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REVIEW ARTICLE

Effects of Altitude/Hypoxia on Single- and Multiple-Sprint Performance: A Comprehensive Review

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Abstract Many sport competitions, typically involving the completion of single- (e.g. track-and-field or track cycling events) and multiple-sprint exercises (e.g. team and racquet sports, cycling races), are staged at terrestrial altitudes ranging from 1000 to 2500 m. Our aim was to comprehensively review the current knowledge on the responses to either acute or chronic altitude exposure relevant to single and multiple sprints. Performance of a single sprint is generally not negatively affected by acute exposure to simulated altitude (i.e. normobaric hypoxia) because an enhanced anaerobic energy release compensates for the reduced aerobic adenosine triphosphate production. Conversely, the reduction in air density in terrestrial altitude (i.e. hypobaric hypoxia) leads to an improved sprinting performance when aerodynamic drag is a limiting factor. With the repetition of maximal efforts, however, repeated-sprint ability is more altered (i.e. with earlier and larger performance decrements) at high altitudes (>3000–3600 m or inspired fraction of oxygen <14.4–13.3%) compared with either normoxia or low-to-moderate altitudes (<3000 m or inspired fraction of oxygen >14.4%). Traditionally, altitude training camps involve chronic exposure to low-to-moderate terrestrial altitudes (<3000 m or inspired fraction of oxygen >14.4%) for inducing haematological adaptations. However, beneficial effects on sprint performance after such altitude interventions are still debated. Recently, innovative 'live low-train high' methods, in isolation or in combination with hypoxic residence, have emerged with the belief that up-regulated non-haematological peripheral adaptations may further improve performance of multiple sprints compared with similar normoxic interventions.

Key Points

Performance of a single sprint in the laboratory environment is generally unaffected by acute exposure to simulated altitude (normobaric hypoxia).

At terrestrial altitude (hypobaric hypoxia), the reduction in air density leads to improved sprinting performance.

Larger alterations in repeated-sprint ability (i.e. with earlier and larger performance decrements) occur in oxygen-deprived environments equivalent to altitudes above ~3000 m or inspired fraction of oxygen <14.4% than at lower elevations.

Innovative 'live low-train high' methods have emerged as 'repeated-sprint training in hypoxia' or 'resistance training in hypoxia' with the belief that up-regulated non-haematological peripheral adaptations may further improve performance of multiple sprints, compared with similar interventions in normoxia.

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1 Introduction

Training and competing in major track-and-field events and cycling races often require the completion of maximal sprints (<30 s); i.e. an 'all-out' ballistic activity that makes the human body move at speeds ranging from 30 to 40 km h^{-1} (running) or >60 km h^{-1} (cycling). The ability to repeat sprints [i.e. repeated-sprint ability (RSA)] describes the aptitude of an athlete to recover and maintain maximal effort during subsequent sprints. Although debated [1], this attribute is considered important to various intermittent activities (e.g. rugby, soccer, Australian football, ice/field hockey, tennis) [2].

Many sport competitions worldwide, including the most prestigious ones, are regularly staged at terrestrial altitudes [i.e. hypobaric hypoxia (HH)] ranging from 1000 to 2500 m. These have included the 1968 Olympic Games in Mexico City [3] and subsequently the FIFA World Cup football tournaments in 1970 and 1986, and more recently in South Africa in 2010 [4]. Every summer, some of the most spectacular stages of the cycling 'Tour de France' are also often decided in mountainous areas during the last 100 m of the final climb. Furthermore, the South African rugby teams and South American soccer squads regularly host international games at altitude [5, 6], occasionally at terrestrial elevations >3500 m (e.g. La Paz, Bolivia). These sporting activities typically involve the completion of single- (e.g. track-and-field or track cycling events) and/or multiple-sprint bouts (e.g. team and racquet sports, cycling races). Here, it is useful to define two different types of exercise involving multiple sprints: 'repeated-sprint' and 'intermittent-sprint' exercises [2]. Repeated-sprint exercise is characterised by the ability to produce short sprint bouts over a series of sprints with brief recovery periods (usually ≤60 s) so that there is a marked performance decrement. In comparison, bursts of maximal power during intermittent-sprint exercise are interspersed with recovery periods long enough (60–300 s) to allow near-complete recovery of sprint performance.

Historically, altitude/hypoxic training has been used by endurance athletes in pursuit of performance enhancement after return to sea level [7, 8] (Table 1). Although unlikely to be the sole factor that drives aerobic performance improvement, haematological changes (i.e. increase in haemoglobin mass) reflect the major physiological adaptation. The use of HH or simulated altitude [i.e. normobaric hypoxia (NH)] while exercising at or near maximal intensity for maximising performance of multiple sprints is increasingly popular, as discussed in the first international symposium on altitude training and team sports held in 2013 [9]. With the development of innovative 'live low-train high' interventions (Fig. 1), the past decade has seen impressive strides in our knowledge of the impact of repeated altitude/hypoxic exposures upon sprint performance with the belief that non-haematological factors may play a more dominant role [10].

The scope of the current review, therefore, was to comprehensively examine acute and chronic hypoxic exposure findings in both NH and HH conditions that are relevant to single and multiple sprints. This review first addresses the effects of acute hypoxic exposure upon sprint, repeated-sprint and intermittent-sprint performance as well as physiological and biomechanical consequences on sea-level residents. Following this, we discuss performance-led investigations and mechanistic advancements that have improved our understanding of the effects of chronic hypoxia on single and multiple sprints This includes altitude-related strategies using both systemic (acclimatisation, 'live low-train high') and local (vascular occlusion) hypoxia. Finally, we provide an updated panorama of hypoxic methods used by athletes and discuss future research directions.

