously being exposed to sun [43].

16.4.2 Exposure to Cold

A cold climate is unavoidable in triathlon. Even in temperate areas, a combination of cold water, wind, wet clothes, fatigue and dehydration can dangerously lower the body temperature and influence the performance of competing athletes.

The physics of heat transfer is the same regardless of whether one is in hot or cold environment. In water, only two of four primary pathways for heat exchange are available, and thus heat loss is principally caused by convective and conductive heat exchange. Despite this, naked individual in cold water will cool approximately four times faster than when exposed to air of the same temperature [44]. As water conducts heat at a much higher rate than the air, in parts of the race on shore, wet athletes' bodies in wet *tri-suits* will cool at much higher speed and consequently can produce an undesired heat loss too.

In a cold environment, our body reacts to cold by trying to keep its core warm (36.5–37.5 °C), leaving behind the thick "shell" to fight a rear-guard action with the elements. Warm blood from the core is diverted from the surface, the temperature of the skin falls, and less heat is lost. If the increase in size of "shell" fails to keep the core temperature, the body turns to its second line of defence; an increase in heat production either by shivering or by exercise. This can increase heat production up to ten times. For an athlete, exercise is the perfect solution; however, problems can arise. If the heat loss is modest, then exercise may produce enough of a net gain in heat to keep the athlete warm, but if the heat loss is rapid the chances are that exercise will be counterproductive. While racing, athletes need their strength and endurance to win the race so using the energy to heat the body can endanger the result of the race.

A foolhardy approach to the cold while swimming in the water or cycling and running on shore negatively impacts performance. When skin temperatures fall below 15 °C, there is also a decline in the strength of handgrip and manual dexterity that could impact the cycling leg of the race. Although the lives of athletes are not directly in danger, this may decrease performance and certainly may have an untoward consequence to the result of the race. This results from the effect of the cold on the muscles, nerves and even brain, if hypothermia is present. Hypothermia in open-water environment can be caused by cool ambient temperatures, wind, currents and cold-water temperature. Exercise-induced asthma (EIA) can be exacerbated by cold temperatures. The combination of cold-water exposure, voluntary apnoea and face immersion (diving bradycardia) may result in increased sympathetic and parasympathetic activities, possibly causing ventricular premature beats [23]. In contrast to the well-described ability to adapt to heat, there appears to be little, if any physiologic ability to acclimatise to cold. "Adaptation" needs to be based on training, experience and the avoidance of those behaviours known to increase the risk of cold illness [22]. However, experiments showed that habituation with repeated cold-water immersions can significantly reduce the magnitude of the cold shock response. It can be reduced by 50% in as few as five 2-min immersions in cold water. Moreover, most of the habituation remains up to a year [45].

In triathlon, lower limit for the water temperature is 12 °C but if outside temperature is lower than 15 °C and the water temperature is lower than 22 °C, then

according to the ITU rules the adjusted value is to decrease the measured water temperature: race will be cancelled if, for instance, water temperature is below 12 °C and air temperature is below 10 °C or water is 22 °C and outside temperature is 5 °C. In order to protect athletes' health and prevent cold exposure, similar rules are developed considering the wetsuit use and maximum stay in the water [13].

Keeping thermal balance is of utmost importance. Wetsuits to protect the body heat are mandatory if water temperature is 15.9 °C and below [13]. But, as overdressing can overheat the body, this should be avoided too. ITU rules are putting limits to use of wetsuits to limit the possibility of overheating. They are 20 or 22 °C and above, depending if the swim length is up to or over 1500 m (22/24.6 for *age group athletes* on same distances) [13].

Aside from using suitable clothes, there are some recommendations to consider:

- Wear a cap in the swimming leg—the head is responsible for one-third of the body's heat loss.
- Wetsuit is crucial to prevent hypothermia.
- Wear only high-tech clothing designed for triathlon.
- Test the clothing in an appropriate environment.
- Always be prepared to recognise the signs of hypothermia.

16.4.2.1 Pre-competition Warm-Up in Cold Environment

When competing in cold conditions or in cold water, pre-competition warm-up or training warm-up is particularly important. Prior to carrying out any medium or high-intensity exercise, it is very important that the muscles that will be used have received the necessary oxygenation for the work they are going to do. This means carrying out wide and progressive low-intensity movements to open up the greatest number of capillaries ensuring that the blood goes to the highest number of muscular fibres.

- Until the race starts, wear additional clothing.
- Under cold conditions, start the warm-up session on shore.
- Session must last between 15 and 20 min.
- It must include all muscles but particularly those that are going to be used more when swimming.
- In transition phase of the race, dry the body if possible.

16.5 Choice of the Right Fluids

Careful choice/treatment of water—whether for drinking, washing, preparing food or swimming, is one of the most important precautions a travelling athlete can take. Water is critical for exercise performance and is the nutrient often neglected.

Fluids are necessary during exercise to allow an athlete's body to function properly to its full potential. With more fluid intake, responses to exercise like heart rate, body temperature and perceived exertion are decreased [46]. Adequate water levels

are also required to store the glycogen goals of carbohydrate loading [47]. In order to maintain a desired level of hydration and thus prevent dehydration, the intake of fluids must be equivalent to the amount of fluid loss [48]. In endurance events, as much as 3 l/h of fluids can be lost through the process of sweating which solidifies the need to replace as one loses [49]. If fluid losses are not replaced, performance will deteriorate, and dehydration can have devastating consequences to the sport performance [50]. Exercise performance is impaired when an individual is dehydrated by as little as 2% of body weight. Losses more than 5% of body weight can decrease the capacity for work by about 30% [51].

Triathlon events may be run for several hours at a time, during the summer months, often during the hottest part of the day. There is the very real possibility that hydration may be compromised during the event or at some point of training session. During exercise, fluid losses are primarily due to sweating and breathing. In triathlon, this is emphasised with unavoidable sun and wind exposure. Uneven body cooling when swimming, running and cycling puts an additional burden on physiological mechanisms of the body's cooling which needs adequate fluid balance to function properly.

Guidelines for drinking in different sports have been heavily debated in recent years [52, 53]. Fact is that hydration requirements will fluctuate with environmental condition and metabolic rate. Fluid volumes to be taken will depend largely on sweat rates, which will be influenced by environmental temperatures, metabolic rates and resulting core temperatures. Because environmental conditions are sometimes not known until after the start off, a potential strategy may be to have a variety of more (gels, cola) and less (weak carbohydrate–electrolyte) concentrated sport drinks available for use during the bike and run phases, to self-administer carbohydrate and fluids according to requirements. The most prudent advice is to drink throughout a long-distance triathlon in accordance with thirst, but in high-level sport where improvisations are not allowed, hydration program of top athletes should be in the hands of nutritional expert [54].

As fluid and food intake tend to be hampered by unpredictable starting times depending on environment conditions, support teams must secure adequate hydration before and during competition, taking into consideration regulations limits for the amount of fluid competitors can carry. Adequate storage space for sports drinks must be secured to give fluids to athletes' racing or during the training sessions.

Use the proper procedures of hydration:

- Team doctor or nutritionist should do the planning.
- Lectures for athletes to explain the importance of hydration should be organised.
- Hydration should be the habit of athletes.
- A protocol of hydration should be established before the triathlon event.
- Sports drinks should be chosen before the triathlon event.
- Athletes should be educated on the hydration protocol before the event.
- During the event, hydration protocol should be laid down and followed strictly.

- Athletes should be weighed before and after the race.
- Daily estimate the body's water balance by measuring the urine specific weight.

In the developed world, the availability of safe water is taken for granted but even there, bad sanitation is a real possibility. In the developing world, water-related diseases remain a major problem. Many important infectious diseases are transmitted by contaminated water but by following some simple rules, that risk can be minimised:

(a) *Recommendations when abroad:*
First-class hotels are no guarantee of adequate water purification.
- Use bottled water only.
- Canned or bottled "carbonated" drinks and beverages made from boiled water are safe.
- Ice should be made from purified water.

(b) *Recommendations while on the field of play:*
- Drink only originally packed sports drinks or those prepared with bottled water.
- Supporting team should carry enough fluids and hand it over to athletes during transitions.
- Store the fluids in an appropriate cool box.

16.6 Choice of the Right Food

Whether at home or abroad, performance of the athletes can be substantially affected by the amount, composition and timing of food intake. Good nutritional practices will help athletes to train hard and recover quickly from the strains of training, travel and competition. A diet that provides adequate energy from a wide range of commonly available foods can meet the carbohydrate, protein, fat and micronutrient requirements of training and competition.

With respect to bioenergetics, success in the triathlon will stem from the ability of the triathlete to oxidise macronutrients at high rates and to transfer this energy to the mechanical energy needed to power the triathlete around the triathlon course [55]. Estimates of energy expenditure approximate 3000 kcal for Olympic triathlon, while long-distance triathlons, including half- and full-distance Ironman events, require prolonged (>4 h) durations of moderate- to high-intensity exercise, which mostly use energy from the oxidation of fat and carbohydrate in the range of 4500 and 11,500 kcal [56–58].

During high-intensity training, particularly of long duration, athletes should aim to achieve carbohydrate intakes that meet the needs of their training programmes and also adequately replace carbohydrate stores during recovery between training sessions and competitions. Dietary protein should be consumed in daily amounts greater than those recommended for the general population. Therefore, meals should consist of 6–10 g/kg/day complex carbohydrates and 1.2–1.8 g/kg/day

proteins [59]. The availability of carbohydrate as a substrate for muscle metabolism and central nervous system support is a critical contributor to long-distance triathlon performance [60]. Our body can only store enough carbohydrate for up to 90–120 min [61]. As the total body carbohydrate stores are limited and are substantially less than the fuel requirements of the long-distance triathlon, in an effort to boost these limited stores, different strategies of carbohydrate loading are developed. Consuming carbohydrate intakes of 10–12 g/kg/day, over the 36–48 h leading up to competition, is often promoted to improve performance [62].

Due to specific timing of triathlon training and competitions, main meals are in the morning and after return from the race/training, usually structured as follows:

- *Breakfast*—1000 kcal
  Athlete should have breakfast 2–3 h before the race. Long races can start early but 1.5 h is minimum. It should be mainly carbohydrate based as our liver glycogen stores decrease over night. Athletes should make sure that they are comfortable with it and involve foods that they are accustomed to, such as toast, cereals and juices.
- *Lunch* (during the race/training)—500–1000 kcal
  Consuming in T1 and T2 while running and cycling is the key, so one should get confident at consuming food and fluid while at a good pace. Athlete will need to intake up to 60–90 g of carbohydrate per hour, during prolonged exercise (over 90 min), to maintain carbohydrate supply to the muscles [61]. Knowing how long he'll be competing, together with his team, he can design the protocol and pack enough nutrition to see him through to the end.
- *Dinner*—1500 kcal
  The capacity of muscles to absorb and store nutrients is increased 30–60 min post-exercise, so it is important to replace carbohydrates and provide protein and electrolytes within this time. Pre-planning the meals or snacks after training ensures that athlete can take advantage of that recovery window. A full carbohydrate-based meal should be taken within 1 h of finishing a tough training session or race. This can be done with food like rice, pasta, bread and protein options like yoghurt, chicken and fish. However, often after exertion, athletes lost appetite so one should find what works for him/her.

Each team/athlete should adopt specific nutritional strategies before and during competition, but it is of the utmost importance that all nutrition protocols are tested before the race day. Those protocols should be developed and designed with the help of a sports nutritionist and they should be followed strictly, especially during competition events.

Proper procedures of sport nutritional practice:

- Team doctor or nutritionist should do the planning.
- Lectures for athletes explaining the importance of proper sports nutrition should be organised.
- Families should be involved in the program of athletes' nutrition.

- Proper nutrition should become a habit of every athlete.
- An aim to reach carbohydrate intake that meets athlete's fuel requirements should be achieved.
- Athletes should be warned against uncontrolled use of dietary supplements.
- Athletes should be banned from improvising with "natural products" that are supposed to enhance their capabilities. They don't work and can contain prohibited substances.

At sport events:

- Food availability at the new destination should be investigated before leaving home.
- Catering provider at new destination should be contacted and in advance informed about team's/athlete's needs.
- Missing food supplies should be taken to new destination.
- A nutritional protocol should be established before the event and followed strictly.
- Protocol should be tested before the event so that athletes can get used to the recipes.
- Care should be taken that athletes are not tempted by the food on offer in self-service restaurants at big events. They should learn in advance what to choose according to their nutrition protocol.

16.6.1 Travellers' Diarrhoea

Travellers' diarrhoea is an illness associated with contaminated food or water that occurs during or shortly after travel. Depending on the length of stay, it may affect up to 50% of travellers [63]. It may be accompanied by nausea, vomiting, abdominal cramps and fever [64]. Simple bout of diarrhoea can seriously endanger the athlete's success in competition. Food spoils rapidly in a hot climate, especially meat, poultry and dairy products. With high humidity and temperature, food becomes an excellent culture media for bacterial growth. Therefore, even light contamination can lead to dangerous bacterial levels within a few hours. In high-risk environments, athletes should stick to food produced in good hotels, in well-known restaurants or self-catering organised by their teams. It is important to keep to the eating plan that is normally used at home and to avoid the temptation to have an "authentic cultural experience".

16.6.1.1 Prevention of Diarrhoea (Short-Term Travel, Up to 3 Weeks)

When abroad, each team/athlete should obey to the rule: *Cook it, peel it or leave it!* Therefore, athletes should eat only fruit and vegetables, which they can peel and wash themselves and eat only hot, cooked food.

1. *To avoid*
   - Using leftovers.
   - Blown tins or "swells" with canned food.
   - Un-pasteurised milk and milk products.
   - Raw shellfish.
   - Food from street vendors.
   - Leaving hot food to stand and cool before serving.
   - Cold meats in restaurants.
2. *To do*
   - Eat all food hot and cooked through (especially meat and seafood).
   - Peel, wash or soak fruit and vegetables before eating (in sodium hypochlorite or some mild disinfectant).

Elite athletes competing in the special events, for example, the Olympic triathlon competition, cannot afford to get ill. In clearly risky situations, preventive use of antibiotics can be considered [65]. This is also necessary for athletes with underlying medical problems. Weighing the risks of the side effects, against the risk of not competing, is subjective and is best accomplished in consultation amongst the athletes and the team doctor.

Antibiotics	
Rifaximin	400 mg daily

Athletes for whom preventive use of antibiotics might be considered:

- Poor "track-record" travellers.
- Low gastric acid:
  - Antacids or H-2 blockers, proton pump inhibitors
  - Gastric surgery
  - Underlying medical problems:
    - Diabetes mellitus

16.6.1.2 Management of Diarrhoea

Estimated rates of *travellers' diarrhoea* during first 2 weeks (i.e. major sport events) in a high-risk destination range from 20% to 50% per trip, meaning that one could find himself in a fight for the medals with 50% of the team having diarrhoea and being dehydrated [63]. Athletes should be reminded of specific ways to avoid *traveller's diarrhoea*.

Unfortunately, if the problem happens, one cannot improvise. If the team doctor is present, he will handle the case, but if athlete is alone, the rules of treatment should be obeyed:

1. *Fluid replacement*
   (a) Discontinue milk products.

(b) Drink commercially prepared medical electrolyte/glucose mixture (e.g. *Gastrolyte, Hydralite*) or commercially prepared oral rehydration salt mixed with the safe water.

(c) If not available, use some sport rehydration drink or prepare rehydration solution using these two simple recipes [66, 67]:

- 1/2 tsp* salt
- 1/2 tsp* baking soda
- 4 tbsp** sugar
- 1 l safe drinking water

or

- 6 level tsp* sugar (25.2 g)
- 0.5 tsp* salt (2.9 g)
- 1 l safe drinking water
  - *tsp: tea spoon = 5 ml
  - **tbsp: tablespoon = 15 ml

2. *Antimotility agents*
   - Loperamide (Imodium)
     4 mg first dose, and then 2 mg dose after each loose stool, not to exceed 16 mg in a 24-h period—alone for mild diarrhoea (tolerable) or with an antibiotic in case of moderate (distressing) to severe (incapacitating) illness [65, 68].
     Note: Antimotility agents should not be used alone in case of severe illness and one should always try to consult with his team's doctor.
3. *Antibiotics* [67]

Levofloxacin	500 mg single dose[a,b] or 3-day course
Ciprofloxacin	750 mg single dose[a,b]
	or 500 mg 3-day course
Ofloxacin	400 mg single dose[b] or 3-day course[a]
Azithromycin	1000 mg single dose[b] or
	500 mg 3-day course
Rifaximin	200 mg 3× daily—3-day course

[a]*Note:* Several scientific papers established a link between quinolone antibiotics and tendon ruptures—seek medical advice before using them [69–71]
[b]If symptoms are not resolved after 24 h, continue daily dosing for up to 3 days

16.6.1.3 Procedures of Epidemic Control

If confronted with a case of diarrhoea, procedures should be initiated to prevent its spread [72–80]. These procedures should be in place before the team's arrival at the new destination and should be designed by medical support team. Some procedures, like regular wiping of the doorknobs with disinfectant, can be applied as preventive measure if the team doctor assesses the new destination as a risky one. Also, enough disinfectant that can be used if this happens should be carried.

These procedures should be initiated the minute the case of diarrhoea amongst members of the team is detected:

1. *Accommodation*:
   - Sick team member should be isolated in a separate room.
   - If several team members become sick, they can stay together in the same room.
   - If isolation room does not have its own toilet, a toilet that will be used only by the sick team member should be designated.
   - Room/toilet should be supplied with disinfectants (e.g. soap and alcohol solution with *chlorhexidine*).
   - All clothes used by the sick team member must be put in a separate bag.
   - Floor and all surfaces should be cleaned according to procedure 3 below.
   - Floor and all surfaces should be disinfected according to the succeeding procedure 4 .
2. *Procedure for the regular cleaning of the rooms during sport events*

Area	Frequency	Cleaning agent	Method of administration[a]
Floors	2× day	Detergent containing *1,3-dihydroxymethyl-5,5-dimethyl imidazoline-2,4-dione*	Prepare solution as in instructions. Soak the mop in the prepared solution. Rinse the mop in another vessel with clean water. Leave to dry.
Other surfaces	1× day		
Walls	1× week		
Toilets	2× day	Solution with *6 g benzalkonium chloride + 3.0 g didecyldimethylammonium chloride + 8.0 g glutaraldehyde*	Spread evenly on the surfaces, after 5–10 min rub and rinse with water. Repeat the procedure if needed.

[a]Always check the instructions by the producers of the cleaning agent regarding preparation of solution and modes of use

3. *Procedure for the cleaning or the rooms in the case of the risk of infection*

Area	Frequency	Cleaning agent	Method of administration[a]
Floors	2× in the morning 2× in the evening	Detergent containing: *1,3-dihydroxymethyl-5,5-dimethyl imidazoline-2,4-dione*	Prepare solution as in instructions. Soak the mop in the prepared solution. Rinse the mop in another vessel with the clean water. Leave to dry.
Other surfaces	1× day		
Walls	1× week		
Toilets	2× in the morning 2× in the evening	e.g. solution with *6 g benzalkonium chloride + 3.0 g didecyldimethylammonium chloride + 8.0 g glutaraldehyde*	Spread evenly on the surfaces, after 5–10 min rub and rinse with the water. Repeat the procedure if needed.

4. *Procedure for the disinfection in the case of illness*

Area	Frequency	Cleaning agent	Method of administration*
Floors	2× day	Disinfection solution with *13.2 g benzalkonium chloride + 6.0 g didecyldimethylammonium chloride + 4.5 g formic acid* or Solution with *4.5 g. didecyldimethylammonium chloride + 1.8 g. isopropyl alcohol*	Prepare solution as in instructions. During disinfection soak he mop in the vessel with the solution. Wipe the whole surface. Mops should be single used. Do not rinse.
Other surfaces	1× day		
Walls	1× week		
Toilets	2× day and immediately in the case of obvious contamination	Disinfection solution of *sodium dichloroisocyanurate*	Prepare solution as in instructions. During disinfection soak the mop in the vessel with the solution. Wipe the whole surface. Mops should be single used.

[a]Always check the instructions by the producers of the cleaning agent regarding preparation of solution and modes of use

5. *Team behaviour*:
   - All team members should be informed in which room the sick team member has been isolated.
   - Contact should be restricted to as few as possible people.
   - Room should be entered only if necessary.
   - Before entering the room, and on exit, hands should be washed and rub in alcohol-based disinfectant.
6. *Sick team member(s) behaviour*:
   - Hands should be washed with liquid soap and water for 60 s and dried with paper towels.
   - After washing the hands, one should disinfect them by rubbing in alcohol-based disinfectant. Then, wait for the hands to dry fully.
   - The steps mentioned earlier should always be carried out after personal hygiene procedures, using the toilet, before taking food, touching anything unclean, etc.
   - Isolation room should not be left except for medical intervention.
7. *Sick team member appliances (cutlery, shaving kit, etc.)*:
   - To be used only by sick team member.
   - Kept separately in isolation.
   - All supplies used must be cleaned with disinfecting solution (e.g. *30% 8 g glutaraldehyde/3 g didecyldimethylammonium chloride/6 g benzalkonium chloride* solution).

16.7 Risk of Travel Fatigue and Overstraining

The effects of airplane travel on athletic performance is relevant in a world that is increasingly reliant on flying for athletic competition. In the need to fulfil the training programs and to compete at the peak of abilities, it is easy to overlook the

problem of *travel fatigue* and the necessity for rest, especially during travel and on arrival to new destination.

Air travel factors, like noise exposure, immobility, sleep loss, dietary changes and even breathing poor-quality air can impact performance in ways that many may not realize. On top of everything, travel is often psychologically stressful due to delays, security and worry over potential lost luggage containing race gear. Regular routines are disrupted when one travels abroad. Athletes are particularly excited about the trip and forthcoming race at the new destination and worried about planning for the departure. Depending on the country to be visited, visas and vaccinations may be required. Professional/national teams usually have arrangements made for them by their administrative staff and they should aim to take of the burden of travel from athletes as much as possible. Basic thing as booking the airplane ticket can make a difference; seats with more leg room should be bought, time of arrival in the evening and direct flights, if possible. These arrangements should extend to coping with formal procedures at departure and disembarkation and avoiding any mix-ups in dealing with ground staff and security controls.

Travel fatigue can be defined as temporary exhaustion or tiredness caused by any long journey irrespective of mode of transport. North–south travel is causing seasonal change, while east–west travel over more than two time zones is causing *jet lag*. Contrary to *jet lag* that can last for a week or more, if properly treated, *travel fatigue* subsides the next day but can accumulate over course of season and can be persistent and impact athlete's capacity to recover and perform, causing behavioural and mood changes with loss of motivation [28]. Problem of lower performance after travel is well documented and can be augmented by poor training regime on arrival [28, 81].

Having arrived safely at the destination, the athlete may suffer not only from *jet lag* but also from *travel fatigue* due to the conditions in travel vehicle, when the passenger simply becomes tired from the journey or stiff after a long stay in a cramped posture. Sitting in a seat for several hours while on the airplane can produce lethargic limbs. The long periods of inactivity during the plane journey may lead to the pooling of blood in the legs and in susceptible people cause a deep vein thrombosis. Moving around the plane periodically during the journey, at least every 2 h, and doing light stretching exercises is recommended [82]. If the coach is present, he should remind athletes to do it and watch over other necessary procedures.

Airplane travel harms athletic performance via the reduced oxygen pressure too. Airplane cabin oxygen pressure is generally maintained at the equivalent of 5000–6000 ft. (1520–1828 m), which is equivalent to an inspired oxygen pressure (PO_2) of 132–127 mmHg. This produces a hypoxic stress and oxygen saturation levels decline significantly after 3 and 7 h of flight [83]. Altitude exposure results in a significant decline in time trial performance in aerobic sports [83–87]. It is plausible that athletes would require a period of adaptation for optimised performance after long flights (>10 h). One may need a day to fully recover their oxygen levels, so it is be recommended that athletes avoid arriving the same day of competition [83].

Aircrafts typically maintain a 20% humidity level, which is well below the body's required 30%. Despite this, long haul flights do not cause significant whole-body dehydration but the dry air in the cabin does, however, influence mucous

membranes of the eyes, mouth and nose and can lead to discomfort [88]. Although the extra body water lost in such circumstances is small, athletes cannot afford it. This means that athletes should hydrate even more than usual. This is important because many people tend to drink less during air travel, which can result in dehydration prior to the race. Athletes should drink about 15–20 ml extra fluid per hour, preferably some sport drink or water [89].

Nutrition is essential for performance and the circadian desynchronisation that contributes to feelings of jet lag also affects gastrointestinal function and digestion [89, 90]. Circadian disruption can cause a delay in the absorption of food from the gastrointestinal tract after eating at night [91]. A large meal eaten late in the evening could lead to bloating and sleep disruption [89]. However, patterns of activity and eating vary in different locations, causing challenges when adapting to new mealtimes and rhythms of activity. Travelling athletes may also have difficulty finding access to palatable foods that are typically included in their usual diet. Thus, appropriate timing of meals may be more important than the energy content of the meal [26, 91]. Small meals before and during flights are better tolerated than large meals [92, 93]. Food served on the airplanes generally is not adequate for athletes but on majority of the airlines it is possible to choose amongst several meals (vegetarian, kosher, Muslim, etc.), so it is possible to choose the one that is closest to what athlete needs and take missing nutrients on board as pre-prepared snacks.

Intestinal gases expand with increase in altitude, which can lead to bloating and pain [94]. This problem is exacerbated by drinking soda or carbonated beverages because the fizz creates more volume. Additionally, although chewing gum or sucking on candy may help relieve ear pressure, both habits can also contribute to bloating. Besides avoiding the food that cause bloating (e.g. legumes), water here plays a pivotal role as remaining hydrated will ensure that the digestive process continues to flow smoothly and help prevent constipation.

Unavoidable noise on board can be cause of fatigue too. Exposure to air travel noise can cause sleep disruption, increase stress and raise blood pressure [95]. Athletes should invest in custom-made ear plugs or noise-cancelling headphones to add a little more peace to their travel.

Moderate- and high-intensity exercise can cause a temporary decrease in immunoglobulins (IgA and IgM) in mucosal secretions [96, 97]. Same can happen after long-haul travel. Although these changes are disputed as direct cause of illness, this immunological change together with dry air in airplane and crowed environment could be associated with an increased risk of respiratory illness. A simple bout of flu at new destination can ruin months of dedicated training.

Some simple things may reduce health problems on new destination [28, 98]:

- Avoid overstraining with intensive training 2 days before the trip.
- When organising the trip, include at least 1 day of rest after arrival.
- Exercise should be light or moderate in intensity for the first few days in the new time zone, because training hard, while muscle strength and other measures are impaired, will not be effective.

- Timing of the endurance training on arrival should not coincide with the circadian nadir 2–4 p.m. and 2–4 a.m. until full adaptation is reached.
- Avoid alcohol or caffeine which act as diuretics and can add to fluid losses.
- Sipping water with electrolytes consistently throughout the day will ensure that the body maintain a balance.
- If possible, make arrangements for dietary selections that are optimal for individual performance. While travelling, eat smaller meals before and during flight; and, upon arrival, time meals to match habits appropriate to the destination.
- Minimise the use of electronic devices.
- Use a neck pillow, eye mask, earplugs and/or noise-cancelling headphones.
- Wear comfortable, loose fitting clothing to aid rest and relaxation and prevent overstimulation.
- Restrain from going out for training if the conditions are too heavy or the athletes are not perfectly well and fully recovered.

Fatigue and overstraining may relate to other health problems. Athletes should never be sent out on training session or competition event if they are not well or are recovering from flu, cold, diarrhoea or other infectious diseases. One should wait a few days until fully recovered and only then can continue safely with the planned activities.

16.8 Water Pollution

Unfortunately, pollution is our reality and triathlon events are often held on racing fields in the major urban areas, not always with appropriate sewage water plants. Near major towns, one should expect a high level of *E. coli bacteria* and potentially harmful chemicals in the water [99, 100]. Infection may result from ingestion or inhalation or contact with harmful microorganisms which may be naturally present, can be carried by people or animals using the water or present as a result of faecal contamination. The most common consequences are diarrhoeal disease, acute febrile respiratory disease, ear infections and infection of the skin lesions [101–103]. Waters in some triathlon areas can also hide leptospirosis bacteria, potentially dangerous algae and other harmful sea organisms like jellyfish.

Athletes should

- Try not to swallow water.
- Wash their eyes and face with available clean water after completing the swimming leg.
- Not sit in wet clothes after completing the race or training.
- Not wash any scratches or grazes with seawater (use clean freshwater).
- Apply antiseptic to cuts and abrasions after washing after the competition/training session.

- Take antibiotics if inflammation develops.
- Take care to minimise contact with polluted water by wearing adequate garment (if allowed, or wear it on training). Freshwater lakes or rivers may additionally harbour pathogens such as schistosomiasis (blood fluke), leptospirosis and amoeba—try not to swallow the water, wear eye protection and dry off quickly to minimise the risk of pathogens entering via the skin, mouth, nose or eyes.
- Clean their garments regularly, after the triathlon event or training session.
- Obtain local advice on the possible presence of dangerous aquatic animals in the area.
- Seek medical advice for
  - Eye discharge (conjunctivitis)
  - Ear infection
  - Red (inflamed) skin or boils
  - Diarrhoea
  - Fever
- Take a shower immediately on return according to the following procedure described below.

16.8.1 Procedures to Reduce Infection [104]

On the training brake at the new destination∗:

- Rub hands and forearms with alcohol-based disinfectant for 3 min, including
  - Both hands, then both forearms and then both hands again for a total of 30 s.
  - Repeat the same 30 s procedure five times, each time with new disinfectant.
- Wait for the hands to dry fully before eating or drinking.
- Rinse your mouth with mouth wash (containing 0.05% chlorhexidine digluconate or *octenidine dihydrochloride phenoxyethanol or* other disinfectant) before eating or drinking.

∗During transitions there will be no time to apply this procedure
After the race or training session:

- Use the water hose to shower immediately on return from the race.
- Your recovery procedure may require you to drink and eat immediately after triathlon. Before you do so, wash your hands with liquid soap and water for 60 s and dry them with paper towels.
- Prior to eating and drinking, rinse your mouth with the mouth wash (containing 0.05% chlorhexidine digluconate or *octenidine dihydrochloride phenoxyethanol or* other disinfectant).
- Wash your *tri-suit* with soap before storing. If competing (training) in heavily polluted water, disinfect it by soaking it in disinfecting solution (e.g. 30% 8 g *glutaral-*

dehyde/3 g *didecyldimethylammonium chloride*/6 g *benzalkonium chloride* solution) for 2 h, then rinse through thoroughly with water before drying and storing.

- Shower thoroughly with liquid soap before changing to dry clothes.

Washing hands properly is the most important measure to avoid the transmission of harmful germs and prevent the infections

16.8.2 Leptospirosis

Leptospirosis caused by *L. interrogans* is found worldwide. Signs and symptoms can range from none to mild such as headaches, muscle pains and fevers to severe, with bleeding from the lungs or meningitis [105]. It is endemic in the tropics, where the climate and soil conditions are favourable for leptospiral proliferation and where animal reservoirs are abundant. Outbreaks may be precipitated by heavy rains and flooding in endemic areas but my also occur in temperate climates during warmer months [105, 106]. Exposure to contaminated water may occur in triathletes [106–109]. Humans become infected through contact with water, food or soil that contains urine from these infected animals. This may happen by swallowing contaminated food or water or through skin contact. Triathlon athletes are at risk in areas that have been shown to contain these bacteria and can contract the disease by swallowing contaminated water, splashing contaminated water into their eyes or nose or exposing open wounds to infected water. As prolonged immersion in water promotes the entry of this bacterium, ingestion of contaminated water during the swimming leg in triathlon is predominant risk of acquiring a disease [109, 110].

Penicillin and doxycycline are recommended for treatment but only doxycycline has demonstrated efficacy as a primary prophylactic agent for leptospirosis. Taken 200 mg/weekly, it shoved efficacy of 95% over 3-week period [111]. As adverse effects of doxycycline include phototoxicity, one should concomitantly use sunscreen preparations and ample fluids with each dose to minimise gastrointestinal disturbances.

In the case of appropriate exposure and appearance of symptoms of leptospirosis while in the sport camp without medical support abroad, self-treatment is an option. Doxycycline in the dose of 100 mg twice daily for 7 days should be started [112].

16.9 Athletes Security Abroad

Of almost 500,000 homicides each year, more than 90% occur in developing countries. Ninety percent of civilian casualties are caused by small arms, and interpersonal violence is a significant risk in many developed countries too [113, 114]. Crime risks fluctuate in tune with economic situation and terrorism is constant wary. Pretravel advice to each team should include not only information about risks of acquiring disease in the places that will be visited but also security information. That type of advice can be obtained from government agencies, departments of foreign affairs or private agencies that are providing such a service [115].

One must:

- Dress inconspicuously (team colours in some countries can make one a target!).
- Look confident—don't look lost.
- Leave expensive jewellery and watches at home.
- Avoid night or solo travel.

- Avoid to flash money.
- Use alcohol in moderation.
- Leave the scene if feeling threatened by the mood and tone set by another people's behaviour.
- Know where the fire escape is—hotel fires are too common.
- If lift looks unsafe, probably it is.
- Remove obvious rental car markings.
- Know the local police number.
- Know where the high-risk areas are.
- Avoid using mobile phone on the street while walking—stop and put back to the wall.
- If mugged, comply; mugging is "economic transaction".

16.10 Dangers of Transportation

Motor vehicle accidents are the leading cause of accidental deaths of long-term travellers living in the third world. Significant portion of those deaths can be attributed to accidents involving motorcycles [116, 117]. The possibility for being injured in a car accident while in foreign towns is the most reason of injury to all travellers abroad, including athletes. This has to be stressed.

Unfamiliarity with road signs, local customs and driving habits and especially driving on the "wrong side of the road" are hazards to drivers and pedestrians alike. Simply learning how to say "slow down" to the taxi driver can save a life. In countries where "rules of the road" are not enforced or are non-existent, there are several common-sense recommendations which should be followed [118, 119]:

- Secure reliable local transport before arrival.
- Use a driver (taxi)—look for reputable company.
- Hire a large vehicle if possible.
- When renting a car, check for seatbelts, good tires and brakes.
- Always wear a seatbelt even if not compulsory by law.
- Be aware of possible car-jacking hotspots and keep the doors locked.
- Avoid riding on motorcycles.
- Avoid driving under the influence of alcohol or jet lag.
- Avoid rural travel by road after dark.
- Avoid overcrowded, overweight or top-heavy buses or vans.
- Carry a cell phone and know how to obtain emergency help in case of accident.

Although developed to provide medical help to the general population, the approach, the systems and the doctrine of *travel medicine* can help every athlete to solve some of the travel-related problems. This approach formatted as medical guidelines can be effectively used in the training of athletes in the form of health manuals and easily administered as a part of pre-competition service given to the athletes in training camps or sport events.

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Nutrition Strategies for Triathlon

17

Louise M. Burke and Gregory R. Cox

17.1 Introduction

Over the past 60 years, sports nutrition has evolved from a collection of anecdotes about the dietary strategies of successful athletes, supplemented by laboratory-based studies typically involving recreational competitors, into a credible science underpinning the health and performance of all athletes [1]. The continual updating of consensus statements and position stands by expert groups on global areas of sports nutrition [2], as well as individual topics within its umbrella [3–5], illustrates the pace with which new information is evolving. The specific nutrition needs and practices of importance to athletes, even within a single sport, are diverse. However, all athletes share some common goals: to train as hard as possible with optimal adaptation and recovery, to remain healthy and injury free, to achieve a physique that is suited to their event and to perform at their best on the day(s) of peak competitions.

This chapter will present a summary of eight key themes in contemporary sports nutrition with an examination of their relevance and implementation within triathlon.

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17.2 Theme 1: Nutrition Goals and Requirements Are Neither Static Nor Universal

Early position statements and reviews on sports nutrition provided quantitative recommendations for nutrient targets for all athletes [6], focusing on single issues (e.g. daily replacement of glycogen) and apparently addressing all training phases and subpopulations of athletes with the same advice. These recommendations have gradually evolved to recognise that macronutrient targets should not be set in absolute amounts or as ratios of energy intake; instead, guidelines should encompass the differences in body sizes, training loads and energy goals across athlete populations [7, 8]. More recently, however, there has been specific attention to the need for each athlete to develop a personalised, periodised and practical eating plan that supports their unique and changing needs. Although more detail around these principles will be provided subsequently, it is important to highlight a key insight that underpins these features.

Contemporary sports nutrition guidelines embrace the philosophy of periodisation that governs the athlete's annual training plans [9]. Although various models of training periodisation exist, the fundamental theory is that exercise sessions should be strategically manipulated (mode, frequency, intensity and duration) within a sequence of cycles to gradually achieve the attributes (e.g. physical, biomechanical, physiological, neuromuscular, psychological) needed for success at chosen competitions. Periodised nutrition involves the organisation of nutrient intakes and dietary strategies around each session, microcycle (e.g. week), mesocycle (e.g. month) and macrocycle (e.g. yearly plan) of training to help address the individual athlete's gaps to achieving these event-specific attributes. The diversity and complexity of the different attributes for success, even within a single sport, means that many aspects of nutrition may change or differ between athletes.