2 Literature Search Methodology

A comprehensive computerised search of the electronic databases PubMed, MEDLINE, SPORTDiscus and Web of Science was performed through September 2016 employing the following keywords: 'sprint performance', 'sprinting', 'all out effort', 'repeated sprint ability', 'intermittent sprints', 'multiple sprints', 'altitude

Table 1 Historical perspectives on the implementation of altitude/hypoxic training methods

Interventions	Acronyms	Date	Targeted athletes	References
Live high-train high	LHTH	1960s	Endurance trained	[148]
Intermittent hypoxic training	IHT	1960s	Endurance trained	[149]
Live high-train low	LHTL	1997	Endurance trained	[150]
Resistance training in hypoxia	RTH	2000s	Power trained	[151]
Repeated-sprint training in hypoxia	RSH	2013	Team/racquet sports	[72]
Live high-train low and high	LHTLH	2015	Team/racquet sports	[85]
Live high-train high and low	LHTHL	2015	Endurance trained	[152]

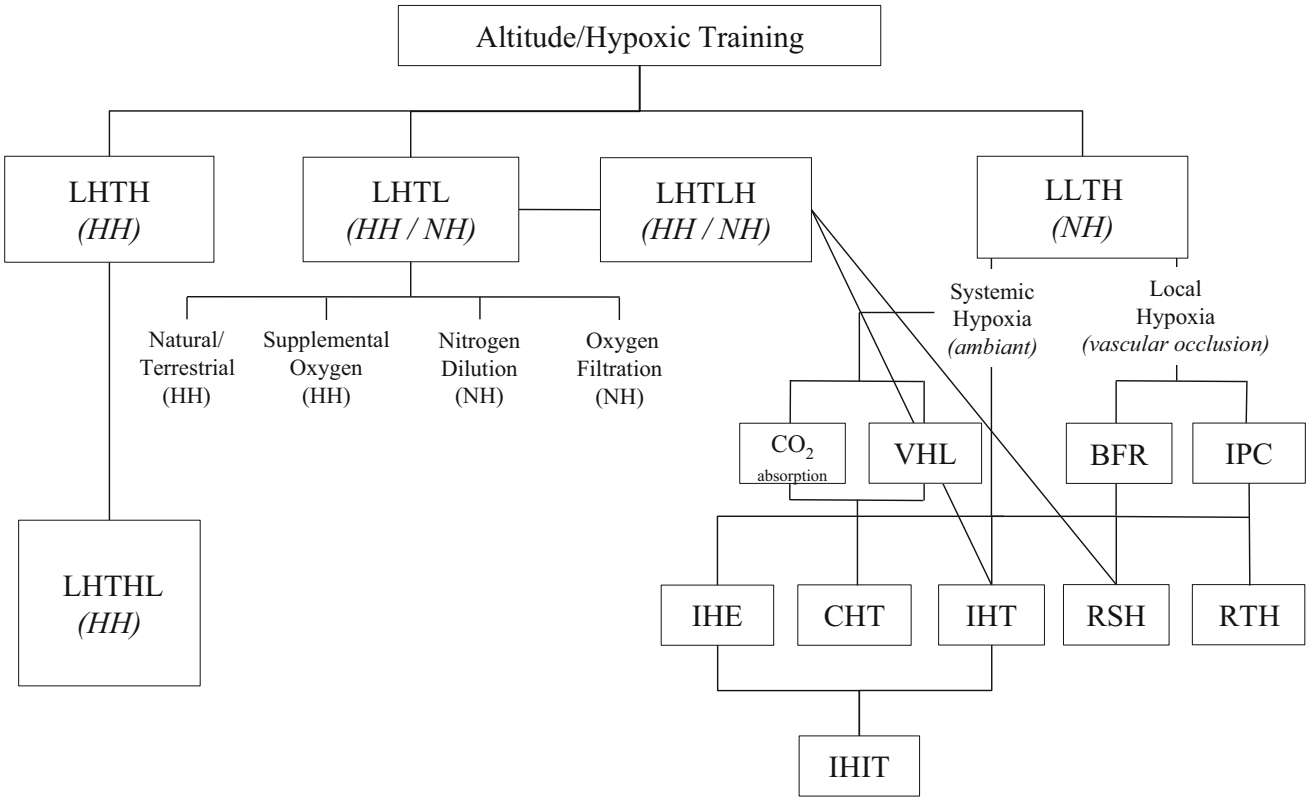

Fig. 1 Updated panorama of the different hypoxic/altitude training methods used for a range of athletes. Adapted from [147]. *BFR* blood flow restriction, *CHT* continuous hypoxic training, *CO_2 absorption* rebreathing with a mask, *HH* hypobaric hypoxia, *IHE* intermittent hypoxic exposure, *IHIT* IHE during interval-training, *IHT* interval hypoxic training, *IPC* ischemic pre-conditioning, *LHTH* live high-train high, *LHTL* live high-train low, *LLTH* live low-train high, *LHTHL* live high-train high and low, *LHTLH* live high-train low and high, *NH* normobaric hypoxia, *RSH* repeated sprint training in hypoxia, *RTH* resistance training in hypoxia, *VHL* voluntary hypoventilation at low lung volume

training', 'acute hypoxia', 'chronic hypoxia', 'normobaric hypoxia', 'hypobaric hypoxia', 'simulated altitude', 'natural altitude', 'live high-train low', 'intermittent hypoxic training', 'repeated sprint training in hypoxia', 'resistance training in hypoxia', 'acclimatization', 'blood flow restriction', 'ischemic preconditioning', and 'fatigue'. Initially, all abstracts were reviewed, and those meeting the full inclusion criteria were obtained for further evaluation. An article was eligible for inclusion if it met the following criteria: (1) sprint duration of $\leq$45 s; (2) an acute exercise study including at least one physiological or biomechanical response to sprinting; (3) a chronic training intervention consisting of at least four sessions to allow sufficient time for measurable performance gains; and (4) at least, pre- and post-performance measures of single and/or multiple sprints must be provided for chronic studies.

3 Performance-Led Investigations in Acute Hypoxia

3.1 Single-Sprint Performance

3.1.1 Normobaric Hypoxia

Performance of 'all-out' efforts <45 s in a laboratory NH environment [i.e. a decrease in inspired fraction of oxygen (F_iO_2)] is relatively unaffected by large hypoxic-related reductions in aerobic power, even at simulated altitudes above $\sim$5000 m (FiO_2 <10.5%) [11, 12]. Visual inspections of the first (or one of the first) repetition of various running [13, 14] or cycling [15–17] repeated-sprint exercises typically reveal unchanged single-sprint performance for simulated altitudes equivalent to $\sim$1000–3600 m (FiO_2 18.5–13.3%).

Even for efforts of longer durations, power output can be maintained over the first sprints in hypoxia: power output produced by university soccer players during 40-s Wingate-like tests was similar at ~1900 and 3700 m (FiO_2 16.4 and 12.7%, respectively) compared with normoxia (FiO_2 20.9%) [18]. As four individuals completed 15 'all-out' treadmill efforts lasting from 15 to 180 s under both normoxic and NH (~3600 m or FiO_2 13.3%) conditions, they were able to run just as fast for bouts of up to 60 s and nearly as fast for efforts of up to 120 s [19]. Such single-sprint performance maintenance for efforts lasting 30–60 s [18, 19] occurs in NH conditions, despite considerable reductions in the aerobic energy availability for sprinting in oxygen (O_2)-deprived environments [12, 20]. The prevailing rationale is that rates of anaerobic energy release, estimated from increase in O_2 deficits (as much as 18% [19]) or muscle lactate concentration ([La]) [11, 12], compensate for the reduced aerobic adenosine triphosphate (ATP) production [21].

3.1.2 Hypobaric Hypoxia

The impairment in aerobic power with altitude ascent (~1% for every 100 m above 1500 m [22]) is mainly owing to the drop in the partial pressure of O_2 (and subsequently arterial O_2 saturation), resulting from the decrease in barometric pressure in HH conditions. Competing above a 1000-m terrestrial altitude is particularly detrimental to a running distance of 800 m or longer [23, 24], or to key performance indicators (i.e. chasing the ball, controlling long passes; the latter being related to the lower air density in HH conditions), affecting the overall team-sport performance and the chances to win matches (i.e. soccer [4, 5] or rugby [6]). Efforts of shorter durations (<45 s) require relatively low contributions of energy from aerobic metabolism [25], and therefore are less affected because a large fraction of the total energy requirement is covered anaerobically. Studying short-term acclimatisation to a 3200-m terrestrial altitude, Burtscher et al. [26] failed to observe any change in Wingate performance 1–3 h and 3 days upon altitude ascent. When athletes are tested on stationary cycle ergometers or treadmills, however, any changes in air volumetric mass (i.e. reduced air density) associated with HH is not considered, which may not reveal the true effect of terrestrial altitude on single-sprint performance.

The decision to hold the 1968 Olympic Games in Mexico City at a terrestrial altitude of 2340 m (barometric pressure 580 mmHg) above sea level has aroused interest in the effects of HH on athletic performance. Anecdotally, the substantial effects of HH were exemplified by the establishment of world records in track events at all distances up to 800 m (100–400 m, 4 × 100-m and 4 × 400-m relays). In fact, Jokl et al. [3] quantified that these winning performances were 1–4% faster than the existing world records for those events. Increasing terrestrial altitude (i.e. HH) reduces air density (~1% for every 100 m [27]), which modifies the aerodynamic drag and facilitates high-speed movements (e.g. running, [23] speed skating [28]), and decreases the energy cost of running at high speeds without the detrimental effect of reducing energy availability [29]. Interestingly, acute exposure to a terrestrial altitude of 2300 m as opposed to NH conditions leads to gains in 1 repetition maximum movement speed and power during the execution of a force-velocity curve in a bench press movement [30]. Accordingly, marginal improvements of ~0.2% at terrestrial altitudes of 500–999 m were seen in elite athletes competing in sprint events (100–400 m) between 2000 and 2009 [24]. Similarly, terrestrial altitudes at or above 1000 m produce faster times (0.1–0.5%) in 100-m sprint to 400-m hurdles events [23].

3.1.3 Models of Single-Sprint Outcomes at Altitude

Numerous comprehensive statistical and theoretical modelling studies have been published to quantify the effects of 'altitude assistance' on the 100-m [31, 32], 200-m [33, 34] and 400-m [35] sprint. The general consensus is that lower air density associated with HH produces a time advantage and will improve performance between 0.03 s [33] and 0.12 s [35] for each 1000-m increase in terrestrial altitude. This implies that advantages are greater (0.07–0.27 s) in Mexico City compared with sea-level conditions [31]. The reduced energy required to overcome air resistance when exposed to HH (i.e. 12–13% of demand at sea level, 10–11% at 2000 m and 8–9% at 4000 m) preserves energy availability for acceleration, in turn improving 100-m running times [32]. Peronnet et al. [29] have calculated the effect of altitude on running performance and concluded that for all running events up to 400 m, there is a progressive improvement in sprint times with increasing HH up to 2500 m. For shorter sprints (100 and 200 m), this improvement is sustained even at altitudes up to 4000 m [29]. Because the air resistance is approximately proportional to the square of the speed [36], athletes engaged in short sprint events (100 vs. 400 m) are likely to benefit more from the reduced air density associated with HH, owing to their faster running speed.

3.2 Repeated-Sprint Performance

3.2.1 Cycling Studies

To quantify the kinetics and influence of fatigue experienced during repeated-sprint exercise, researchers have

tended to use one of three indices (i.e. RSA is better interpreted using the three scores): initial or best sprint performance, fatigue scores (fatigue index or sprint decrement score) and cumulated/averaged sprint performance [2]. Balsom et al. [15] were among the first to demonstrate an impaired ability to maintain power output towards the end of each exercise bout (i.e. −8.5% in the last sprint bout) in HH corresponding to 526 mmHg (~3000 m) compared with normoxia. More recent NH studies have confirmed that simulated altitudes of ~2000–3000 m have been associated with earlier (after a few repetitions only) and larger (5–10%) reductions in either peak or mean power output compared with normoxia during typical repeated-sprint exercises (6–10 × 5–10-s cycling sprints—20–30 s recovery) [16, 17, 37]. The consensus is that, unlike single sprints (i.e. unchanged or even slightly improved), HH or NH vs. normoxic conditions report inferior RSA (i.e. lower averaged mean/peak power output often with a larger sprint decrement score or fatigue index [2]).