17.2.1 Application to Triathlon

Although triathlon is considered an endurance sport, it encompasses a range of events from 20 min to 8 h + in duration, with different requirements for success [10]. Specific preparation for each race format will require different training programmes, protocols and goals, leading to overall differences between event specialists or changes across the season in the same triathlete as they prepare for different races. Even within a triathlete's training programme, daily or weekly fluctuations in training load and goals should be matched by changes in energy and nutrient intake. Furthermore, specialised nutrition strategies that optimise performance or adaptation or mimic race-day practices should be integrated around key training sessions or training phases. Table 17.1 provides some examples of nutrients and nutritional strategies that may be periodised within different programmes commonly undertaken by triathletes. The deliberate implementation of this targeted and individualised approach to sports nutrition is a highly specialised task that should involve the input of the triathlete, coach, sports scientists and sports nutrition expert,

Table 17.1 Examples of the periodisation of nutrient intakes and dietary strategies for triathletes

Nutrient/ issue	Background	Scenarios of training cycle or daily periodisation	Scenarios of within-day periodisation
Energy	Key determinants of energy needs include BM and the training load. Energy intake may also need to be manipulated to support growth or targeted changes in body composition	• Energy intake should be increased on days of heavier training load or during phases of high-volume training	• It is likely that distributing energy over the day to support training sessions will lead to better metabolic outcomes than irregular or concentrated (e.g. evening-focussed) energy
CHO	CHO provides a key fuel for the muscle and brain/central nervous system. Muscle glycogen also acts as a regulator of many metabolic processes and responses to exercise Daily CHO intakes should vary according to the exercise workload and the goals of the session. Examples of possible daily targets include 3–5 g/kg BM: light training 5–7 g/kg BM: higher workload training 6–10 g/kg BM: heavy workload or desire to restore/load glycogen 8–12 g/kg: very high workload or maximal glycogen supercompensation Consideration to the triathlete's body composition should be given when interpreting these guidelines	• CHO intake should be generally increased during periods of high-volume training • CHO intake should be increased on days of heavy training load or key training sessions where high-quality/intensity outputs are required • CHO intake should be increased on the day before heavy training or to "load" for longer races to ensure appropriate muscle glycogen stores • CHO intake may be decreased during periods or on days with "light" training • CHO intake may be avoided/decreased around occasions when there is a desire to exercise or recover with low CHO availability	• When there is a desire to "train low", the triathlete can refrain from consuming CHO before and during a morning training session or in the interval between two training sessions • When there is a desire to "train high" or race well fuelled, the triathlete can consume CHO (1–4 g/kg) in the hours prior to an exercise session, especially in the morning after an overnight fast • When there is a desire to "train high" or race well fuelled, the triathlete should consume CHO during the exercise session, with targets ranging from 30 to 60 g/h for sessions 75–150 min to maximal targets of 60–90 g/h for prolonged races and "gut training" • When rapid recovery of muscle glycogen is required between exercise sessions, the triathlete should commence CHO intake soon after the first session, targeting an intake of ~1 g/kg/h until normal eating patterns are resumed

(continued)

Table 17.1 (continued)

Nutrient/ issue	Background	Scenarios of training cycle or daily periodisation	Scenarios of within-day periodisation
Protein	Dietary protein intake interacts with exercise to promote whole body protein synthesis, including targeted muscle proteins. Protein needs of athletes in focused training are greater than those of sedentary people	• Triathletes in committed training should aim for daily protein targets of at least 1.4–1.7 g/kg • Protein targets should be increased to 1.8–2.4 g/kg/day to preserve lean mass during periods of active loss of body fat/mass	• Protein intake should be distributed over the day at 3–5 meals/snacks, each providing at least 0.3–0.4 g/kg BM • When there is active promotion of lean mass gain or adaptive response to exercise, it may be useful to add a pre-bed protein-rich snack • The target for each eating occasion should be increased to 0.4–0.5 g/kg in scenarios of active fat/mass loss or when the meal is large and likely to have delayed absorption
Water	Daily water intake from drinks and water-rich foods must cover obligate losses (e.g. urine production and respiratory water losses). The major determinant of fluid needs for athletes is daily sweat losses associated with exercise or environmental conditions	• Fluid needs are increased in warm weather, at altitude or during periods of heavy training associated with high sweat losses • Although thirst may generally guide daily fluid intake, it is useful to check fluid status (urine characteristics, acute changes in BM) when there is a change in characteristics around fluid balance	• Hyperhydration before exercise may help to offset large sweat losses which can't be replaced during the session; this may assist race performance in extreme heat • An individualised and adjustable fluid plan during exercise should aim to keep dehydration to acceptable levels by balancing opportunities to drink with sweat losses • There may be some advantages to periodic training with a deliberate (moderate) fluid deficit to enhance acclimatisation processes • Proactive rehydration including replacement of adequate fluid volume (~125% of the deficit) and electrolyte losses may be useful in some situations of recovery • It may be counter-productive to attempt full rehydration after evening training sessions if the need to urinate during the night interferes with sleep

Iron	Iron requirements are increased in athletes, particularly females and endurance athletes to accommodate increased iron losses and increased needs for synthesis of new tissues and iron-containing compounds Strenuous exercise interacts with the iron regulating hormone, hepcidin, to reduce iron absorption and recycling	• Iron requirements may be increased during periods of high training volume • Iron status should be checked and supported during periods of altitude training to ensure that erythropoietic responses are optimised	• To maximise iron absorption, iron-rich meals should be consumed at times in the day that best avoid the interference associated with post-exercise increases in hepcidin
Caffeine	Caffeine enhances exercise capacity and performance, especially to mask fatigue and perception of effort	• Race-day caffeine strategies should be practised in training to identify successful protocols • Caffeine intake may assist the performance of fatiguing training sessions, allowing the triathlete to train harder; this may be most useful for sessions involving greater metabolic stress (e.g. “train low”)	• Daily rituals of “social” intake of caffeine may be organised to contribute to performance uses of caffeine • Caffeine intake over the day should be organised to avoid negative effects on sleep

with continual fine-tuning according to feedback and experience. Menu plans and nutrition strategies should also take into account an individual's food preferences, cultural eating practices and special dietary needs including food restrictions and intolerances, as well as food availability in the athlete's environment which is ever changing with the global nature of triathlon racing.

17.3 Theme 2: Energy Availability Sets an Important Foundation for Health and Performance Success

Energy intake is the foundation of an athlete's diet since it supports optimal body function, determines the capacity for intake of macronutrient and micronutrients and assists in manipulating body composition. Conventionally, nutritionists have focused on energy balance and how each side of the energy equation (dietary energy intake vs. total daily energy expenditure) can be manipulated to store or utilise body fat and protein and thus achieve desirable changes in physique (e.g. gain in muscle mass, loss of body mass [BM]/body fat). However, energy availability (EA) is a newer concept in sports nutrition which examines the energy that is left to address the body's many non-exercise needs once the energy cost of training and competition is removed [11]. EA is defined operationally as dietary intake minus exercise energy expenditure, normalised to the body's fat free mass (FFM) which represents the most metabolically active tissues:

- EA = (energy intake = exercise energy expenditure)/FFM [11].

It is self-evident that the energy that is committed to an athlete's exercise programme is not available for other functions. Importantly, if a mismatch between energy intake and exercise load leads to a reduction in EA, the body adapts to conserve energy by reducing expenditure on non-exercise functions. There is sophisticated laboratory evidence that when EA is decreased from a "healthy" level (~45 kcal/kg FFM or 189 kJ/kg FFM) below a threshold (typically set at ~30 kcal/125 kJ/kg FFM), many hormonal and metabolic processes are impaired by such energy conservation [12]. Low EA (LEA) underpins the problems of impaired bone health and menstrual function identified in the female athlete triad syndrome [13], but new insights over the last decade have identified its occurrence in male athletes and its impact on a range of other body systems and performance factors [14]. Indeed, the concept of relative energy deficiency in sport (RED-S) was developed to address this expanded concern, including the sequelae of functional hypothalamic amenorrhea (females), reduced testosterone levels and libido (males), poor bone health, increased risk of illness and injuries, gastrointestinal disturbances and cardiovascular disease and impairments of haematological status, training capacity and performance [5, 14]. The main causes of LEA are disordered eating, misguided weight loss programmes and inadvertent failure to recognise or address a high-energy expenditure associated with training/competition [11]. Although general risk factors for these issues can be identified in various sports (e.g. weight-sensitive

and weight category sports) or sporting scenarios (e.g. sudden increase in training load, change in food environment), it appears that other sport- and culture-specific characteristics contribute both to the development of LEA and the manifestation of its effects on health and performance [14]. Programmes that promote prevention and screening/early intervention are important to prevent long-term secondary health consequences. The primary treatment is to increase EA and often requires a team approach including a sports physician, sports dietitian, physiologist and psychologist.

17.3.1 Application to Triathlon

Triathlon involves a number of risk factors for the development of LEA as well as the progression of impaired body function into tangible problems around health and performance. Given the long gestation from junior ranks to elite race winner (typically late 20s), a significant loss of time to injury and illness will delay and, in many instances, prevent a junior triathlete from developing into an elite-level performer. The heavy training loads required to master three different sports, and the preparation for the longer events within triathlon, often tally 20–30 h per week. Both the energy expenditure associated with such a programme and the significant number of hours committed to training and recovery activities create a challenge in meeting energy needs within the available opportunities. Athletes should be vigilant to capture opportunities before, during and after training to replenish energy needs and to manipulate food and fluid selections to reflect training schedules. The desire to be as light and lean, often misguided in relation to both the goals and time frame of achieving/maintaining them, adds a separate contribution to the development of LEA and RED-S, which is discussed subsequently.

17.4 Theme 3: The Achievement of the Body Composition Associated with Optimal Performance Is Challenging and Requires a Long-Term Plan

Various attributes of physique (body size, shape and composition) are considered to contribute to success in various sports. The assessment and manipulation of some features (e.g. BM, body fat, lean mass) may assist in the progression of an athletic career. High-performance endurance athletes are typically light and lean as a result of the genetic factors that have predisposed them to being successful in their sport as well as the modifying effects of training and diet. However, the specific muscular strength and power required in many sports may require targeted gains in lean mass. BM and composition can be assessed via a number of techniques, with surface anthropometry (e.g. measurements of girths, breadths and subcutaneous [skinfold] fat) being a popular field technique and dual X-ray absorptiometry (DXA) being a technological tool of increasing popularity [15, 16]. Both techniques can provide valuable information when they are used, according to standardised techniques by

trained personnel, to track changes over time in the same athletes. The interpretation of these assessments should always account for the precision of the technique, while goals around manipulating body fat/lean mass should consider what is optimal for an individual athlete's long-term health and performance goals rather than rigid prescriptions of an "ideal" formula for a specific event. Effective strategies for loss of BM/fat should achieve an energy deficit that avoids unnecessary restriction of EA and takes advantage of the meal satiety and lean mass preservation associated with a higher protein intake [17]. Gains in lean mass are achieved by an appropriate training programme, supported by the spread and total intake of energy and protein [18]. Unfortunately, many athletes set unreasonable goals and methods to achieve more rapid results, leading to compromises of health and performance.

A more successful approach to management of body composition is to achieve periodisation over the yearly training plan, using the experience gained over an athletic career to permit gradual refinement of ranges that are best suited to the individual athlete. Indeed, this periodised approach was well illustrated in a recently published 9-year case history of a female Olympic middle-distance runner [19]. During the general preparation phase of each season, optimal EA was prioritised, with the athlete being ~2–4% over her ideal "race weight" and body fat levels. Optimisation of body composition prior to each racing season included the implementation of an individualised energy deficit over a specific timeframe, using various metrics (BM, performance and hunger) to guide the process. This approach targeted peak performance at identified times while minimising injury risk and maximising training adaptation and long-term athlete health through management of EA.

17.4.1 Application to Triathlon

Junior development and age-group triathletes are often misguided in their desire to achieve rapid shifts in "leanness" and enthusiastically employ strategies that target reductions in fat mass without considering the larger implications around diet quality and overall health. Reducing energy intake to drive a reduction in fat mass will likely reduce EA with the risk of contributing to health and performance concerns associated with RED-S [4]. Restricted energy intakes or extreme diets may also lead to a suboptimal intake of nutrients needed for health and adaptation. The disadvantages of unsound weight/fat loss strategies may manifest in terms of increased prevalence of illness an injury and as an inability to train consistently or to gain the expected adaptations and performance improvements. The concept of improving performance through the optimisation of the "power-to-weight" ratio is well known in triathlon, but many triathletes fail to realise that a gain in lean mass can also assist with this goal and is a hallmark of developing into an elite performer. In essence, triathletes should adopt eating strategies that support daily training outcomes as well as promote increases in protein synthesis to achieve favourable body composition changes that align with world-class performances.

17.5 Theme 4: Guidelines for Nutrition Need to Consider Training Load, Body Size and Timing of Intake

Although recommended dietary intakes for the general population are typically provided as simplistic (absolute) nutrient targets or ranges, the specific needs of athletes merit greater consideration particularly to account for the variability imposed by their exercise demands and the potential importance of the timing of nutrient intake in relation to an exercise session. The complexity of contemporary guidelines for the carbohydrate (CHO) and protein intakes of endurance athletes is well illustrated by their application to the needs of triathlon.

CHO is an important dietary nutrient since body stores are relatively limited and can be acutely manipulated by daily intake and even a single training session within a triathlete's programme [20]. CHO is a key fuel for the brain and central nervous system and provides a versatile muscle substrate because it can support exercise over the large range of intensities seen in a triathlon due to its utilisation by both anaerobic and oxidative pathways [20]. There is robust evidence that the performance of prolonged sustained or intermittent high-intensity exercise is enhanced by strategies that maintain high CHO availability, defined as matching endogenous (e.g. muscle glycogen) and exogenous (e.g. CHO consumed during exercise) sources to the fuel demands of exercise [21, 22]. Meanwhile, depletion of these stores is associated with fatigue in the form of reduced work rates, impaired skill and concentration and increased perception of effort [20–22]. These findings underpin various race nutrition strategies, discussed subsequently, that supply CHO before, during and in the recovery between events to enhance CHO availability but should also be extended to training scenarios where high-quality outputs or race simulation is desired.

Guidelines for daily CHO intake now target the variable fuel costs of training [8] as well as the importance of high CHO availability for performance and support for other body systems, such as immune [23] and, potentially, bone [24] health. Although the actual substrate cost of typical training sessions undertaken by endurance athletes is (surprisingly) poorly characterised [25], some general ranges of CHO intake are suggested according to parameters that account for the workload (e.g. intensity, duration of the session) and the triathlete's BM (as a proxy for the size of the working muscle). Table 17.1 includes a summary of guidelines for various scenarios in which there may be benefits to achieving high CHO availability, providing daily targets which can be fine-tuned according to individual experience and noting that the timing of intake of CHO in relation to the exercise session plays a role in manipulating CHO availability [8, 21]. Integration of these guidelines into a race plan is summarised in Table 17.2 and discussed in more detail within a subsequent theme. It is important also to note that many training scenarios do not require promotion of CHO availability or intake, allowing the triathlete to manipulate their food choices to meet other goals. Furthermore, as discussed subsequently, benefits from deliberate strategies to undertake training sessions with low CHO availability may also be possible. Thus, CHO intakes may vary significantly between athletes and from day to day in the same athlete [21].

Table 17.2 Nutritional strategies for high-performance athletes in key triathlon races

Event and typical finish times of competitive triathletes	Mixed relay (~20–25 min)	Sprint (~55–75 min)	Olympic (1:45–2:15 h:min)	Half ironman (3:30–4:30 h:min)	Ironman (8:00–10:00)
Pre-race refuelling: • Normalisation of glycogen = 7–12 g/kg/day for 24 h • CHO loading = 10–12 g/kg/day for 36–48 h	Glycogen normalisation	Glycogen normalisation	Glycogen normalisation	CHO loading, while maintaining typical fibre intake	CHO loading, especially with low-residue diet
Pre-race meal • 1–4 g/kg CHO in 1–4 h pre-race • Reduced fat, fibre and protein according to risk of gut issues	Familiar pre-race meal + CHO after warm-up	Familiar pre-race meal + CHO after warm-up	Familiar pre-race meal + CHO after warm-up	Familiar pre-race meal + CHO after warm-up	Familiar pre-race meal + CHO after warm-up
Opportunities for in-race nutrition: Includes race feed zones and participant's own supplies	Nil—not needed		Transition areas Participants can carry bike-mounted bottles and sports foods	Transition areas Participants can carry bike-mounted bottles and sports foods Feed zones are provided on bike and run course	Transition areas Participants can carry bike-mounted bottles and sports foods Feed zones are provided on bike and run course
In race fuelling goals • 45–75 min: mouth rinse/small CHO amount • 1–2.5 h: 30–60 g/h • >2.5 h: up to 90 g/h	N/A	N/A	Trial CHO mouth rinse up to intake of 30–60 g from CHO drinks or gels/confectionery	30–60 g/h CHO; consider trialling intakes up to 90 g/h using CHO drinks and concentrated gels/confectionery	Target intakes up to 90 g/h using CHO drinks, gels/confectionery and real foods on bike. Run leg may have fewer practical choices and need lower targets

In race hydration goals • Aim to keep net fluid deficit <2–3% BM, especially in hot weather	N/A	N/A	Cost: benefit analysis may show that time cost of drinking larger volumes may negate benefits in elite triathletes	Plentiful opportunities for frequent small intakes of CHO drinks towards race fluid plan	Plentiful opportunities for frequent small intakes of CHO drinks towards race fluid plan
Special issues for hot-weather events Pre-cooling and hyperhydration may be added to more aggressive fluid plans	Consider pre-race pre-cooling with ice slurry in addition to external cooling strategies if significant thermal challenge is anticipated		Consider pre-race pre-cooling with ice slurry in addition to external cooling strategies if significant thermal challenge is anticipated Consider pre-race hyperhydration if large fluid deficit is anticipated		
		Where possible, adjust fluid intake during event, independently of CHO intake, in view of increased sweat losses			
Special comments for non-elite competitors		Do not overdrink by consuming fluid in excess of sweat losses			

Despite earlier debate, contemporary perspectives on protein intake for athletes have moved beyond the population guidelines, typically set at 0.8–1.0 g/kg BM in most countries, both in terms of the total daily target as well as the advice around how it should be consumed over the day. Allowances which target the absence of protein insufficiency in largely sedentary populations do not address the needs of high-performance triathletes who need to optimise the adaptive response to training and to achieve the physique attributes of lean-mass-to-body-fat ratio needed for successful performance in their events [26]. Dietary protein interacts with exercise, providing both a trigger and a substrate for the synthesis of contractile and metabolic proteins in the muscle [27, 28], as well as enhancing changes in non-muscle tissues such as tendons and bones. Planned intake of dietary protein can enhance the sustained (~24 h) increase in contraction-stimulated synthesis of muscle protein that occurs after strength or endurance exercise, by supplying leucine to further upregulate the mammalian target of rapamycin complex 1 (mTORC1) pathway [27, 28] as well as the amino acid building blocks for the synthesis of new proteins. High-quality leucine-rich protein sources, when consumed in amounts equivalent to ~0.3–0.4 g/kg of rapidly digested protein at 4–5 eating occasions per day, can optimise the training response when athletes are in optimal EA [18]. Meanwhile, a goal of 0.4–0.5 g/kg might be required in scenarios of energy deficit/weight loss in which rates of muscle protein synthesis are suppressed [18] or in the case of mixed meals with slower protein digestion/absorption kinetics [18, 29]. Overall daily targets of 1.3–1.7 g/kg/day are likely to meet the physique and adaptation goals of weight-stable triathletes. Meanwhile, triathletes who wish to achieve effective weight loss, which promotes the retention or even an increase in lean mass, are advised to engage in resistance exercise and to consume dietary protein in quantities of 1.6–2.4 g/kg [18]. Table 17.1 includes a summary of the current recommendations for protein intakes for high-performance athletes according to their major goals.

17.5.1 Application to Triathlon

A high daily training commitment requires the triathlete to incorporate strategies that promote optimal performance in the current session while simultaneously facilitating recovery from the previous one. While heavy training loads accentuate this challenge for the elite competitor, recreational triathletes have the added complexity of managing their specific nutritional needs around other commitments such as work, family and study. Age-group triathletes typically "sandwich" their training into concentrated weekend blocks often incorporating two or three disciplines into the one session (i.e. swim + cycle or cycle + run). This situation highlights the importance of athletes being well planned to have suitable foods and fluids on hand, which cater to the practical considerations as well as their desire to eat.

The requirements for CHO and protein on heavy training load days contrast with those of a sedentary work or rest days, necessitating vastly different eating patterns and food/fluid choices. For training sessions that reflect the demands of racing, additional CHO intake can be achieved via pretraining snacks based on familiar and

well-tolerated CHO-rich foods and fluids; in addition to increasing overall intake, this strategy specifically promotes high CHO availability and performance benefits for these key sessions. As will be discussed in Theme 6, there are different benefits to undertaking lower-intensity (aerobic) sessions with strategies that achieve low CHO availability, such as training fasted or withholding CHO early in a session. However, this strategy also decreases total daily nutrient/energy intake unless the triathlete can schedule additional snacks and/or increases in meal size to offset the energy reduction. Such dietary approaches should be incorporated with the assistance of expert dietary input from a skilled sports nutrition professional.

During extended training sessions (i.e. long training runs or cycling sessions or combination "brick" sessions), there may be benefits to consuming food/fluid choices that provide protein to assist in the maintenance of positive protein balance and promotion of bone remodelling [30, 31] as well as CHO for fuelling. When sessions are performed below race intensity, athletes typically have improved tolerance for wholesome, nutrient-rich foods. While these food options might not be suitable for racing, inclusion in training will support favourable adaptations to training while better supporting health and well-being.

17.6 Theme 5: Competition Nutrition Strategies Target the Limiting Factors in Optimal Performance

Competition requires the athlete to perform at his or her best, or at least at a better level relative to other competitors. This goal rightly focusses attention to the factors that can cause fatigue at various timepoints throughout and especially in the latter stages of a race, where fatigue is defined operationally as a loss of the work outputs (e.g. speed, power) and skill/technical prowess that otherwise underpin success. The causes of fatigue are specific to the race, the environment/scenario in which it is undertaken and the individual athlete and include depletion of muscle glycogen fuel, dehydration and electrolyte imbalances, hypoglycaemia and suboptimal central nervous system fuelling, gut discomfort/upset and disturbances to acid–base balance. Dietary practices in preparation for and during a race can reduce or delay the onset of these fatigue factors.

17.6.1 Application to Triathlon

With total race distances ranging from 20 min to 8+ h for top competitors, individual segments of each race ranging from 4 min to 4 h and environments scaling from cold water to extreme heat on bike and run courses, race nutrition in triathlon must tackle a variety of issues. Race preparation should include strategies to store muscle glycogen in the amounts that suit the fuel needs of the event. For shorter races (Olympic distance and below), where the fuel cost is spread across different muscle groups, it is probably sufficient to normalise the superior glycogen concentrations associated with endurance training; this is typically achieved with CHO intakes of 7–10 g/kg

BM for 24 h [8]. For long-distance/half Ironman and above, where glycogen may become limiting for race performance, protocols which supercompensate glycogen are beneficial. The contemporary CHO loading protocol is an abbreviated version of the original, involving 36–48 h of CHO intakes targeting 10–12 g/kg/day [8, 32]. This is often undertaken in conjunction with a low-residue (fibre) diet, which may reduce the risk of gut issues during the race but also achieve a small reduction in BM to partially offset the mass of the additional muscle glycogen and stored water [32]. Further enhancement of fuel availability is provided by a CHO-rich pre-race meal and CHO-rich snack during/after the race warm-up, noting that is impractical to consume further supplies during the swim leg of a race. Pre-race CHO intake is important for morning events where it can restore liver glycogen following an overnight fast as well as provide an ongoing supply of CHO from the gut during the first part of the race [32]. Event characteristics and individual preferences will dictate the amount, type and timing of pre-race intake; this should be well practised to develop a personalised protocol. Athletes should also consider fluid needs to achieve optimal hydration status for the race and its specific environmental conditions.

Many triathlons offer an opportunity for athletes to consume fluid and CHO during the race to further address physiological limitations around these factors (Table 17.2). CHO ingestion during longer-distance events (e.g. long distance and above) can maintain high rates of CHO oxidation in the face of dwindling endogenous stores [33], with a systematic review of studies of endurance protocols showing substantial benefits to performance [22]. Whereas older guidelines [6] recommended that distance athlete experiment with hourly CHO intakes within the range of 30–60 g to find a beneficial strategy, contemporary recommendations now suggest a sliding scale of intakes with smaller amounts for shorter duration events and higher rates of intake for longer distance and above [8]. These new guidelines are based on the mechanism of likely benefits to performance as well as the recognition that athletes can learn to tolerate and utilise substantially higher amounts than previously considered possible. Although the intestinal absorption of CHO consumed during exercise, plateauing at ~60 g/h, was thought to limit the contribution of exogenous CHO to muscle fuel use during exercise [34], current advice is that intakes of 90 g/h and even higher can be of benefit to very long races (e.g. Ironman). A combination of the use of mixtures of CHO using different absorption routes (e.g. fructose and glucose) and enhancement of gut tolerance and absorption [34, 35], possibly involving the upregulation of the sodium-dependent glucose transporter (SGLT1), with chronic "gut training" [34] can increase the muscle CHO supply for better performance.

However, even in shorter events in which muscle glycogen stores are not challenged, performance benefits may be associated with intake of smaller amounts of CHO. This is now known to be a central nervous system activation associated simply with mouth exposure to CHO (the so-called mouth rinsing effect). Indeed, there is robust evidence that the detection of CHO by receptors in the oral cavity activates centres in the brain which enhance perceptions of effort and pacing decisions [36]. Table 17.2 summarises the suggested approaches to CHO supply in various events, noting both the targets and the opportunities to achieve these during transitions and

the cycle and run legs. A range of sports drinks, gels, confectionery and whole foods can be used to meet various targets, both in training and racing, around taste, practicality, fluid vs. CHO requirements, use of transportable CHO sources, electrolyte replacement and supplementation with caffeine. Access to these supplies and the practicalities of consuming them vary according to the mode of exercise (e.g. the cycle leg is more accommodating than the run), the event organisation (e.g. placement of feed zones) and race tactics.

Meanwhile, there is interest in nutritional strategies including chronic or periodised exposure to high-fat, low CHO (LCHF) diets that may allow triathletes in ultra-endurance (Ironman and above) events to increase their ability to oxidise fat as a muscle fuel in view of its relatively unlimited pool size and capacity to support exercise at intensities up to ~75–80% VO_{2peak} [37]. However, although targeted adaptation to a high-fat diet with CHO restriction is associated with very high rates of fat utilisation across a range of exercise intensities, this comes at a cost of a greater oxygen demand during exercise (lower speed for a given oxygen supply or greater oxygen requirement for the same speed) [38] as well as a downregulation of the capacity of CHO oxidation pathways [39]. Such adaptations have been shown to impair performance of shorter endurance races [38], or selected segments within a longer race conducted at higher exercise intensities (>80–85% VO_{2peak}) [40], probably limiting the utility of LCHF diets to selected individuals, events or scenarios [41]. Modelling of the total demands and rates of use of fat and CHO substrates in the Ironman event has created dissention about whether enhanced fat use would be sufficient to meet the needs of elite competitors [42–44]. Meanwhile, case histories have shown that periodisation of CHO availability within a chronic LCHF diet might offer some performance advantages to some athletes and event characteristics [45], while chronic LCHF was unsuitable for performance of another elite Ironman competitor [46]. Further investigation is needed; however, it is acknowledged that this is a polarising topic and any promotion of high-fat diets needs to be balanced against evidence that higher rates of CHO intake during ironman racing is associated with faster performances [47].

Fluid intake to address sweat losses is important during longer events, especially those conducted in hot environments. Typically, a fluid deficit equivalent to >2–3% BM loss is considered to increase the perceived effort, raise core temperature and cause reductions in performance, especially in the heat [48]. However, fluid intake should involve an individualised and well-practised plan which balances the time cost of drinking, the risk of gut upsets and the potential advantages of being lighter, with the benefits of better hydration. Although sports drinks are commonly used to address fuel and fluid targets simultaneously, triathletes should consider their needs for CHO and water replacement separately in different environmental conditions. For example, an increase in total fluid intake during Olympic-distance triathlon races in hot weather will lead to a greater CHO intake from such drinks [49]. While this outcome can address the slight increase in CHO oxidation rates in hot conditions, higher CHO intakes can also increase the risk of gastrointestinal upset [47]. A personalised drinking plan can be adjusted to all levels of triathlete, including recreational competitors who may drink in volumes exceeding their sweat rates and who should be warned about the dangers of developing hyponatremia [50].

Major triathlons are often held consistently (e.g. the Kona Ironman World Championships) or uniquely (e.g. Tokyo 2020 Olympic Games) in hot and/or humid environments. The performance and health challenges associated with hot-weather racing should be addressed by strategies such as acclimatisation, appropriate pacing and pre-cooling activities [51]. Nutrition practices include the adjustment of the race fluid plan including pre-race hyperhydration via consumption of large amounts of fluid together with an osmotic agent (e.g. glycerol or sodium) to offset some of the in-race fluid deficit [52, 53]. The integration of ice slurries within pre-cooling strategies to reduce pre-race core temperature via the "heat sink" created by the phase change from ice to water may also be beneficial [54]. All such practices should be well trialled before implementing in a race.

17.7 Theme 6: Nutrition for Adaptation and Recovery Is a Targeted Tool

A strenuous (prolonged and/or high intensity) session of exercise causes stress to the body including the depletion of body fuel stores, loss of fluids and electrolytes, inflammatory and redox responses, damage or increased breakdown of proteins and disruption to homeostasis. Recovery between exercise sessions may have two separate but overlapping goals: restoration of body losses/changes caused by the first session to restore performance levels for the next session and promotion of adaptive responses to the exercise stress to gradually enhance desirable body features/functions (see Fig. 17.1). Over the past decade, post-exercise nutrition has been integrated into a highly promoted "recovery industry" that often targets athletes with one-size-fits-all strategies for universal application. While there is no doubt that such strategies are of benefit in many situations, particularly for scenarios involving multiple sessions of training each day, athletes should apply critical thinking to identify the importance of proactive recovery nutrition and the various issues involved (see Fig. 17.1). The timing, amount and type of nutrient support provided after exercise should be determined by the specific conditions caused by the first session, the duration of the recovery period and the goals of the next session. Such attributes and goals of training are highly specific to the sport and individual athlete.

17.7.1 Application to Triathlon

Although the scheduling of several training sessions in a day is a core feature of most high-performance sports, triathlon poses a unique challenge in requiring the mastery of three disciplines and the need to practise the sequencing of these sports as they occur in a race. Needs around recovery and adaptation are of high importance to maximise the outcomes of the training load.

When restoration of function and performance is a priority, particularly around racing or key training sessions, it can often be useful to quickly supply nutrients without which some elements of recovery cannot happen effectively. For example,

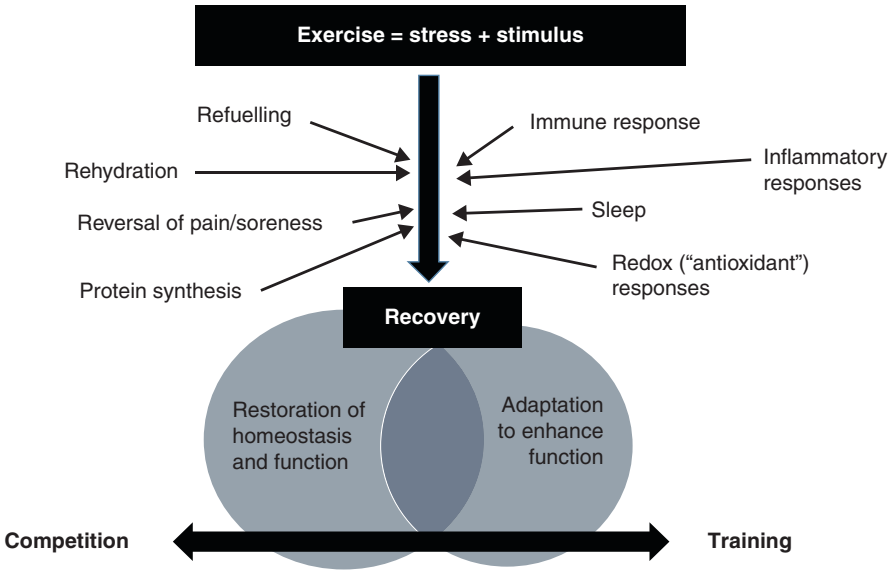

Fig. 17.1 A representation of the various processes in recovery, integrating adaptation and restoration of function/homeostasis

when it is important to refuel between sessions, early intake of CHO (up to a threshold of 1 g/kg/h) can maximise glycogen storage, including potentially taking advantage of a small period of enhanced synthesis in the hours after exercise [55]. Restoration of hydration status requires the replacement of the fluids and electrolytes lost in sweat; this often needs deliberate intake of sodium as well as a volume of fluid equivalent to ~125% of the net fluid deficit to account for ongoing sweat and urine losses during the recovery period [56]. The specific case for post-exercise protein synthesis has been made previously (Theme 4) and the protein content of recovery meals/snacks can also be justified as a general contribution to the optimal distribution of protein intake over the day as well as an enhancer of glycogen synthesis when the CHO content of recovery eating is suboptimal [57]. It is important to realise that not all post-exercise situations call for such aggressive CHO and fluid intake strategies; indeed, in some situations, this may lead to unnecessary intake of kilojoules, reliance on nutrient-poor choices or interrupted sleep due to overnight urination needs (see Table 17.1).

More importantly, there is growing recognition that the *lack* of nutrient support during and after exercise may accentuate the training stimulus and/or increase the adaptive response. Although there is interest in the concept that acclimatisation to exercise in hot weather may be enhanced by deliberately undertaking workouts with a moderate degree of dehydration [58], the principle of enhanced adaptation via altered nutrient availability is best demonstrated via the manipulation of muscle glycogen stores. Exercise undertaken with reduced CHO availability, particularly endogenous CHO stores, selectively modulates gene expression and intracellular

signalling within the muscle. Mechanisms of these effects include alterations in cell osmolality and increased activity of molecules within the regulatory CHO-binding domain of the AMP-activated protein kinase, as well as perturbations to circulating FFAs and hormones in concert with plasma glucose and insulin concentrations [59]. A range of different strategies to augment the training response with low CHO availability ("train low") are available; these include commencing training with low exogenous CHO availability (e.g. overnight fasted and/or withholding CHO during a session) or the more potent strategy of deliberately commencing selected training sessions with lowered muscle glycogen stores (e.g. using a first session to deplete glycogen, then training for a second time after withholding CHO to prevent glycogen restoration [21, 57]).

Although there is clear evidence of enhanced cellular responses to train-low strategies, the translation to performance enhancement has been less certain. Early investigations failed to detect superior performance outcomes due to the impairment of training intensity when "train-low" sessions were overemphasised in the training programme [60]. Such sessions need to be appropriately integrated into a periodised programme to complement high-quality training. A recent sequencing of practices has been investigated: this involves a performance-promoting session ("train-high" with high intensity), followed by restriction of CHO during recovery to prolong the post-exercise increase in cellular signalling and post-transcriptional regulation associated with depleted glycogen stores [1, 21]. The next session of exercise is then undertaken as a moderate-intensity "train-low" session to complete the sequence [1]. In sub-elite populations of triathletes and cyclists, this better integration of train-low and train-high sessions into the training sequence has been associated with superior performance compared with the same training undertaken with normal CHO availability [61]. So far, however, this does not seem to be the case in studies involving elite endurance athletes [38, 62], although it is often incorporated into real-world training sessions [63].

A single bout of exercise causes immune, inflammatory and anti-oxidant responses, with chronic exercise (i.e. training) promoting adaptations in the systems involved. Unlike the situation with the previously discussed issues in post-exercise recovery, specific nutritional strategies to promote or preserve optimal anti-oxidant and immune function in athletes are not well described. It is beyond the scope of this chapter to fully interrogate our current understanding of the role of food-derived antioxidants or anti-inflammatory nutrients in recovery and performance. However, there is evidence that supplementation with large quantities of anti-oxidant supplements (e.g. vitamins C and E) might interfere with adaptation via a dampening of the redox-related signalling processes associated with exercise [64], despite offering some advantages to the immediate recovery of oxidative status. There is also interest in foods containing high amounts of phenolic compounds with anti-inflammatory properties (e.g. various types of cherries and berries), particularly to reduce the post-exercise soreness and impaired muscle function often seen after strenuous sessions [65, 66]. Although further investigation is needed to fully understand any benefits from these recovery nutrition strategies, it is generally suggested that food forms of phytochemicals might provide

a more balanced approach to nutritional support and that approaches might be needed to address goals around immediate restoration of function versus longer-term adaptation.

17.8 Theme 7: High-Performance Athletes Walk a Tight-Rope Between Adequate Training Stimulus and the Risk of Illness/Injury

Injuries and illness are a challenge to success in high-performance sport and, ironically, athletes are potentially at higher risk of developing these as their calibre and training levels increase. Performance is likely to be directly affected if the athlete is sick and/or injured at the time of competition, but these issues can indirectly affect race outcomes by reducing the consistency of training. Injuries may occur to skeletal muscle, to bone, especially stress fractures, and to tendons and ligaments, especially in relation to high or constantly varying training loads, to repetitive low impact modes of exercise (e.g. running) and to weaknesses in the tissues resulting from inadequate nutrient status or support. Given the high prevalence and costs involved, it is not surprising that there has been a great deal of interest in factors that may reduce the injury risk or decrease the recovery time should an injury occur. Bone stress fractures are frequently associated with LEA which should be corrected in both the prevention and treatment of such problems. Attention to vitamin D status and intake of protein and calcium should also be considered. Nutrition goals during injury rehabilitation include adjustment to new energy requirements and distribution of daily protein intake to minimise loss of lean mass and increase muscle repair [67]. The prevention and treatment of injuries to tendons and ligaments is an area of recent active research with initial data on the role of nutritional support from collagenous proteins and micronutrients (e.g. vitamin C, copper) showing some potential [68].

Physically demanding bouts of exercise reduce the metabolic capacity of immune cells, with this transient immunodepression lowering the resistance to pathogens and increasing the risk of subclinical and clinical infection and illness. This may be juxtaposed with an environment of high risk of exposure to pathogens, including travel to a location with different hygiene standards or just close encounters with other people as happens in training squads, long-haul flights and group dining or living scenarios. Optimising training load management (e.g. excessively large training volumes and/or sudden changes in training) and adequate sleep play a major role in reducing the incidence of illnesses, as do good hygiene practices around exposure to surfaces, food/water and people who may harbour pathogens. However, good nutrition status may also help to combat exercise-induced immunodepression, with important considerations including EA, and adequate intakes of protein, CHO, fatty acids and micronutrients (iron, zinc, magnesium and vitamins A and D). It is of interest that training with low CHO availability is associated with greater acute perturbations of cytokines that are important for immune status [23], iron metabolism [69, 70] and bone metabolism [24]. Although it is unclear whether chronically training under such scenarios or, by contrast, chronically using strategies that promote

higher CHO availability during training have an impact on long-term health and injury profiles, athletes who are already at higher risk of illness and injury should be aware of this finding.