3.2.2 Running Studies

Similar to cycling-based literature, exacerbated impairment in RSA associated with HH or NH has been reported when maximal or 'all-out' efforts are repeated on an instrumented sprint treadmill [14, 38]. For instance, a larger sprint decrement score (8 vs. 3%) and shorter cumulated distance covered (111 vs. 117 m) over 5 × 5-s sprints (25-s recovery) at a simulated altitude of ~3600 m (FiO_2 13.3%) compared with near sea level has been concluded [38]. Whereas, single 30-m sprint performance improved by ~4% in amateur rugby players after 12 h at a 1550-m terrestrial altitude and RSA (6 × 70 m sprints—30-s recovery) declined by ~2% [39]. This latter result likely reflects the contrasting effect of HH on aerodynamic drag (faster unique sprint) and maximal aerobic power (worsened RSA).

Investigating the NH dose response, Goods et al. [40] reported the effects that three sets of repeated sprints (9 × 4 s—16–36 s recovery and 3-min inter-set rest) on a non-motorised treadmill completed near sea level and at simulated altitudes equivalent to ~2000, ~3000 and ~4000 m (FiO_2 16.3, 14.4 and 12.6%, respectively) had on RSA. Results indicated that each increase in NH severity was associated with a significant reduction in mean power output beyond the first set (i.e. sets 2 and 3). Contrastingly, others have argued that poorer RSA in NH conditions is solely observed for simulated altitudes in excess of ~3500 m (FiO_2 13.4%) [13, 14]. Although non-motorised treadmill sprint performance (10 × 6-s treadmill sprints—30-s recovery) was relatively resilient to NH conditions (~2500–3000 m or FiO_2 15.4–14.4%), fatigue development was only significantly exacerbated (sprint decrement score of 9.9 vs. 4.9%) at the most severe hypoxic level corresponding to a simulated altitude of ~4100 m (FiO_2 12.0%) compared with normoxia [13]. In another study, impairments in RSA (8 × 5-s treadmill sprints—25 s recovery) at a simulated altitude equivalent to ~3600 m (FiO_2 13.3%) exceed those observed in normoxic and moderate NH conditions (~1800 m or FiO_2 16.8%) [14]. This suggests that RSA decrements with increased severity of NH may not necessarily follow a monotonic (i.e. linear) pattern. Hence, the low O_2 tension does not significantly affect RSA until reduced arterial O_2 pressure reaches a critical point (i.e. highly individual but suggested to be <75%, at least for 5-km time trials [41]) when it declines below the plateau portion of the oxy-haemoglobin dissociation curve and encroaches upon the steeper portion.

In addition to the level of hypoxia [13, 14, 40], the design of the RSA testing protocol [i.e. number/duration of sprints/rest periods, difference in the exercise modes (overground vs. treadmill vs. cycle ergometer), nature of the hypoxic exposition (HH vs. NH) and participants' background (training status) among others] will influence the ability to maintain sprint performance. During a clustered running RSA protocol (four sets of 4 × 4-s non-motorised treadmill sprints—26-s recovery and 146-s inter-set rest) in a NH environment corresponding to ~3200 m (FiO_2 14.0%), amateur team-sport athletes were unable to match the peak speed reached in normoxia during sets 3 and 4, while impaired acceleration was only seen for set 4 [42]. Contrastingly, in highly trained professional Australian footballers, peak and mean speeds were not impaired during completion of ten 6-s sprints with 24 s of recovery at NH corresponding to ~3200 m (FiO_2 14.0%) vs. normoxia [43]. Well-rounded athletes, compared with their less-fit counterparts, possess the concomitant ability to maintain a high rate of energy production (i.e. matured anaerobic energy systems) and to rapidly recover (i.e. high maximal aerobic power), thereby postponing the reliance on anaerobic glycolysis during repeated sprinting in O_2-deprived environments.

3.3 Intermittent-Sprint Performance

Compared with repeated sprinting, the effects of HH and NH upon intermittent-sprint performance are less evident. For instance, performing 4 × 30-s 'all-out' cycling bouts (4-min rest) caused reduction in bouts 3 and 4 compared with bout 1, but these changes were of similar magnitude across moderate-to-high NH conditions (~2000 and 3400 m or FiO_2 16.4 and 13.6%, respectively) and normoxia [44]. In one study, Feriche et al. [45] examined repetitive 400-m sprints near sea level and at a terrestrial

altitude of 2320 m with recovery periods lasting 1–5 min. Individuals were able to run 10–15% faster near sea level than at altitude with 1- or 2-min recovery periods between sprints; however, this difference was eliminated when a 5-min period between sprints was allowed [45]. Similarly, 5–6 days at a terrestrial altitude of 4350 m (barometric pressure 452 mmHg) did not significantly impair the restoration of muscle power during two repeated supra-maximal 20-s cycle efforts separated by 5 min of rest [46]. Finally, maximal performance during intermittent-sprint running (20-s runs with 100-s recovery) was not affected by exposure to HH equivalent to 2500 m (barometric pressure 560 mmHg) in trained middle-distance runners in an environmental chamber [20]. These data illustrate that the completion of intermittent sprints in O_2-deprived environments may not be associated with large performance decrements when recovery is of sufficient duration. Sprint duration per se, number of completed efforts, exercise-to-rest ratio or severity of hypoxia likely (but unknown) also have the potential to differently affect metabolic accumulation, depletion of energy stores and/or neuromuscular function, and thereby intermittent-sprint performance.

Team sports are characterised by intermittent physical activities, where sprints only represent a small percentage of the total time (1–3%) compared with walking and slow and moderate running (35, 40 and 20% of total playing time, respectively) [47]. The repetition of these activities throughout a 90-min soccer game, for instance, could lead to neuromuscular fatigue and hinder straight sprinting, the most frequent action preceding a goal [48]. The effects of altitude on the performance of multiple sprints have been recently documented in match-play data (see next section). However, the game-to-game reliability for sprinting during soccer games [49] is quite low and therefore it is inaccurate and difficult to quantify the influence of altitude per se from actual competition data. To more accurately quantify altitude-mediated decrements on realistic team-sport game situations (i.e. preventing the adoption of pacing strategies), several authors applied effort simulations in a laboratory environment where intermittent sprints were completed [50, 51]. Reportedly, athletes are at a disadvantage during an 80-min (2 × 40 min) cycling intermittent-sprint protocol at a simulated altitude equivalent to ~1600 m (FiO_2 17.0%), as evidenced by lower values for peak (~4%) and mean power output (~5%), as well as for total work completed (~5%) compared with normoxia [50]. Despite SpO_2 (−8%) and heart rate (+3%) alterations at ~1000 m NH (FiO_2 18.4%) compared with normoxia, an exacerbated decline in treadmill sprint distance covered only occurred during the final 15 min of a 90-min match simulation protocol in an O_2-deprived environment [51].