Iron status is an important factor in health and performance, but compromised iron status is a common occurrence among endurance athletes, particularly females. This occurs due to factors from both exercise (e.g. haemolysis and alterations to the iron regulatory hormone hepcidin) and non-exercise origin (e.g. inadequate iron intake, heavy menstrual blood losses). Issues around iron status are covered in greater detail by Cairo [71]. Routine screening of iron status according to standardised protocols and treatment of suboptimal iron stores is recommended and should be incorporated into the annual training year, especially around travel and exposure to altitude. Options include dietary counselling to improve iron intake, oral iron supplements and, in the case where the athlete is unresponsive or where faster approaches are needed, an intramuscular or intravenous treatment under the supervision of a physician.

17.8.1 Application to Triathlon

Triathletes face an increased risk of illness and injury from several characteristics, that is, high and varying training loads across three different exercise disciplines, and regular racing programmes both domestically and internationally. Consistent or lengthy interruptions to training will unravel the athlete's development and either delay or derail their movement along the elite athlete pathway. The most common injuries experienced by elite triathletes relate to bone stress and require an extended rehabilitation period. It is important that nutrition plans incorporate strategies that optimise bone remodelling such as maintaining adequate EA, consuming adequate calcium, ensuring vitamin D sufficiency and strategically planning CHO and protein intakes around daily training. While the daily training loads of recreational triathletes don't meet those of their elite athlete counterparts, the busy nature of daily triathlon training alongside work and family commitments can interfere with opportunities to incorporate adequate rest, recovery and sleep as well as incorporate appropriate food and fluid choices. Triathletes should be vigilant with their daily food and fluid choices to ensure they align with daily training requirements. Coaches and performance support staff play an important role to ensure athletes are reminded of the importance of incorporating appropriate nutrition strategies to support health and well-being.

17.9 Theme 8: A Pragmatic Approach to Supplements and Sports Foods Is Needed

Athletes represent an enthusiastic audience for the numerous supplements and sports foods which make up a multibillion-dollar industry [72]. Of course, they also reflect the interests of the general community where the majority of people

report supplement use. Although a "food first" philosophy is promoted in relation to nutritional needs, athletes often require medical supplements as part of a treatment or prevention plan for nutrient deficiencies; this includes iron deficiency [71] and vitamin D deficiency [73]. In addition, sports foods may assist in meeting nutritional goals or nutrient targets in scenarios where it is impractical to eat whole foods. Meanwhile, the largest supplement categories with the most heavily marketed claims include performance products that directly enhance exercise capacity and supplements that provide indirect benefits through recovery, body composition management and other goals. While the majority of these products are not supported by robust evidence, there are five (caffeine, creatine, nitrate/beetroot juice, beta-alanine and bicarbonate) which may contribute to performance gains, according to the event, the specific scenario of use and the individual athlete's response [74]. Specific challenges include developing protocols to manage repeated use of performance supplements in multiple events or training sessions in the same day, the interaction between several products that are used concurrently and the individual experiences of athletes with regard to benefits and side effects [75, 76].

Potential disadvantages of supplement use include expense, false expectancy, side effects and safety issues associated with poor manufacturing processes and the risk of ingesting substances banned under the World Anti-Doping Agency's list, which are sometimes present as contaminants or undeclared ingredients [77]. Strict liability codes mean that a positive urine test can trigger an Anti-Doping Rule Violation with potentially serious impact on the athlete's career, livelihood and reputation, despite unintentional intake or minute (ineffective) doses. Despite earlier reluctance, many expert groups, including the International Olympic Committee [3], now pragmatically accept the use of supplements which pass a risk–benefit analysis of being safe, effective, legal and appropriate to an athlete's age and maturation in their sport. Expert guidance or supervised use should be considered and all supplements should be trialled thoroughly by the individual before committing to using them in a competition setting. Third-party auditing of products can help elite athletes to make informed choices about supplement use but cannot provide an absolute guarantee of product safety [3].

17.9.1 Application to Triathlon

Several supplements and supplement strategies are worthy of considered use across the various triathlon disciplines (Table 17.3). Sports foods provide an important aid in meeting high energy and nutrient needs, by providing a convenient and practical option for intake before, during and after training and races. Although triathletes rely heavily on fellow athletes, coaches and media outlets for information regarding supplement use, they should be directed to receive professional advice, particularly in the case of medical supplements.

Many triathletes are interested in the use of performance supplements on race day; indeed, caffeine is widely used in triathlon races [78]. However, even when such products can be justified, they should not take priority over the development

Table 17.3 Supplements and sports foods of potential value to triathletes and triathlon performance [79]

Type	Product	Characteristics and evidence-based uses
Sports foods	Sport drinks	• CHO-containing fluid (typically 6–8% concentration) which supports fuelling and hydration goals during and after workouts and races. • Provides moderate levels of key electrolytes (e.g. ~20 mmol/L sodium) to help replace sweat electrolyte losses and increase voluntary intake of fluid.
	Sport gels and confectionary	• Convenient and compact CHO source to assist with fuelling goals during workouts and races • May contain multiple transportable CHO sources to aid intestinal absorption when intakes >60 g/h.
	Liquid meal supplements	• Convenient, portable, and easy-to-consume source of CHO, protein and micronutrients. • Situations of use include as a well-tolerated pre-event meal or post-exercise recovery snack or during travel when access to food is limited.
	Protein supplements	• Concentrated protein from high-quality foods (e.g. milk) or isolated protein fractions (e.g. whey). • Provides convenient option to meet protein intake target when it is impractical to transport, prepare or consume food sources of protein (e.g. immediately post-exercise, during travel).
	Electrolyte supplements	• Provides larger concentrations of electrolytes (e.g. ~50 mmol/l sodium) for more targeted replacement. • Situations of use include pre-exercise hyperhydration before races in hot conditions, proactive electrolyte replacement in long races by individuals with large sweat/sodium losses and assisted rehydration following moderate to severe dehydration/electrolyte loss.
Medical supplements	Iron supplements	• Supplemental form of iron for prevention and treatment of diagnosed cases of reduced iron deficiency. Should be taken under the supervision of a medical practitioner and sports dietitian and in conjunction with dietary intervention.
	Calcium supplements	• Supplemental form of calcium for prevention and treatment of poor bone status when diet is unable to meet calcium requirements. Should be taken under the supervision of a medial practitioner and sports dietitian and in conjunction with appropriate medical and dietary intervention (e.g. to address LEA).
	Vitamin D supplements	• Supplemental form of vitamin D for prevention and treatment of vitamin D insufficiency/deficiency due to inadequate sunlight exposure. Should be taken under the supervision of a medical practitioner.

Performance supplement	Glycerol for hyperhydration	• Total body water may be increased when glycerol is consumed (1–1.2 mg/kg) as an osmotic agent with a large bolus of fluid (~20–25 ml/kg) in the hours before a race. • Hyperhydration strategies might be useful to reduce overall fluid deficit in races undertaken in the heat in which large sweat losses cannot be practically replaced during the event. • Should be undertaken under the supervision of a sport scientist and with appropriate experimentation during training.
	Caffeine	• Highly biologically active compound with many effects including masking of pain/fatigue/perceived effort and enhanced recruitment of muscle motor units. • Small to moderate doses (~3 mg/kg BM) appear to be as effective as larger doses (5–6 mg/kg BM) in enhancing the performance of races of 1 h and greater. • Further studies should investigate the range of triathlon events that benefit from caffeine intake and various effective doses and consumption protocols (e.g. intake before, during or toward the end). • May be consumed in cola, energy drinks, sport gels/confectionary, tablets and gum. There are problems with using coffee as a source of caffeine due to the variability of the caffeine dose.
	Nitrate (beetroot juice)	• Inorganic nitrate works with enterosalivary system to produce nitric oxide through an alternative and oxygen-independent pathway to arginine–NO production. • Associated with improved exercise economy (reduction in oxygen cost of submaximal exercise) to improve endurance exercise performance and enhanced skeletal muscle contractile function to improve muscle power and sprint exercise performance. • Typical dose = ~8 mmol nitrate taken 2–3 h pre-race, especially with chronic intake for 3+ days pre-trial, but intake during longer races may sustain the effect. • Effect of nitrate supplementation on endurance events is inconsistent and may involve individual responsiveness, including observations that it seems less effective in elite athletes.
	Bicarbonate loading (acute) B-alanine (chronic) supplementation	• There is potential, but inconsistent evidence, that an increase in blood-buffering capacity might enhance the performance of short higher-intensity races of up to 30–60 min duration (e.g. relays, sprint triathlons) where disturbances to acid–base balance occur due to reliance on oxygen-independent glycolytic pathways of ATP production. • Acute increases in extracellular buffering may be achieved by consuming 300 mg/kg BM bicarbonate, 2 h pre-race. Further field studies are needed with high-level triathletes to confirm benefits. Risk of gastrointestinal problems should be noted, especially in combination with high-intensity running. • Increased intracellular buffering may be achieved by increases in muscle carnosine content via chronic supplementation with B-alanine (e.g. 6–10 w at 3–6 g/day). Split doses over the day or sustained release preparations may reduce the common side effects of paraesthesia (tingling).

and practise of fuelling and hydration strategies. In fact, numerous sports foods and fluids contain caffeine, which should be accounted for in the race-day nutrition planning. Given the extended nature of several triathlon races and the delicate balance of consuming adequate CHO and fluid, the incorporation of performance supplements into a race nutrition plan should be well rehearsed and carefully considered. Trialling the use of sports foods and fluids alongside performance supplements, within sessions that mimic race elements, should form a regular component of the training programme.

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Part V

Training

Quantifying the Training Load in Triathlon

18

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18.1 Concept and Components of Training Load

A training load is a dose or stimulus produced by a workout or a part of a workout. From a coach's perspective, it is specifically an *external load*, that is, what the coach prescribes. This external load is made up of the exercise as well as the volume, intensity and recovery (Box 18.1).

Moreover, *internal load* is the physiological impact generated by the stimulus, dose or workout in a triathlete. In other words, how a particular session "affects" the triathlete, or its perceived strain or stress.

Box 18.1 Components of the External Load

1. Exercise
2. Volume
3. Recovery
4. Intensity

Exercise in endurance sports is usually a mode of locomotion (swimming, pedalling, running, rowing, etc.). Strength exercises are also taken into account

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to quantify global training load. Furthermore, we must remember that exercises can significantly differ in complexity, both in strength and endurance. The latter is described in more detail later based on a particular model.

The *volume* is the quantitative part of the load, usually measured in terms of distance, time or exercise repetitions. To set individual doses in a group of triathletes of varying levels, it is best to dose in terms of time. If the training is taking place on a difficult terrain or under unfavourable weather conditions, time is also preferable. It also allows to reduce the triathletes' obsession with distances, as they compete based on distance. However, if the triathlete is a top-level triathlete, or if a given triathlete is undergoing a longitudinal follow-up programme, it will be just as suitable or more relevant to observe or measure distances. This is considered for endurance-type workouts. When applied to strength training, then use repetitions.

It is necessary to distinguish *volume* from *duration*; although it sometimes comes to the same, *duration* refers to the total time, from the moment the workout starts to the moment it ends, and the session sometimes includes a large amount of pauses. So one measure is how much time the session "lasts" and another is how much time is spent on "net effort time". For example, we spent 2 h in the gym, where we swam 2000 m in 45 min and then ran for 30 min. At first sight, it seems that the workout only lasted 45 + 30′ = 1 h 15 min (instead of 2 h), but if we count pause time during the 2000 m, it may actually have been 40 min. Therefore, the volume is the net exercise time, which in this latter case would be 40′ swimming and 30′ running. As we will see, the meaning of volume changes as we consider different types of exercises (though always counted as "time"). Following the scenario in the example, clearly, it is not possible to measure strength based on the session's duration, since the net dose cannot be correctly measured using time anyway. The strength volume is usually measured by the number of repetitions of a specific movement (lifts, jumps or throws).

Intensity is the qualitative aspect of the load, usually measured using internal variables (such as heart rate or subjective perception of the effort) and external variables (such as pace, velocity or power). We recommend that beginner or intermediate triathletes focus on internal variables during much of their training because, due to their lower energy efficiency, it will not be equivalent to a given external intensity when fatigue appears. External variables become important for top-level triathletes or for all levels, when competitions are approaching, to establish forecasts and race strategies. Please note that internal variables to monitoring intensity (such as HR or RPE) is different to the concept of internal load, which is basically a perception of the training strain.

Recovery is the pause duration between stimuli, as an "incomplete" or "complete" recovery can be intended (in fact it is difficult to distinguish, and it would be more accurate to call it "short or long" recovery). In the aforementioned training method examples, we sometimes write *r* to refer to short pauses and *R* to long pauses. Technically, the term "pause" would implicitly include the concept of short recovery and "recovery" the long recovery.

Density is the ratio between repetition time and pause time. There are different ways to calculate it (Box 18.2). The term "repetition" refers to a specific effort that starts and stops. For example, swimming 4 × 400 m comprises four repetitions. It does not refer to the number of strokes. However, in strength training, the "repetitions" do refer to the number of lifts, jumps or throws.

Box 18.2 Ways of Expressing or Calculating Training Density in Terms of Repetitions of Cycles

1. Σ of all effort times/Σ of all pauses (Σ = SUM)
2. The ratio of repetition time with respect to the next pause time

Example: 7 × 4 min zone 4 r' = 1 min

Method 1. 7 × 4 = 28; 6 × 1 min = 6 (intermediate pauses are counted); Density: 28/6 = 4.666

Method 2. Formally expressed as 4:1. The result is 4/1 = 4

Note: Although the first expression is more accurate, it is easier and faster to mentally evaluate the second. Colloquially, coaches refer to Method 2, but we will use Method 1 in the following model, as it takes the full real time into account.

To finish, *frequency* is the number of sessions over a certain period, for example, "per week", referred to as "sessions per week". Therefore, there could be more than seven sessions if double sessions take place on several days, even when including whole days of rest.

18.2 Load Quantification Models

It is impossible to quantify the training load perfectly. Internal load cannot be monitored beyond subjective scores, which are also logically influenced by factors such as rest hours, nutrition and stress. Since all of these influence the extent to which the training is assimilated, it is probably more appropriate to take this complexity into account rather than the analytical perspective of quantifying the external load only.

Several models have taken the internal load into account [1]. Foster's et al. 2001 [2] model considers the triathlete's score on the Borg CR10 scale shown in Table 18.2, though used in this case to evaluate the "overall session stress", approximately 20–30′ after it had ended. This would be an internal load score up to this point. However, Foster's model multiplies it by the session duration, making it a "mixed" model. On the other hand, it considers that all types of training can weigh the "stress × duration" and thus includes the quantifying of strength training. The latter is a serious conceptual mistake, since duration is taken into account and not the specific strength training variables (mainly mechanics, intensity, volume of repetitions).

Tudor Bompa, a major sport training theorist, established a scale, from 0 to 5, for coaches to assess how hard a session is overall [11].

Based on this scale, we propose to use the scale in Table 18.1 so that triathletes indicate, once again about 20–30′ after the training has ended, "how hard"

Table 18.1 Scale to assess subjective load equivalents (ECSs by its Spanish acronym) [12]

Value	Type of effort
0	**Rest**
0.5	
1	**Light** load
1.5	
2	**Medium** load
2.5	
3	**High** load
3.5	
4	**Very high** load
4.5	
5	**Competition** (also **exhaustive training** or **test** as hard **as a competition**)

After 20–30 min of training, triathletes are asked to assess their overall perception of how hard the session was from 0 to 5. "Mid-point" scores are allowed

the session is perceived. Since it allows to assess any type of session and refers to a "perceived stress" scale, we refer to "subjective load equivalents".

When there are several sessions per day, each session is rated. The weekly hardness can thus be calculated more accurately (Σ of all scores/weekly frequency).

Despite the benefits of the internal load and such simple scoring, it would be a mistake to consider this load only: different people may reflect the same scores though they are performing highly differentiated external training loads. Coaches mainly quantify the external load for planning purposes (although it is good practice to also generally consider what is pressumed to be the athlete's ECSs score).

Another conceptual difficulty relating to internal load is that not only sensations of hardness should be reported but also fatigue markers, such as biochemical, neural and neuromuscular (urea, CPK, etc.) indicators. The latter are difficult to monitor outside research scenarios.

To understand how to quantify the external load more in depth, we will focus in this book on a specific model that has been used in research and practice over the last decade. The main reason is space limitation in the text. It is worth noting, moreover, that this manual is designed for 3 different modes of endurance exercise, and no other models consider the equivalences between (swimming/cycling/running), or integrate strength training based on exercise mechanics (EM), to cite the main differences.

We do not intend, in doing so, to undermine other models—summarised in Table 18.2—which are generally helpful because they are simple to use. We must remember that the problem is unsolvable, as mentioned at the beginning of this chapter. Essentially, coaches should quantify what they ask for and what the triathlete does, or they will be unable to determine the relationship between training and performance. But they must seek "the least bad method", according to the method's underlying concept and ease of use.

The model we describe is called objective load equivalents (hereon ECOs by its Spanish acronym), described for the first time by Cejuela and Esteve in 2011 [12].

Table 18.2 Classic external load quantification methods

Method	Considerations	Load example	Calculation example
Time × RPE (Norwegian Olympic Committee)	Uses RPE scale 0–10. Multiply time in RPE range by its value	20′ in RPE 3 + 20′ in RPE 5	(20 × 3) + (20 × 5) = 160
Edwards [3]	Multiply time in HR range by coefficient (1–5) 1–50–60%; 2–60–70%; 3–70–80%; 4–80–90%; 5–90–100%	20′ in 55% + 20′ in 65%	(20 × 1) + (20 × 2) = 60
TRIMPS "De Lucía" [4]	Uses the three-phase model of two metabolic thresholds and multiplies the time in phase by 1/2/3	20′ in Phase 1 + 20′ in Phase 2 + 10′ in Phase 3	(20 × 1) + (20 × 2) + (10 × 3) = 90
Global demand index [5]	Multiplies duration of effort by the average of the HR made in % of maximum HR or of reserve, and to divide it by the total time of the effort	20′ to 60% of the maximum HR + 10 × 2′ to 85% HR Máxima	20 × 60 + ((10 × 2) × 85)/20 + 20 = 72.5
Units of training [6]	Multiplies coefficient × time in zone of swim speed. Recovery (0); aerobic light (1); aerobic medium (2); anaerobic threshold (3); intense aerobics (4); anaerobic (5); speed (8); competition (10)	Totals per zone: Ae Lightweight: 1000 Ae medium: 800 Anaerobic threshold 2000 Speed 200	(15 × 1) + (11 × 8) + (26 × 3) + (2 × 8) = 197
Session RPE [2]	Multiplies perception of overall hardness of the session (in RPE scale 0–10) for the total duration of the session	Varied session of 55′ duration	Rated "5" 55 × 5 = 275
TRIMPS Banister [7], Morton [8][a]	TRIMP = minutes × (factor to × HR ratio × Exp (Factor B × HR ratio)) HR ratio = (HR Average – HR Repose)/(HR Max – HR Repose) Factor A = 0.86 and factor B = 1.67 for women. Modification Morton et al. [8]: TRIMP = minutes × HR ratio × 2718 Exp (Factor B × HR ratio)		

(continued)

Table 18.2 (continued)

Method	Considerations	Load example	Calculation example
TRIMPS "of Hayes and Quinn" [9][a]	Add to the original concept the consideration of the fractional training and pauses through different equations		
training stress score TSS [10][a]	Calculation TSS = (sec × NP® × IF®)/(FTP × 3600) × 100 where "SEC" is the duration in seconds, "NP" is normalized power, "IF" is intensity factor (% of FTP), "FTP" is functional threshold power (functional threshold power or your best estimated 1 h average, which can be estimated from 20-min tests). The "3600" comes from the number of seconds in an hour.		

[a]No Calculations are displayed for the complexity and extent of space

A more advanced version of the model is presented in this book, and it now considers density and other elements that had not been contemplated previously. Thus, to avoid confusion, the model is now called ECOs, and the 2011 model is called the "original model". To make it easier to use, you can access our app with the following QR code and calculate the external load value based on the current model premises explained below:

18.3 Considerations and Criteria for Quantifying the Objective Load Using the ECOs Model for Endurance Training

The original ECOs model is simplified in Fig. 18.1. Basically, the time in minutes accumulated in each zone is multiplied by an intensity factor (IF) and by an exercise factor (EF) or mode of locomotion. The total sum of the loads in zones gives the session load.

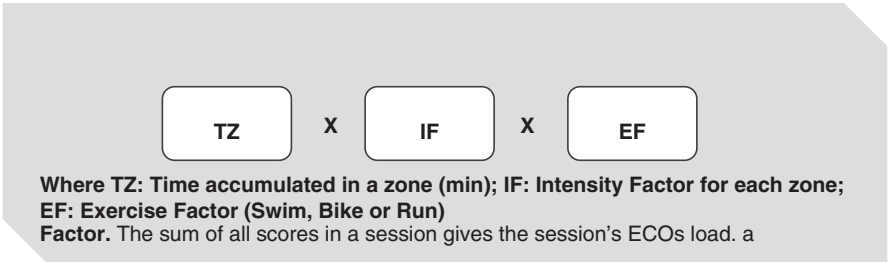

Fig. 18.1 Original ECOs model (2011)

Table 18.3 ECOs model: zones and their IF

Zone	IF
1	1
2	2
3	3
4	4
5	6
6	9
7	15
8	50
9	150
10	300

These coefficients are based on two concepts. The first is the general standard lactate kinetics (up to zone 7), which was based on the 1996 model of Mújika et al, to obtain a nonlinear trend. The second is the obtention of coefficients that are equal to different training loads perceived as equivalent throughout the training, considering "minutes × IF". This was based both in short and long distance events, based on typical workouts for a given performance level. Finish, the values were rounded to make them easier to remember, and it is understood that they could be modified (within the range) a little downward or upward in "odd" zones, for example, zone 5 could be an IF between 5 and 8

18.3.1 Training Zones and IF

There are ten training zones. The IFs are shown in Table 18.3.

For decades, applying systematic evaluation procedures to personalised zones has enabled coaches to observe how enormous the extent of individualisation can be regarding standards. That is, many triathletes are far from the standards we calculate, whether in some zones or in all of them. Moreover, when zones that are "real and unified according to different variables" are transferred "to the real terrain", they deserve to be qualified so as to reflect all external conditions (climate, weather, trajectory, equipment, etc.) and internal conditions (accumulated fatigue, momentary health status, situations affecting measuring instrument reliability, etc.).

Therefore, the laboratory data must be transferred to the field, and the field data is incomplete if it does not include sufficient physiological measurements.

The coach must understand all this and inform the triathletes: they make quicker progress when they follow more accurate workouts, and this accuracy is not only due to the measurement instrument but also to the procedures followed by the workout control instruments.

The more information, the better. The idea is not so much to define what is best [13] but to know how to "triangulate" the heart rate, velocity/power and pulse variables, integrate them and know when to listen to one data or another, as well as know how to recognise the sensations associated with personal physiological zones.

Box 18.3 Criterion for Identifying which Zone Has Been Worked in Each Part of the Session

Criteria to become familiar with the zones

1. Perform an objective test to establish zones and control variables (heart rate, pace or power).
2. Check/adjust the zones during daily training, including respiratory perceptions and ability to maintain a conversation (*talk test*) whenever possible. The latter is referred to as perceived zone.

As one becomes more familiar, the following criteria will be followed:

Continuous Efforts of Low or Moderate Intensity (Zones 1–3)

1. Initial criterion	Main reference (for the most part of the session)	Secondary/additional criteria
2. Perceived Zone	Perceived Zone Eventually pace/power	HR Pace/power

Fractionated and/or High Intensity Efforts (Zones 3–8) or Competition (Any Zone)

1. Initial criterion	Main reference (for the most part of the session)	Criteria secondary/ complementary
2. Pace/ power	Pace/power and Hazard Score	Perceived Zone

Hazard Score: consider "how I feel about what remains", although it can be done without calculating the variable previously explained variable, as an added criterion

Note: In the case of swimming, more specifically in crawl swimming in a pool and without equipment, these zones will be worked on mainly based on pace associated to a recent test. In other conditions (open water, other styles outside the specialty, use of fins, paddles, pull, endurance swimsuit, etc.) work would be based on zone sensation

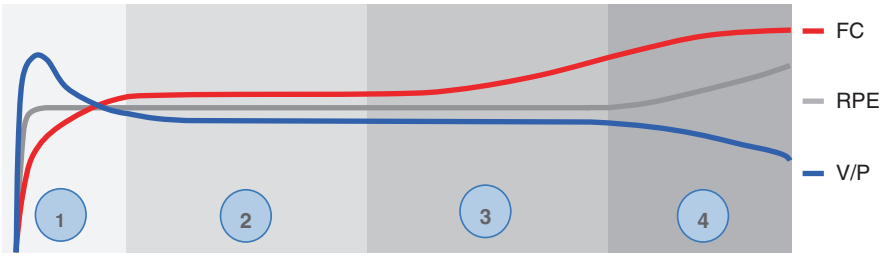

Fig. 18.2 General dynamics of intensity control variables. HR: heart rate; RPE: rating of perceived exertion; V/P: velocity/power

Box 18.3 can help you to prioritise one or more variables depending on the type of session.

To summarise, zones are ranges of intensity within you exercise at a physiological domain a physiological state. The correct location of the zones allows you to train a certain factor much more than when determined by performance.

The zone (perceived zone) is usually applied at first moments of the periodisation, and the performance variables (speed/power) at the end, on the understanding that a zone may be skipped from a metabolic viewpoint, but it must be tolerable, however, because the objective is a (reasonably achievable) performance in absolute terms.

Let's look now at Fig. 18.2. It can be transferred to daily training and cover stages 1–4 or only the first 2 or 3. An initial stable perceived exertion is considered in order to observe its effect on the heart rate has been "fixed" to observe its effect on the heart rate (HR) and the velocity or power (V/P) in fatigue situations. At stage 1, the athlete seeks the expected velocity or power as soon as possible and makes several adjustments (start fairly fast and then slow down or start slowly and then accelerate). The RPE refers to the current intensity. It has been simplified because it is not reached at once. At stage 2, the necessary adjustments have been made and all the variables are lined up, because there is no fatigue. At stage 3, the HR starts to drift (due to the slow component, it could also be associated to the accumulation of lactate in high zones), but the feeling is still the same in terms of effort intensity and it allows to maintain speed or power. But if the effort is extended to a considerable degree, the perception inexorably increases, and since previous fatigue signs are already present, velocity or power should be reduced to ensure that the perception does not increase sharply. Once a certain point is reached, the latter will be inevitable for both variables (high perception and lower velocity).

When evaluating performance factors, we locate at least two critical points such as two metabolic thresholds, maximum power or aerobic velocity (associated with VO_{2max}) and the maximum power or velocity in *sprint*. If one considers those points as zones for which those factors should be improved (which is logical), it should be assumed there a range of intensities around those points (since in practice, it would

be almost impossible to maintain oneself precisely at that concrete level of heartbeat, power or speed).

Once this range is established applying arbitrary criteria (± something), we obtain the zones usually identified by "pair" numbers (specifically zones 2, 4, 6 and 10).

Other zones now remain "in the middle" (3 and 5) or "below the first threshold" (zone 1). These are the "odd" zones.

Two more physiological peaks should be taken into account: maximum power and glycolytic anaerobic metabolism, which is zone 8, within the range of anaerobic speed reserve (ASR) or anaerobic power reserve (APR). It can be estimated based on average speed or power effort lasting around 1 min, for example, swimming 100 m (the usual distance, though it depends on the level), cycling for 1 min or running 300–500 m (depending on the level). It can also be evaluated based on 120% of Maximal Aerobic Speed (MAS) or Maximal Aerobic Power (MAP) in the case of triathletes who do not take part in "supersprint" or "ultra endurance" extremes: in this case, we consider an average range (30–60%) of their ASR/APR.

The other odd zones (7 and 9) are associated with the ability to repeat submaximal efforts related to glycolytic or phosphagenolysis anaerobic powers, called, in the field of training, "lactic" or "alactic" anaerobic capacity. Here too, there are big differences in highly "long distance" or highly "sprinter" cases: either % of MAS/MAP or range of ASR/APR apply. The most practical option is to select the appropriate distances and work by % of personal best in the distance used in the training session. When using our app, one of these options must be chosen.

Tables 18.4 and 18.5 show the formulas to be applied to each segment.

To make it easier to calculate zones, the following QR code is provided:

Table 18.4 Heart rate zones of the ECOs model

Zone	HR Range
1	(HRZ2 × 0.85)–(HRZ2 − 3)
2	(HRZ2 − 2)–(HRZ2 + 2)
3	(HRZ2 + 3)–(HRZ4 − 3)
4	(HRZ4 − 2)–(HRZ4 + 2)
5	(HRZ4 + 3)–(HRZ6 − 3)
6	(HRZ6 − 2)–([a] or [b])
7–10	No need to use HR

[a]If the person has a maximum theoretical HR over HRZ6 according to the formula 207–(0.7 × age), indicate this as the upper limit

[b]If the person shows a real maximum HR over the last year above the HRZ6 reported in the evaluation, indicate this as the upper limit

Table 18.5 Zones of the ECO model according to speed pace and power

Zone	Running or swimming speed range	Cycling power range (W)	Swimming zones (100 m pace)
1	(SZ2 × 0.85)–(SZ2 × 0.97)	(PZ2 × 0.85)–(PZ2 × 0.97)	(PaZ2 × 1.15)–(PaZ2 × 1.03)
2	(SZ2 × 0.98)–(SZ2 × 1.02)	(PZ2 × 0.98)–(PZ2 × 1.02)	(PaZ2 × 1.02)–(PaZ2 × 0.98)
3	(SZ2 × 1.03)–(SZ4 × 0.97)	(PZ2 × 1.03)–(PZ4 × 0.97)	(PaZ2 × 0.97)–(PaZ4 × 1.03)
4	(SZ4 × 0.98)–(SZ4 × 1.02)	(PZ4 × 0.98)–(PZ4 × 1.02)	(PaZ4 × 1.02)–(PaZ4 × 0.98)
5	(SZ4 × 1.03)–(SZ6 × 0.97)	(PZ4 × 1.03)–(PZ6 × 0.97)	(PaZ4 × 0.97)–(PaZ6 × 1.03)
6	(SZ6 × 0.98)–(SZ6 × 1.02)	(PZ6 × 0.98)–(PZ6 × 1.02)	(PaZ6 × 1.02)–(PaZ6 × 0.98)
7	(SZ6 × 1.03)–(SZ6 × 1.10) or 10 to 50% ASR. That is, ((10%*(SZ10–SZ6))+SZ6) to ((50%*(SZ10–SZ6))+SZ6) or 80–85% of the personal best in the repetitions	(PZ6 × 1.03)–(PZ6 × 1.10) 10 to 50% APR. That is, ((10%*(PZ10–PZ6))+PZ6) to ((50%*(PZ10–PZ6))+PZ6) or 80–85% of personal best in 1 min average Normalized Power	(PaZ6 × 0.97)–(PaZ6 × 0.90)
8	(SZ6 × 1.11)–(SZ6 × 1.20) or 51 to 73% ASR. That is, ((51%*(SZ10–SZ6))+SZ6) to ((73%*(SZ10–SZ6))+SZ6) or 86–100% of the personal best in the repetitions	(PZ6 × 1.11)–(PZ6 × 1.20) 51 to 73% APR. That is, ((51%*(PZ10–PZ6))+PZ6) to ((73%*(PZ10–PZ6))+PZ6) or 86–100% of personal best in 1 min average Normalized Power	(PaZ6 × 0.89)–(PaZ6 × 0.80)
9	95–97% S Max 74 to 85% ASR. That is, ((74%*(SZ10–SZ6))+SZ6) to ((85%*(SZ10–SZ6))+SZ6) or 95–97% of the personal best in the repetitions	74 to 85% APR. That is, ((74%*(PZ10–PZ6))+PZ6) to ((85%*(PZ10–PZ6))+PZ6) or 95–97% of personal best in 5 sec average Normalized Power	Apply same procedures as set in the Speed column considering a personal best in the distance of the workout repetitions (95–97%) or submaximal perception
10	98–100% S Max 90 to 100% ASR. That is, ((90%*(SZ10–SZ6))+SZ6) to ((100*(SZ10–SZ6))+SZ6) or 98–100% of the personal best in the repetitions	90–100% APR. That is, ((90%*(PZ10–PZ6))+PZ6) to ((100*(PZ10–PZ6))+PZ6) or 98–100% of personal best in 5 sec average Normalized Power	Apply same procedures as set in the Speed column considering a personal best in the distance of the workout repetitions (98–100%) or maximum perception

For swimming, the 100-m time will be indicated in seconds. The pace is expressed by zone such as PaZ2 for "pace zone 2", PaZ4 for "pace zone 4" and PaZ6 for "pace zone 6". For example, "104" refers to the pace to do 1 min with 44 s every 100 m

Zones 9 and 10 are highly variable according to the sports speciality, as explained earlier. This is why the % option is given.

Table 18.6 Coefficients per segment to quantify the ECOs

	S	B	R
Difficulty maintaining the technique	****	*	**
Delayed onset muscle soreness (DOMS)	*	*	****
Usual session density[a]	*	**	***
Energy cost[b]	***	**	***
Sum total (1–4)	9	6	12
(%)	75	50	100
Ratio/relative coefficient	0.75	0.5	1

S swimming, *B* bike, *R* run

[a]Apart from fractional efforts, which are greater in swimming, it is generally understood that there are more frequent pauses in swimming during workouts; there are also more frequent pauses in road cycling training, due to the uneven orography though to a lesser extent, and much less in running, or never in the continual method

[b]In the case of sessions with no *drafting* nor prior fatigue, based on published data and equations, applied to competition speeds of the same level of relative performance [11–22]. Interactions and fatigue are not taken into account because they are presumed to be coefficients for isolated sessions with no drafting. Consult other coefficients for other situations

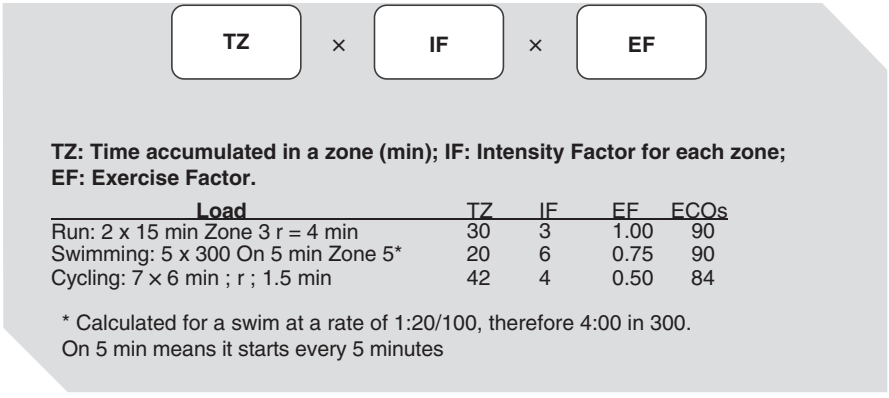

Fig. 18.3 Original ECOs model (2011). Calculations of a load (session fractions are used for simplicity). The parts of the entire session will be summated

18.3.2 Exercise Mode and EF

To weight the gross ECOs score (time × IF), ECOs are considered equivalent to the value of the run; this value being equal to 1. For swimming, the coefficient is equivalent to 0.75, and for the cycling, to 0.5. The justification is based on a comparative analogue scale from 1 to 4, based on the overall studies regarding the sections in Table 18.6.

Figure 18.3 shows examples of calculations using the original model.

18.3.3 Density

In exception to the Hayes and Quinn's model [9], no other model took the pause effect into account to calculate the load. This would mean that there is no difference between performing 8 × 5 min and 40 min of zone 4. Therefore, it prevents us from quantifying the degree of load progress in a particular zone: it would lead to errors when comparing two fractionated training sessions, as it assumes that everyone recovers more or less in the same way. However, the main difference between higher level triathletes and lower level ones is known to usually be their recovery capacity, not only between efforts but also between training sessions, allowing them to train at more frequently during the week. A good-level amateur, for example, can endure an elite workout but perhaps not with such short breaks nor undergoing such a large number of weekly sessions.

Essentially, load is increased or reduced compared to the original model according to two criteria:

- Load percentage (LS). Considers the % of pause time taken during a predominantly continuous effort with respect to the total duration. That same load % is subtracted.

Box 18.4 Application of the Load Substraction

Example 1: 1 h 40 min of cycling in zone 1. The gross load or load in a continuous mode would be 100 min × IF 1 × FE 0.5. This is 100 × 1 × 0.5 = 50 ECOs. In the case of a 3-min pause, 3 out of a total duration of 103 min would be calculated as 2.91%; so instead of evaluating 50 ECOs, you subtract the 2.91% and the result is 48.54 ECOs (rounded to 48 or 48.5).

Example 2: Effort during a run in a main part of the session of 3 × 15′ zone 3 *r*′ = 2′. Total net time was 45′, and the pauses added 4′. Therefore, 4′ out of 49′ (45 + 4) represents 8.16%. Since the gross load would be 135 ECOs (45 min × IF 3 × EF 1), calculating 8.16% of 135, we obtain 11.02. We subtract those ECOs (135−11), estimating a final 124 ECOs. If the sequence had variable repetitions, the same net times and total pause times would be added and calculated in the same way.

- Density weight with respect to a standard density (SD) for high-intensity efforts. Allows a load increase (LI) from SD up to a limit (LIL).

18.3.3.1 Load Percentage

LS is used with zones 1, 2 and 3, considered to be eminently directed towards being developed by means of continuous methods. For this reason, the percentage value of gross load—based on pause time with respect to total task duration—will be subtracted. In other words, we use only a "negative" LI, which can be subtracted, and which we call LS. Examples are given in Box 18.4.

18.3.3.2 Weighting Density in High-Intensity Workouts

Weighting of density is mainly applied to zones 4–8. These latter zones are considered to be usually training with fractionated methods. Thus, the recovery doesn't require "subtracting" ECOs but will depend on how much recovery takes place.

Since certain ratios are usual for each zone between working time and pause time, an SD per zone is established as a criterion, where nothing is added to, nor subtracted from, the value of the gross ECOs (those obtained from the result of the time × zone IF). In this calculation, if we assume the volume is equal in a certain zone, recovering excessively will decrease the total load, while achieving a recovery level below the SD would increase the value of the gross ECOs. Table 18.7 shows the SD, examples of extreme density (XD) in each zone and the equation to calculate the LI according to Density calculated with the Method #1 of the 18.2 box. The XD establishes the equation's load increase (LI) limit, since the workouts are assumed to take place with a minimum development load.

Density is considered to be the sum of repetition times/sum of recovery times. See method #1 at the 18.2 box. In the case of sets and repetitions, long and short recovery are summated.

Example Cycling, one part of the session with 7 × 4 min Zone 4 $r = 1$ min (active recovery, logically, which, in cycling, is considered to added to zone 1). This load is 6 (min) × 1 (IF) × 0.5 (EF) = 3 ECOs. On the other hand, the density will be of $(7 \times 4)/(6 \times 1) = 4.66$. We must remember that pauses are calculated according to "number of repetitions minus 1". For zone 4, the SD is 5.71. Therefore, the load will be weighted to calculate the LI using the specific equation, in this case $LI = 29.304\ln(D) - 50{,}791$ (see Table 18.7). The calculations give −5.65.

Table 18.7 SD per zone, XD and equation for LI calculation

Zone (#)	SD Σ work time/Σ pause time	XD Σ work time/Σ pause time	Equation for LI
8	0.30	1.2	$89{,}914(D) - 27{,}562$
7	0.46	1.4	$51.027\ln(D) + 45.471$
6	1.38	3.5	$37.085\ln(D) - 6.2192$
5	2.48	5.5	$40.257\ln(D) - 35{,}627$
4	5.71	13	$29.304\ln(D) - 50.791$

SD based on Seiler and Tonnesen [23], Stepto et al. [24], Vuorimaa et al. [25], Billt et al. ([26]; [27]), Stepto et al. [28], Macdougall et al. [29], Babineau and Leger [30], Laursen et al. [31], Lindsay et al. [32], Polishuk [33]

Therefore, the gross load will be reduced by 5.65%. This gross load is 28 (min) × 4 (IF) × 0.5 (EF) = 56 ECOs. The 5.65% reduction is 3 ECOs. Therefore, we set 53 ECOs as the "standard". The duration of the repetition is also taken into account, according to what is explained below (18.3.4). As 4 min apply to a STL of 60 and a LIL of 25%, 1.66% will be added. Therefore, 0.70 ECOs (rounded to 1) are added to the "standard" load (54 ECOs so far). We also add the 3 ECOs from the active recoveries. Final result: 57 ECOs.

18.3.4 Weighting the Effort Duration against the Standard Time Limit in a Zone

When planning workouts using various methods (short or long intervals, continuous methods, etc.), different training loads with the same effort time net value in one zone may take place with longer or shorter repetitions and similar density. Several studies have showed that different metabolic variables (lactate, ammonium, slow component, etc.) could be amplified by conducting longer repetitions despite having similar densities. That is why triathletes who perform longer repetitions, even with the same density, are rewarded.

Furthermore, single efforts can be scheduled in a control session, so the model should also reward the possibility of getting closer to a zone's sustainable time. Its corresponding criterion in the model is called Standard Time Limit (STL) (Table 18.8).

Repetition time (RT) is thus weighted against STL with respect to the zone's STL. It is known as the RT/STL ratio and an LI is applied to each zone. In the case of single efforts (1 repetition), where the STL equals or exceeds 1/3 of STL, the LI is directly applied.

Example 1 Run, workout 5 × 3′ zone 6 $R' = 3$. The RT/TLC calculation is 3/6, i.e. 0.5, multiplied by 40 in this zone. So we would initially obtain a 20% LI. On the other hand, density is (5 × 3)/(4 × 3) = 1.25. When applying the equation, in this case LI = 37.085ln (D) − 6.2192, the result is an LI of 2.1 (which we should add to the

Table 18.8 STL by zones and equation for calculating the LI

Zone (#)	STL (min)	Equation for LI
1	900	As from 50% of STL, 10% is applied
2	360	
3	150	
4	60	25 × RT/STL
5	33	35 × RT/STL
6	6	40 × RT/STL
7	4	60 × RT/STL
8	1.9	75 × RT/STL

Based on Beneke [34], Billat et al. [35], Péronnet and Thibault [36]. STL is not included for zones 9 and 10, although it would be about 4 to 6 sec in zone 10
RT repetition time, *STL* time limit criterion

gross ECOs, i.e. 135). We thus obtain an LI of 20% and another of 2.1%; therefore, a net result of 22.1%. We multiply 135 ECOs × 122.1%, and the result is 165 ECOs.

Example 2 If we perform a 14 × 400 m run at 1:15 r' = 1′, on a basis of 10, this represents 14 times 1.25 min (therefore 17.5 min) on a basis of 10. Thus, the gross ECOs would be 17.5 (min) × 9 (IF) × 1 (EF) = 158. Calculating the LI using the *D* and the specific equation, we obtain a result of 4.8%. That is why the load would be 165 ECOs. By adding the LI for the effort duration, the result would be 8.33%, finally obtaining 178 ECOs.

Furthermore, if we performed a single 6-min repetition in zone 6, it would be 6 (min) × 9 (IF) × 1 (EF) = 54 ECOs, to which 100% of LI (40%) would be added, therefore a total of 76 ECOs.

18.4 Other LI for Special Cases: Transition (bricks), Indoor Cycling, Draft and Drag, Zone Subdivision, Progressive Workouts, Specific Strength, Extreme Conditions

The objective of the model is to embrace the range of training modes, means and methods. A number of special situations will be broken down in the succeeding texts. In some cases, the coach's judgement is needed. The rationale is developed in Sect. 18.4 for a more fluent reading.

Triathlon transitions. Performing a mode of locomotion that is "preceded by" to another demands greater energy costs. Previous studies have justified the LI to apply on the load value of the second exercise. Table 18.9 synthesises the LI.

Example Running, after cycling for 2 h on a bike in zone 1, I immediately get off to run 30 min in zone 2. That 30 min run would be 60 ECOs (30 min × IF 2 × EF 1).

Table 18.9 LI for a transition's second segment

Transition	LI (%)
Swimming–**Cycling**	10
Cycling–**Running**	15
Running–**Cycling**	10

The second segment undergoes an ECOs LI (in bold)
Based on McCole et al. [37], Hue et al. [38], Millet et al. [39], Millet and Vleck [40], Vercruysseen et al. [41]

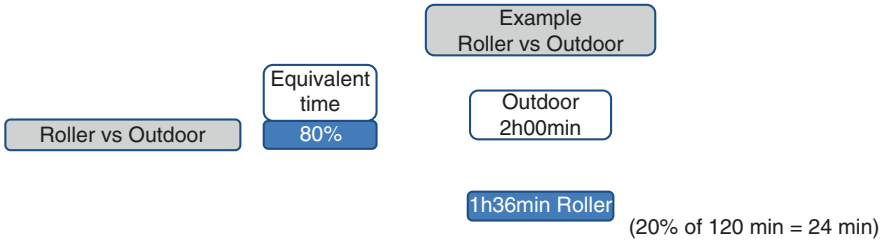

Fig. 18.4 LI for roller workouts

However, I add 15%, resulting in 9. So there are 69 ECOs + another 60 ECOs on the bike, totalling 129

Indoor cycling. When training on an indoor trainer, referred as "roller", or a spinning bike, pedalling is more constant than when cycling on a road, where it is necessary to sometimes stop pedalling, either due to the characteristics of the road or to ride behind another cyclist. *Outdoor* cycling is generally estimated to lead to 80–90% of pedalling time. Thermoregulatory capacity is also limited when training *indoors* [42]. For this reason, the same load can be applied to a roller training, completed in 20% less time compared to the same outdoor workout (Fig. 18.4).

Draft and drag effect. Training while closely following behind another swimmer or another cyclist is known to be easier: the term *drafting* refers to the effect of reduced aerodynamic resistance and *drag* refers to its "suction" effect.

It is possible to go faster while spending less energy. Different studies have calculated this saving, which amounts to 11–21% in swimming and 25–40% in cycling. Therefore, the EF value—0.75 for swimming and 0.50 for cycling—could increase in those situations, according to the coach's judgment (since it can be complex to determine how much session time is affected). For example, the fact of cycling by "alternatively cycling in first position of the group and drafting" entails energy cost oscillations, and this causes additional muscular soreness. Another example is that the latter is not comparable either to being constantly alone, such as in long distance training and under windy conditions, or on a highly wavy track (Table 18.10).

Example Cycling, 3 h alone in zone 1. The normal calculation would be 180 min × IF 1 × EF 0.5 = 90 ECOs. If we apply an IF of 0.60, we would obtain 108 ECOs. When swimming 80 min in zone 1 in the sea, without following anyone, the calculation would be 80 min × IF 1 × EF 0.88 = 70.4 (instead of 60 ECOs in the standard model).

Subdivision of zones. Some methods are programmed in "high or low" zones. For example, we may be brought to work higher or lower in a controlled manner in odd zones. The training value could be qualified here, and therefore the coach would be allowed to adjust the IF. For example, running 8 × 1000 min zone 5. If conducted in a "low" zone 5, I could maybe take on a much bigger volume than in a "high" zone 5. In other words, if we deliberately work in "low" zone 5, the IF should be less than 6, maybe 5. If we would exercise in the "high" part of zone 5, which is closer to 9, perhaps 7.5. This can allow the coach to better determine the load stress. The same applies to zone 3. There is a big difference between medium, high, and low; so the IF could simply be adjusted to 2.5 or 3.5.

Table 18.10 EF modified by draft/drag situations

Situation	Corrected EF	Standard EF
Swimming *without* drafting	0.88	0.75
Constant cycling *without* drafting	0.58–0.60	0.50

Based on Miyashita [19], Bassett et al. [43], Chatard and Wilson [15], Pendergast et al. [20], Zamparo et al. [22]

Progressive efforts. Making an intense effort following a recovery is not the same as making an intense effort during a continuous effort. Therefore, more value is given to the same minutes in zones above zone 1 performed during variable progressive continuous training. By several estimates (energy cost per slow component and increased intensity, muscle damage, dehydration, etc.), and each section that is not in zone 1 is evaluated at 20%.

Example Running 1 h 30 min in zone 1, where the last 15 min are in zone 2. The standard calculation would be 75 min × IF 1 × EF 1 = 75, added to 15 min × IF 2 × EF 1 = 30. The total standard would be 75 + 30 = 105. But if those 20′ of zone 2 have a 20% higher load value, 30 × 0.2 = 6 ECOs more, thus totalling 111.

Specific strength. Cycling on a plain surface, in the same zone, is quite different from cycling over a mountain pass. In cycling, gears can be adjusted, and if the slope is manageable (2–5%), repetitions in zone 3 can be even more easy as well as shifting to harder gears. However, muscle soreness is greater in an uphill run (or downhill), or when running with weights, or pedalling imposing a harder-than-usual gear (low pace), or swimming in a drag bathing suit, etc. These loads should be weighted regardless of the calculations applied for strength training.

Because of the notable level of complexity, a 20% increase is generally applied to the load in these situations, as for progressive efforts.

Extreme conditions. A same workout conducted under different conditions of heat, humidity, extreme cold, etc., should be weighted. Coaches are responsible for estimating the LI, from 10% to 20%. The ability to prolong the effort is indeed going to be limited, so ultimately it is possible that no additional load be accumulated but rather compensated in some way.

18.5 Considerations and Criteria to Quantify the Objective Load Using the ECOs Model for Strength Training

Among the different quantification models for endurance sports, the session (RPE) method of Foster et al. [2] is the only model to include strength training. This model also considers how hard the session is overall. Although other authors have supported the model [44], this method basically weighs the session duration, which is conceptually debatable as to its ability to quantify strength training. Theoretically, we should compute displacement, time or total weight deployed in a workout or speed; but this cannot be applied to all actions and training control practices.

Among all the sport training proposals, worth noting is that of Naclerio [45] who advanced the so-called percent volume, where sets are multiplied by repetitions by the % of 1 maximum repetition (RM). This model—which does not differentiate between mechanics and exercise and could not include other exercises such as jumps or throws—originated, together with the comments of Naclerio and Faigenbaum (personal communication), the concept of an integral model, which is finally adapted to the ECOs.

In line with the rationale applied to the ECOs in Swim, Bike and Run, the energy cost in the Multiple Activity Compendium Tables published by Ainsworth et al. [14] for the ACSM was taken into account for strength. This energy cost is shown to be variable, but we can consider that it is usually lower in the case of strength than in any other activity, even cycling.

The difficulty to maintain the technique is regarded as very low, precisely because of abundant pauses and the low number of repetitions, as well as the wide variety of possible movements (from free to guided).

On the other hand, muscular soreness can be considered to be similar to running or not, but it is evaluated as such since the concept of neuromuscular interaction comes into play: it will always be very high in the case of strength, more than in any other segment.

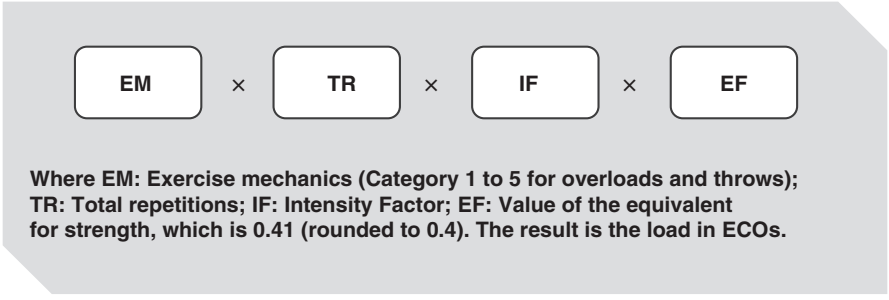

Fig. 18.5 ECOs model for strength training

Table 18.11 Categories of EM of the ECOs model for resistance exercises

Category	Characteristics	Examples
5	Maximum power (Full body power)	Snatch Clean and Jerk
4	Power (Power with multijoint)	Push press 1/4 Explosive squat Dead Lift with jump
3	Multijoint movements of all the upper or lower body (Maximum strength moderate speed)	Bench press Squat Bulgarian Pullups Rope climb Climbing movements
2	Auxiliary multijoint movements ("functional") (Functional strength endurance)	Dumbbell row Single leg squat Multijoint free weight Multijoint elastic bands
1	Pelvic girdle, single joint, low to moderate speed in Variable Endurance machines Any training with low weights and controlled speed Technique	Biceps, triceps, knee extension, knee flexion, calves machine… + Low-intensity technique

Table 18.12 EM categories of the ECOs in jumping and throwing exercises

Category	Characteristics	Examples
5	Maximum power/high impact (Maximum intensity)	High jumps Maximum distance long jumps Fast upper-body movements, with a wide range of fast movements and fast brake Complex tasks at maximum intensity
4	Fast brake/change of direction (Max/high intensity)	High-intensity complex tasks technique Upper body plyometrics technique Maximum height jumps landing at the same height Long jumps nearing maximum distance Single leg complex plyometrics
3	Complex task basis, global exercises Technique, just upward falls (Medium intensity)	Basic complex task movements (2 feet–2 hands) Full body medicine ball training Vertical jumps falling up Basic single leg movements
2	Exercises of full upper body or full lower body, dynamic skills technique (Low Intensity)	Full lower or upper body medicine ball training Move jumps (2 feet or alternating feet at low intensity)
1	Mono/bi-articular on the spot, Technical (Low intensity)	Medicine ball training mono-/bi-articular exercises Jumps on the spot (2 feet)

Table 18.13 IF in simplified strength exercises. Resistance (any kind of weightlifting exercises, including free weights, machines, even own body weight exercises)

Resistance	
Exercise Category	Values (category × IF × 0.4)
5	1.50
4	1.12
3	0.84
2	0.24
1	0.08

Note: "Final" IF are considered by exercise category, and the value of the 0.4 ECO-F is already included. Only those factors should be multiplied according to the category by the number of total repetitions of the exercises in the same category. For lifts, IFs: 0.75/0.70/0.70/0.30/0.20 are taken into account respectively, and 0.95/0.85/0.70/0.50/0.20 for jumps

Density, comparing Swim, Bike or Run, is considered extremely low, so it is not taken into account (e.g. it can be at least 2:1 in S, but it is always below 1:2 in strength; very much below in fact, by more than 1:10.).

Therefore, the ECO is 0.41 for strength (rounded to 0.4).

The model proposes to multiply the value of ECOs × exercise. Category × sets × repetitions × IF. The density is included according to the category of each exercise. All the latter is multiplied by the EF (in this case, 0.4). Figure 18.5 synthesises the concept.

The model generally differs for two types of exercises: resistance vs jumps or throws. Based on this, five categories of EM are differentiated in each. Table 18.11 shows the model for resistance exercises, and Table 18.12 shows the jumps and throws model. The method is the same, but it is used to identify the exercises according to their mechanics and assign them a category.

The intensity is weighted by an IF. It is also possible to link the intensities to various criteria for strength training; see Tables 18.13 and 18.14 in this regard. The training load can already be calculated on that basis, using the product of EM × RT × IF × 0.4.

As argued in Sect. 18.4, the medium or high weight value range in the resistance exercise does not greatly alter the value used to multiply sets × repetitions. We must remember that we have to multiply sets × repetitions × 0.4 to complete the calculation. This is because there is an implicit load range for each goal for which an exercise category is selected.

Table 18.14 IF in simplified strength exercises. Jumps and Throws

Jumps and Throws	
Exercise Category	Value (category × IF × 0.4)
5	1.90
4	1.36
3	0.84
2	0.40
1	0.08

Note: "Final" IFs are considered by exercise category, and the value of the 0.4 ECO-F is already included. Only those factors should be multiplied according to the category by the number of total repetitions of the exercises in the same category. IFs 0.75/0.70/0.70/0.30/0.20 for lifts are taken into account and 0.95/0.85/0.70/0.50/0.20 for jumps, respectively

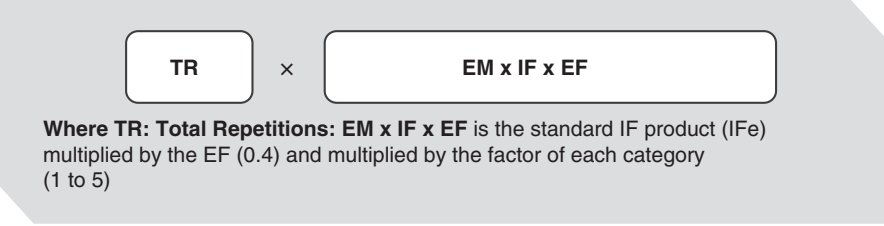

Fig. 18.6 Simplified ECOs-F model

Box 18.5 Examples of ECOs Calculations in Different Sessions

Session Example	Calculations	Load
Bench press: 4 × 10 × 70%	3 × ((4 × 10 × 70)/100)	= 33.6
Squat: 5 × 8 × 75%	3 × ((58 × 75)/100)	= 36
Pullups: 4 × 8 × 75%	3 × ((4 × 8 × 75)/100)	= 28.8
Triceps: 2 × 15 × 50%	1 × ((2 × 15 × 50)/100)	= 6
Biceps: 2 × 15 × 50%	1 × ((2 × 15 × 50)/100)	= 6
Core: 6 × 25	1 × ((6 × 25 × 20)/100)	= 10
Total training load (ECOs)		**= 120.4**
Total training load (simplified)		**= 108.9**

Session Example	Calculations	Load
Snatch: 2 × 3 × 90%	5 × ((2 × 3 × 90)/100)	= 10.8
¼ Explosive squat: 2 × 4 × 80%	4 × ((2 × 4 × 80)/100)	= 10.24
Pullups: 4 × 6 × 70%	3 × ((4 × 6 × 70)/100)	= 20.16
Bench press: 4 × 6 × 70%	3 × ((4 × 6 × 70)/100)	= 20.16
Push press: 2 × 4 × 70%	4 × ((2 × 4 × 70)/100)	= 8.96
Core: 8 × 30	1 × ((8 × 30 × 20)/100)	= 19.2
Total training load (ECOs)		**= 89.5**
Total training load (simplified)		**= 86.4**

Box 18.6 Examples of ECOs Calculations in Different Sessions

Session Example	Calculations	Load
Core: 6 × 20	1 × ((6 × 20 × 20)/100)	= 9.6
Split: 2 × 12	2 × ((2 × 12 × 30)/100)	= 5.76
Own Body Weight Single Leg Squat: 2 × 10	2 × ((2 × 10 × 30)/100)	= 4.8
Clean technique: 2 × 5	2 × ((2 × 5 × 40)/100)	= 2.4
Clean technique: 2 × 5	2 × ((2 × 5 × 40)/100)	= 2.4
Squat: 2 × 6 × 70%	3 × ((2 × 6 × 70)/100)	= 10.08
Pullups: 2 × 4 × 75%	5 × ((2 × 4 × 75)/100)	= 12
Clean: 2 × 3 × 80%	5 × ((2 × 3 × 80)/100)	= 10.68
Harmstrings: 3 × 10 × 70%	1 × ((3 × 10 × 70)/100)	= 8.4
Knee extension: 2 × 10 × 70%	1 × ((2 × 10 × 70)/100)	= 5.6
Calves: 4 × 10 × 70%	1 × ((4 × 10 × 70)/100)	= 11.2
Total training load (ECOs)		**= 82.9**
Total training load (simplified)		**= 64.7**

Session Example	Calculations	Load
Core: 6 × 20	1 × ((6 × 20 × 20)/100)	= 9.6
Split: 2 × 12	2 × ((2 × 12 × 30)/100)	= 5.76
Own Body Weight Single Leg Squat: 2 × 10	2 × ((2 × 10 × 30)/100)	= 4.8

Session Example	Calculations	Load
Clean technique: 2 × 5	2 × ((2 × 5 × 40)/100)	= 2.4
Clean technique: 2 × 5	2 × ((2 × 5 × 40)/100)	= 2.4
Squat: 2 × 6 × 70%	3 × ((2 × 6 × 70)/100)	= 10.08
Clean: 2 × 4 × 75%	5 × ((2 × 4 × 75)/100)	= 12
Snatch: 2 × 3 × 80%	5 × ((2 × 3 × 80)/100)	= 10.68
Box jumps: 2 × 5	3 × ((2 × 5 × 70)/100)	= 8.4
Two-feet hurdle jumps: 2 × 5	2 × ((2 × 5 × 50)/100)	= 4
Step Jump: 2 × 5	5 × ((2 × 5 × 95)/100)	= 19
Total training load (ECOs)		**= 89.1**
Total training load (simplified)		**= 87.4**

Therefore, clarifying the original model in Box 18.4, it is possible to simplify according to Fig. 18.6.

Considering the difficulties in identifying a % of real 1RM, the calculation plus the nature of certain typical exercises (i.e power exercises are related to a certain amount of theorical % at each EM cathegory), the calculation could be simplified using the values in Table 18.13, with an estimated error of ±2–20%, according to the examples shown in Fig. 18.6 and Box 18.5.

Boxes 18.5 and 18.6 show quantification examples, indicating the ECOs model and the same simplified model.

18.6 Special Cases

Exercises using variable endurance machines are poorly addressed in the simplified model. In such cases, especially when working with medium or high weights, the simplified model greatly reduces the ECOs value compared to the original model. Coaches should consider what they prefer in this case: to consider such exercises as category 2 or to use the original quantification of category 1.

Box 18.7 Estimation of the ECOs in Metabolic Circuits: Total Value Per Circuit

Session example: 3 circuits of 30 sec with a 30 sec recovery

(Estimating 25 repetitions in most exercises every 30 sec, except repeated jumps)

Exercises	Calculations	Load
1. Burpees	2 × ((25 × 20)/100) × 0.4	= 6
2. ¼ Squat jumps	2 × ((25 × 20)/100) × 0.4	= 6
3. Clean	2 × ((25 × 20)/100) × 0.4	= 6
4. Skipping	2 × ((25 × 20)/100) × 0.4	= 6
5. Weightlifting	2 × ((25 × 20)/100) × 0.4	= 6

Exercises	Calculations	Load
6. Split jumps	2 × ((25 × 20)/100) × 0.4	= 6
7. Squat + military press	2 × ((25 × 20)/100) × 0.4	= 6
8. Repeated Jumps	1 × ((40 × 20)/100) × 0.4	= 3.2
Total training load (ECOs-F)		**= 45.2**
(Data per circuit, therefore 3 circuits are a total of **135.6**, the same result whether simplified or not)		

Example of session: 3 circuits of 30 sec with a 30 sec pause

(Estimating 25 repetitions in most exercises every 30 sec, except spring jumps), and counting Bike strength Bike / Run ECOs

Exercises	Calculations	Load
1. Cycle 30″ MAP	0.5 × 9 × 0.5	= 2.25
2. ¼ Squat jumps	2 × ((25 × 20)/100) × 0.4	= 6
3. Cycle 30″ MAP	0.5 × 9 × 0.5	= 2.25
4. Clean	2 × ((25 × 20)/100) × 0.4	= 6
5. Run 30″ MAS	0.5 × 9	= 4.5
6. Weightlifting	2 × ((25 × 20)/100) × 0.4	= 6
7. Cycle 0″ MAP	0.5 × 9 × 0.5	= 2.25
8. Military 8-squat + press	2 × ((25 × 20)/100) × 0.4	= 6
9. Cycle 30″ MAP	0.5 × 9 × 0.5	= 2.25
10. Run 30″ MAS	0.5 × 9	= 4.5
Total training load (ECOs-F)		**= 42**
(It is 42 ECOs per circuit, therefore 3 circuits are a total of **126** in total, the same result whether simplified or not)		

Quantifying "metabolic" type circuits, considered in principle at a metabolic level rather than a neuromuscular level, is a pending aspect and difficult to quantify. The circuits would have to be calculated based on the assumption that they were all category 1 (as they are closer to "technique" than to "power"); a number of estimated repetitions (20–30) would have to be established; and metabolic stress of reference needs to be assessed. The latter has already been evaluated in a preliminary way in the course of different reproductions of this circuit type as zone 4 for "Circuit Training" type formats and zone 6 for "Oregon" or "intermittent" formats. Box 18.7 summarises approximate calculations per circuit and therefore would have to be multiplied by the number of circuits to estimate the value of ECOs. In this case, the values produce the same result whether the original or simplified calculations are applied.

A controversial aspect can be the classification of some exercises according to the learning phase, the overload and movement speed and ultimately the strength demonstration. For example, learning a Bulgarian split squat, or using it in a general own body weight exercises circuit, should be classified as category 2. They are commonly used, however, to build up strength (category 3). In the case of snatch

or clean-and-jerk, it could be related to category 2 in a metabolic circuit, with very little weight, or to category 5 to build up maximum power. This consideration is left to the coach's judgment.

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Science-Based Criteria to Identify Talent Among Triathlon Athletes

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Human talent is an uncommon and extremely valuable resource in society that is largely wasted at each generation [1]. This waste of talent is due to a great lack of understanding [2]. Over the years, the fact that talent has been defined in multiple ways points unmistakably to the complexity of the notion and the lack of consensus on the subject. The wide range of terms to designate sports talent generates confusion regarding methodological aspects. Some widely used terms in the literature such as talent detection, identification and selection are not in fact analogous. Detecting and identifying sports talents lie at the heart of the subsequent development of sporting talent. Identification and detection are terms used to describe initial signs of early skills. However, some authors [3, 4] consider that talent detection consists in discovering the potential of young athletes before starting a specific sport, while identification is limited to the process of recognizing athletes already introduced to a sport with the potential to become elite athletes. The word "potential" appears in both definitions, which is significant, as it implies a focus on development and not on innate capacities. When identification or detection is associated with specific tests, problems of validity and generalizability arise. When referring to human potential, we should understand *what can be* and not *what is*, while evaluation procedures refer to *what is* and not *what can be* [5].

Talent identification programmes aim at screening athletes ready to receive extra support to maximize their performance [6]. This identification can be conventional or genetic.

On the one hand, understanding individuals' genetic architecture may represent notable progress in talent development programmes, especially in sports. According to Ahmetov and Fedotovskaya [7], sport performance characteristics is approximately 66% hereditary, especially in the case of endurance sports such as triathlon. Remaining performance would be due to environmental factors [8]. Genetic

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identification programmes differ from their conventional forerunners: since genetic information is specific to an individual and does not change throughout life, it can be applied in theory at any time without considering maturation or peak shape [6]. In addition, a single test would provide the required multidisciplinarity, not achievable through conventional tests [6]. However, some authors have questioned the utility of genetic tests as talent identification programmes. Frazer et al. [9] argue that 100,000 elite athletes in a specific sport would be necessary to recognize a phenotype associated with talent in 15% of cases. What sport has that amount of elite athletes? Is 15% a sufficiently significant percentage?

Camporesi and McNamee [10] argue that statistical associations do not necessarily indicate cause–effect relationships and that there are transferability problems, as tests are generalized to different populations (children, race, etc.) according to standard variables (adults, ethnic groups, etc.). For the time being, whether we they like it or not, genetic tools should be considered as the last resort to direct athletes' early specialization. Athletes tested negatively in their favourite sports would be excluded from promotions, despite their willingness to train hard [6]. According to ethical standards, this fact represents a violation of a person's autonomy [10, 11]. As far as sports are concerned, the Australian Sports Institute [12] urges both family members and coaches to be wary of genetic testing, for its lack of validation and replicability. Not only do genetic tests fail to predict future sporting performance [10] but genetic tests violate child athletes' rights to an open future (ROF), by limiting their career to a single sport [11].

Furthermore, conventional identification programmes are designed to recognize superior capacity [13] or to identify young athletes who possess extraordinary potential for absolute sports success [14]. The challenge of these programmes is to produce specifically designed valid and reliable tests that identify young athletes' capabilities and help their development [15] regarding anthropometric, physiological, psychological, social, and technical, etc., variables. However, the first difficulty, especially in triathlon, consists in selecting sensitive, valid and reliable tests adjusted to a particular sport's performance requirements [13, 16, 17]. Conventional means of identification in triathlon typically use specific tests that measure given aspects of the performance, usually per race segment or per combination of segments and/or in conditions outside the competition itself. This is an added difficulty, given that triathlon is a multidisciplinary sport and, consequently, a talented athlete in one segment can compensate low abilities in another: this is called the compensation phenomenon [18]. In addition, talent may be missed because these identification tests do not reflect athletes' true potential [19]. Therefore, extraordinary talent or aptitude can be identified by looking at the pace of learning rather than the level of skill [20] using, for example, a triathlon performance indicator [21].

Researchers often differentiate between athletes' performance levels by classifying them as follows: (1) elite or not elite; (2) expert, amateur and novel; (3) international, national and local; etc. Each study establishes its own classification, making subsequent comparisons difficult. Therefore, a consensus must be reached on how to identify talent. If we also consider athletes' intrapersonal characteristics, especially in the case of young people (maturation, years of experience, relative age,

etc.), identifying triathlon talent can turn into a very complex and multidisciplinary process.

Factors related to performance in adult triathletes have been studied at length for decades [22–29]. However, there is a shortage of literature on performance factors in young triathletes who have different physical, psychological, social and competitive characteristics [21, 30, 31]. Generally, talent is identified between the ages of 6 and 15 years [14, 32–35], depending on the sport. In addition, aspects such as relative age, maturation, anthropometric characteristics and peak shape must be considered to conduct conventional comparisons between athletes.

19.1 Sports Specialization in Triathlon

Villaroel et al. [36] analysed correlations between chronological age and performance in male triathletes who participated in World Cups, World Series and Olympic Games during the 2007–2010 period. The authors found that age correlated positively with performance and suggested that the 26–32 years age range corresponded to optimal performance in Olympic triathlon. In addition, they observed that participants' ages differed across Olympic Games (28 years for Athens and 30 years for Beijing) compared to the World Cups from 2007 to 2010 (27, 27, 28 and 27 years, respectively), the former athletes being older and, therefore, more experienced. In this line, Knechtle et al. [37] also observed that the peak of Olympic distance performance was reached at 27 ± 5 years for men and 27 ± 4 years for women.

Malcata et al. [38] observed the trajectory of elite triathletes (337 women and 427 men) over a 5-year period (419 sprint and Olympic competitions) to predict performance in the London 2012 Olympic Games. Triathletes' levels were categorized according to their position in the competition, including the athletes who finished a world championship (from 2000 to 2012) or Olympic Games (from 2008 to 2012) in the *top 3*, *top 16,* etc., for the rest of triathletes. The authors suggest that a graphical representation of triathletes' progress can help detect critical performance moments.

Lepers et al. [39] observed that while performance in triathlon decreased with age in all three disciplines, it fell less steeply in the cycling segment. In addition, cycling and running performance decreased less sharply with age in short-distance than in long-distance triathlons. Even the type of triathlon (mountain or road) affected age-related performance: performance decreased more in mountain triathlons.

19.2 Relative Age in Triathlon

The chronological age difference between a subject born in early January and another born at the end of December, within the same year, can be of almost 12 months. Furthermore, in minor category groups competing in triathlon, this difference can increase to 2 years, for example, in the case of Spanish age categories: *prebenjamín* (6–8 years), *benjamín* (8–10 years), *alevín* (10–12 years) and *infantil* (12–14 years),

and up to 3 years in the *young* category (14–16 years). This fact can trigger notable differences, both intellectual and physical, between triathletes, resulting in erroneous interpretations when comparing several individuals. The phenomenon is known as the *relative age effect*. It can lead to inequality of opportunities, since two or more athletes are considered to be sporting equals when, in reality, they are not.

Grouping subjects by chronological age is the most practical and functional solution. Sports federations, especially those that manage minority sports, need to establish categories based on an age scale according to which an acceptable minimum number of athletes can participate, even if the group lacks biological homogeneity. Werneck et al. [40] studied relative age in professional triathletes. They analysed the birth quartiles of 111 triathletes (55 male and 56 female) who participated in the 2012 London Olympics. The authors observed almost significant prevalent participation ($p = 0.07$) of the first 2 quartiles with respect to the next two. In addition, they found a significant dependence between the birth quartile and participation in the games, though only in men.

Ortigosa-Márquez et al. [41] related the selection tests carried out by the Spanish Triathlon Federation (100 and 1000 m in swimming and 400 and 1000 m in the race) over the 2013–2015 period with relative age in 919 boys and 402 girls. For male triathletes, participation frequency of triathletes in the selection races was similar across the different quartiles, though somewhat lower in the last quartile. In all the categories (except in second-year junior, due to a limited sample), significant differences were found between quartiles in some race performances, especially between Q1 and Q4. In all cases, athletes born in Q1 performed better. In addition, the authors observed how the cadet category was more sensitive than the junior category to the effect of relative age. In the case of female triathletes, the results were less consistent: it seemed that relative age sensitivity was found only for swimming, especially in second-year cadets.

Ferriz [21] related relative age with selections in the triathlon technification programmes, as well as with the medals won in the Sports Games of 160 cadet triathletes. The results showed, on the one hand, that a relative age effect existed when triathletes were selected for technification programmes, both in men and women (see Fig. 19.1). In addition, a relative age effect was also observed regarding medal achievement (see Fig. 19.2), especially gold medals.

19.3 Optimal Morphology of Triathletes

One of the explanatory factors of triathlon performance differences is morphology [42]. This factor, however, does not affect the three segments in the same way [39]. Therefore, triathlon should be considered an integral sport, composed of three disciplines and two transitions. The different nature of each discipline makes it difficult to reach a consensus regarding which anthropometric factors optimize performance. If we analyse the specific somatotypes of swimmers, cyclists and runners, dissimilar values are found [22, 43, 44]. It is difficult, therefore, to derive an optimum triathlete somatotype.

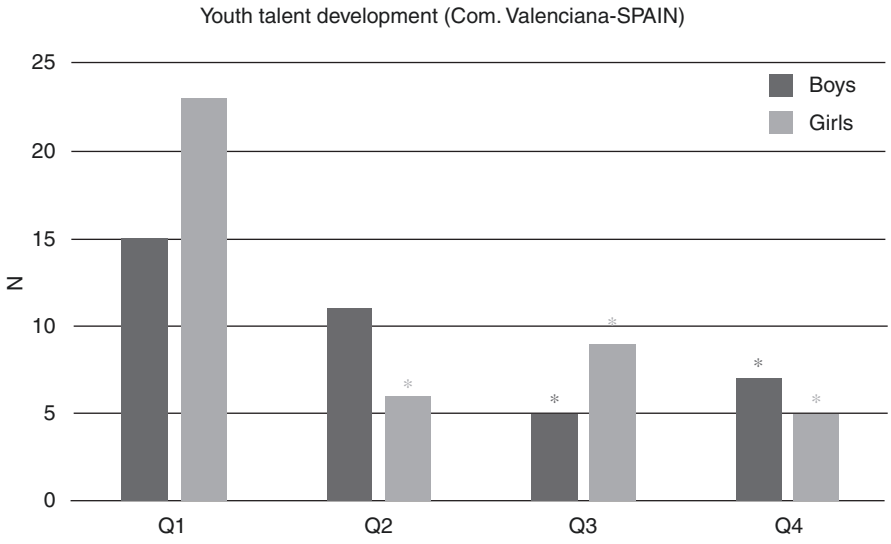

Fig. 19.1 Youth talent development (Valencia Community, Spain)

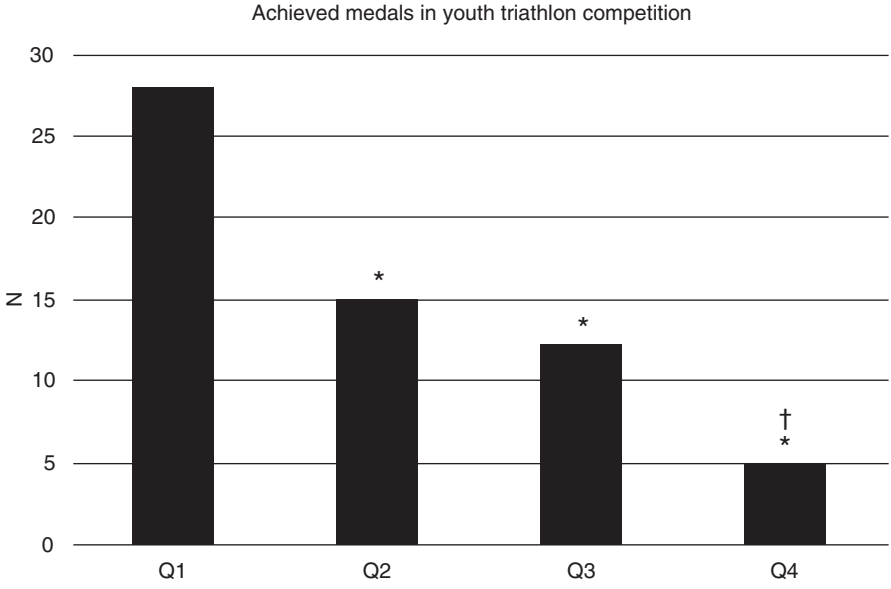

Fig. 19.2 Sports Games Medals won and birth quartile

Triathletes are now trained from an earlier age than a few years ago, so changes can be observed in their body morphology [39]. High stature has generally been related to triathlon performance, since both a greater length of the lower limbs favours racing performance [45] and a greater length of upper limbs are an advantage

for swimming [25, 46]. In addition, triathletes seem to be smaller than swimmers and are more similar to road cyclists and runners [42].