3.4 Match Play at Terrestrial Altitude

As previously mentioned, the physical performance of many team sports requires players to complete an intermittent exercise pattern. Owing to the large distance covered in a match and the numerous brief explosive efforts (e.g. accelerations, changes in pace and direction, sprints) to be performed, competing at altitude is likely to exacerbate fatigue development in team-sport athletes [47, 52]. Data collected during official games of the 2010 FIFA World Cup [4] and in preparation for the 2011 FIFA under-20 World Cup [53] display reductions in total distance covered, averaging 3–9% at 1200- to 1750-m terrestrial altitudes compared with near sea level. More specifically, using a 5-min rolling sample period for analysis gave an indication of transiently lower output, and possibly fatigue, during matches. As a result, there were a 7, 21 and 20% reduction in total distance covered, high-speed running and acceleration frequency, respectively, in the 5 min subsequent to the peak 5-min period at a terrestrial altitude of 1600 m compared with sea level [53]. Similar observations were made during youth soccer games played at an elevation of 3600 m [54]. This may indicate a transient neuromuscular fatigue, likely reducing the 'willingness' of players to repeat intense exercises during a game [52].

Non-acclimatised athletes may be at a disadvantage when required to perform at altitude. Low- (i.e. <15 km h^{-1}) and high-speed running (i.e. 15–36 km h^{-1}), as expressed in meters per minute of match time, declined by 8 and 15% during matches played 4 days after arrival at a terrestrial altitude of 1600 m [53]. These observations indirectly support the presence of altitude/hypoxia-related anticipatory modification of pacing, possibly owing to altered effort perception [55], in addition to the well-known decline in maximal aerobic power when O_2 availability is reduced [22] to prevent excessive fatigue during games. In fact, Tucker et al. [56] proposed that, rather than acting just as a measure of exercise intensity, the conscious perception of effort may play a regulatory function to ensure that the work rate remains at an intensity that can be safely sustained for the expected duration of the exercise. In the face of environmental challenges, such as heat stress [57] or in O_2-deprived conditions [53, 54], match activity pattern would be adjusted (low-intensity activity) to preserve homeostasis and the ability to undertake high-intensity activities (sprinting characteristics).

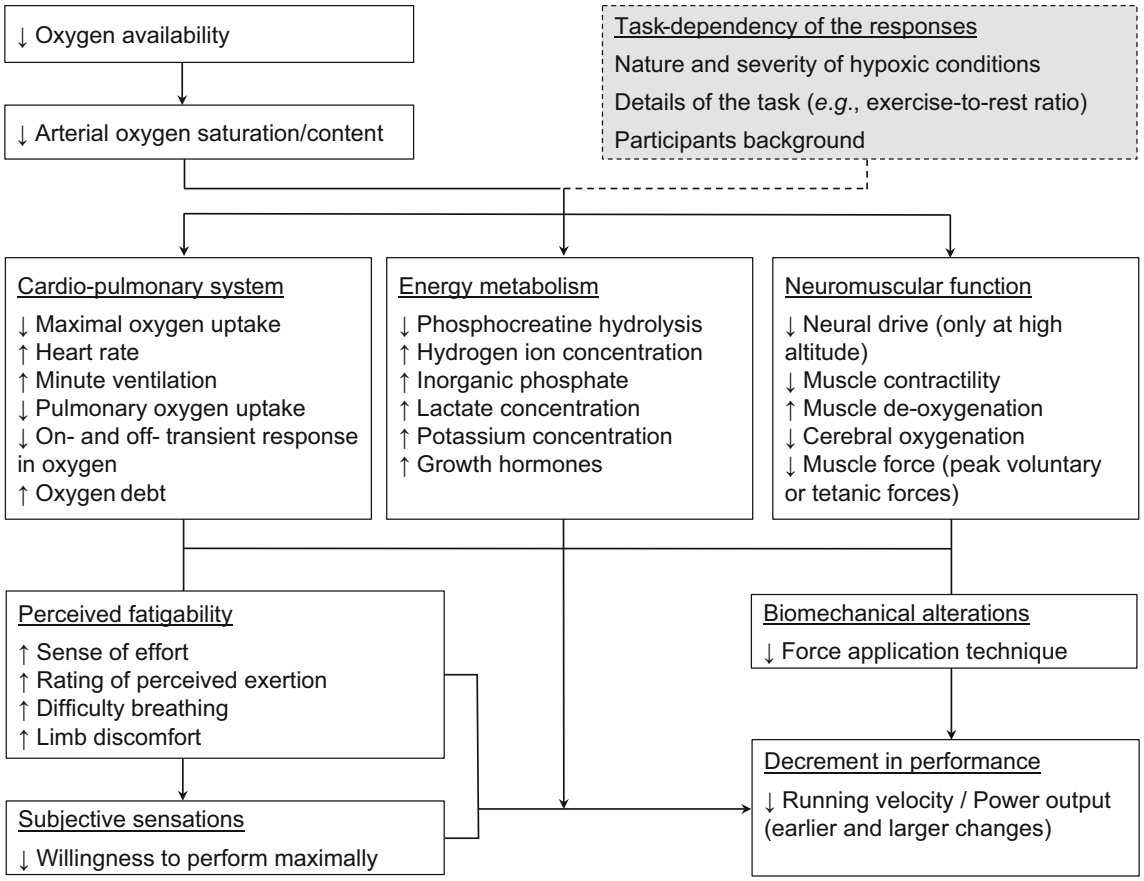

Fig. 2 Main acute responses of reduced oxygen availability (hypoxia) to multiple-sprint exercise. Note that the list of modulating factors is not intended to be all inclusive, but rather it provides an initial set that can be expanded as new experimental findings emerge