Landers et al. [25] compared the morphology and performance of 71 absolute and junior triathletes all participating in the 1997 Triathlon World Championships. The authors took 28 anthropometric measurements, which were grouped into four parameters: muscle mass, fat mass, bone mass and length of body segments. By applying regression equations, they verified that lesser fat mass was the factor that correlated most with overall success in the race. Length of body segments was also important for performance, especially in swimming.

Later, Landers et al. [47] compared the morphology of junior triathletes in1997 with others in 2011 to check whether junior triathlete morphology had changed during that period. Both men and women triathletes in 2011 obtained significantly higher ectomorphic values. In addition, both men and women were lighter, with a smaller circumference of tight arm and thigh, and a smaller femur diameter. In contrast, only men had greater body segment lengths and lower endomorphic values than their peers in 1997. The authors attribute these differences partly to changes to the competition, as drafting was allowed. In addition, shorter competition distances for juniors means the foot race plays a more important part.

Canda et al. [42] determined triathletes' complete anthropometric profile according to sex, category (senior, under 23 and junior) and performance level (Levels 1 and 2) of 108 men and 45 women, over the 1999–2009 period. On the one hand, they found that in the male category junior triathletes had lower body mass, lower height and lower sitting height than senior triathletes. In addition, junior triathletes presented higher values in iliac crest, supraspinal, biceps, triceps, thigh, leg and pectoral folds and, therefore, greater value of the sum of eightfolds, fat percentage and endomorphic values. They also had smaller chest, waist and relaxed and tight arm perimeters. In girls, junior triathletes presented higher values of iliac crest, supraspinal, abdominal, biceps, triceps, thigh and leg folds. Similarly, junior triathletes showed a greater sum of eightfolds, fat percentage and endomorphic values. On the other hand, when they compared performance levels in boys, they observed differences only in the anterior thigh fold, as it was lower for triathletes categorized as Level 1. In contrast, in girls, they observed differences in all the folds, except in the pectoral folds, as they were lower for Level 1 triathletes. Consequently, Level 1 triathletes presented a lower percentage of fat, a lower sum of eightfolds and lower endomorphic values. In addition, these higher-level triathletes presented a higher percentage of muscle.

Pion et al. [44] evaluated a sports orientation programme in Belgium, applying a range of tests to assess physical condition and motor coordination. They also took 22 anthropometric measurements. Their aim was to relate the indicators with the greatest potential in one of the nine sports evaluated. For this, they examined 141 elite athletes (who represented Belgium in an international event, including triathlon). The authors found that the triathletes' differentiating factors were lower percentage of body fat and endurance during an aerobic test. Detection reliability reached an adequate rate of 91.4%.

In a study on young triathletes, basic anthropometric measures (height, body mass, weight, etc.) were not related to overall triathlon performance based on triathlon performance indicators [21]. In contrast, the percentage of fat in girls was negatively related to performance in the competition race segment. In addition, this percentage of fat in girls was a sensitive factor for distinguishing between levels of performance, so it was highly significant concerning triathlon talent development.

19.4 Physical and Physiological Factors

A widely accepted measurement of aerobic fitness related to success in endurance events such as triathlon is VO_{2max} [48–50]. Other authors, nonetheless [51–53], have determined that maximum work peak in cycling and maximal aerobic running speed predicted overall performance in triathlon to a greater extent than VO_{2max}.

If VO_{2max} is considered to determine performance, Kovárová and Kovár [54] suggest treating VO_{2max} relative to an individual's mass. Ballesteros [55] indicated that athletes with a VO_{2max} below 50 ml/kg/min are unlikely to be successful in endurance sports because international triathletes have values around 75 ml/kg/min, significantly higher than common or amateur triathletes [56]. However, when applied to trainee athletes, who are still growing and have not yet exceeded their peak growth, VO_{2max} cannot be used as a reference to determine an individual's potential. Rowland (in [57], p. 407) found that several studies highlighted that physical training programmes did not improve maximum aerobic power. One of the explanations, according to Katch and Katch [58], is that lack of hormonal response (growth hormone, sex steroids, etc.) before puberty is critical. The other hypothesis is that VO_{2max} or aerobic function cannot improve if the system's efficiency is already maximum at those ages. Confirming this finding, MacDougall et al. [59] found that VO_{2max} relative to weight (ml kg min) was higher in young children than in older children at the same running speed. Rowland's example (in [57], p. 410) illustrates the case of a 14-year-old boy who is able to run twice as fast as a 5-year-old while both their oxygen consumption levels relative to weight were analogous.

Some studies [60, 61] had already reported that the parameters that evaluate submaximal exercise are also good predictors of triathlon performance. The reason is that two athletes with equivalent VO_{2max} values may obtain different performances due to the amount of time they can maintain a certain percentage of their VO_{2max} during the exercise. Some of these variables are the second ventilatory threshold (VT_2) and economy.

VT_2 together with relative VO_{2max} are the two factors that best predict aerobic performance [28]. An essential triathlon performance parameter is the length of time an athlete is able to maintain a VO_2 close to its maximum [62]. This parameter is determined by VT_2, which can vary in all three disciplines. Millet et al. [52] found that the VT_2 of Olympic distance running elite triathletes was at 83.9% of VO_{2max}. However, the authors stated that this variable (measured as percentage of VO_{2max}) is not the most sensitive variable for monitoring performance improvement in elite athletes. In line with this result, Lucia et al. [63] observed that this variable did not

change during the professional cyclists' season. Ishiko [64] examined the value of VT_2 in triathletes and found no significant differences in cycling and running values. They did, however, observe that the VT_2 was higher in those with superior rankings in triathlon, which would imply that it was a good performance indicator. On the other hand, during a training season with Olympic distance triathletes, Galy et al. [65] observed that the VO_{2max} value remained relatively constant compared to greater changes to VT_2. We can thus deduce that it is sensitive to specific training.

Genetic, physiological, anthropometric and training factors are known to also influence economy [66]. For example, race economy was different compared to isolated sport in elite triathletes and in mid-level triathletes [67]. In female triathletes, the energy cost was higher in junior triathletes than in senior triathletes when economy was evaluated after cycling, though this was not observed in men [51]. Globally, it seems that there are no differences in economy between triathletes and long-distance runners [68].

19.5 Performance Indicator in Triathlon

It is easier to evaluate performance in other sports than in triathlon, for example, in swimming or athletics, where influential environmental factors—temperature, humidity, wind, etc.—are controllable or more stable. Performance is usually measured based on the evaluation of personal style and/or distance. Records are even set. In triathlon on the other hand, there are no records and no personal marks, because environmental factors are not only going to be uncontrollable but they are also going to largely condition performance during the event.

The nature of triathlon makes it difficult to use absolute time as a comparative variable of reference. Each triathlon takes place in specific meteorological conditions, with subtle oscillations in distances or circuit, in different tactical positions, etc. For this reason, it is a real challenge to monitor talent in triathlon, especially that of trainee athletes.

Most studies relate performance to the athlete's position in the race, as mentioned earlier. However, performance is not always well represented by triathletes' desired position. When an athlete reaches the finishing line 1 min or 10 s after the first to arrive, we cannot consider that the same performance has taken place. The performance of the athlete coming first may be the same, but the performance of the athlete coming second is not. If the variable is defined as the position in race, the same performance is found in both cases. Another possible case is that both triathletes finish together, and one of them wins the race in the final sprint. For sporting purposes, there is an obvious winner, but from a scientific viewpoint, both athletes have demonstrated an equal performance: the race lasts approximately 2 h and one hundredth of a second is irrelevant in the analysis.

For this reason, a triathlon performance indicator is needed to analyse young triathletes' performance and identify performance factors in order to develop triathlon talent [21]. This indicator can be calculated for the three segments separately and for the overall result in the competition. In addition, results are registered through a

chip-based timing system, which allows to obtain highly accurate individual performance according to portions of the race.

The variable is expressed from 0 to 10,000 where 10,000 is the best segment time, thus the best performance. Remaining individual performance is calculated as a proportional part of that best time score.

$$\text{TPI} = \frac{\text{Winner time}}{\text{Personal time}} \times 10,000 \tag{19.1}$$

Calculation applied by the triathlon performance indicator [21] (Eq. 19.1).

This performance indicator in triathlon [21], unlike that proposed by Clotet and Perez [69], considers the winner's partial time rather than the average time of all participants, since, on the one hand, it establishes the race's best performance and, on the other, data are only assumed to be parametric after having conducted a standardizing test race.

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Altitude Training and Endurance Performance

20

Paul Robach and Carsten Lundby

20.1 Introduction

The decision to organize the 1968 Olympics in Mexico City at an elevation of 2250 m has created a general interest in the effects of altitude on physical performance. This event is also considered the starting point for scientific research investigating the potential beneficial effects of altitude training on physical performance, primarily with a focus on aerobic performance. The hypothesis was that prolonged exposure to altitude hypoxia could promote an improvement in endurance performance, based on the well-established blood response associated with high-altitude acclimatization, i.e. an increase in red blood cell synthesis leading to a higher blood oxygen carrying capacity. Such hypothesis is consistent in light of the strong relationship existing between the total amount of circulating red blood cells (a parameter assessed either by total red blood cell volume (RCV) or total hemoglobin mass (Hb_{mass})) and aerobic performance, as shown in blood manipulation studies [1, 2] and also cross-sectional data indicating 30–40% higher red blood cell volumes in endurance athletes [3]. Presumably the first study on altitude training, published in 1965, reported that training and acclimatization to around 2000 m could indeed improve aerobic performance [4]. The authors commented that "altitude and acclimation can bring back" normal "capacity for maximum aerobic work at altitude", and furthermore that "within one week after return from this altitude, performances in the endurance-type events show considerable improvement". This study was a

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first evidence to support the notion that altitude training could be useful for athletes competing either at altitude or at sea level upon return from altitude.

Both because of the 1968 Olympics and the concomitantly promising scientific results, altitude training has become popular among endurance sports. The method's popularity has probably been reinforced by the many examples of outstanding performances and records achieved by athletes who prepared at altitude, including the world's most successful female cross-country skier [5]. The enthusiasm for altitude training has also generated a great amount of scientific research over the past 50 years, to better understand the mechanisms underlying performance effects, to provide practical recommendations to athletes, and/or to test new methods to optimize the use of hypoxia in association with training.

It is however important to emphasize that today altitude training remains a controversial topic despite the research effort made over the past decades. Not only opinions can diverge between coaches' observations and experimental data, but also the results may be contrasted between scientific studies. In this chapter, our objective is to provide the reader with a critical analysis of the scientific literature, detailing each of the methods currently used by endurance athletes. We also discuss the methodological difficulties inherent to the scientific evaluation of altitude training. Finally, we propose some recommendations to triathletes wishing to use altitude in their training plan.

20.2 A Few Reminders About the Altitude Environment

Exposure to altitude, whether natural (mountain) or simulated (hypoxic mask, room or tent) is associated with a decrease in oxygen availability in the air, and consequently in the blood and tissue. At the cellular level, hypoxia induces the accumulation of transcription factors, the hypoxia-inducible factors (HIFs). The HIF signaling cascade mediates the effects of hypoxia on the cell and upregulates several genes to promote adjustments to low-oxygen conditions, such as those involved in erythropoietin (Epo) production. At the systemic level, among the many physiological responses to hypoxia, three important mechanisms improving oxygen transport during exercise can be mentioned: (1) an increase in pulmonary ventilation via carotid body activation (within minutes of exposure to hypoxia), (2) a decrease in plasma volume leading to the increase of the hemoglobin concentration (within days), and (3) an increase in the total number of red blood cells (within weeks) triggered by an early rise in Epo secretion. Regarding exercise capacity, a very important consequence of hypoxia is that aerobic performance decreases at altitude, as shown by the diminution of maximal oxygen uptake (VO_2max). This well-known phenomenon, which occurs at altitudes as low as 1000 m, and furthermore is found accentuated in endurance athletes, has two practical implications in endurance sports: (1) any endurance performance is systematically decreased during a competition or a performance held at altitude; (2) during any training session performed at altitude, a given absolute working intensity produces a more severe stimulus than at sea level.

The magnitude of the physiological responses to hypoxia depends on the severity of the hypoxic stimulus and the time spent at a given altitude. In practice, altitude training involves exposure to moderate altitude, usually between 1800 and 3000 m (even if higher altitudes are sometimes applied). The main reason for using moderate altitude and therefore mild hypoxia is that altitudes above 3000 m (corresponding to the lower end of the high-altitude domain) may not be well tolerated by athletes, neither at rest because of sleep disturbances, nor during exercise because of performance limitations. Another reason is that altitude training facilities are rare above this altitude. The drawback of using moderate altitude is that expected physiological responses may be less pronounced than those observed at high altitude. To compensate for this, a sufficiently long period at moderate altitude is necessary, so that in practice altitude training camps usually extend over several weeks.

20.3 The Different Methods of Altitude Training

Three main methods of altitude training are available: (1) the "historic" or "classic" altitude training method, developed in the 60s and known as the "live high -train high" (LHTH) method, consists of living and training in the mountains at elevations generally comprised between 1800 m and 2500 m during 2–4 weeks. When using this technique, the athlete is continuously submitted to mild hypoxia during several weeks; (2) a variation of the LHTH method—developed in the late 70s—consists in living at moderate altitude but descending on a daily basis to train at low altitude. This method, called "live high—train low" (LHTL), requires the athlete to be intermittently exposed to hypoxia; (3) a third method, namely live low—train high (LLTH), also known as hypoxic training, conversely consists in superimposing hypoxia to exercise, the athlete otherwise living in normoxic conditions. Other methods, such as intermittent exposure to hypoxia or combined methods using LHTL and hypoxic training are briefly discussed in this chapter.

For each of the three methods, we succinctly review the possible mechanisms of action and we comment on the available evidence in favor or against the method's ergogenic effects on endurance performance at sea level. Our aim is not to provide an exhaustive review of the literature in the field but, as far as possible, to highlight controlled studies.

20.3.1 Live High—Train High (LHTH)

Altitude training facilities, mountain resorts, and/or glacier ski resorts allow the athletes from various endurance disciplines (e.g. running, cycling, swimming, Nordic skiing) to carry out altitude training camps to improve their exercise performance. The "historic" or "classic" LHTH method is probably the most popular among athletes, likely because of the numerous anecdotal reports from world-class athletes demonstrating outstanding competitive results after physical preparation in the mountains. Scientific evidence indicating that LHTH may actually increase

RCV or Hb_{mass} even in elite endurance athletes, such as cyclists [6], swimmers [7, 8], or cross-country skiers [9] reinforce the notion that LHTH approach is worthwhile. However, the hematological advantage conferred by LHTH may not systematically translate into performance improvements since most of the few controlled studies failed to demonstrate any clear benefit on endurance performance in highly-trained individuals [10]. An often-mentioned argument to account for the lack of positive effect after LHTH is the difficulty in managing training intensities in a hypoxic environment where the athletes' aerobic capacity is impaired. This problem had already been emphasized by Adams and co-workers [11], explaining the lack of positive effect of LHTH on VO_2max and 3000-m running performance in conditioned middle-distance runners. Indeed, during LHTH, the athlete has to choose between two strategies. The first one is to maintain altitude training intensities at the same absolute level as in normoxia, provided the level of intensity is sustainable at altitude. This option, which increases the overall training load, potentially augments the risk of chronic fatigue. The second one is to maintain altitude intensities at the same relative level as in normoxia (therefore reducing absolute speed/power). Beyond the question of a possible detraining effect with the second alternative, decreasing speed may be also quite disturbing in sport disciplines in which pacing control strategies during training are rather invariant (such as running or swimming). In those athletes, even slight changes of effort perception during hypoxic exercise may negatively alter their training habits and therefore progression. In practice, as a precaution against overreaching or overtraining, coaches generally prefer to rely on the second option, i.e. reducing training intensities, even if some sessions may be performed at high intensities. Although this cautious alternative makes sense, particularly in athletes with no prior experience of altitude training, it is not excluded that the commonly applied reduction in training intensities during LHTH may be indeed inappropriate, explaining at least in part the lack of positive effect reported in previous studies. Conversely, maintaining the same absolute training intensities as at sea level might induce positive effects [10]. At least this strategy has no negative influence on the overall training response when using the hypoxic training approach (see below) [12]. Nevertheless, this view is speculative in the absence of data comparing different training interventions during LHTH.

In summary, there is still a gap between science and practice regarding LHTH ergogenic effects. Several controlled studies in highly trained individuals suggest no advantage, whereas anecdotal evidence and the adherence of many athletes to the method instead suggest a benefit. Today, the evidence for or against LHTH benefits may not be robust enough to draw a definitive conclusion. The high variability existing between individuals in response to altitude training [13] may explain to some extent the discrepancies between science and practice. Further, well-controlled LHTH studies are therefore warranted, keeping in mind that methodologically, it may be extremely difficult to isolate the role of hypoxia when comparing two groups training either at natural altitude or at sea level, i.e. in very different environmental conditions. In practice, applying the same training stimulus in this context seems very challenging, which may bias the results.

20.3.2 Live High—Train Low (LHTL)

As indicated above, a major disadvantage inherent to "live high—train high" altitude training is that athletes cannot fully compensate for the limited oxygen and therefore cannot train at their maximum ability. To overcome this problem, an alternative approach, namely "live high—train low" (LHTL), was proposed in the late 70s (see Gledhill & Froese) and further developed in the early 90s [14]. The idea was to take advantage of the beneficial effects of moderate hypoxia on erythropoiesis while avoiding the potential problems associated with exercising at altitude. In their landmark study published a few years later, Levine and Stray-Gundersen demonstrated that living at moderate altitude for several weeks and training near sea level improved endurance performance in collegiate runners [15]. Besides the fact that this compelling study has allowed the LHTL method to spread among endurance athletes—without however supplanting the popularity of the historical LHTH method—it has also generated substantial scientific production. One reason is probably the ease of implementation of the LHTL method, which does not require sports facilities in the mountains and can even be implemented at low altitude by using hypoxic rooms or tents producing normobaric hypoxia (instead of hypobaric hypoxia at natural altitude). Another reason may be the convincing nature of the hypothesis, which is based on an effective stimulation of erythropoiesis at rest in intermittent hypoxia [16], coupled with training under normal oxygenation conditions which is much easier to implement than at altitude. After almost three decades of research on the LHTL method, the most widespread view suggests that improvements in endurance performance may be achieved after such preparation. Although the reported increases of 1–2% can be considered small, they are nonetheless worthwhile for elite athletes in whom training adaptations have reached a plateau. The most established mechanism underlying LHTL improvement is related to a 3–4% RCV expansion [17] potentially boosting oxygen transport during aerobic exercise. An alternative mechanism related to an improvement in skeletal muscle function and leading to augmented exercise economy may also play a role on exercise performance [18]. However, this hypothesis has been challenged by a subsequent work [19–21]. At the practical level, the abundant literature on LHTL has also led to establish recommendations for athletes and coaches to optimize the hypoxic intervention (see below).

Another point to consider for the LHTL approach is the choice of the hypoxic stimulus, which can be either hypobaric hypoxia (natural altitude) or normobaric hypoxia (simulated altitude). In practice, using natural altitude during LHTL offers the opportunity of a stimulating mountain environment. However, daily commuting between altitude and plain may induce fatigue. Using simulated altitude resolves the problem of transportation but implies substantial confinement which may not be well tolerated by some athletes. At the physiological level, the two interventions produce the same level of partial pressure of O_2 in inspired air, which is commonly presumed to be the principal physiological stimulus for adaptation to hypoxia. However, since barometric pressures are different (i.e. lower at natural altitude), it has been proposed that adaptive responses to prolonged hypobaric hypoxia may differ from those induced by normobaric hypoxia [22]. Nevertheless, this question

remains controversial [23]. Data comparing hypobaric versus normobaric LHTL in triathletes and indicating no difference in the Hb_{mass} response or performance changes suggest that both interventions produce similar effects [24].

The widespread view that LHTL favors sea-level endurance performance is however still a matter of debate, as illustrated by contrasted reviews on the topic, concluding either to benefits [17, 25–27] or to a lack of effect [28, 29]. The most recent LHTL studies in endurance athletes do not reconcile views either, reporting either positive [8, 24, 30] or no effect on endurance performance [20, 31]. The most recent review on LHTL concludes that "the scientific evidence for both hypo- and normobaric LHTL to improve sport-specific exercise performance seems to lack strong scientific support" [32], highlighting potential confounding factors in the methodology and/or data interpretation. In particular, training effects may bias the results since hypoxic and training stimuli coexist during any LHTL intervention, and training itself can be a powerful stimulus for performance enhancement, even in highly trained individuals. To adequately isolate the specific role of hypoxia on performance, it is therefore extremely important to apply identical training regimen between altitude and control athletes. In a recent hypobaric LHTL study in cross-country skiers where we systematically mixed the athletes from both groups during training to minimize this bias, we observed similar performance improvements between groups, suggesting an overall training effect but no altitude acclimatization effects [20].

In summary, there is still no scientific consensus regarding the ergogenic effects of LHTL in endurance athletes. Outstanding performance attained by some athletes following LHTL preparation suggests that the method is effective in certain individuals, but there is insufficient evidence to suggest that LHTL promotes robust improvements in sea-level endurance performance in endurance athletes. As for LHTH, the high variability existing between individuals in the response to altitude training [13] may explain to some extent the discrepancies between science and practice about LHTL, and between the studies themselves. As pointed out in recent reviews [32, 33], well-designed and controlled LHTL studies are therefore warranted to clarify the specific effects of hypoxia in highly-trained endurance athletes.

20.3.3 Live Low—Train High (LLTH)

Also known as "hypoxic training", this method aims to improve exercise performance by combining exercise and hypoxic exposure during training. Superimposing a hypoxic stimulus to exercise is thought to induce a higher metabolic stress on the skeletal muscle, in turn promoting stronger adaptive responses leading to performance enhancement. Such assumption is supported by the fact that hypoxia evokes rapid cellular responses via hypoxia inducible factors (HIFs), and in particular an increase in vascular endothelial growth factor expression, potentially improving muscle capillary network and therefore oxidative capacity. In practice, LLTH can be implemented during specific training sessions by using normobaric hypoxia, either via a mask connected to a device delivering hypoxic air, or breathing into a hypoxic

room. Various protocols have been tested in terms of altitude (2500–4000 m), exercise intensity (moderate to high) or exposure duration (30 min–1 h). Although attractive due to the ease of implementation, LLTH yielded contrasted results, both in untrained and trained individuals. A review on this method [34] highlights the difficulty to establish clear effects of the LLTH method on (1) the respiratory capacity of the skeletal and (2) systemic oxygen transport and aerobic performance, leading the authors to conclude that "a functional benefit for competition at sea level cannot be expected with a training intervention using hypoxic training as an adjunct to normoxic training in athletes". More recent research on LLTH also failed to demonstrate a robust effect on sea-level aerobic performance or skeletal muscle function, as we have seen in a double blind study [35]. The fact that angiogenesis has not been demonstrated in human skeletal muscle following hypoxic training [33] does not support the assumption that HIF-induced cellular changes translate into functional changes into muscle tissue, which may explain why the LLTH method does not confer benefit on VO_2max.

In summary, the lack of scientific evidence supporting the LLTH approach probably explains why endurance athletes may not frequently use this technique. The fact that expected improvements following LLTH primarily relates to "peripheral" muscle changes rather than "central" hematological alterations may also explain the lack of attractiveness for endurance athletes, who may be more interested in methods yielding systemic improvements in oxygen transport. Recently, an alternative LLTH approach using very high-intensity exercise in hypoxia—namely repeated-sprint training in hypoxia—has been proposed to improve exercise performance. Although supporting evidence is debated [29], and furthermore, the mechanisms governing potential performance changes are unclear, data nonetheless indicate that this method can improve repeated-sprint performance at sea level [36]. However, since repeated-sprint training in hypoxia is not reported to boost VO_2max at sea level [36], it seems difficult to recommend this approach for endurance athletes. Interestingly, very recent data indicate that high-intensity resistance circuit-based training in hypoxia (15% O_2) improves VO_2max in moderately trained subjects (51 $ml \cdot min^{-1} \cdot kg^{-1}$) [37]. Again, the mechanism of this improvement remains elusive. Nevertheless, if such finding can be confirmed in endurance-trained athletes, resistance circuit training in hypoxia could be considered for endurance athletes.

20.3.4 Other Methods

Resting exposure to severe hypoxia (4000–5500 m) during intermittent (few minutes, repeated) or prolonged (<3 h/day) hypoxic exposure has been proposed to improve sea-level performance. In the absence of any evidence showing significant physiological changes potentially associated with improving athletic performance at sea level, those methods cannot be recommended for preparation of endurance athletes [38].

Recently, it was reported that combining LHTL and repeated-sprint training in hypoxia in hockey players (baseline VO_2max: 52 $ml \cdot min^{-1} \cdot kg^{-1}$) substantially improved

running performance (through indirect assessment of aerobic capacity) [39]. Although it seems difficult to recommend such approach for endurance athletes based on this finding obtained in team-sport players, further research might test this model via direct assessments of VO_2max and endurance performance in endurance athletes. It is important to note, however, that in such intervention which combines three factors (hypoxia, regular training, and repeated-sprint training), data interpretation is complex and the actual role of hypoxia on performance may be difficult to establish.

20.4 Altitude Training for Competing at Altitude

In the previous section, we have highlighted that altitude training is widespread among endurance athletes, which is in contrast to the absence of scientific consensus supporting its benefits for sea-level endurance performance. In this context, scientific recommendations for sea-level performance should remain cautious. Things are somewhat different for performance at altitude since experts concur that a period of altitude training is beneficial, if not necessary, to adequately prepare an endurance competition held at moderate altitude. This makes sense as altitude alters endurance performance and thus the athlete has to "integrate" these changes to minimize the risk of counter-performance. It is well-established that acclimatization to high altitude is associated with robust physiological changes concurring to improve oxygen transport at rest and during exercise. While acclimatization to high altitude has little positive effect on the initial VO_2max impairment, this process progressively allows endurance capacity to re-increase, without however reaching initial sea-level value. Physiological responses are somewhat different at moderate altitude, since 3 weeks of LHTL at 2340 m progressively restores VO_2max and endurance performance at 2320 m in highly-trained cyclists [40]. This data strongly support the notion that altitude training may be crucial before competing at altitude, and furthermore that athletes are advised to expose themselves to this altitude for at least 14 days before the competition. On the other hand, it was recently shown that endurance athletes considering LHTL before competing at 1780 m should live at the altitude of the competition and not higher, since higher altitudes (2454–2800 m) may require more than 3 weeks of acclimatization to minimize performance decrements [41]. Finally, recent data suggest that classic altitude training at 1380 m during 14 days or normobaric LHTL at 3000 m during 14 days may both allow the athletes to achieve reasonable acclimatization before the competition at 1380 m [42]. By contrast, in another report, normobaric LHTL was not found to improve VO_2max at 2500 m [43]. Nevertheless, a majority of studies support the view that a period of 2 weeks of classic altitude training or LHTL can be recommended before competing at altitude.

The potential beneficial effect of LLTH for performance at altitude has received less attention. LLTH may lead to adaptive responses, ultimately minimizing the performance decrement at altitude. However, its benefit remains unclear [34]. We have recently reported that hypoxic training might facilitate endurance capacity at moderate altitude [35] but this issue needs to be further examined before specific recommendations can be made for endurance athletes.

20.5 Altitude Training for Performance in Triathlon

Performance in triathlon is complex since it depends on several key physiological determinants of endurance exercise capacity, which are (1) VO_2max, (2) exercise economy during various exercising modes (swimming, cycling, and running), and (3) lactate threshold. While there may be minor potential for further improvement in VO_2max with training in elite endurance athletes [9], it is not excluded that altitude training—despite inconclusive scientific evidence—may to some extent boost VO_2max, and thus endurance potential in triathletes. In support of this empirical notion is the long tradition of LHTH altitude training among elite triathletes, frequently using altitude training centers such as Font Romeu (1800 m, France), Sierra Nevada (2320 m, Spain), St. Moritz (1822 m, Switzerland), Flagstaff (2106 m, Arizona) or Snow Farm (1520 m, New Zealand). Recurrent examples of medals obtained at the Olympics or world championships by these athletes after LHTH again illustrate the "gap" between science and practice. LHTH in triathlon generally consist in training camps lasting up to 3 weeks, the training load being reduced during the first days, then returning to high training volumes comparable to sea-level training. Upon return to sea level, tapering is generally observed during 2–3 weeks before the competition. LHTH is used by triathletes during final preparation before competing but also during general preparation in an attempt to boost training capacity. Polarized training seems to be widespread among triathletes using LHTH, which makes sense given the very high training volumes attained by elite triathletes (35 h/week).

Natural or simulated LHTL is also considered by elite triathletes. Although this practice is less common than LHTH, examples of outstanding performance obtained by elite triathletes after simulated LHTL (using for instance the hypoxic room facilities in Prémanon, France) also suggest that this approach could be worthwhile for some athletes. However, implementing LHTL in triathletes is further complicated by the very high training volumes required each day, which potentially collide with daily exposure to hypoxia which should be the longest possible. Using collective living spaces in hypoxia in complement to hypoxic sleeping rooms may help resolve this issue. By contrast, sufficient daily exposure to hypoxia may be difficult to attain when using hypoxic portable tents, as previously highlighted in triathletes [31].

Hypoxic training, i.e. LLTH, does not seem to be widespread practice among triathletes, perhaps because of the uncertainty as to the method's benefits.

Although beyond the scope of this chapter, another issue potentially impacting triathlon performance is the hot air/water environment encountered during some competitions (this is for instance a concern for the next summer Olympics to be held in Tokyo in 2020). Heat training, as well as heat acclimatization, which may help to minimize performance decrements in the hot environment [33], will likely be recommended for the next Olympics. Recently, it was proposed that combining heat and hypoxic exposure may have the potential to improve exercise performance, based on the "cross-adaptation" concept. Whether this approach may have some ergogenic effects on endurance performance remains unknown in the absence of

robust scientific evidence or compelling mechanism [44]. Accordingly, to date, it cannot be scientifically recommended for triathletes to superimpose hypoxic exposure to a heat training program.

Finally, since there is no major triathlon competition held at altitude, world-class triathletes are not required to consider altitude training in their final preparation before events such as the Olympics or the World Triathlon Series. It should be noted however that a certain number of world-class triathletes use altitude in their final preparation before these competitions. On the other hand, important triathlon events are held at altitude (e.g. Colorado), including popular very-long distance triathlon competitions (e.g. Embrun Ironman). In view of the potential benefits of altitude training for competition at altitude, it is thus highly recommended for triathletes to consider altitude preparation before attending such events.

20.6 Practical Recommendations

Making firm scientific recommendations to optimize performance enhancement after altitude training remains a difficult exercise given the scientific controversy existing over the method's effectiveness. Most of the available recommendations are therefore based on empirical evidence from the coaching experience (with the exception of Hb_{mass} for which there is more evidence). Here below is a summary of the main recommendations.

20.6.1 Is There an Optimal Hypoxic Dose for Altitude Training?

In the absence of conclusive evidence regarding endurance performance enhancement after altitude training, the notion of an "optimal hypoxic dose" proposed by Levine et al. [45] mostly refers to the dose that results in a significant increase in red blood cell volume or Hb_{mass} following altitude training in athletes. The hypoxic dose depends on three parameters, which are (1) the level of hypoxia, (2) the duration of daily exposure to hypoxia, and (3) the number of consecutive days in hypoxia. The optimal range for natural altitude is generally comprised between 2000 and 2500 m [27], while higher simulated altitudes $\leq$3000 m may be applied during normobaric LHTL [46]. Classic altitude training is sometimes implemented at altitudes lower than 2000 m, raising the question of the sufficient erythropoietic stimulus. Daily hypoxic exposure during LHTL intervention should be as long as possible, i.e. up to 20–22 h/day [27] even if durations of 16 h/day are also considered adequate [26]. Shorter durations of 8–11 h/days are also used but they are not currently recommended. Finally, the duration of an altitude training camp should be at least 3 weeks to expect significant Hb_{mass} expansion [27]. Shorter durations of 2 weeks are also recommended [17], which may be relevant for athletes who cannot afford the time for the recommended 3–4 weeks, provided they are able to "physiologically" respond quickly enough. To simultaneously account for elevation and time, a model named "kilometer hours" has

recently been proposed [47]. This model, predicting for instance a 3% increase in Hb_{mass} following 700 km·h of hypoxia, can be a valuable tool to avoid implementing inadequate altitude training camps.

20.6.2 What Is the Timing of Return from Altitude Training for Optimal Sea Level Performance?

"By no means is the issue of timing an altitude training camp prior to competition a trivial matter" [48]. This statement in a recent review well summarizes the complexity of an issue that coaches and scientists have been trying to answer for a long time. Although many recommendations are available today, it is important to remember once again that these are based on experience feedback rather than science, as highlighted in the above-mentioned review's conclusion: "Ultimately, the best time to return from altitude training prior to a major competition for peak performance remains undocumented from a physiological standpoint" [48].

While recommendations on timing may vary between experts or sport disciplines, the general trend seems to indicate that competition should be planned either immediately (i.e. within 48 h) after return to sea level to benefit from altitude-induced physiological effects which are known to decline fairly quickly, or after 2–3 weeks of sea level training after the descent (in this case tapering is often recommended). Between these two "optimal windows", there seems to be a period of potential fatigue unfavorable for competition. Although the option to compete immediately after return from altitude makes sense from a physiological standpoint, its practical implementation may be complicated for the athlete, considering travel constraints. Competing 2–3 weeks after return is probably a safer option, but since most of altitude-induced physiological effects have disappeared at that time, this raises the question of the actual effect of hypoxia on the observed performance improvements. Indeed, well-conducted intensive training camps (at altitude or not) followed by adequate tapering have the potential to improve endurance performance, even in highly-trained athletes.

20.6.3 Is Iron Supplementation Required During Altitude Training?

Sufficient iron stores are required to support red blood cell synthesis. The view that low iron store is a limiting factor for erythropoiesis during altitude training is suggested by retrospective data indicating that iron supplementation during altitude training camp may improve Hb_{mass} production in athletes with low pre-altitude iron stores [49]. Prospective data show that iron supplementation either boosts [50] or does not increase the Hb_{mass} response during altitude training [51]. To increase the chance of getting a positive Hb_{mass} response, it is generally recommended for endurance athletes to initiate oral iron supplementation before starting natural or simulated altitude training, until the end of the camp. However, in the absence of robust

evidence supporting the notion that iron supplementation is necessary to optimize the Hb_{mass} response to altitude training in iron-repleted athletes, it may be appropriate to supplement first those athletes who are significantly iron-depleted.

20.7 Conclusion

Although altitude training—a common practice among endurance athletes—is generally perceived as beneficial for sea-level endurance performance, to date there is no scientific consensus on this issue. This can be explained since altitude training induces at best minor performance improvements, which makes it scientifically difficult to dissociate the real effects of hypoxia compared to those of training. Furthermore, placebo effects should not be totally overlooked in an area where there is a positive a priori. On the other hand, there is no data showing that altitude training has a negative influence on performance, suggesting that this training practice is not deleterious for athletes. The large individual variability existing in the response to altitude in humans also applies to altitude training, perhaps explaining why some individuals may benefit from this preparation and others do not. Since it is not possible to finely predict the individual response to altitude training, endurance athletes wishing to include altitude in their preparation should test their own tolerance during a preliminary altitude training camp, using either LHTH or LHTL. Careful physiological and performance monitoring at this stage will help the athlete to better assess the potential constraints and benefits of altitude training.

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The Overtraining Syndrome

21

Davide Susta

21.1 Definition

A clear clinical definition of the overtraining syndrome is still lacking despite decades of investigations, hypotheses, debates and a joint ECSS-ACSM consensus statement [1]. In this chapter, the OTS is defined as a systemic, multifactorial, clinical condition affecting athletes exposed to excessive training load/insufficient recovery and presenting with mood and behavioural disturbances, pathophysiological responses to exercise and reduced sport specific performance. To be able to more accurately assess and to manage triathletes, it is important to put emphasis on the physiological fact that a temporary reduced performance is part of the normal training plan, based on the 'supercompensation' principle: after a period of overload training (lasting few weeks), the human body needs to recover to better adapt to more demanding workloads and an active recovery period, known as 'taper', is administered so that after tapering performance will increase. This drop in performance is a physiological condition known as 'functional overreaching' (FOR) and it should not last for more than 4 weeks. To proficiently manage their athletes, a multidisciplinary team of practitioners (coaches, trainers, psychologists and physicians) should be able to differentiate between FOR and 'non-functional overreaching' (NFOR), which is characterised by a longer recovery time needed to fully restore performance level. NFOR has to be considered as the first level of pathophysiological adaptations associated with a combination of excessive training and insufficient recovery. NFOR can be caused by a sudden training load increase (volume and/or intensity), as well as by additional, i.e. non-training related, psychological stress triggering or precipitating maladaptive responses (e.g. personal circumstances affecting athlete's life). It is important to emphasise

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here that the currently accepted definitions of FOR, NFOR and OTS are to be seen as different stages of an adaptations' spectrum, ranging from physiological to pathological, reflecting systemic responses to physical and psychological stressors. NFOR and OTS are maladaptations due to acute and chronic disruptions of many processes involved in preserving an athlete's body homeostasis and well-being.

21.2 Epidemiology

Worldwide triathlon participation is very large with more than 2 million people competing in events across the world year-round. Because of the intrinsic endurance/ultra-endurance nature of the sport, and also because of the three different sports specific movements involved (swimming, cycling and running), triathletes are highly exposed to both mechanical and metabolic overloading. Triathlon-related injuries in both professional and amateur triathletes have been reported for the last two decades [2, 3]: risk factors have been identified and suggestions to minimise mechanical overloading leading to overuse injuries have been proposed [4]. On the other hand, despite triathlon being a high physiologically demanding sport requiring high training volumes, epidemiological data on OTS among top athletes, as well as in other categories (amateur, young elite, senior) are lacking. There is a general agreement among practitioners that OTS is a relatively rare condition among elite triathletes, whereas it is usually under diagnosed among amateur athletes and young triathlon prospects.

Birrer et al. [5] reported data on NFOR/OTS among elite Swiss athletes (63 male, 76 female, 26 sports, 31 disciplines) and only one triathlete out of 139 elite athletes reported symptoms suggesting functional overreaching, without symptoms/signs of NFOR/OTS. Another epidemiological study providing interesting insights about OTS has been conducted by Matos et al. [6] in elite young English athletes. This study did not include triathlon. Nevertheless, their findings are useful to better understand the role of exercise intensity and the effect of non-training sources of stress in young elite athletes experiencing symptoms of NFOR/OTS. Competitive level, which is usually associated with higher training volumes and higher metabolic intensities, seems to be a major determining factor: at club level, 21% of athletes experienced NFOR/OT, while at international level, the percentage was 45%. Being involved in individual sports is also associated with higher risk of developing NFOR/OTS symptoms (37% vs. 17% in team sports athletes) or staleness as reported by Kenttä et al. [7] (48% in individual sports vs. 30% in team sports athletes). Interestingly, even if only partially relevant to triathletes, is the percentage of young athletes playing 'low intensity' sports and experiencing NFOR/OTS (34% according to their survey), thus indirectly suggesting a major causative role in developing OTS for psychological stress/mental overloading, such as pressure from relatives and sponsors, anxiety in relation to their future as top athletes, personal/social life stress, school/work-training balance, etc.

21.3 Pathogenesis

A detailed description of the OTS various pathophysiological hypotheses is beyond the scope of this chapter (please see [1] for further details). Nevertheless, it is important to highlight two theories relevant to manage triathletes. Firstly, Autonomic Nervous System (ANS) imbalances are common findings in athletes diagnosed with OTS and, in addition, ANS changes has been reported in studies on athletes with FOR and NFOR/OTS [8–10]. Secondly, hypothalamic-pituitary-adrenal and hypothalamic-pituitary-gonadal hormonal axes dysfunctions were suggested as pathophysiological hypotheses and studies have confirmed their role in athletes developing FOR/NFOR/OTS [3, 11–14].

21.4 Diagnosis

The OTS diagnosis-making process may appear straightforward because of the current definition centred on a long-lasting drop in performance, but the complex pathophysiology of this syndrome should not be underestimated because of the implications on triathletes' overall well-being and ultimately their athletic career. Few factors affect the diagnosis-making process. Firstly, 'Overtraining Syndrome' as definition of this clinical condition is now widely accepted but a definitive list of symptoms presenting at the same time and making it a syndrome is still lacking, despite a general agreement on OTS signs and symptoms as typical responses to various forms of stress, both sport specific and non-sport specific. Secondly, the diagnosis process is challenging because the current OTS definition is not clear, clinically speaking, and thresholds between different stages (FOR, NFOR, OTS) are inadequately defined to guide the diagnostic process; Thirdly, 'prolonged reduced performance' as 'clinical' outcome does not always correlate with usual clinical blood biomarkers (metabolic, hormonal, haematological) and specific clinical tests, thus making it more difficult to follow the usual clinical processes to confirm the OTS as diagnosis and to plan a therapeutic intervention designed to counteract the hypothesised causative factors.

21.4.1 Medical History

The first step in approaching an athlete presenting with a potential OTS is to carefully investigate not only his/her medical history but also training related aspects. In particular, it is useful to ask questions to identify recent changes in training volume/intensities, loss of motivation to practice and to compete, willingness to practice, enjoyment of training, perception of performance level, perception of increased effort while exercising, perception of practice efficacy/frustration, mood disturbances, anxiety, irritability during practice sessions, loss of appetite, decreased libido, overall sleep quality, clinical depression symptoms, unexplained weight loss,

increased vulnerability to viral/bacterial infections especially of the upper respiratory airways (more frequent than usual cold or flu virus symptoms), recent exposure to environmental stressors such as high altitude hypoxia, extreme hot or extreme cold (including participation to competitions taking place in such environments). Because of its complexity, OTS diagnosis is usually made by first ruling out systemic conditions associated with signs and symptoms of 'chronic fatigue', and by confirming OTS features as second step. Systemic conditions such as bacterial, viral and parasitic infections (e.g. mononucleosis), heart and lungs pathologies (including bacterial infections and flu virus), nutritional deficiencies (e.g. iron) and eating disorders, autoimmune diseases, endocrine and neuro-endocrine diseases (e.g. hypothyroidism), depression and other psychological disorders, sleep disorders, chronic inflammation and blood diseases (including anaemia and leukaemia) should be considered in making a differential diagnosis.

21.4.2 Specific Tests

There is evidence that exercise testing provides useful information to identify FOR. In general, a blunted cardiac response to submaximal (as well as maximal) power output has to be considered a useful sign to identify an initial maladaptation to training overload. In a study conducted by Le Meur et al. [15], it has been shown that sport specific performance was reduced and other physiological parameters were concurrently reduced after a period of deliberate training overload (consisting of a practice session duration increase by 40%). Experienced triathletes were asked to complete a maximal incremental test by running on a tartan track, for 3 min, at selected constant speed (starting at 11 km/h and increasing speed by 1 km/h each stage) and rest for 1 min in between bouts at increasing speeds. While the tested triathlete was resting between bouts (1 min), blood samples were collected (earlobe) to measure blood lactate concentrations at the end of each stage (running speed). Heart rate was also recorded at each running speed stage. These two parameters, heart rate and blood lactate concentration recorded during an incremental test administered before and after a period of deliberate overload training, are combined in the following equation to generate an OR index. This index provides a tool to classify a triathlete as OR and to discriminate between normal response and initial maladaptation to training loads:

$$\text{OR index} = 0.17\,\Delta HR + 0.88\,\Delta\left[\text{La}\right]_{\text{b}} + 1.36$$

where ΔHR and $\Delta[\text{La}]_{\text{b}}$ are the variations recorded pre- and post-overload training.

A negative value of the OR index suggests maladaptations.

The only test sensitive to differentiate between NFOR and OTS has been proposed by Meeusen et al. [12]. Ten athletes diagnosed with NFOR (5, one female) or OTS (5, one female, one male was a triathlete) were asked to complete a two-bouts

incremental graded exercise test until exhaustion administered 4 h apart on the same day. Blood samples were taken pre and post each maximal test with the aim of measuring hormonal responses to maximal exercise until exhaustion. After the second maximal incremental test, both adrenocorticotrophic hormone (ACTH) and prolactin (PRL) were increased in NFOR group and were unchanged (compared to baseline values) in OTS group. Despite being a sensitive and specific test, it is difficult to be implemented, mainly because of the protocol (two maximal tests during the same day) and also because of specific blood hormones analyses costs and constraints. An additional information provided by this study is worthy of note: again, similarities between OTS and stress-related disorders have been proven, putting emphasis on the interplay between athletes' body and mind, between exercise-related mechanical/metabolic overload and neuro-endocrine maladaptations, thus justifying a psycho-behavioural assessment of the triathletes at risk.

21.5 Treatment

When an athlete is diagnosed with FOR, the best advice is to adjust training load so that the body can quickly adapt and physiological responses to sport specific exercise are restored, a strategy known as 'active recovery' leading to restored/improved performance within few weeks. When a triathlete is diagnosed with NFOR, the best treatment is rest, so that the body can correct imbalances caused by overload training, the athlete can resume training and restore performance within many weeks or months. Unfortunately, no other options are available despite many OTS pathogenesis hypotheses, suggesting, for example, a potential role for pharmacological interventions. So far, none of the proposed pharmacological therapies has been validated in a population as special as the athletes. A promising approach to restore performance in NFOR/OTS athletes has been recently proposed by Susta et al. [10]. This is based on repeated exposure to normobaric intermittent hypoxia and hyperoxia, combined to light intensity exercise: each session consisted of repeated exposures to hypoxia (O_2 at 10%) and hyperoxia (O_2 at 30%) delivered as 6 to 8 hypoxia-hyperoxia cycles (total time 45 min to 1 h), three times a week, delivered 1.5–2 h after a low-intensity exercise session (2 bouts of 30 min, running at 50% of VO_{2max} with 10 min rest between bouts) over 4 weeks. This novel strategy has to be further validated in larger athletic populations before being prescribed.

Behavioural strategies and a flexible approach to triathletes' training plan can be helpful. Stress management education/therapy, psychological support, minimising/avoiding sudden increases in training loads and always allow for adequate recovery are all viable options. Rescheduling a competitive season is not an easy decision to take but it is often the most sensible choice to minimise the risk of OTS progressing to the state of chronic condition, affecting daily life and not only athletic performance. It is important to design a long term therapeutic strategy, based on psycho-behavioural and functional outcomes, instead of looking for shortcuts too often leading athletes to waste their time and confidence.

21.6 Triathletes Monitoring

Adaptations to training loads have to be seen as a continuum encompassing acute and chronic responses to increasing stimuli and resulting in FOR, NFOR and OTS, mainly depending of recovery time and quality after overloading periods. Since it is difficult to differentiate between NFOR and OTS using metabolic biomarkers commonly used in internal systemic medicine, detecting early signs and symptoms of maladaptation is of paramount importance. To this purpose, there is a growing emphasis on athletes' monitoring with the aim of identifying patterns/trends suggesting potentially concerning changes in an athlete's health status. Performance monitoring is nowadays a common practice among professionals and semi-professional athletes, thanks to the many sport scientists involved in coaching, in strength/power conditioning and in performance analysis. Accurate, individual training programmes are a common practice, so the risk of exposing athletes to inappropriate workloads is minimised.

Various psychological and physiological measures have been suggested in the last decades to more accurately detect early stages of FOR/NFOR and by monitoring both external load (training) and internal load (metabolic responses) practitioners should be able to act in a timely manner and protect an athlete from progressing along the pathophysiological pathway leading to OTS.

21.6.1 Psychological Monitoring

There is good scientific evidence supporting the regular use of questionnaires to profile triathletes' psycho-behavioural responses to training loads during different phases of their conditioning programme. Psycho-behavioural changes should be considered as early signs that a triathlete is 'under excessive stress'. A recent systematic review by Saw et al. [16] highlighted that subjective measures of well-being for routine assessment are cheaper, more easily undertaken and more sensitive than objective measures to detect mood changes suggesting the onset of maladaptations potentially leading to NFOR/OTS. In particular, to subjectively measure athletes' well-being, questionnaires have been developed: the Profile of Mood States (POMS), the Recovery-Stress Questionnaire for Athletes (RESTQ-S), the Daily Analyses of Life Demands of Athletes (DALDA), the Multicomponent Training Distress Scale (MTDS), to mention only few. According to Saw et al. [16], subjective measures do not correlate with objective measures except for aerobic capacity measures (VO_{2max}) and 'Vigour' (strong evidence for a positive association) and 'Fatigue' (moderate evidence for negative association). In relation to FOR/NFOR/OTS, more relevant findings are that subjective measures are more responsive to training than objective measures and in particular (1) subjective measures identify impaired well-being resulting from an acute increase in training load and improved well-being resulting from a reduction of the training load and that (2) subjective measures also identify impaired well-being resulting from chronic overload. Questionnaires collect self-reported measures and their main advantage (compared to objective measures)

is that they can be self-administered daily with minimal disruption to the training schedule. As suggested by the systematic review's authors, the MTDS is more convenient than the RESTQ-S questionnaire because it includes mood disturbance, but RESTQ-76 S is more robustly validated among athletes [4].

The RESTQ-76 Sport is a reliable psychometric tool to monitor athletes' psychosocial status, to identify potential sources of stress and to assess recovery [17]. This questionnaire consists of 76 statements divided into 7 general stress scales, 3 sport-specific stress scales, 5 general recovery scales and 4 sport-specific recovery scales. Stress statements are: general stress, emotional stress, social stress, conflict/pressure, fatigue, lack of energy, physical symptoms, injury, emotional exhaustion, disturbed breaks. Recovery statements are: social recovery, physical recovery, general well-being, sleep quality, success, being in shape, personal accomplishment, self-efficacy, self-regulation. Each scale investigates 4 items using a Likert scale ranging from 'never' to 'always' in 6 steps.

In triathletes exposed to 4 weeks training overload and 2 weeks taper, the RESTQ-76 Sport scores accurately mirror training loads and this questionnaire has been proven to be sensitive and accurate to differentiate between overreaching and non-overreaching triathletes [18]. DALDA can also be used to monitor acute well-being in athletes as 'current well-being' is compared to 'normal', thus avoiding the repeated measures needed to define an individual's baseline in questionnaires (such as RESTQ-S) using Likert scales.

21.6.2 Performance Monitoring

Since the OTS definition is based on 'underperformance', regularly assessing sport specific performance should be part of athletes' routines as this approach provides coaching staff with useful information to early detect a prolonged reduction in power output. In addition, sport specific ergometry provides other performance-related physiological parameters, thus allowing for timely adjustments to training and recovery.

There is limited evidence about which test should be performed on a regular basis. Coutts et al. [18] proven that a 3-km running time trial (3-km RTT) administered once a week is a useful tool to best manage training loads. Before the test, each triathlete was required to warm-up by jogging for 800 m, and to complete an incremental warm-up consisting of 3 bouts of a 2-min shuttle running over 20 m. Triathletes were tested individually to avoid any influence of pacing and they were verbally encouraged to perform at their best without receiving feedback about their ongoing performance (lap splits). In the group of deliberately overloaded triathletes (4 weeks of progressive overload training) a significant decrease (3.7 ± 7.5%) in 3-km RTT performance was recorded. After 2 weeks of taper, performance was not different between the intervention and the control groups.

In a similar study, Hausswirth et al. [19] investigated cycling performance among triathletes exposed to overload training for 3 weeks (practice sessions duration was increased by 30%) with the aim of detecting sleep disturbances induced

by FOR. Cycling performance, measured as maximal power output in a incremental test (5 min at 100 W as warm-up, 5 min at 150 W followed by 5 min at 200 W and + 25 W every 2 min afterwards until exhaustion) was significantly reduced by 10 ± 4 W, from 371 ± 38 W to 361 ± 37 W. Perceived fatigued increased as well, from 3.9 ± 3.3 to 12.7 ± 5.3 arbitrary units i.e. fatigue item in POMS questionnaire.

21.6.3 Physiological Monitoring

The multifactorial nature of the OTS and its complexity have been investigated from many different clinical perspectives and a variety of OTS pathophysiology hypotheses have been suggested.

Since one of the NFOR/OTS pathogenesis hypothesis is an imbalance of the autonomic nervous system (ANS) leading to a parasympathetic overdrive, as suggested by physiological responses to exercise such as reduced maximal heart rate and blood lactate concentrations after maximal exercise, monitoring Hear Rate Variability (HRV) was suggested as strategy to prevent the onset of ANS imbalances associated with FOR/NFOR and OTS. The use of HRV is still controversial but a relatively recent study by Le Meur et al. [9] provides useful information on how to monitor HRV to be able to detect changes among FOR triathletes. The use of portable/wearable technologies now allows for convenient and reliable HRV monitoring in athletes, and daily HRV recordings are now easier than ever before to collect. HRV was recorded on a daily basis and a progressive increase in the parasympathetic modulation of heart rate in FOR athletes was detected. As pointed out by the authors of this recent study, isolated, once a week HRV recordings may not detect training-induced autonomic modulations in FOR athletes, mainly because of a wide day-to-day variability. Instead, the weekly mean of each HRV parameter showed a larger change in indices of parasympathetic tone in the FOR group than the control group.

A systematic review of 38 studies investigating hormonal profiles correlated to FOR/NFOR/OTS and acute hormonal responses to exercise, showed that there is no evidence to support the use of blood hormones as biomarkers of NFOR/OTS. In athletes diagnosed with OTS a blunted response of ACTH and PRL to maximal exercise are cautiously suggested as potentially useful, the evidence being limited by a limited number of studies published, the use of different exercise protocols (maximal test and two bouts test), the small sample size of the experimental groups and by contradictory results [20].

In the past, Free Testosterone/Cortisol Ratio (FTCR) was suggested as useful to prevent the onset of overtraining but there is growing evidence that FTCR does not correlate with performance changes so it is not useful to early detect NFOR [21]. Instead, should a triathlete fail to restore performance and/or psychological well-being, FTCR could be useful to confirm an overloading status or to detect a risk of developing OTS after a period of intense training, according to Banfi and Dolci [5] who followed up for two seasons professional football players. A Free Testosterone/

Cortisol Ratio (FTCR) higher than 0.8 reflects an athlete's ability to cope with training loads whereas a value lower than 0.5 suggests the athlete is at a high risk of NFOR/OTS and he/she has to rest and to focus on recovering. Markers of increased oxidative stress were altered at rest and after exhausting exercise in OTS athletes compared to healthy controls and this was associated with poorer antioxidant protection, but further evidence is needed to suggest these measurements as part of blood biochemistry routine assessment [22].

In conclusion, monitoring athletes exposed to high mental, metabolic and mechanical loads, administered as part of a conditioning programme with the aim of enhancing performance, seems to be not only the best way to optimise a sport specific training, but also the most promising preventive strategy to avoid to 'wait until is too late', by prompting practitioners to timely take the right decisions to minimise the risk for triathletes to develop OTS.

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Part VI

Special Considerations

Triathlon Race Day Medical Care

22

W. D. B. Hiller

22.1 Introduction

Nothing is more important than the welfare of the athlete.

The purpose of this chapter is to provide an understanding of concepts, issues, and solutions on medical care at triathlon races for medical and race organizational personnel. It is dedicated to the many thousands of medical volunteers around the world who make our sport safe and possible.

Providing care for a race which includes all of the medical risks of open water swimming, bicycle racing, and running is a very challenging task [1–4]. Races vary from a few hundred meters for each discipline to the Olympic distance (1.5 k swim, 40 k Bike, and 10 k run) to Ironman distance (3862 m swim, 180.2 k bike, and 42.2 k run) to ultradistance races and everything in between [1, 2, 5–7].

Triathlons happen in a huge variety of climates, terrains, altitudes, distances, and weather conditions.

Weather conditions on race day can include extreme heat or cold, sudden storms, strong winds, torrential rain, and lightning. Rapid changes in ambient temperature can make the race itself and caring for athletes very challenging.

The Race Medical Director (RMD) is typically responsible for every aspect of race day medical care for the athletes (Appendix 1) [1, 2, 6, 7].

The scope and time of care to be provided must be explicit, detailed, and in writing. The details of the medical tent(s) size, location, access for athletes and ambulances, water and electrical connections, and responsibilities of the logistics for all of this must once again be explicit and in writing. Over the years, RMDs have occasionally found to their horror that the tent is not what was promised or where it

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was supposed to be, or that there is no water, no cooling tubs, no electricity, and no plan to remedy the situations (Appendix 1) [1, 2, 5, 7, 8].

Recruiting the medical and paramedical personnel for the race is essential. The RMD should start the process 6 months or longer before the race and should regularly communicate with and whenever possible meet with the rest of the Race Committee (RC).

The goal of the medical team is to recognize and to be prepared for whatever may happen in a particular race.

22.2 Pre-Race Planning

The RC or Local Organizing Committee (LOC) is responsible for pre-race planning and for executing the plan on race day.

The size and composition of these groups is dependent on the length and complexity of the venue, the number of competitors, and the number of events or days of events planned [1, 2, 5–7].

Even in venues with annual events, there are changes in road closures and conditions, and availability of life guard, police, ambulance and hospital services, and many other things. Extensive detailed and constantly updated planning prevents poor outcomes.

There must be an overall disaster plan for the race which involves all aspects of the race organization. This includes many other things, such as security, evacuation plans, communication, chain of command, and plans for triage [1, 2, 9].

Almost every function in a race is dependent on every other function in the race in some way. For this reason, it is essential that the medical team be closely integrated into the race structure from the beginning of the planning cycle. Personal relationships with the race management and race directors are keys to success. For example, the RMD needs to know who will be setting up the medical tent, delivering water, cots, and radios. Who and where the spotters are on the course, who will clean up, package up, and return or dispose of the contents of the medical tent. These can go from small questions to big problems without proper relationships (Appendix 1) [1, 2, 7].

22.3 Communication on Race Day

Communication on race day is essential. There must be dedicated channels for the medical team to communicate between the RMD and medical personnel on the field of play, as well as between the RMC and local hospitals and ambulance services. There, of course, should be a clear communication between the medical team and the RC. In larger races, there are medical personnel dedicated to communications. Well marked course maps—physical or electronic—should be immediately available to the RMC, and field of play medical personnel (Appendix 1) [1, 2, 6, 7].

A formal arrangement for contacting family or friends of injured athletes must be established. This is usually managed by the RC.

There should be a clear plan for dealing with bikes and personal effects of injured athletes as well.

22.4 Role of the RMD

The RMD must be a physician who is experienced in the care of athletes at mass events. In the case of inexperience, the RC should see that prior to the race, the RMD gets to go to a similar well-managed race to observe the medical program in person.

It is very important for the RMD to be involved in the planning for the race in the months leading up to the race itself. Creating and managing a good triathlon is a bit like being a part of an orchestra practicing to play a symphony: everyone and everything needs to be on time, in tune, and in synch for the music to work.

RCs typically have regular meetings in the months preceding the race. These can range from monthly initially to weekly as the race day approaches. Attending these meetings allows the RMD to develop friendly working relationships with the other key players in the race.

Essential arrangements with local hospitals and ambulance providers must be arranged far in advance in coordination with the RC (Appendix 1) [1, 2, 6, 7].

The few days befor the race are very busy and sometimes chaotic.

Promises and plans frequently do not coincide with reality at these races without the active intervention of the RMD.

The RMD or designated substitute should be present when the medical tent goes up to ensure the size, location, and orientation of the tent. Plans for electrical and water supply for the medical tent should be approved by the RMC and RMD months prior to the race and the results should be inspected the day before the race. Water and food for the athletes and staff should be in place before the race begins (Appendix 1) [1, 2].

Equipment and medical supplies should be in place the day before the race.

On race day, the RMD should be continuously available to coordinate care for the entire race. This means that he/she should not have direct patient care responsibilities. Quick consults on patients under another provider's care is reasonable, but should not interfere with overall race medical management [1, 2].

The RMD must have the authority, in cooperation with the technical officials, to disqualify an athlete for medical reasons (Appendix 1) [1, 2, 5, 7].

It is recommended that the RMD work with the RC to include a medical questionnaire for age group athletes. For an unconscious or confused athlete with serious allergies or medical problems, this information can potentially and significantly alter triage and medical care [1, 2, 7].

22.5 Medical Staffing for the Race

Recruiting physicians, nurses, and paramedical personnel should begin at least 6 months before the race. ER doctors, sports medicine, family and internal medicine doctors, orthopedic surgeons, anesthesia providers, and others are well suited to the task. Physicians Assistants and Nurse Practitioners are good additions to the team. Nurses are essential to take good care; ER and ICU nurses are appointed for quick studies in the medical tent. For races which provide IVs

in the medical tent, there should be designated IV starters (Appendix 1) [1, 2, 5–7].

22.6 International Triathlon Union (ITU) Staffing Requirements

- Physicians have the authority to withdraw an athlete at any point for safety or health reasons.
- One physician per 200 athletes with a minimum of four physicians.
- One nurse per 100 athletes with a minimum of six nurses.
- Two paramedics per 100 athletes.
- Two physicians must be present and on duty for the entire event. One doctor should be in the medical tent and another must be mobile (ITU) [3, 5].
- There should be meetings with the medical team prior to the race to go over clear and specific protocols for the treatment, work flow in the medical tent, and the list of medications with designation of which providers may prescribe and administer which medications under what conditions (Appendix 1) [1, 2].
- The meeting hopefully held the day before the race should establish the final schedule for the medical team, for times, locations, and responsibilities.
- Clear lines of communication and authority should be established. There is typically a final meeting on race morning an hour or so prior to the start.
- If there are work shifts, clear time and hand of protocols must be established.
- Communication among medical staff at different locations must be formalized. This may be done using radios supplied by the RC or cell phones.
- Race venues are usually quite loaded near the finish line and at the main medical tent. So, phones should have earbuds and mics, and be set to vibrate as well as on maximum ringer volume (Appendix 1) [1, 2, 6, 7].

It is very important that the medical staff be immediately and clearly identifiable. The classic medical team shirt in many places is a scrub shirt with a pocket with "Medical" (or corresponding symbol) printed small in front and large on the back. However, in a survey of 300 medical volunteers at the 2018 Hawaii IRONMAN Triathlon World Championship by Dr. Bob Laird, the overwhelming request was for a Tee shirt with front pocket, marked as above (Appendix 1) [1, 2, 6, 7].

22.7 Race Day Conditions

The RMD should be involved in decisions which potentially affect the safety of the athletes (Appendix 1) [1, 2].

Triathlons are held in a wide variety of climates. There are races with water so hot that swim courses have to be shortened or cancelled. There are swims in water cold enough to require wetsuits, shortened swim courses, and occasionally cancelled swims.

Guidelines for using wetsuits, shortening, or cancelling the swim portion of races are based on water temperature have been developed and adopted by ITU and USAT (Appendix 2) [1, 2].

The ITU has adopted internationally accepted guidelines for swim water quality testing [4]. These guidelines were meticulously followed, for example, for the Rio Olympic and Paralympic Games, media reports to the contrary.

Air temperature (wet bulb) is typically measured on race morning, and in ITU races, it is measured hourly throughout the day. Again, see the ITU guidelines for water and air temperature (Appendices 1 and 2) [7, 10].

Weather conditions on race day can include storms, and there are times when crosswinds, torrential rain, lightning, or sudden temperature changes can threaten the race and athletes. While making decisions on the continuation or modification of the race, the RMD should be involved (Appendix 2) [1, 2].

The guiding principle when the race is in question should always be "Nothing is more important than the welfare of the athletes."

22.8 Swim Course Safety

Water conditions can be very unpredictable. Storms or strong tides and currents, bad visibility, red tides, stinging jellyfish, sharks, or innumerable other hazards can present themselves on race day. When conditions potentially endanger the athletes, the RC and RMD sometimes have to make hard decisions: even at world championships swim courses have been modified or cancelled. (e.g., 2008 BG ITU World Championships, Vancouver, Canada) [1, 2].

Although rare, deaths do occur in the swim portion of triathlons [1–3].

There should be a medical team equipped with automated external defibrillators and prepared to perform cardiac resuscitation at the swim finish. It is highly desirable to have a dedicated ambulance at the swim finish (Appendix 1) [1, 2].

Depending on the primary medical tent location, race course layout, and potential swim course dangers, there is a need for a small medical tent at the swim finish.

Typically, there are lifeguards on the swim course on jet skis, paddle boards, and inflatable boats. There should be direct communication between the lifeguards, the medical team, and ambulance(s) at the swim finish. Some races have medical teams on the water (Appendix 1) [1, 2, 6].

22.9 Post Swim Medical Problems

If there are sharp objects around the swim start in shallow water around the course or at the swim finish, lacerations to the feet, hands, or elsewhere can occur.

Corneal abrasions and facial contusions are not uncommon. Bruising of the arms, legs, and chest can occur, and rib fractures can rarely occur [1, 2, 5, 7, 11].

Pulmonary aspiration can happen with significant consequences.

Exhaustion, hypo or hyperthermia, arrythmias, panic attacks, headaches, nausea, and vomiting can occur after the swim.

Jellyfish stings, sea urchin spines, and sunburn are found in tropical venues (Appendix 1) [1, 2, 5–7, 11].

22.10 Bike Course Safety

Bike courses are unsurprisingly the source of trauma, minor, and occasionally otherwise.

Many of the accidents happen at predictable locations—sharp turns on descents, bad pavement, pot holes, gravel on road surfaces, wet or oily road surfaces, pedestrian crossing, and otherwise are danger points.

For this reason, it is important to have spotters who can communicate with medical team covering the entire bike course and medical coverage at danger points. It is bad for the race to have an unattended injury.

There must be very clear and prospectively determined emergency vehicle access to the bike and run course. The emergency vehicles must enter at a safe place and move only in the direction of the racers.

When an injured athlete is removed from the course, there should be a clear protocol for dealing with and documenting the dispensation of the bike.

If T2 (bike/run transition) is not by the main medical tent, there should be another medical tent and medical team at T2 (Appendix 2) [1, 2, 6, 7].

22.11 Run Course Safety

The entire run course should be monitored by spotters who are in contact with medical team. There should be medical personnel situated around the course in such a manner that quick access to any part of the course is insured. If there is a long out and back run course, there should be a medical team and tent appropriately placed (Appendix 1) [1, 2, 6, 7].

22.12 Finish Line Medical Care

The finish line is the most common place for athletes to collapse, and the interval after finishing the most common time to present to the medical tent [1, 2, 6, 7, 11]. In large races, there can be a huge flow of athletes into the medical area at peak finish times. This can amount to multiple athletes per minute needing care for hours at some races.

The goal of medical team at the finish line is to ensure that athletes get the care they need and at the same time to stay out of the way of the finish line team and, if possible, field of view of the media. This all should be done with the full support and cooperation of the finish line crew. There should be a place for the medical team members to stand out of the way, but with immediate access to athletes as needed.

Very clear, mutually understood and explicit indications for medical intervention should be shared between the RMD and finish line Director.

An experienced finish line crew can signal medical team when needed for a particular athlete, and experienced medical crew can decide who needs to go to the medical tent.

Depending on the distance to the medical tent from the finish line, wheel chairs, stretchers, wheeled stretchers, or modified golf carts may be indicated. The transport path should be as direct and private for the athletes as is practical (Appendix 1) [1, 2, 6].

22.13 The Medical Tent

The location of the medical tent(s) is quite important and can be contentious. The finish line is where most medical issues occur (Appendix 1) [1, 2, 6, 7] and where many other essential race functions live. Around the finish line typically includes the grand stands, timing, race officials, communication area, security, VIP/race sponsor areas, water and food for athletes, doping control, and race announcing. Nearby are the athletes' personal gear, medal pick up, massage area, and family areas.

All of this being considered, nothing is more important than the welfare of the athlete. The medical area needs to be close enough to the finish line to provide immediate unobstructed access and transportation to the medical tent by the medical staff. The medical tent must provide real privacy for the athletes. There must be direct vehicular access to and from the medical tent for emergency vehicles. Athletes who recover should then have easy access to the athlete's areas, tents, and their bikes and other gear.

The tent should be in a clean, dry, and flat area. Footing should be clean and stable. There should be water and or hand cleaning stations in the medical tent (Appendix 1) [1, 2, 6, 7].

Translators should be identified on race day and be available either in person or by cellphone or radio.

Access to the medical tent should be explicitly determined well in advance of the race and agreed upon by the RC. There should be security personnel to politely control medical tent access: when there is an issue regarding the access, the RMD should be consulted. There is no place for the media in the medical tent except in specific cases, usually a victor's interview performed without compromising the privacy of another athlete. This is always done with the explicit consent of the athlete. A policy concerning family members is especially important. Their presence may be helpful to their athlete, but compromises the privacy and potentially the care of other athletes. For this reason, communication between the medical tent and the RC family services liaison is important, so that families do not lose track of loved ones.

Good lighting is always important, especially so in races where athletes will be in the tent in the late afternoon, evening, or night time.

Cots or chaise longs are usually placed with head by the tent wall with good spacing between them, allowing simultaneous access to both sides of each athlete

being treated. It should be easy to put a patient in the Trendelenburg position. There should be wide aisles in the tent to allow for athletes supported on either side, wheel chairs or stretchers to traverse the tent. In hot climates, there should be cooling tubs with ice available. Hyperthermia is potentially fatal, and should be treated immediately upon diagnosis [1, 2, 5, 7, 8].

Each cot should be clearly marked with a letter and/or number, which should go onto the medical record with the patient's bib number and name. Typically each group of 4–6 cots are designated to specific medical team members (Appendix 1) [1, 2].

Medications should be in a controlled area and managed by a pharmacist or nurse with sign out sheets for appropriate medications. There should be a posted list of available medications. For restricted or limited medications, there should be designated access.

Because hyponatremia is a common problem in triathlon, immediate electrolyte analysis for patients at risk is commonly available. ITU races require this, as do many IRONMAN races (Appendix 1) [1, 2, 5, 7].

If offered, IVs should be started by medical team members who are experienced and have current skills. Electrolyte analysis is recommended prior to IV if there is any question of hyponatremia. Typical fluids used are NSS, D5NSS, and 3NSS (the latter for symptomatic hyponatremia; Appendix 1) [1, 2]. The legitimate medical uses of intravenous infusions may not need a therapeutic use exemption in certain settings (hospital treatment, surgical procedures, or clinical investigations). In other situations, such as trauma with or without blood loss, severe dehydration, and intractable vomiting, the athlete should receive appropriate treatment and apply for a retroactive therapeutic use exemption as soon as reasonable following the WADA rules.

After the race, the RMD should follow-up with patients who have been hospitalized until they are discharged from the hospital.

Medical records should be maintained following the race, and a medical report for the RC should be created. This report should include an overview of the type of injuries, where they occurred and disposition of the athlete. This is important both to identify potential areas of improvement for the race and for the protection of the RC and medical staff (Appendix 1) [1, 2, 6, 7].

22.14 Conclusion

Triathlon race day medical care is a complex challenge. It has been evolving over the 40+ years of the modern sport. Successfully providing care at races is an art and science. Planning medical support for a specific race is a unique challenge that becomes more manageable with the experience of decades of effort as expressed in the evolving literature of the sport.

Appendix 1

Medical and anti-doping control management in ITU event. ITU competition rules 2019.

Medical and Anti-doping Control Management in ITU Event

Medical Management

1. General
   (a) The ITU Medical Committee may appoint a medical delegate to oversee the medical operations of the event.
   (b) The ITU Medical Delegate (ITU MD) may conduct one site visit prior to an ITU event.
   (c) The ITU Medical Delegate will liaise with the event appointed RMD. The ITU Medical Delegate reviews, with the RMD all the information relating to medical and doping control requirements for the event.
   (d) The RMD attends the ITU event, the year prior to study and work with the medical team.
2. Medical plan

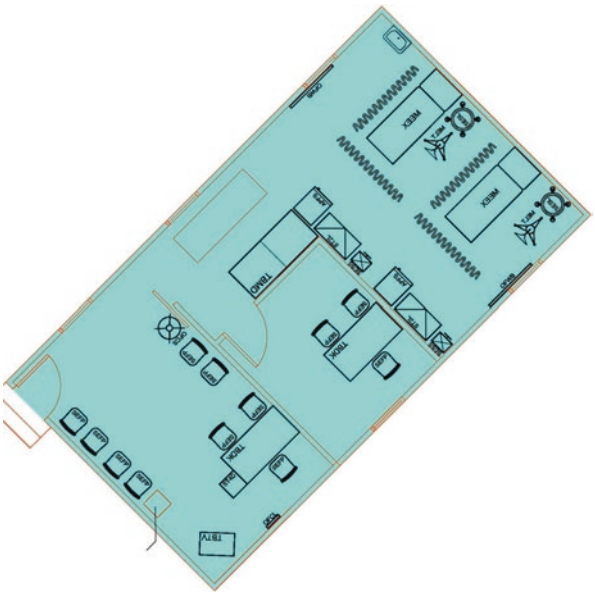
   The LOCs of the World Championship, World Cup, and Continental Championship events should submit a full competition medical plan to the ITU Events Department and to the ITU Medical Committee no later than 1 month before their event. This document should include:
   (a) Onsite medical services (facilities, equipment, and supplies).
   (b) Offsite medical services (facilities, equipment, and supplies).
   (c) Medical coverage per discipline.
   (d) Paratriathlon special services (if applicable).
   (e) Staffing and scheduling.
   (f) Ambulance distribution and medical response maps.
   (g) Communication plan.
   (h) Operational plan and procedures.
   (i) Team doctors Information and registration forms.
   (j) Athletes' waiver.
   (k) Budget.
3. Personnel
   (a) The LOC will appoint a RMD. The RMD is responsible for the overall medical operations of the venue, and should preferably have experience in major sport/endurance events. The RMD is responsible for informing the ITU Medical Delegate (ITU MD) and/or TD about the medical organization of the event.
   (b) The RMD appoints other medical staff; organizes the facilities in cooperation with the LOC; and organizes supplies and equipment.
   (c) Two paramedics per 100 athletes is the minimum.
   (d) There should be 1 physician per 200 athletes with a minimum of 4 physicians.
   (e) There should be 1 nurse per 100 athletes with a minimum of 6 nurses.
   (f) Two doctors must be present and on duty for the entire event. One doctor should be located within the medical facility and the other doctor must be mobile.

(g) Physicians have the authority to withdraw an athlete at any point for safety or health reasons.
(h) Doctors, nurses, and paramedics must be clearly identifiable and have the authority to enter the field of play in the event of medical emergencies.
(i) Medical spotters will be placed along the swim course.
(j) Medical spotters will be placed every 500 m on the bike course and will be supplied with radios and/or cell phones. The spotters will not be on the field of play, but will have access in the case of an emergency.
(k) Medical spotters will be placed on the run course (numbers will be determined based on the course design).
(l) Paramedics and stretchers must be in attendance adjacent to the swim exit, transition area, and at the finish area.
(m) The LOC must ensure that all marshals and other race officials are aware of all medical facilities and their locations.

4. Ambulances and access
   (a) A minimum of three ambulances will be required, plus an additional one for every 500 athletes: one ambulance will be stationed near the finish area and the medical facility, two ambulances will be stationed strategically on the bike course. The final number should be approved by the ITU MD or TD.
   (b) Ambulances will be equipped with the following: direct communication with medical headquarters and direct communication with all necessary cardiopulmonary resuscitation supplies and trained personnel.
   (c) Ambulance emergency access routes must be planned both from the competition site and bike course.
5. Hospitals
   (a) The nearest hospital must be informed of the event well in advance and advised of the possible emergency that may arise.
6. Medical records
   (a) Accurate and complete medical records must be kept on all medical instances. Those records must be submitted to the ITU Medical Delegate or TD.
   (b) The records must be shredded after the events to protect the privacy laws in place in each jurisdiction.
7. Race medical management
   (a) Main principles for an effective race medical management.
      - Split the course in sectors in order to have the same communication code between the TOs, medical staff, and LOC.
      - Place the ambulance at the most dangerous points.
      - Make sure that an ambulance can reach the entire field of play using the minimum of the course.
      - Allocate a number to the dangerous corners for effective communication.
      - Make sure that there will be a number of paramedic bikes for an effective response to the accidents.
      - The volunteers that are found in distance of 100 m from the point of the accident should make warning signals to the following athletes.
      - Report to the VCC immediately.
      - Inform the TD and the medical services.