4 Responses to Sprinting in Hypoxia

4.1 Physiological Adjustments

Laboratory studies have shown that, with reduced O_2 availability or large arterial hypoxemia (low SpO_2), limitations in energy supply (i.e. phosphocreatine hydrolysis, anaerobic glycolysis, oxidative metabolism), metabolites by-product accumulation (e.g. inorganic phosphate, H^+), failure to fully activate the contracting muscles, lower cerebral oxygenation levels and slower on-transient O_2 responses likely occur. This would increase the magnitude of the O_2 deficit incurred during each sprint and thereby place more demand on anaerobic sources to maintain the required rate of ATP provision (Fig. 2) [58]. It is generally described that sprint repetition in O_2-deprived environments elevates heart rate [13], minute ventilation [13], O_2 debt [13, 15, 21], muscle deoxygenation level [13, 17, 37] and growth hormone release [44]; whereas, it lowers SpO_2, pulmonary O_2 uptake [13, 15, 43, 58] and electromyographic signals of active musculature [13, 37]. Additionally, an increase in muscle [La] accounts for a hypoxia-induced reduction in aerobic ATP production [12, 13, 15]. For instance, higher post-exercise blood [La] (10.3 vs. 8.5 mmol L^{-1}) and lower O_2 uptake (3.03 vs. 3.19 L min^{-1}) when barometric pressure in an hypobaric chamber was set to 526 mmHg (~3000 m or ~FiO_2 14.4%) vs. normoxia reflect a greater reliance on the anaerobic energy systems during repeated sprinting in hypoxia [15]. The increased physiological stress associated with lowered O_2 availability can be attributed to a haemodynamic response arising from a reduction in SpO_2.

4.2 Perceptual Responses

In general, arterial hypoxemia contributes towards performance regulation via an effect on the sense of effort: this is reflected by a slightly greater [14, 40] or unchanged perception of effort [37], but reduced completed work in hypoxic vs. normoxic conditions. The linear relationship ($r^2 = 0.62$, $p < 0.05$) observed between changes in SpO_2 and ratings of perceived exertion over 20 × 5-s cycle sprints (25-s recovery) in 15 national-level soccer players illustrates this idea [59]. Perceived fatigue is also

associated with decreased power outputs during intermittent cycling sprints (4 × 30-s 'all-out' sprints—4 min of active recovery), and its association with brain cortical oxy-haemoglobin during 'all-out' bouts and feelings of fatigue and energy during recovery suggests these feelings are integrated in part through the dorsolateral prefrontal cortex [60].

4.3 Neuromuscular Responses

It is now accepted that rather than a single factor, the decline in the performance of multiple sprints involves changes within the muscle cell itself (peripheral factors) and/or alterations in the neural activation of contracting muscles (central factors), while the details of the task determine the relative contribution of the underlying mechanisms (i.e. task dependency) [2]. To date, the neuromuscular adaptations to fatigue during repeated sprinting have mainly been studied in the laboratory environment where most of the influencing variables can be controlled or manipulated. After repeated treadmill sprinting at a high simulated altitude equivalent to ~3600 m (FiO_2 13.3%), the maximal voluntary isometric contractions in knee extensors were significantly decreased, but not less explosive (i.e. contraction speed not altered), compared with moderate NH (~1800 m or FiO_2 16.8%) and normoxic conditions [61]. In this later study, high NH conditions (i.e. 3600 m) induced an increased alteration in maximal torque but not in rapid torque development. Adjustments to the central nervous system occur in an O_2-deprived environment to explain these exacerbated neuromuscular perturbations. In particular, greater attenuation of the surface EMG activity (i.e. a proxi for the central motor drive to skeletal muscles) accompanies a decline in power output across repetitions in high NH conditions [13, 16, 37, 62]. In the repeated treadmill sprints study by Girard et al. [63], however, the fatigue-related decline in activation of the six studied lower-limb muscles was only exacerbated at a simulated altitude of ~3600 m (FiO_2 13.3%) vs. normoxia in rectus femoris and tibialis anterior muscles, highlighting the muscle dependency of NH [63]. Compared with normoxia, NH equivalent to ~3100 m (FiO_2 13.8%) caused significant parallel reductions in quadriceps root mean square activity and in power output during the repetition of cycle sprints (i.e. their magnitudes exceeded those of control situations) [37]. These changes were concomitant to a greater central activation failure, while the amount of peripheral fatigue (assessed via quadriceps twitch force) incurred at exercise termination was similar. Lower RSA in NH conditions corresponding to ~3600 m (FiO_2 13.3%) has been associated with larger cerebral deoxygenation compared with normoxia [16, 37, 62], which leads to a larger decline in muscle recruitment in O_2-deprived environments [16]. In this later study, changes in cerebral deoxy-haemoglobin concentration explained 83% of the variance in EMG activity in hypoxia. This hypoxia-mediated active musculature 'de-recruitment' is consistent with the hypothesis that central motor command and power output during 'all-out' repeated sprints are limited, to prevent excessive locomotor muscle fatigue.

The consequences of arterial hypoxemia can be attributed to an indirect inhibitory effect of the sensory afferent feedback (groups III/IV muscle afferents) originating from the less oxygenated muscles [41]. In addition, any hypoxia-related reduction in cardiac output will jeopardise arterial O_2 delivery to the exercising muscle (i.e. as a consequence of lower muscle blood flow [64]), provoking a greater reliance on anaerobic energy contribution, potentially accelerating the decline in ATP and phosphocreatine levels and aggravating muscle [La] and H^+ accumulation [65]. Hypothetically, there is also the possibility of a direct effect of brain hypoxia on muscle recruitment, outside any influences related to peripheral muscle fatigue and its associated afferent feedback [66]. This may occur if hypoxia is of a severe nature (<75% SpO_2), and would likely be associated with pronounced reductions in RSA. In this context, the hyperventilation response, inducing an arterial hypocapnia and causing cerebral vasoconstriction [67], might also be involved. Reduced air density and larger hypoxemia, owing to an altered ventilatory pattern (i.e. higher breathing frequency and lower tidal volume) in HH conditions, could induce these responses [68]. This scenario, however, is unlikely to happen in a large majority of athletes given the elevations (1000–2500 m) of most of the popular competition grounds. Interestingly, acclimatisation to a high altitude for 14 days attenuates the impact of acute hypoxia on the development of central fatigue but fails to improve the exacerbated development of peripheral fatigue associated with constant-load cycling exercise in acute hypoxia [69]. Direct measurements of cerebral and venous blood gases, under hypoxic conditions that athletes would face during competitions, are required to experimentally verify the consequences of changes in cerebral metabolism and oxygenation on performance of multiple sprints.

4.4 Metabolic Responses

The non-invasive technique of near-infrared spectroscopy offers real-time measurement of oxygenation and haemodynamics in tissues, and thus, constitutes a relevant tool to further explore the cerebral and muscular determinants of RSA. De-oxygenation levels quickly plateaued and were relatively similar in NH equivalent to ~3600 m (FiO_2 13.3%), and in normoxia during completion of 10 × 10-s

cycling sprints (30-s recovery) [16]. In contrast, peak deoxy-haemoglobin at the end of each repetition progressively increased and was lower through 10 × 6-s running sprints at a simulated altitude of ~4100 m (FiO_2 12.0%) vs. normoxia [13]. Furthermore, slower re-oxygenation rates during recovery periods between efforts have been incriminated in the performance decline in O_2-deprived environments [17]. Greater amplitudes of muscle blood perfusion variations, suggesting enhanced muscle blood flow after hypoxic training for instance [70, 71], would reflect a greater O_2 use by fast-twitch fibres [72]. Because the re-oxygenation rate of the skeletal muscle has similar recovery kinetics to phosphocreatine after a single exercise bout [73], it has been postulated that reduced phosphocreatine re-synthesis post-exercise might be the result of sub-optimal re-oxygenation of the active skeletal muscle elongating the recovery time between high-intensity exercise bouts [74].

4.5 Biomechanical Manifestation of Fatigue

Empirical field-based observations made by coaches and recent research findings indicate that faster individuals attain higher running speeds by applying more forward-oriented, but not necessarily greater, magnitude of total forces to the ground [75]. Reductions in horizontal force production (i.e. braking and pushing force peaks) exceed those in the vertical direction when 40-m run-based sprints were repeated, which subsequently alters ground impact parameters (i.e. slower stride frequency or longer contact time decreasing vertical stiffness) as measured from a 5-m long force plate system [76]. To date, there is no field-based investigation on the terrestrial altitude influence on mechanical determinants of multiple-sprint performance. Our current knowledge of sprint-induced biomechanical manifestation of fatigue (as discussed below) is limited to laboratory-based findings in NH.

The recent modification of an instrumented treadmill for sprint use (i.e. the so-called ADAL treadmill), which allows athletes to run and produce speed ''freely'', i.e. with no predetermined belt speed imposed, allows the continuous measurement of both valid and reproducible ground reaction forces during accelerated runs [77, 78] (Fig. 3). In a pilot study, the nature and extent of fatigue-induced alterations in running kinetics (i.e. reduced average horizontal forces and propulsive power), kinematics (i.e. longer contact/swing times and slower step frequency) and spring-mass characteristics (i.e. lower vertical and leg stiffness) during repeated sprinting (5 × 5-s—25-s recovery) on this instrumented treadmill were similar between a simulated altitude of ~3600 m (FiO_2 13.3%) vs. normoxia, despite larger NH-induced decrements in RSA [38]. Comparing the effects of different altitude levels on mechanical alterations, Brocherie et al. [14] further revealed that at a simulated altitude of ~3600 m, impairments in RSA (8 × 5-s—25-s recovery) and the magnitude of fatigue-induced kinetics and kinematic alterations exceed those observed near sea level or at ~1800 m, while there was no or minimal difference between these two later conditions. Specifically, high hypoxia accentuated the fatigue-related inability to effectively apply forward-oriented ground reaction force (alteration in the force application technique) and to maintain vertical stiffness and stride frequency when completing a large number of sprint repetitions. This is taken to reflect progressively shorter and less effective acceleration phases across repetitions under high hypoxia. However, NH had no residual effect on performance and associated neuro-mechanical alterations (i.e. similar fatigue pattern across conditions), after resting for 6 min near sea level, during subsequent repeated sprints in normoxia [63]. Interventions (i.e. ergogenic aids, horizontally oriented exercises) maximising RSA tolerance when decrement in performance is high (i.e. hypoxia exposure) warrant further investigation.

5 Residence at Altitude/Acclimatisation

An unprecedented growing number of athletes regularly engage in prolonged (>10 days) periods of altitude exposure, i.e. 'traditional' altitude training camps in the form of 'live high-train high' or 'live high-train low' protocols, in the belief that it can promote greater physiological adaptations compared with sea-level training, and thereby maximise endurance-based performance under both normoxic and hypoxic conditions [7, 8, 79] (Fig. 1). There are, however, relatively few articles that have investigated the effect of such altitude training on the performance of multiple sprints. This is not surprising, considering that the theoretical rationale supporting the use of chronic altitude/hypoxic exposure for improving sprint performance is less compelling. That said, scientific evidence supporting the benefits of altitude training for improving anaerobic performance is not novel. More than 25 years ago, for example, Karvonen et al. [80] reported that 2 weeks of strength and speed training at a terrestrial altitude of 1860 m in five national-level sprinters significantly improved 150-m normoxic sprint times compared with a similar programme near sea level. Similarly, 10 days of specific training at a similar altitude by 400-m runners also resulted in a significant improvement in Wingate test performance on the first 2 days near sea level [81]. Ten days in an altitude facility at a simulated altitude of ~2150 m combined with normoxic training outdoors resulted in a ~0.8% improvement in 400-m race time at sea level, but no change occurred after living and training in normoxic