- The ambulance should enter from the nearest intersection and park close to the side of the road. The volunteers should continue to inform the other athletes.
- The ambulance should exit from the nearest crossing point. The ambulance should move on the FOP according to the athletes' flows.
- When there is a need for a simple transport of a patient from the spot of the incident to the venue, then the ambulance may follow the course to the athletes' area. In case of an accident of involving many athletes during the bike course, we should ensure first the athletes' integrity who is involved in the accident as well as the other athletes, and those needing medical attention will be sent to the nearest medical services. The actual facility is arranged prior to the event by the ITU Medical Delegate or TD.

8. Athlete medical
   (a) Area specifications.
   - Finish area tent size: 3 × 6 m for World Cup/9 × 9 m for World Championships.
   - Location of tent: adjacent and accessible to finish area.
   - Four tables.
   - Twelve to twenty chairs.
   - Two wheel chairs.
   - One carry chair.
   - Six stretchers.
   - Access to toilets.
   - Radio communication and medical records area.
   - Must be located in a secure area with direct access to the competition finish and must not be accessible to media.
   - Emergency access and ambulance placement must be planned.



9. Medical supplies
   - Cardiopulmonary resuscitation.
   - Medication for acute cardiac care, asthma, and allergy.
   - ECG machine 12 leads.
   - Defibrillator.
   - Blood glucose monitoring equipment.
   - Sodium level analyzers.
   - Oxygen.
   - Thermometers and rectal thermometers.
   - IV fluids (NS or 5% dextrose in NS, 3% Nacl).
   - Ice, ice buckets, and ice water tubs.
   - Blankets.
   - Towels.
   - Dressing material wound care.
   - Hospital to be notified.
   - Medical emergency vehicles on site and on course with planned access routes.
   - Bikes for mobile medical spotters.
   - Medical records (all medical treatments must be recorded and stores for records).
   - LOC must ensure that all athletes sign the medical waiver and report any allergies or medications that are being taken.
10. Massage facilities
    (a) General requirements.
        - A massage facility should be placed adjacent to the athletes lounge, but not in the medical facility area.
        - The massage facility should be available at tent or other such covered facility.
        - Massage personnel should be determined by the number of athletes and the level of services offered.
        - Massage is not a requirement, but is recommended as a service to the athletes.
11. Spectator medical
    (a) 10-m^2 tent should be provided for spectator medical personnel with limited facilities as above.
12. Cold water conditions' preparation

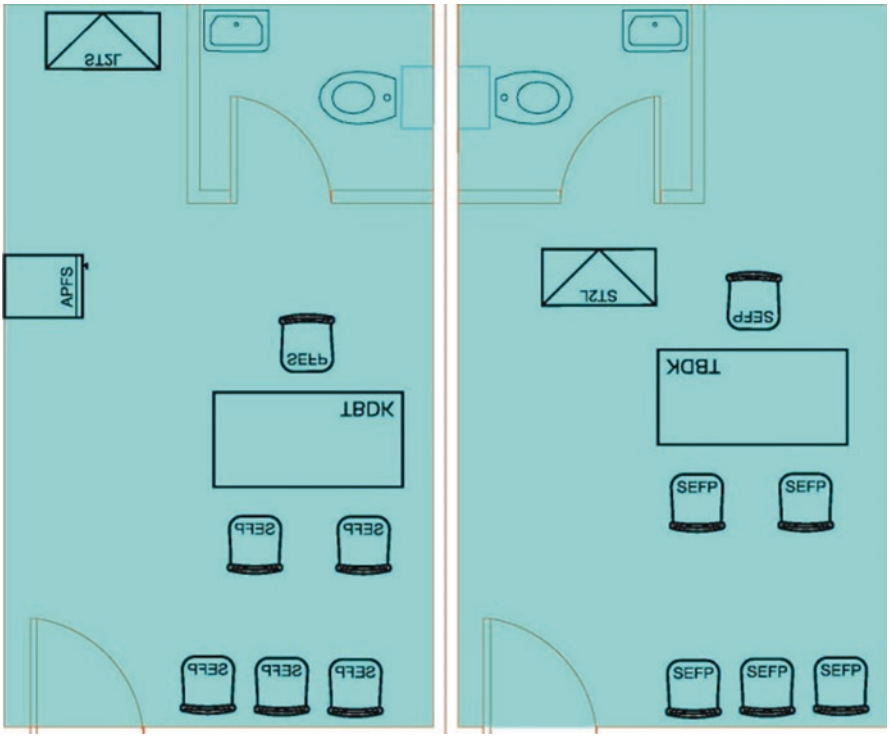
    It is often organized in events where water temperature below 15 °C. Although the final distance of the swim leg is a decision that has to be made by the TD, the ITU Medical Delegate, the LOC Medical Team, and the Lifeguards, the LOC should provide the following in any case:
    (a) Shower with ambient water temperature at the swim exit.
    (b) Advise the athletes to have a proper swim warm-up in order to immerse their bodies and heads prior to starting the competition to acclimatize.
    (c) Blankets, and so on to combat hypothermia should be ready.

(d) Increase the number of the safety boats and equip them with space blankets.
(e) Create medical stations around the course where athletes with hypothermic symptoms can stop.
(f) Provide heaters and blankets at the recovery area.

Doping Control

1. General
   (a) It is the responsibility of the LOC to make sure that anti-doping control is conducted at the event. The LOC should contact the proper anti-doping organization and arrange for the appropriate number of tests to be conducted. The number of tests needed will be stated in the contract. A minimum of 10 urine tests should be conducted at any ITU event.
   (b) ITU complies with WADA on all doping rules and regulations (See ITU website for all current information on anti-doping control). All tests should be conducted using best practices of all international standards.
   (c) The results of all tests and the anti-doping control forms should be forwarded to ITU as soon as possible.
   (d) Provisions should be made to accommodate anti-doping control at the event. This will include at a minimum private waiting areas, secure washroom facilities, processing rooms, and bottled water.
2. Personnel
   (a) The anti-doping control agency will require a number of doping control chaperones. The exact number will depend on the number of tests being completed and the event schedule. Both male and female chaperones will be needed. In some countries, the national federation may have certain obligations to the national anti-doping agency, please check with your NF on this issue.
3. Doping control
   (a) The LOC of each ITU event must have provisions for a minimum of 10 in-competition urine tests. The exact number and who will be tested will be communicated to the TD by the anti-doping organization conducting the tests.
   (b) The LOC must contact the anti-doping organization associated with the national federation in their country or region. If you are unsure, please contact ITU Anti-Doping Director for an agency in your area.
   (c) Once doping control has been confirmed with the appropriate agency, please inform the ITU Anti-Doping Director.
   (d) Anti-doping control facility onsite requirements: the exact size will be determined by the number and type of tests being conducted. The anti-doping organization conducting the tests will be able to specify exactly what requirements they will need to have to conduct the tests according to the international standards of testing:

- A 3 × 3 m area (preferably not a tent) completely private area, away from the public and media.
- Two double toilets to accommodate the testing procedure.
- Two tables.
- Twelve chairs.
- Bottled water, sport drink, and replenishing food for the athletes (sealed).
- Minimum of 10 volunteers to work as drug testing chaperones.
- Security personnel to ensure that only doping control personnel and athletes, with their designated personnel, are allowed in the anti-doping control area.



Appendix 2

ITU swimming: general rules, wetsuit use, modifications. ITU competition rules 2019.

Swimming ITU Competition Rules

1. General rules
   (a) Athletes may use any stroke to propel themselves through the water. They may also tread water or float. Athletes are allowed to push off the ground at the beginning and the end of every swim lap.
   (b) Athletes must follow the prescribed swim course.
   (c) Athletes may stand on the bottom or rest by holding an inanimate object, such as a buoy or stationary boat.
   (d) In an emergency, an athlete should raise an arm overhead and call for assistance. Once official assistance is rendered, the athlete must retire from the competition.
   (e) Athletes may sportingly maintain their own space in the water:
      - Where athletes make accidental contact in the swim and then immediately afterwards move apart with no penalty incurred.
      - Where athletes make contact in the swim, and an athlete continues to impede the progress of the other athlete without moving apart, this action will result in a time penalty.
      - Where athletes deliberately target another athlete to impede their progress, gain unfair advantage and potentially cause harm will result in disqualification and may be reported to World Triathlon Tribunal for potential suspension or expulsion.
   (f) Rules about water quality are outlined in Section 11.
2. Wetsuit use
   (a) Wetsuit use is governed by the following tables:

Elite, U23, junior and youth athletes:		
Swim length	Forbidden	Mandatory[a]
Up to 1500 m	20 °C and above	15.9 °C and below
1501 m and longer	22 °C and above	15.9 °C and below
Age group athletes		
Up to 1500 m	22 °C and above	15.9 °C and below
1501 m and longer	24.6 °C and above	15.9 °C and below

[a]When mandatory, the wetsuit must cover at least the torso

3. Maximum stay in water

Swim length	Elite, U23, junior and youth	Age group
Up to 300 m	10 min	20 min
301–750 m, below 31 °C	20 min	30 min
301–750 m, 31 °C and above	20 min	20 min
751–1500 m	30 min	1 h 10 min
1501–3000 m	1 h 15 min	1 h 40 min
3001–4000 m	1 h 45 min	2 h 15 min

4. Modifications
   (a) The swim distance can be shortened or even cancelled according to this table.

Original swim distance	Above 33.0 °C	32.9–32.0 °C	31.9–31.0 °C	30.9–15.0 °C	14.9–14.0 °C	13.9–13.0 °C	12.9–12.0 °C	Below 12.0 °C
Up to 300 m	Cancel	Original distance	Original distance	Original distance	Original distance	Original distance	Original distance	Cancel
750 m	Cancel	Cancel	750 m	750 m	750 m	750 m	750 m	Cancel
1000 m	Cancel	Cancel	750 m	1000 m	1000 m	1000 m	750 m	Cancel
1500 m	Cancel	Cancel	750 m	1500 m	1500 m	1500 m	750 m	Cancel
1900 m	Cancel	Cancel	750 m	1900 m	1900 m	1500 m	750 m	Cancel
2000 m	Cancel	Cancel	750 m	2000 m	2000 m	1500 m	750 m	Cancel
2500 m	Cancel	Cancel	750 m	2500 m	2500 m	1500 m	750 m	Cancel
3000 m	Cancel	Cancel	750 m	3000 m	3000 m	1500 m	750 m	Cancel
3800 m	Cancel	Cancel	750 m	3800 m	3000 m	1500 m	750 m	Cancel
4000 m	Cancel	Cancel	750 m	4000 m	3000 m	1500 m	750 m	Cancel

- Note: the temperatures above are not always the water temperature used in the final decision. If the water temperature is at or below 22 °C and the air temperature is at or below 15 °C, then the adjusted value is to decrease the measured water temperature according to the next chart:

		Air temperature (value in °C)										
		15	14	13	12	11	10	9	8	7	6	5
Water temperature (value in °C)	22	18.5	18.0	17.5	17.0	16.5	16.0	15.5	15	14.5	14.0	Cancel
	21	18.0	17.5	17.0	16.5	16.0	15.5	15.0	14.5	14.0	13.5	Cancel
	20	17.5	17.0	16.5	16.0	15.5	15.0	14.5	14.0	13.5	13.0	Cancel
	19	17.0	16.5	16.0	15.5	15.0	14.5	14.0	13.5	13.0	12.5	Cancel
	18	16.5	16.0	15.5	15.0	14.5	14.0	13.5	13.0	12.5	12.0	Cancel
	17	16.0	15.5	15.0	14.5	14.0	13.5	13.0	12.5	12.0	Cancel	Cancel
	16	15.5	15.0	14.5	14.0	13.5	13.0	12.5	12.0	Cancel	Cancel	Cancel
	15	15.0	14.5	14.0	13.5	13.0	12.5	12.0	Cancel	Cancel	Cancel	Cancel
	14	14.0	14.0	13.5	13.0	12.5	12.0	Cancel	Cancel	Cancel	Cancel	Cancel
	13	13,0	13.0	13.0	12.5	12.0	Cancel	Cancel	Cancel	Cancel	Cancel	Cancel

   (b) If other weather conditions dictate, that is, high winds, heavy rain, changing temperature, current, and so on, the Technical Delegate in consultation with the Medical Delegate (if applicable) may adapt limits of the swim length or adopt provisions about the use of wetsuits. The final decision will be made 1 h before the start and will be clearly communicated to the athletes by the Technical Delegate;
   (c) For aquathlon events (normally run-swim-run), the LOC should plan for a swim-run where the water temperature is expected to be below 22 °C. Where

a run-swim-run aquathlon has been planned, but on competition day the water temperature is below 22 °C, the format will change to swim-run;

(d) Water temperature must be taken 1 h prior to the start of the event on competition day. It must be taken at the middle of the course and in two other areas on the swim course, at a depth of 60 cm. Where the average measured figure is 27 °C or below, the lowest measured temperature will be considered as the official water temperature. Where the average measured figure is above 27 °C, the highest measured temperature will be considered as the official water temperature.

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Triathlon Marine Plan in the Olympic Games and in the ITU World Triathlon Series

23

Thanos Nikopoulos and Sergio Migliorini

23.1 The Marine Operation Plan

The Triathlon Competition follows International Federations protocols and as such will be run in accordance with ITU Competition Rules and the Event Organizers Manual. The Organizing Committee (LOC and LOCOG) should prepare a Marine Operation Plan and submit to the ITU Technical Delegate (TD) and the Medical Delegate (MD) for approval that should include the following:

- Detailed description of the swim courses along with the GPS coordinates of the buoys set up;
- Complete list of the marine/rescue staff along with the equipment (boats, kayaks, boards, etc);
- Communication process between marine staff, local authorities, and ITU;
- Schedule of activities along with the check-in and check-out timelines of involved personnel;
- Normal operating procedures with a clear description of the movement of each marine personnel (before, during, and after the event);
- TV-press presence;
- Water quality data;
- Environmental data;
- Emergency action plan.

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23.2 Emergency Action Plan

- The LOC should be in a position to verify the number of athletes who entered and exited the swim course at every moment of the event through the use of timing mats. The plan should include the basic steps:
- Confirm that there is a pre-start timing that captures the athletes' timing chip.
- Record the athletes who are not wearing a wetsuit.
- Manually count the number of athletes who come out of the water.

If an athlete is missing, an emergency action plan should be activated. The main steps in this process should be:

- Check all chip-timing records.
- Check lifeguards manual records.
- Check with the transition officials to see if all the bikes have exited transition.
- Confirm that the athlete was racing with or without a wetsuit.
- Perform a visual check of the swim course by the life guards.
- If the athlete was wearing a wetsuit and a search on the water surface has not found the athlete, the issue will be referred to the control center for further actions.
- If the athlete was not wearing a wetsuit and there is no sign of the athlete on the swim course, a water based search has to be conducted.
- The underwater search will commence if a hasty surface search does not locate the athlete.
- Evacuation procedure with a detailed plan on both individual and mass evacuation.
- Mass evacuation, which required all competitors are evacuated from the water.
- *Major incidents* shall include: casualties that are not fully conscious or so exhausted that they are unable to keep their head above water or support themselves onto a kayak; casualties that have any form of respiratory/cardiac difficulty; and casualties that are seen to disappear/sink below the water surface.
- *Minor incident* shall include all incidents or casualties that are not categorized as major incidents or causalities.

A full dress rehearsal should be conducted with the presence of the ITU Technical Delegate and ITU medical Delegate.

23.3 The Swim Course: Field of Play Medical Plan

The swim course operating procedures document details of all the rescue procedures for athletes in the water and comprehensive details of general water safety.

Medical staff will service the swim section of the competition in three areas:

On rescue boat: one doctor to provide simple medical treatment to athletes in the water or emergency life-saving treatment during delivery of the casualty to

the evacuation pontoon. The doctor will wear a lifejacket and will be a competent swimmer.

On evacuation pontoon: one field of play team leader and four field of play team members, one or two ambulance staff from primary ambulance response vehicle to provide definitive medical care and emergency life-saving procedures as required once casualty has been delivered to the evacuation pontoon. All will wear life jackets.

Once the casualty has been retrieved from the water, will be placed on the evacuation pontoon by the water safety team, and will be handed over to the medical team.

Ambulance: one or two parked near the evacuation pontoon. Ambulance emergency access routes must be planned both from competition site and bike course.

The medical team leader will:

- Assess the casualty—primary survey.
- Communicate injuries and treatment required to the field of play team and radio forward as required.
- Commence primary survey treatment (immediate saving).
- Re-assess casualty—secondary survey.
- Commence relevant secondary survey treatment.
- Prepare the casualty for evacuation from the pontoon to the athlete medical room or directly to the hospital.
- Communicate directly with the venue medical manager regarding the medical condition of the casualty and the proposed medical management.

The medical team members will:

- Await report from field of play team leader.
- Team members called forward by team leader onto the pontoon.
- Team member 1 should bring red medical bag onto the pontoon and assists the team leader (see above).
- Team members 2 and 3 should bring scoop and basket stretcher onto pontoon (if appropriate) and assist the team leader (see above).

The ambulance team will:

- Move on to the pontoon if requested to assist by the team leader.
- Assist in the packaging of the casualty and removal from the pontoon to the athlete medical room or hospital.

23.3.1 Medical Treatment

The field of play team will provide immediate life-saving treatment on the evacuation pontoon for clinical stabilization before packaging the casualty for transfer. The

casualty will be transferred either to the athlete medical room or to the hospital via the field of play retrieval ambulance.

The athlete will be transferred either by:

- Walking casualties: transfer by walking or by wheel chair.
- Minor injury: non-walking transfer by wheel chair or basket stretcher and trolley bed.
- Major injury (including unconscious): transfer by scoop, basket stretcher and trolley bed.

The decision has to be taken by the field of play team leader together with the field of play team whether the casualty be taken to athlete medical room or directly to the hospital. It will be communicated to the venue medical manager via radio, who will make the necessary out-of-venue arrangements. This radio call will be logged in the VCC (Venue Command Centre) and the relevant FA (functional area) will be contacted. Further medical treatment will be carried out either in the medical room or in the field of play retrieval ambulance. For minor injuries, the casualty may be returned to his team, athlete lounge, or transferred by car to the LOCOG polyclinic in the OG.

23.3.2 Transferring to Hospital

For major injuries, where athletes need to be transferred straight to the hospital, athletes will be taken to either:

- Non-life threatening—transfer by ambulance either to the polyclinic in the Olympic Village or to the designated athlete hospital.
- Life threatening: transfer to the nearest major trauma center.

The sport athlete service team will be responsible for ensuring the athletes equipment is looked after on the venue and transported back to the athletes using sport vehicles post competition. One passenger can travel with the athlete in the ambulance to the hospital.

23.3.3 Medical Resilience

- The field of play retrieval team will return to the pontoon as soon as possible.
- The field of play team leader will either continue the treatment of the casualty or hand over management to a member of the field of play team or the sports medicine doctor (or may request the sport medicine doctor to take a field of play position).
- Equipment and drugs: additional full sets of equipment (red bags) and drugs will be available to replace those used on the field of play.

23.4 Swim Course Personnel

(a) Staff

- All the positions of the swim course personnel are described in the marine operation plan-games level competitions (Appendix Section ITU Event Organizer's Manual).
- *Technical officials*. The number of technical officials assigned to the swim course will be determined by the TD based on the number of athletes in the competition and the swim course layout.
- *Boats and their drivers*. Five surf boats (inflatable) will be available. The boat drivers must be aware of the rules concerning proximity to the athletes in the water (expected instruction from ITU technical official).
- *Lifeguards*. Ten lifeguards will attend the event to assist in controlling safety and direction on the swim course (FOP). The lifeguards should ensure the following:
  - In case of false start, carefully stop the course.
  - Athletes round the turn buoys without cutting the course and without endangering themselves underneath the turn buoys.
  - In the event of an emergency, the chief lifeguard becomes the swim coordinator and controls the FOP. All personnel on the FOP must follow the directions of the chief lifeguard.
- *Scuba divers*. A team of six scuba divers will be on the FOP to ensure the safety of the athletes from under the surface, as well as to perform a final check of the turning buoys and markers pre-race.
- *Jet ski drivers*. A team of three jet ski drivers will be on the FOP to ensure the safety of the athletes. The jet skis will carry rescue sleds and they will interfere in case of an emergency.

ITU Triathlon Water Quality Statement

24

Sergio Migliorini

24.1 International Triathlon Union (ITU) Medical Documents

Triathletes are very intense exposed to the contaminated water during training and triathlon competitions. They can become infected due to water ingestion and/or due to skin or mucous membrane contact with the water (Schep 1992) [1]. It is very plausible that, during a competition, some athletes are more prone to develop infections than under normal circumstances. There are several physiological mechanisms during exercise that may lower the resistance to infections during and shortly after an endurance effort like a triathlon. It is mainly based upon three mechanisms:

1. The hormonal stress reaction.
2. The drop in some specific immune parameters such as the concentration and activity of natural killer cells.
3. The possible failure of local protective mechanisms of the gastrointestinal tract and of the skin.

24.2 Water Quality Problems

The most important health problem due to contaminated swimming water is associated with microbiological contamination.

For triathlon, the most relevant pathogens are in order of importance associated with the following risk factors:

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1. ***Human and animal faecal contamination***
   There is an association with the following pathogens: enterococci/faecal streptococci, *Escherichia coli* (*E. coli*), faecal coliformi, total coliform, salmonella, shigella, campylobacter, vibrio cholera, gastro enteritis viruses, enteroviruses, parasitic protozoa, some other exotic parasites, Aeromonas and plesiomonas.
2. ***The occurrence of rodents***
   Most relevant are brown rats and muskdeer, who can pass leptospirae in their urine into the water when they are infected. A determination of leptospirae in surface water is very complicated and not feasible. The most important reason is the very low dose that is necessary for an infection (below the limit of laboratory detection) and the fact that the concentration of leptospirae in the water is almost always extremely low.
3. ***Eutrofication of the water***
   Some pathogenic bacteria such as Aeromonas, plesiomonas, cyanobacteria can actually multiply themselves in surface water. This multiplication is stimulated if the water contains more nutritious matter (eutrofication). People may be exposed to blue-green algae or red tide algae toxins by drinking or bathing in contaminated water.
4. ***Bird colonies***
   Since birds can be infected with plesiomonas, Campylobacter and salmonellae, and they may contaminate the water with those organisms.
5. ***Industrial cooling water installations***
   It is proven that naturally occurring and potential severely pathogenic amoebas can grow in a hot environment like the plume of an industrial cooling installation.

24.3 Risk Factors

The most important risk factor is faecal contamination due to human faeces. Animal faeces is less important in harboring human pathogens. Human faeces can come into the water directly (bad sanitation, in many countries discharge of non-purified sewage and by discharge from house boats). However, also, purification of sewage is not able to control all the disease-causing organisms. Dependent on the flow and the extent of the discharge, there can be large fluctuations in the quality of the water.

Faecal contamination can harbor many potential pathogens. It is not feasible to test all those microorganisms. Further on, there is also a lack of data considering the quantitative relationship of the concentration of many of those pathogens and the risks of disease. Especially the viruses are the most hazardous.

24.3.1 The Commonly Used Indicator

Bacterial indicators of faecal contamination considered are enterococci/faecal streptococci, *E. coli*, faecal coliformi and total coliform. Thermotolerant coliform bacteria are present in human and animal faeces. However, they also occur in other environments that bear no relationship to faecal contamination (discharge from

paper industry, brewery's, and the like). *E. coli* is a species of faecal coliform that is specific for faecal material from humans and other warm-blooded animals. Enterococci are a subgroup within the faecal streptococcus group and are distinguished by their ability to survive in salt water. Faecal coliforms as a group was determined to be a poor indicator of the risk of GI illness [2].

Water quality control consists of:

(a) ***Sanitary inspection****:* Quite obviously measurable entities are not, however, so specific indicators of the water quality, such as:
- The color of the water that may not change in an abnormal way.
- The absence of oils that float on the water or that cause smelling.
- The absence of a smelling of phenol.
- A transparency of the water of more than one meter. When the diminishing transparency is caused by algae, this can be considered as a sign of eutrofication and of an indicator of inferior water quality.
- The absence of the blue-green algal bloom or the red tide algal-bloom.
- The absence of rodents.
- The absence of industrial cooling water installations.
- The absence of garbage.
- Sewage discharge or the occurrence of house and boats.

(b) ***Bacterial and or viral tests****:* Those tests are a measure for faecal contamination, and they are very important regarding swimming associated health risks.

Traditionally, the microbiological quality of waters has been measured by the analysis of indicator microorganisms. Human enteric virus are the most likely pathogens responsible for waterborne diseases from recreational water, but detection methods are complex and costly for routine monitoring and so the main parameters analyzed for compliance with the directive are indicator organisms.

EEA Standards

The ***Directive 2006/7/EC*** [3] reduced the number of parameters from 19 to 2 key microbiological parameters, and the faecal contamination is assessed by determination of two mandatory indicator bacteria:

- *E. coli*
- *Enterococci*

The choice of microbiological parameters is based on available scientific evidence provided by epidemiological studies conducted by the WHO and health institutes in Germany, France and The Netherlands.

For some characteristics, *E. coli* may be considered a more useful indicator than faecal coliforms and it has been included in all recent laws regarding fresh, marine and drinking waters [4, 5]. *E. coli* and enterococci are adequate indicators of GI illness in marine water. In fresh water, *E. coli* is a more reliable as consistent predictor of GI illness than is enterococci. *E. coli* was superior to enterococci at predicting illness, and the *E. coli* guideline level was supported because exposure below presented no significant risk, whereas exposure above were associated with an elevated and statistically significant increased risk of GI illness [6–8]. The body of literature

does support the use of enterococci and *E. coli* as useful predictors of GI illness in marine environment [9]. Also, there are standards such as salmonellae and enteroviruses; however, one will not determine those microorganisms on a routine basis.

EEC has a guideline limit that indicates the value that one should try to realize excellent swimming water.

A	B	C	D
	Excellent quality	Good quality	Sufficient quality
For sea and transitional waters			
(1) Enterococci ufc/100 ml	100[a]	200[a]	185[b]
(2) *E. coli* ufc/100 ml	250[a]	500[a]	500[b]
For inland waters			
(1) Enterococci ufc/100 ml	200[a]	400[a]	330[b]
(2) *E. coli* ufc/100 ml	500[a]	1000[a]	900[b]

[a]According to percentile 95°
[b]According to percentile 90°

(c) ***Weather forecast****:* The microbiological contamination could improve where the event is running under rainy conditions.

24.4 Standards Under Discussion

It is very difficult to prove that an infection is due to an exposure with swimming in a surface water since:

1. The symptoms are often quite vague and a specific. So the disease in often not properly diagnosed.
2. Viral examinations are very difficult, because there can be so many possible viruses responsible for the disease.
3. Numerous epidemiological studies of waterborne illness in developed countries indicate that the common aetiological agents are more likely to be viruses and parasitic protozoa than bacteria [10].
4. It is very difficult to establish the relationship with the exposure to the swim water because there is no routine testing for those viruses and because the symptoms in general only start several weeks after the exposure.
5. No treatment is available for those viral illnesses, so a practical working doctor is no so eager to find out which virus was the cause of the disease. On the contrary, when a doctor considers a possible leptospirosis infection that may look quite similar, it has much more practical implications for the treatment to determine whether or not the patient is infected.

We learn from this that it may very well be possible that the health problems associated with faecal contaminated swimming water do not restrict themselves to the rather innocent gastroenteritis.

24.5 ITU Water Quality Rules

Since 2010 ITU adopted the EEC standards 2006/7/EC of bathing water quality.

Especially for international competitions, it is absolutely necessary to get a clear view on the water quality of such competitions. Since in some countries, there is a risk for some very serious diseases causing health problems [11].

24.5.1 Water Quality Tests Submit to ITU

According to ITU rules, the Local Organizing Committee (LOC) must submit water quality tests:

- Two months before the competition
- Seven days before the competition
- on the first competition day of the event for statistical purposes only.

Samples of the water collected from three different locations on the swim course will be separately analyzed and the poorest results will determine if the swim can take place. The swim will be allowed if the following values are below the level of tolerance in the different types of water.

(a) ***Sea and Transitional Waters***:
- PH between 6 and 9.
- Enterococci not more than 100 per 100 ml (ufc/100 ml).
- *E. coli* not more than 250 per 100 ml (ufc/100 ml).
- Absence of positive visual evidence of red tide algal bloom.

(b) ***Inland Waters***
- PH between 6 and 9.
- Enterococci: not more than 200 per 100 ml(ufc/100 ml).
- *E. coli* not more than 500 per 100 ml(ufc/100 ml).
- The presence of blue-green algal bloom/scum (cyanobatteria) with more than 100,000 cells/ml [12].
- This test is only acquired in case of positive visual evidence of blue-green algal bloom. Because of the potential for rapid scum formation, daily sanitary inspection is mandatory by the LOC medical director in the two weeks before the competition in the area prone to scum formation. Where not scums are visible but the water shows strong greenish discoloration,

turbidity, and the transparency is less than 0.5 m, the cyanobacteria test must be performed.

ITU recommend to organize only triathlons in swimming water that falls in the Excellent water quality category. If the water quality test shows values out of the tolerance limits as indicated above the swim will be cancelled, unless the ITU Medical Committee permits the Good Water Quality category.

The water quality results delivery may vary from 48 h to 96 h depending on the methodology that the laboratory is using; therefore, making a decision on competition delay based purely on data that is not reflecting the current conditions 100 percent is not advisable. Particularly in cases where the event is running under raining conditions, ITU uses approach recommended by WHO [13] to guide decision-making (water quality decision matrix) [14] by combining:

- water quality analysis
- sanitary inspection
- weather forecasts

Water quality decision matrix—sea and transition water

		Two past results *E. coli* < 250 Enterococci < 100	Last results *E. coli* 250–500 Enterococci 100–200	Two past results *E. coli* 250–500 Enterococci 100–200	Last results *E. coli* > 500 Enterococci >200
Sanitary inspection category	Low	1	2	2	4
(Susceptibility to fecal influence)	Moderate	1	2	3	4
	High	2	3	3	4

Key for Levels

1. *Very good water quality*: *E. coli* < 250 ufc/100 ml or enterococci < 100 ufc/100 ml with no or potential visual pollution during sanitary inspection or forecasted heavy rain;
2. *Good water quality*: *E. coli* < 250 ufc/100 ml or enterococci < 100 ufc/100 ml with poor visual pollution during sanitary inspection or forecasted heavy rain;
3. *Good water quality*: *E. coli* 250–500 ufc/100 ml or enterococci 100–200 ufc/100 ml with no or potential visual pollution during sanitary inspection or forecasted heavy rain;
4. *Fair water quality*: *E. coli* 250–500 ufc/ml or enterococci 100–200 ufc/ml but with potential or poor visual pollution during sanitary inspection and/or potential for forecast heavy rain;

5. *Poor water quality*: *E. coli* >500 ufc/ml or enterococci >200 with any visual pollution during sanitary inspection and /or potential for forecast heavy rain.

Water quality decision matrix—inland water

		Two past results *E. coli* < 500 Enterococci < 200	Last results *E. coli* 500–1000 Enterococci 200–400	Two past results *E. coli* 500–1000 Enterococci 200–400	Last results *E. coli* >1000 Enterococci >400
Sanitary inspection category	Low	1	2	2	4
(Susceptibility to fecal influence)	Moderate	1	2	3	4
	High	2	3	3	4

Key for Levels

1. *Very good water quality*: *E. coli* < 500 ufc/100 or enterococci < 200 ufc/100 ml with no or potential visual pollution during sanitary inspection or forecasted heavy rain;
2. *Good water quality*: *E. coli* < 500 ufc/100 ml or enterococci < 200 ufc/100 ml with poor visual pollution during sanitary inspection or forecasted heavy rain;
3. *Good water quality*: *E. coli*500–1000 ufc/100 ml or enterococci 200–400 ufc/100 ml but no or potential visual pollution during sanitary inspection or forecasted heavy rain;
4. *Fair water quality*: *E. coli* 500–1000 ufc/100 ml or enterococci 200–400 ufc/100 ml but with potential or poor visual pollution during sanitary inspection and/or potential for forecast heavy rain;
5. *Poor water quality*: *E. coli* > 1000 ufc/100 ml or enterococci > 400 ufc/100 ml with any visual pollution during sanitary inspection and/or potential for forecast heavy rain.

ITU recommend to organize only triathlon in swimming water that falls in the Decision Matrix Very Good Water Quality category. If the water quality decision matrix show values out of the ITU tolerance limits, the swim will be cancelled, unless ITU Medical Committee permits the Good Water Quality category.

4/02/2019

Dr. Sergio Migliorini

ITU Medical Committee Chair

On behalf of the ITU Medical Committee

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Part VII

The Paratriathlon

ITU Paratriathlon History

25

Eric Angstadt Torres

The growth and development of paratriathlon inside the International Triathlon Union (ITU) has a history of more than 20 years. There are some known isolated participations in 1983 and 1984 who may be the very first ones inside recognized triathlon competitions in the USA. Slowly, it became more popular till the number was big enough to have them kind of structured in categories based on impairments and racing together with the age group waves. In 1996 in Cleveland, this recognition led to the first official World Championships for paratriathletes inside ITU. It was named as AWAD (Athletes with a Disability) and was run under this name till 2005 when the term Paratriathlon was set. It was run under the Olympic distance till 2009, when it was changed to Sprint distance and has remained like this ever since.

One of the main goals since 2008 and led by newly elected ITU President Marisol Casado was to achieve inclusion in the Paralympic Games (PGs). There has been an active Paratriathlon Committee also elected in 2008. Inclusion at the PGs was achieved at the end of 2010 conditional to ITU changing the existing classification system to one that would be sport-specific and evidence based. A specialized staff was hired to help in this process and intense research started at the end of 2011 and has been an ongoing activity since then. A first iteration of a new system was introduced in 2014 and used for the Rio de Janeiro 2016 PGs. A second revision was introduced immediately post-Rio and will potentially be reviewed post Tokyo 2020.

Paratriathlon has experienced a huge growth in numbers and spread worldwide, it has increased the number of competitions with a model of full inclusion inside the ITU and the able-body races in order to maximize the resources of existing venues and high-level events and athlete overall experience. There is a limit of participation in the different level of races to ensure safety and to manage the events effectively with high standards.

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Since 2017, live streaming of major paratriathlon events was introduced and since 2014, the interval start system was applied for classes in the Visually Impaired medal events and since 2017 in the PTWC (paratriathlon wheelchair class) that offer exciting competition and more inclusion with certain impairments.

There is still a lot of growth potential in Asia and Africa as well as South America and numbers continue to grow worldwide.

- 1980s—first Paratriathlon held.
- 1996—Cleveland (USA) First official World Championships for Paratriathlon as part of the age group waves with separate rankings.
- Annual World Championship under ITU as part of the age group and monitored by the Athletes with a Disability Commission until 2005.
- From 2005 to date, separate specific waves for the paratriathlon categories.
- 2007—first formal classification (Hamburg).
- 2008—British Triathlon Federation led development of profile-based classification system, later amended and adopted by ITU in 2009.
- December 2010—accepted into the 2016 Rio de Janeiro Paralympic Games, conditional to developing a sport-specific evidence-based classification system.
- 2011–2013—research conducted to develop an evidence-based classification system.
- 2013 London—198 athletes at the World Championships.
- 2013—33 National Federations (NFs) and 305 athletes.
- 2014—new classification system and education program for classification seminars by ITU facilitators were introduced (since 2014 to date—12 seminars in four continents).
- Since 2014, participation quota at ITU Paratriathlon events limited by class.
- 2016—42 NFs and nearly 500 athletes.
- 2016 debut at the 2016 Rio de Janeiro Paralympic Games—three classes per gender—60 athletes total.
- 2017—revised classification system, nine sport classes (three for visual impairment, two for wheelchair classes, and four for ambulant paratriathletes).
- 2017—introduction of the high level World Paratriathlon Series races, including live streaming.
- 2018—55 NFs and nearly 600 athletes worldwide.
- 2020—Tokyo PGs: four medal events per gender and 80 athletes.

Physiological Considerations for Paratriathlon Training and Competition

26

Ben Stephenson and V. L. Goosey-Tolfrey

26.1 Introduction

The sport of paratriathlon made its Paralympic Games debut in Rio de Janeiro, September 2016. As noted by Mujika et al. [1], the inclusion of the sport at the Paralympic Games has set clear performance targets, which have resulted in a rise in professional paratriathletes. Despite the growing status of paratriathlon, there remains very little literature concerning the sport. To date, the case study of a single, male, below-knee amputee paratriathlete's training habits by Mujika et al. [1] remains the only source of peer-reviewed information. However, whilst the work of Mujika et al. [1] provides a starting point for research in paratriathlon, the report lacks applicability to many as the paratriathlete profiled was competing in Olympic distance races, not the sprint distance typical of paratriathlon.

The lack of literature centred on paratriathlon opposes that of Olympic triathlon. The physiology [2], training habits [3] and strategies to improve performance in the heat [4] have all been investigated in the able-bodied (AB) variant of the sport. However, these topics are severely understudied in paratriathlon. Furthermore, the transferability of the findings from Olympic triathlon to paratriathlon is to be questioned due to certain consequences of physical disabilities such as spinal cord injury (SCI), cerebral palsy (CP), amputation (AMP) or visual impairment (VI). Thus, it is pertinent for those working in the sport to have a better understanding of the aforementioned topics and to learn how transferable findings are between AB and Paralympic triathletes. Specifically, there is a need to understand how this population responds to training from a physiological perspective and how training can be manipulated to attenuate any performance decrement and thermoregulatory strain when competing in the heat.

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26.2 The Paratriathlete

Paratriathlon is a multi-disability sport with athletes' impairments typically consisting of SCI, CP or other neurological disorders, AMP or VI. Accordingly, coaches and practitioners working in the sport must have an appreciation of the many consequences of athletes' impairments that may affect performance or training capabilities and how they may differ for AB athletes. The following sections will outline the consequences of specific impairments before discussing how they may relate to paratriathlon training and/or performance.

26.2.1 Spinal Cord Injury

The spinal cord, enclosed in the vertebral column, is the major channel through which afferent and efferent information is relayed between the brain and body [5]. Damage to the spinal cord, and subsequent injury, can occur through the application of excessive forces (e.g. motor vehicle collision) or via degenerative and congenital disorders. Spinal cord injury (SCI) can be defined as either tetraplegia or paraplegia, depending on the level of injury. Tetraplegia refers to injuries of the cervical region of the spinal cord and results in a loss of function in all four limbs. Paraplegia, meanwhile, refers to injuries of the thoracic, lumbar or sacral regions of the spinal cord and causes a loss of function in the trunk, organs and legs below the lesion level. Commonly, paratriathletes with an SCI compete in the PTWC category (Fig. 26.1). However, ambulant athletes with an incomplete SCI also can compete in ambulant paratriathlon categories depending upon the functional capacity assessed during classification.

Spinal cord injury is the most extensively researched physical impairment with respect to athletic performance [6]. A great deal is known from other Paralympic sports concerning the physiological disparities of athletes with an SCI compared to AB athletes [7, 8]. These include, amongst others, lowered active muscle mass, lower stroke volume as a consequence of an impaired muscle pump in the lower limbs and a potentially lowered maximum heart rate (HR) [6]. All consequences of SCI are proportional to lesion level and completeness such that athletes with a high level, complete lesion display greater impairment, whereas those with a lower level, incomplete lesion will display physiological responses to exercise more akin to AB athletes [7].

Athletes with an SCI display mechanical limitations such as impaired trunk function, as a result of muscle paralysis, that reduces coughing efficiency [9]. Furthermore, this population group may be prone to immunoendocrine deficiencies due to thedecentralisation of the autonomic nervous system [10]. Additionally, muscle paralysis commonly necessitates the use of a wheelchair for daily ambulation, resulting in significant use of the upper limbs during daily life and paratriathlon training [11]. Furthermore, athletes with an SCI display diminished muscle mass in comparison to AB athletes, resulting in greater peripheral fatigue [12]. This may interact with athletes' perceptions of effort during, or after, exercise as peripheral

Fig. 26.1 Example of a PTWC athlete with a spinal cord injury. Image courtesy of British Triathlon

cues outweigh central cues. Injury to the spinal cord also results in a lack of afferent input to the thermoregulatory centre with a loss of vasomotor control and sweating capability below the lesion level [13]. This is further exacerbated by a loss of venous muscle pump to redistribute blood from paralysed muscle [14]. A supplementary consequence of significant muscle paralysis is that athletes commonly display high proportions of body fat [15, 16]. For example, in their case study of a PTWC H1 paratriathlete with an SCI, Graham-Paulson et al. [16] reported a body fat of 25.4%, as assessed via duel-energy X-ray absorptiometry. This is considerably higher than 15.1 ± 5.6% shown by Mueller et al. [17] in their descriptors of male AB triathletes, employing similar methods.

26.2.2 Cerebral Palsy

Cerebral palsy (CP) is a postural and movement disorder caused by central brain injury at a young age which results in altered neuromuscular physiology and diminished exercise capacity [18]. CP presents three main impairment profiles: hemiplegia, where one side of the body is affected; diplegia, where two limbs are affected (typically the lower limbs); quadriplegia, where all four limbs are affected [19]. Athletes with CP signify a large proportion of the Paralympic athlete population, including participation in paratriathlon. In paratriathlon, athletes with CP may compete in the PTWC category if they are wheelchair users, whilst ambulant

Fig. 26.2 Example of a paratriathlete with right-side hemiplegia cerebral palsy. Image courtesy of British Triathlon

athletes with CP compete in PTS2 to PTS5, depending on their impairment severity (Fig. 26.2). Despite the commonality of athletes with CP in Paralympic sport, and their impairment presenting several areas for consideration [18], research is emerging only now to help support athletes.

Runciman et al. [18] studied the pacing strategies of track and field athletes with CP in comparison to an AB group over a distance-deceived (1000 + 600 m) and non-deceived shuttle run test (1600 m). During the distance-deceived trial, athletes were under the assumption the trial would be completed at 1000 m, thus paced themselves accordingly. The authors noted that athletes with CP commonly underperformed in comparison to the AB group through overly conservative pacing as a potentially protective mechanism. Specifically, the CP athletes displayed a flatter pacing profile during the distance deceived trial. Runciman et al. [18] also found that for the same relative intensity athletes with CP had a tendency to report lower rating of perceived exertion (RPE) values in comparison to the AB group. As such, there are potential considerations regarding pacing and perception of effort during longer duration tasks in this impairment group.

Athletes with CP display significant movement inefficiencies (Blauwet et al. [20]). Specifically, the presence of athetosis, spasticity, or ataxia may all increase the energy cost of movements [20]. As such, the internal workload required to produce a set absolute intensity is significantly greater in athletes with CP, thus increasing the susceptibility to excessive overload. Moreover, it has been shown, albeit in children, that the metabolic heat production for an absolute workload is elevated

in this population group due to the aforementioned movement inefficiencies [21]. Additionally, athletes' high muscular tone impairs venous return via muscle pumps and negatively impacts blood flow distribution [22].

26.2.3 Amputation

Although athletes with an amputation (AMP) represent a large proportion of Paralympic athletes, very little research has focused on the physiology of these athletes, with most literature centred on biomechanical properties [23]. In paratriathlon, athletes with an AMP may compete in wheelchair or ambulant classes, depending on the level of their impairment (Fig. 26.3). Athletes with an AMP contend with the same issue of reduced active muscle mass as athletes with an SCI [24]. Similarly, wheelchair athletes with an AMP face the common issue of excessive upper limb use, due to the associated large loads of daily locomotion and competitive performance during the inefficient movement of chair propulsion [11]. Likewise, ambulant upper limb amputees display a similar propensity for upper-body overuse syndromes due to excessive loads from residual limb overcompensation [25], which is likely further exacerbated by the swimming component of paratriathlon training. Furthermore, similar to athletes with CP, ambulant athletes with an AMP also suffer from movement inefficiencies [20, 26]. The energy cost of ambulation at a set workload is significantly greater for amputees than AB individuals with ascending levels of AMP creating increased metabolic demand [26].

Fig. 26.3 Examples of wheelchair-dependent and ambulant paratriathletes with a lower-limb amputation. Images courtesy of British Triathlon

A considerable uniqueness to athletes with an AMP is the lower body surface area to volume ratio as the result of missing limbs [27]. A lower body surface area diminishes the capacity for evaporative and convective heat loss, increasing heat storage in this population. Furthermore, socket liners and prostheses act as an insulating barrier, limiting heat dissipation [28] and promoting stump skin injuries [28, 29]. Moreover, skin grafts on amputees, or other athletes, removes a portion of the skin capable of sweating and cutaneous vasodilation [30].

26.2.4 Visual Impairment

Individuals with a VI may participate in paratriathlon, in the PTVI category, whilst displaying a range of impairment severities (Fig. 26.4). Athletes can be categorised in one of the following three levels: B1 are considered blind athletes; B2 are considered to have severely impaired vision; B3 are considered to have moderate to poor vision. VI includes athletes with albinism, an impairment caused by genetical mutations that can result in diminished melanin synthesis affecting the skin, eyes and hair [31]. Whilst athletes with VI may be physiologically similar to AB athletes, they still require several considerations as a consequence of their impairment.

Firstly, athletes with VI are restricted in their ability to utilise visual feedback [32], that may have implications in motor learning, proprioception and developing

Fig. 26.4 Example of a female paratriathlete with a visual impairment (right), competing in the PTVI (B3) category, and her able-bodied guide (left). Image courtesy of British Triathlon

robust techniques (e.g. swimming or running) to tolerate training loads (TLs) [33]. Additionally, restricted visual feedback has been proposed to impact the ability to monitor personal pacing [32]. It has been shown that swimmers with a VI display a significant alteration in their pacing profile compared to AB athletes, suggested to be related to an undermined pace awareness [32]. Furthermore, compromised visual feedback not only relates to sporting movements but will also present difficulties in self-monitoring hydration through urine colour or volume [11]. Finally, athletes with albinism are more prone to sunburn in situations of high radiant load due to the lack of skin pigmentation [31].

26.2.5 Conclusion

In summary, there are several consequences of Paralympic athletes' impairments that must be considered by those working within paratriathlon. The following sections discuss how these consequences affect paratriathlon training and competition, with particular focus on athletes' physiological response to TL and thermoregulation during competition.

26.3 Training Considerations

26.3.1 Considerations for Training Load Quantification

Management of training is fundamental to coaches, athletes and sports scientists to prevent both under- and overtraining in an attempt to optimise performance outcomes and manage injury and/or illness risk [34]. To manipulate training for performance enhancement, training must first be quantified as a TL [35]. Several physiological, psychological or external measures have been utilised in the quantification of internal or external TL.

Whilst methods of TL quantification have been assessed in AB triathlon [36], there has been little work in paratriathletes. This is despite paratriathletes representing a population potentially at greater need of TL quantification and monitoring. For example, athletes with an SCI are particularly prone to overuse injuries and illnesses [37, 38], with the latter resulting from coughing inefficiencies and/or immunoendocrine deficiencies [9, 10]. PTWC athletes with an SCI, CP or an AMP are at heightened risk of shoulder overload due to the stresses of daily wheelchair use [11, 37]. This is further aggravated by paratriathlon training modalities whereby the upper limbs are in use extensively. Moreover, ambulant athletes with CP, or an AMP, display movement inefficiencies, increasing the metabolic cost of locomotion [20, 26], augmenting the susceptibility to excessive TLs. Finally, athletes with a VI may be more susceptible to TL intolerance and overuse injuries as a consequence of an inability to learn robust techniques [33]. However, quantifying TL in groups of athletes with physical impairments raises certain issues that must be considered. This includes potentially limited sympathetic responses to exercise in athletes with

an SCI, limiting the use of HR as a training tool [39]. Furthermore, there may be limits to the use of session RPE [40] in some populations due to altered perceptions of effort, such as athletes with CP [18].

26.3.2 Training and Illness Risk

Whilst it is well accepted that athletes' TLs must be of a sufficient level to stimulate a positive training adaptation [34], it has also been acknowledged that high TL increases the likelihood of illness or injury [41]. Specifically, the most common medical complaint is illnesses of the upper respiratory tract (URI) [38], with evidence of high training stress supressing the ability of the innate immune system to combat pathogens and increasing URI incidence [41–43]. Contraction of URI in athletes is likely to have a direct effect on impairing performance or limit training availability [42]. Thus, URIs are an unwanted outcome of insufficiently managed TLs making any information of the relationship between TL and URI valuable for coaches, medics and sports scientists.

Though the interaction between training-imposed physiological stress and immune function is multi-faceted [44–46], one area which has received a great deal of attention is the suppression of mucosal immunity. Over 90% of infections involve the mucosa [47], and a key antibody in host defence of the upper respiratory tract, and the first line of defence in mucosal immunity, is the salivary secretory immunoglobulin A (sIgA). The role of sIgA in defence against URI has been well studied with research tracking AB athletes over prolonged periods noting for evidence that lower sIgA levels relates to URI incidence [43, 44, 48].

The proposed mechanism through which depressions in sIgA, and resultant URI incidence, occur is through chronically high TLs with insufficient recovery [45]. Specifically, it has been speculated that prolonged sympathetic nervous system activation and elevated cortisol, via regular intense exercise, may down-regulate sIgA synthesis. Furthermore, sIgA secretion may be attenuated through the inhibitory effects of sympathetic nervous system activation and cortisol on sIgA transcytosis through the epithelium [46]. Support for the notion of TL negatively effecting sIgA levels has come from the findings of several authors [43, 44, 48]. As such, it is proposed that high TL suppress mucosal immunity, i.e. sIgA, which, in turn, heightens the risk of URI.

Despite the great deal of attention interactions between TL and mucosal immunity have received in the AB population, there is a scarcity of research in Paralympic athletes. This is despite athletes potentially being at heightened risk of URI due to the propensity for excessive overload, and therefore TLs, caused by movement inefficiencies. Furthermore, Paralympic athlete populations may display a greater propensity for illness [9, 10]. Leicht et al. [49] presented a negative correlation between TL and sIgA secretion rate in wheelchair rugby players with an SCI over a five-month period. They noted that the effects of TL on mucosal immunity were not different from those of AB athletes [49]. Conversely, Edmonds et al. [50] found no changes in sIgA concentration over 14 weeks of periodised training in a group

of swimmers of mixed disabilities. From a paratriathlon perspective, preliminary evidence suggests that sIgA secretion rate is impaired by high weekly training durations, but not internal or external TL [51]. Furthermore, there appears to be no evidence of a relationship between sIgA parameters and URI incidence, although in 50% of URI incidences, training availability was impaired, thus highlighting the need to minimise illness risk [51].

26.3.3 Intensified Training

Whilst coaches and sports scientists aim to longitudinally manage athletes' TL to minimise illness or injury risk, it is commonplace to plan intentional, acute, overload of training in an attempt to maximise beneficial outcomes. Typically, these periods of intensified training (IT) involve progression of session duration and intensity of frequency [52], thus elevating TL above normal levels; these are often undertaken during training camps. During periods of IT, it is possible for athletes to become overreached, whether it be functional or non-functional. In fact, some coaches view OR as a deliberate part of the training process due to the potential for improved performance after a taper period [53]. Functional OR is defined as an accumulation of training and/or non-training stress resulting in short-term decrement in performance capacity with or without related physiological and psychological signs and symptoms of maladaptation in which restoration of performance capacity may take from several days to several weeks [54]. If IT were to continue for athletes displaying functional OR, the development of non-functional OR or even overtraining syndrome is possible whereby any recovery in performance may take several weeks, months or years [54]. As such, it is important for coaches and sports scientists to be able to monitor athletes for any signs of potential OR prior to significant performance decrement.

As the definitions of Meeusen et al. [54] allude to, the most accurate way of assessing OR in athletes is through testing performance, preferably at a maximal, sport-specific level [54]. However, the use of maximal performance tests brings a certain level of disruption to athletes' training routine either by invoking further fatigue or through excessive rest and subsequent detraining [55]. Consequently, efforts have been made to identify markers of OR, after IT, that are less taxing to athletes [56].

To date, several non-invasive parameters have been studied in relation to IT and potential OR. Two of the most commonly assessed variables are the responses of resting cortisol and testosterone. Cortisol, a major catabolic hormone, is secreted from the adrenal cortex, via the hypothalamic–pituitary–adrenal axis and increases in response to stressors [57]. Testosterone, meanwhile, is the primary steroid hormone within the androgen family and its secretion is regulated by the hypothalamic–pituitary–gonadal axis [57]. Due to the steroid structure of both hormones, they freely diffuse into saliva which can offer a less invasive medium for sampling than plasma [57]. Salivary cortisol and testosterone represent the biologically active portion of the body's hormones, with the use of both together representing the body's

catabolic:anabolic balance [52]. It had been proposed that after periods of IT there is an increase in the biologically active, free cortisol with a concomitant decrease in free testosterone; thus, representing a more catabolic state as the body fails to maintain homeostasis with a challenging stress-recovery balance [58]. Whilst salivary cortisol and testosterone have commonly been assessed in AB athletes with respect to IT, this has not been regularly explored in Paralympic athletes. This is despite athletes being at greater risk of OR as a consequence of their physical impairment heightening the internal load of external workloads. Furthermore, immunosuppression is another aspect of athletes' responses to IT that has received attention. It has been well documented that individuals are more susceptible to illness after periods of IT [46] and when overreached [59]. Thus, Coutts et al. [56] proposed that sIgA may also be a sensitive marker in response to IT due to the relationship between training stress, mucosal immunity and URI incidence.

Sleeping habits have recently been assessed in relation to IT and OR. Sleep is a vital part of recovery where many biological regenerative processes occur; furthermore there are close links between sleep, immune function and mood state [59, 60]. A recent review concluded that sleep quality is typically impaired during training camps [61], with Hausswirth et al. [59] evidencing this by presenting greater wake times and movement, as measured by wrist actigraphy, during a period of IT in AB triathletes. However, it has been shown that sleep quantity may increase during IT as greater TL augments the requirement for sleep [62]. As such, sleep responses to IT may be specific to the parameter measured. Interestingly, whilst sleep quality was objectively impaired in the study of Hausswirth et al. [59], subjective sleep quality was not altered. Recently, Caia et al. [63] state that although athletes may be able to accurately perceive sleep duration, subjective sleep quality is a poorer indicator of sleep efficiency in comparison to actigraphy. It is worth noting that the responses of any sleep parameters to IT have been understudied in athletes with a physical impairment. This is despite the review of Gupta et al. [61] stating that levels of insomnia symptoms can be up to 70% greater in Paralympic athletes, which may relate to thermoregulatory impairments or altered melatonin secretion in athletes with an SCI [64]. For example, Silva et al. [65], in their study of Paralympic track and field athletes, reported 83% of athletes presented excessive daytime sleepiness and had poor sleep quality, albeit from subjective assessments.

One area in the responses to IT that has received a great deal of support is psychological measures. Typically, psychological measures are in the form of self-report questionnaires of mood state, such as the Profile of Mood States (POMS), or subjective stress and recovery such as the Recovery-Stress Questionnaire for Sport (RESTQ-S). POMS is a questionnaire that provides a method for assessing transient, fluctuating mood states. It includes five negative affect scales: fatigue, depression, tension, anger, confusion and a positive affect scale, vigour. Participants are given a score for each of the six mood states and as a total mood disturbance [66]. RESTQ-S measures include scales which assess various stressing agents of a

general nature and general recovery activities and additional sports-specific scales. Outcome measures include total, general or sport-specific stress and recovery [66]. Several studies have shown support for the use of psychological questionnaires and subjective wellness measures to detect OR after IT [52, 53, 55, 56, 66] with suggestions that psychological changes are the first manifestation of OR [66].

Although the effects of IT and markers of OR have been studied in many sports the sport of triathlon has enabled researchers to deliberately overreach athletes [53, 55, 56, 59]. This is partly due to triathletes' habitually high TLs and, therefore, 'at risk' status of OR [55]. Despite the focus IT has received in triathlon, there is little evidence in paratriathlon; this is in spite of reports that paratriathletes may be undertaking large TLs with potential IT periods [1]. As Zeller et al. [67] state, to avoid the risk of overtraining, it is important that scientific knowledge in the area of disability sport be developed. This is due to physical disabilities placing Paralympic athletes at greater risk of excessive overload, thus OR, and the potential for performance impairment. Unpublished observations in paratriathlon suggest training camp environments can actually invoke beneficial adaptations despite large increases in TL. By minimising external life stresses, and through careful management of athletes' training, improvements in perceived recovery, stress and sleep can occur, without negative consequences on hormonal (salivary cortisol and testosterone) and immunological (sIgA) parameters [51].

26.3.4 Conclusion

In summary, coaches and sports scientists must consider how paratriathletes' TL may impact upon their likelihood of URI or OR incidence. Whilst paratriathletes display physical impairments that may increase their susceptibility to illness or excessive overload, preliminary evidence from our research group suggests that this population is robust to changes in TL. Nonetheless, caution should be applied when generalising these findings due to the great individuality in paratriathletes and differing impairments representing differing risks to training unavailability.

26.4 Thermoregulation and Preparing for Competition in the Heat

Whilst coaches and sports scientists aim to manage paratriathletes' TL, to minimise the likelihood of training unavailability through URI or OR, the overarching goal is for athletes to perform to their maximum potential in competitive situations. However, it is common for competitive events to be held in environments that challenge athletes' endurance exercise performance due to high ambient temperatures or humidity (Table 26.1). Thus, it is pertinent to understand the thermoregulatory toll imposed by competition in the heat and how to optimally prepare.

Table 26.1 Historic weather data at selected paratriathlon

Date	Event	Location	Temperature (°C)	Relative humidity (%)
10th July 2015	ETU European championships	Geneva, Switzerland	29	18
18th July 2015	ITU world Paratriathlon event	Iseo, Italy	35	31
1st August 2015	ITU world Paratriathlon event	Rio de Janeiro, Brazil	31	52
11th September 2016	Paralympic games	Rio de Janeiro, Brazil	28	66
8th October 2017	ITU world Paratriathlon cup	Sarasota, USA	30	80

26.4.1 Thermoregulation and Competition in the Heat

It is well understood that endurance exercise performance is impaired in hot and humid environmental conditions, with several studies reporting a diminished performance with added thermal load [68–70]. Endurance events, such as triathlon or paratriathlon, require high relative exercise intensities to be sustained for extended periods of time. This increases the likelihood of athletes developing substantial hypohydration, cardiovascular strain, elevated whole-body (core and skin) temperature, reliance upon carbohydrate metabolism, RPE and thermal strain, resulting in fatigue, under-performance or even heat illness [70–73].

Raised core (T_c) and skin (T_{sk}) temperatures, with a narrowing of the temperature gradient between the two parameters, has been attributed to performance decrements in the heat. This is through alterations of the central and peripheral nervous systems via elevated cardiovascular strain and decreased neural drive to maintain an external workload [74]. Thus, due to the influence of body temperature on performance, there has been great interest in determining the thermoregulatory toll imposed by endurance exercise in the heat, specifically in real-world athletic events.

Due to event durations, and the relative intensity of effort, studies have presented the thermoregulatory responses to triathlon races of varying lengths, including half-Ironman and Ironman events. Research has shown that AB triathletes' T_c can reach 38.4 ± 0.7 °C [75] or 38.8 ± 0.7 °C [76] at the end of half-Ironman races in the heat. Furthermore, by acquiring in-race T_c readings, Baillot and Hue [75] presented a trend in T_c changes during racing. The authors showed that T_c: rose during swimming as a consequence of a limited capacity for heat dissipation in warm water; plateaued on the bike due to significant convective cooling through a high cycling velocity and drafting illegality increasing air flow; rose again when running as a result of limited convective cooling and a high metabolic rate [75]. Moreover, a similar trend has been presented in laboratory studies concerning sprint [77] and Olympic distance events [78]. However, data is still restricted to infrequent T_c sampling during field-based research. Moreover, there has been no field-based research detailing the

thermoregulatory strain of sprint distance triathlon events. Additionally, few studies have researched Paralympic athletes, despite athletes being at elevated risk of heat-related performance impairments.

Athletes with an SCI display well-acknowledged impairments in skin blood flow and sweat responses below their lesion level [13], reduced venous return [14], whilst the high body fat percentages will further increase the heat storage for a given workload [79]. Consequently, the finite literature regarding thermoregulation in Paralympic athletes has focused on those with SCI. In their case study of a male handcyclist with an SCI, Abel et al. [80] present a peak rectal temperature of 40.4 °C at the end of a 42 km race (1:48:54 h) in temperate (20.0 to 22.0 °C) conditions. Moreover, the athlete's T_c increased continuously throughout the race, highlighting the level of thermoregulatory disruption. In a larger study, Griggs et al. [81] revealed peak T_c values of 39.3 ± 0.5 °C for wheelchair rugby players with an SCI at the end of a competitive wheelchair rugby match in temperate conditions. Additionally, Griggs et al. [81] stated that the rate of T_c change was significantly greater in athletes with SCI in comparison to non-SCI, indicating impairment-specific responses. Similarly, Veltmeijer et al. [82] found greater increases in T_c in wheelchair tennis players with an SCI (0.6 ± 0.1 °C; $n = 2$) than players without SCI (0.3 ± 0.1 °C; $n = 4$) during simulated match play also in a temperate environment. However, research is even more limited in other impairment groups, despite athletes also being at risk of thermal strain.

Due to the greater metabolic heat production of locomotion in athletes with CP [21], they are at elevated risk of thermal strain and performance impairment than AB athletes [83]. Furthermore, this will be exacerbated by their high muscular tone impairing venous return [22], increasing the cardiovascular strain and relative intensity of exercise [84]. Additionally, athletes with CP's impaired pace awareness [18] may increase their risk of thermoregulatory strain. When competing in the heat, AB athletes typically progressively down-regulate their intensity of effort to redistribute work in a manner that allows them to complete the required task in the context of the accumulating heat strain [85]. This relates to not only physiological adjustments but also behavioural alterations to account for the cognitive interpretation of the environment, thermal state or perceived effort [86]. If athletes with CP are unable to effectively process the aforementioned factors, they may increase the risk of heat illness and/or performance impairment as a consequence of maintaining an inappropriate workload for the environmental context.

As a consequence of missing limbs, athletes with an AMP display limited body surface area for evaporative and convective heat loss, increasing heat storage in this population [27]. This also leads to a closer coupling of T_c and T_{sk} which is further exacerbated by socket liners and prostheses limiting heat dissipation [28]. Moreover, skin grafts remove a portion of the skin capable of sweating and cutaneous vasodilation [30], augmenting the impairment in thermoregulatory capacity. Consequently, athletes with an AMP may experience considerable thermal discomfort whilst exercising in the heat due to elevated T_{sk}. Also, similar to athletes with CP, those with an AMP display significant gait asymmetries [26], elevating metabolic heat production for a given workload. The thermoregulatory challenge posed

by individuals with an AMP was described by the case study of Andrews et al. [87]. The authors showed that an individual with a lower limb AMP completed a 16 km run in 12.0 °C slower, yet with a greater T_c (38.4 °C), than a non-amputee (37.9 °C). This was proposed to be due to the aforementioned effects of AMP on movement inefficiencies, metabolic cost of locomotion and impaired heat loss.

As athletes with a VI are limited in their capability to use visual feedback, this can directly impact pacing [32]. As previously mentioned, an inability to self-pace and down-regulate the intensity of effort when competing in the heat will increase the thermoregulatory toll and, thus, negatively impact performance. Furthermore, difficulties in self-monitoring hydration through urine colour or volume may cause issues when training or competing in the heat [11]. Ensuring proper hydration is a well-acknowledged method of alleviating heat stress when competing in thermoregulatory challenging environments [73]. Therefore, if athletes with a VI cannot self-regulate their hydration based on visual feedback, they increase their risk of thermoregulatory strain in hot environments. Finally, athletes with albinism are prone to sunburn in situations of high radiant load [31]. This exacerbates thermal sensation and limits thermoregulation during exercise through a locally mediated effect on sweat gland responsiveness and capacity [88].

Recent work has sought to characterise the thermoregulatory strain imposed by paratriathlon competition in the heat [51]. Collecting T_c data from paratriathletes in competition at the 2017 Iseo-Franciacorta WPC and 2018 Iseo-Franciacorta WPS, our research group present initial findings on this subject. It was noted that athletes' T_c peaked at temperatures significantly greater than previous reports from half-Ironman events [75, 76], with a mean of 39.50 ± 0.56 °C noted. Furthermore, 22 athletes displayed a peak $T_c \geq 39.5$ °C (Fig. 26.5). This was hypothesised to be

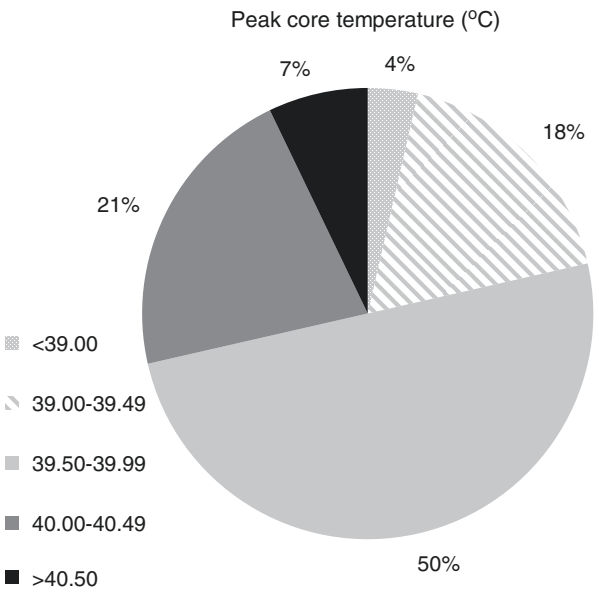

Fig. 26.5 Peak core temperature distribution during paratriathlon racing. Adapted from Stephenson [52]

due to the aforementioned consequences of paratriathletes' impairments increasing the thermoregulatory strain. Furthermore, by sampling T_c at 30 s intervals across the race, we noted a trend for T_c to continually rise when considering the average T_c across swim, bike and run sections. Lastly, there was an impairment-specific interaction as T_c when running was greater for PTVI athletes than those in PTWC. This is presumably due to the considerably disparate race demands across paratriathlon, depending on athletes' race category. Whilst not statistically significant, PTWC athletes displayed a greater change in T_c during cycling compared to ambulant race categories. This is likely to result from a lower surface area for convective heat loss, a closer proximity to the road surface for radiant heat gain and longer segment durations when handcycling. However, during the run segment, PTWC athletes in a racing wheelchair utilise less-active musculature and travel at a greater velocity, thus are exposed to greater air flow for convective heat loss, compared to ambulant runners. Therefore, it is not surprising that changes in T_c were significantly greater for PTVI athletes when running compared to those in PTWC as heat dissipative potential was lower and heat production was higher.

26.4.2 Preparing for Competition in the Heat

As explained, endurance exercise performance is impaired in environments that significantly stress athletes' thermoregulatory systems, yet, paratriathlon events are regularly held in such conditions. Thankfully, performance impairments can be ameliorated through chronic heat exposures and processes of heat acclimation (HA) or heat acclimatisation [71]. HA refers to repeated exposure to artificial environments, such as the use of heat chambers or hot water immersion, whereas heat acclimatisation concerns exposure to natural environments including warm-weather training camps.

Heat acclimation has been extensively researched and has been described as the most important intervention one can adopt to reduce physiological strain and optimise performance in the heat [89]. It is well understood that HA results in beneficial thermoregulatory, cardiovascular and perceptual adaptations that lead to attenuated impairments when performing in hot and humid conditions [71]. Specifically, HA has been shown to reduce T_c and $T_{sk,}$ improve cardiovascular stability, increase whole-body sweat rate and lower the threshold for sweating onset, decrease sweat electrolyte content, increase skin blood flow, induce plasma volume expansion, and decrease muscle glycogen use and blood lactate accumulation [71–73, 90].

To induce beneficial adaptations, HA typically consists of 5 to 16 d of heat exposure, with individual sessions of 1 to 2 h in temperatures ≥35.0 °C [71, 91, 92]. It has been proposed that heat exposures must be of sufficient duration and thermal strain to increase T_c, T_{sk} and sweat rate, the main drivers for adaptation in AB athletes, above a set threshold [90, 92–94]. It has commonly been reported that a significant proportion of HA adaptations occur within the first week of chronic heat exposures [95]. Hence, short-term HA (<8 d) has been commonly researched as it may be more applicable to elite athletes as it is potentially less disruptive to training regimes

[96]. Nonetheless, there is evidence that moderate-term HA (8–14 d) is required for full adaptation of several parameters such as sweat rate and end-exercise T_c and HR, which may be particularly meaningful for the preparation of athletes competing in endurance events such as triathlon or paratriathlon [71, 72, 90]. As such, although moderate-term HA induces greater thermoregulatory adaptations, strategies must be sought to minimise interference with athletes' training regimes to be efficacious.

Commonly, protocols have consisted of fixed intensity exercise over the acclimation period [97–99]. However, it has been speculated that this approach results in diminishing adaptations during the intervention as the relative thermal strain imposed gradually lessens [90, 94, 96]. Consequently, isothermic approaches have been employed. During isothermic HA, athletes maintain a set thermal strain, commonly a T_c of ~38.5 °C [96, 100, 101], over the HA period whilst external workload gradually increases concurrent to thermoregulatory adaptation. This provides a series of sufficiently overloading thermal stimuli to invoke continued adaptations [94]. It is worth noting, however, that isothermic HA presents challenges that limit its applicability to the elite athlete setting. Due to the requirement to continually measure T_c, there are issues with the financial cost of telemetric T_c pills, potential discomfort from rectal or oesophageal temperature instrumentation and inaccuracies of tympanic temperature [94]. Thus, alternative methods to regulate exercise intensity during HA have been proposed. One such method is by controlling the relative intensity and using HR to dictate the external workload performed [73]. Utilising a HR associated with a percentage of peak oxygen uptake or maximum HR would provide a constant cardiovascular stimulus as athletes acclimate [73]. Support for this proposed regimen comes from evidence of unchanged HR during isothermic HA [92, 96, 102]. As such, the use of HR rather than T_c may provide greater real-world application to elite athletes and coaches looking to acclimate to the heat [73, 90].

Despite isothermic HA providing a constant thermal strain for adaptation, its applicability for athletes, especially pre-competition, has been questioned [92, 103]. This is due to the impact successive days of exercising heat stress may have on athletes' fatigue and internal TL [93]. This is particularly pertinent in the multi-modal sport of triathlon or paratriathlon whereby coaches and athletes must balance the distribution of swim, bike and run training. Therefore, the efficacy of passive HA has been studied. Passive HA concerns exposing the body to environmental heat stress without the additive exercise strain, examples of which include the use of hot water immersion [92, 103, 104] or sauna exposure [91, 105]. These methods have been employed immediately after exercise in temperate environments, thus allowing individuals the opportunity to train without impairing session intensity whilst also invoking HA. Furthermore, prior exercise results in elevated T_c, T_{sk} and sweat rate before the commencement of passive HA, thus reducing the required heat exposure duration. Passive HA has been shown to induce positive thermoregulatory adaptations [91, 92, 104, 105], with some evidence of improved endurance performance [92, 105]. Nonetheless, the use of passive HA has also been questioned as it is unclear if adaptations are similar to that of active HA [71]. Moreover, it seems logical that prior to competition in hot environments, athletes should be perceptually

aware of the added strain of exercise and heat stress. This is particularly relevant in endurance sports whereby familiarisation with the heat, even without HA, induces pacing alterations to protect performance [86].

Acknowledging the respective merits of active and passive HA, the use of combined methods has been proposed as an approach to optimally acclimate in a time-efficient manner [72]. From preliminary evidence, it appears mixed active and passive HA may be capable of inducing beneficial thermoregulatory adaptations, such as a 7.1% expansion of plasma volume whilst increasing whole body sweat rate and decreasing resting and exercising tympanic temperature, HR and rate of oxygen uptake [103]. Additionally, due to the structure of the protocol, the logistical and physical demands of the athlete were reduced but potentiating stimuli for adaptation were maintained [103]. This approach may be particularly appealing to elite athletes, pre-competition, whereby best practise guidelines are rarely attainable within the confines of physical preparation [93].

To date, very few studies have researched HA in Paralympic athletes. Castle et al. [106] studied a small group of target shooters with tetraplegia ($n = 2$) or paraplegia ($n = 3$), undertaking a seven-day consecutive HA intervention consisting of 20 min moderate intensity, isothermic arm crank ergometry and 40 min rest. As HA resulted in a decrease in resting and exercising aural temperature, a decrease in RPE and thermal strain and a small increase ($1.5 \pm 0.6\%$) in plasma volume, this was the first evidence of beneficial adaptations in Paralympic athletes. Nonetheless, due to the lack of change in exercising HR or whole body sweat rate, the responses were deemed only partial acclimation [106]. As no sweat responses were noted during HA, as a consequence of athletes' impairments, it was speculated that the temperature of hot cerebral-spinal fluid perfusing the hypothalamus may be of greater importance for adaptation than peripherally derived, neural, thermal afferents [106]. This proposal is of particular relevance for athletes with impairments to peripheral thermal afferents, such as the Paralympic athlete population.

Despite the study of Castle et al. [106], research is still severely limited for Paralympic athletes and is confined to those with SCI. As described earlier, Paralympic athletes are likely to be at heightened risk for performance decrements in the heat as a consequence of the following: a loss of vasomotor control and sweating capability [13], reduced venous return [14, 22], movement inefficiencies increasing metabolic heat production [20, 26], impaired pace awareness [18, 32], limited surface area for heat loss via missing limbs or skin grafts [27, 30], limb insulation from socket liners [28], or a propensity for hypohydration [11] or sunburn [31]. Recent work, however, has aimed to bridge this knowledge gap. By utilising an 8-day mixed, active and passive, HR-controlled HA intervention, we have been able to demonstrate partial HA in seven paratriathletes of mixed impairments [51]. Specifically, these responses were: a reduction in exercising T_c, blood lactate concentration, RPE and thermal strain at a fixed intensity with concomitant plasma volume expansion. Furthermore, the inclusion of a physiologically matched AB group permitted comparisons to the Paralympic cohort whereby additional adaptations were noted including T_{sk}, HR and sweat rate changes. This is the first evidence

of differences in thermoregulatory variable changes to the same protocol between physiologically similar Paralympic and AB athletes and also the first support for the efficacy of mixed HA, utilising HR to control the relative intensity of effort.

26.4.3 Conclusion

Paratriathletes display significant thermoregulatory strain when competing in the heat with T_c noticeably greater than previous reports in AB long-distance triathletes. This is likely due to the myriad impairments that may diminish the thermoregulatory capacity in Paralympic athletes. Furthermore, impairment-specific temperature responses may occur to the differing nature of paratriathlon racing across race categories. Nonetheless, strategies may be put in place to attenuate the thermal strain of competition, namely HA. HA is an effective strategy prior to competition in the heat and may be manipulated to complement the multi-modal nature of paratriathlon, pre-competition. Moreover, recent evidence suggests paratriathletes are capable of displaying positive thermoregulatory adaptations that may ameliorate the physical demands of racing in the heat.

26.5 Summary

There are myriad training and competition considerations that should be made when working with paratriathletes. Due to the consequences of their impairments, athletes may be at a heightened risk of excessive internal TL caused by movement inefficiencies increasing the metabolic cost of absolute workloads [20, 26]. This, as well as immunoendocrine deficiencies [10], may result in a greater propensity for mucosal immunity suppression and URI incidence [38], with associated training and performance impairments, or the genesis of OR. However, athletes' impairments also present issues with TL quantification which must be acknowledged. Nonetheless, negative consequences of elevated TLs can be alleviated through careful training management and a minimisation of external life stresses.

Concurrently, athletes' impairments result in a significantly elevated susceptibility for heat-related illness or performance decrements. Specifically, paratriathletes may display: a loss of vasomotor control and sweating capability [13]; reduced venous return [14, 22]; movement inefficiencies increasing metabolic heat production [20, 26]; impaired pace awareness [18, 32]; limited surface area for heat loss via missing limbs, skin grafts or wearing socket liners [27, 28, 30]; or a propensity for hypohydration [11] or sunburn [31]. Preliminary evidence highlights the level of thermoregulatory strain imposed by paratriathlon competition in the heat, with athletes' T_c significantly greater than AB athletes reported elsewhere [75, 76]. However, it is possible for athletes to appropriately prepare for competition in the heat via HA. HA may invoke beneficial thermoregulatory adaptations in both AB and Paralympic athletes to attenuate performance decrements in challenging environments.

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