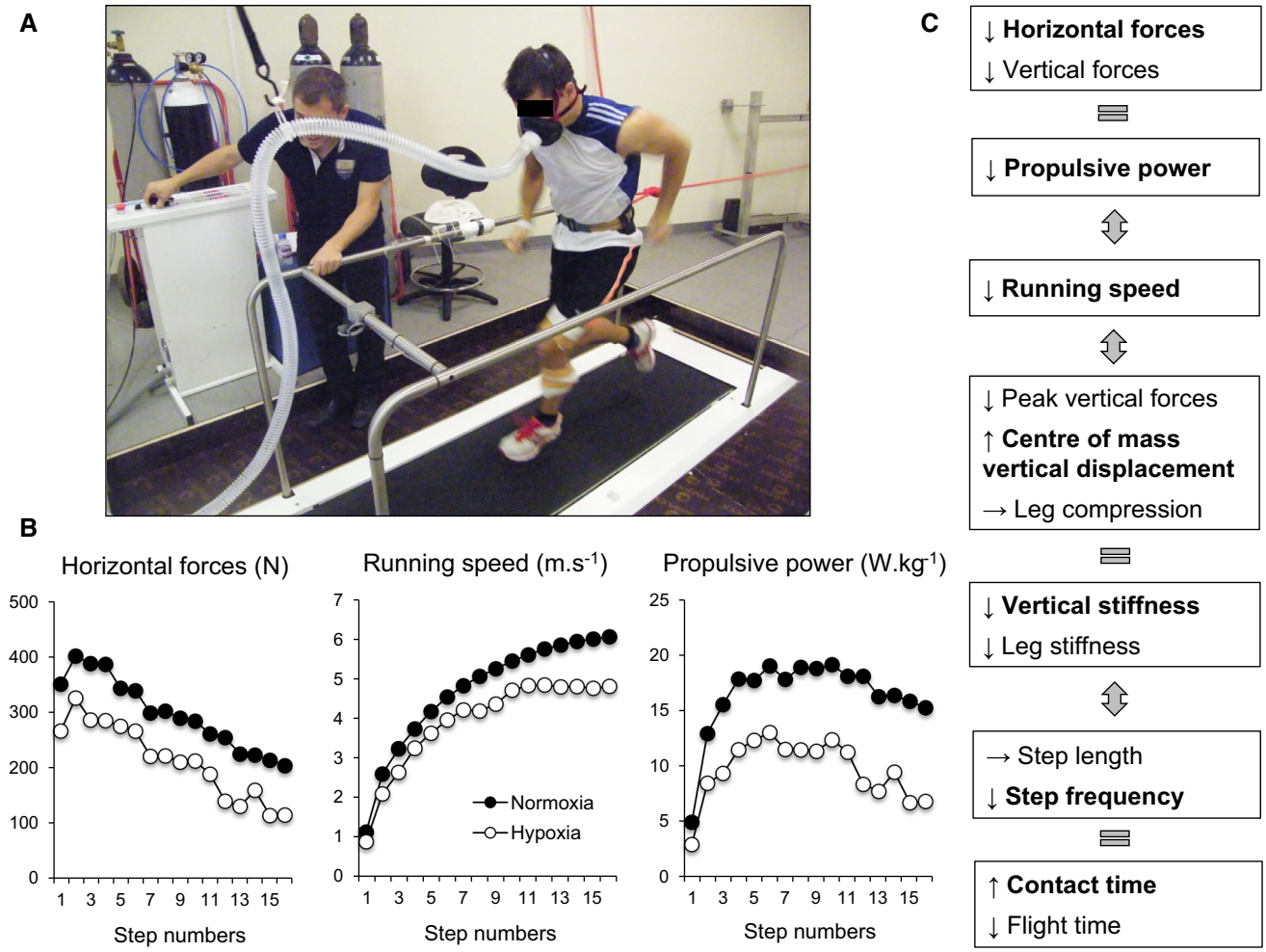

Fig. 3 Illustration of the ADAL instrumented sprint treadmill with the participant breathing a reduced-oxygen air mixture (**a**), typical curves of propulsive power, running speed and horizontal forces averaged for each step during the last effort over 5 × 5-s sprints (25-s recovery) at a simulated altitude of ~3600 m (inspired fraction of oxygen 13.3%) (*black*) compared with normoxia (inspired fraction of oxygen 20.9%) (*white*) (**b**) and overview of the main fatigue-induced changes in running mechanics across repetitions with hypoxia-related alterations marked in *bold* (**c**) (adapted from [14])

conditions [82]. In an uncontrolled study by Fornasiero et al. [83], sea-level RSA increased by 9.6% in national-level road cyclists exposed to 7 days of living and training at 1600 m followed by 10 days of residence (nitrogen house) at 2700 m.

It is noteworthy that increments in haematological-related factors (e.g. in haemoglobin mass) associated with chronic altitude exposure [84] are physiological adaptations, which probably do not significantly benefit nor deteriorate sprint performance [85], unless efforts are repeated. Altitude training, however, may boost performance of multiple sprints via an enhanced ability to deplete large amounts of high-energy phosphates at a faster rate (i.e. greater improvement in anaerobic capacity) [86], as well as beneficial changes in acid-base homeostasis and lactate metabolism [82]. In support of this proposition, increases in maximal accumulated O_2 deficit have been reported either after 15 days spent at a simulated altitude of 2650 m and training at 610 m (+10% [87]) or after 14 nights spent at a terrestrial altitude of 2100 m and training at 2700 m (+29% [88]). In elite cyclists, muscular and systemic capacity for maintaining pH and K^+ balance during exercise were similar before and after 4 weeks of placebo-controlled normobaric living high (3000 m; 16 h d^{-1}) and training low (800–1300 m) [89]. Accordingly, 30-s 'all-out' cycling performance was similar before and after the intervention. An effective tolerance to repeated-sprint training in hypoxia has also been observed: despite higher hypoxia-induced perceptual strain during the first out of six sessions during a 2-week 'live high-train low' training camp, perceptual responses improved thereafter when sprinting repeatedly in hypoxia, which was not

concluded following similar training in normoxia [55]. Finally, several weeks of continuous exposure to 4520–5050 m in HH did not affect the average mechanical power output during 10- to 30-s 'all-out' cycle exercises in lowlanders [90, 91]. A possible limitation of these later studies, however, is that sprint performance as measured at the end of the altitude camp depends both on physiological changes associated with HH (i.e. likely negative) and acclimatisation to altitude (i.e. likely positive).

6 'Live Low-Train High'

The development of hypoxic facilities (e.g. O_2-filtration or nitrogen-dilution chambers, normobaric hypoxic tents or 'altitude' masks) within the last two decades has prompted the implementation of the 'live low-train high' methods. While living at sea level, athletes receive exposure to hypoxia either at rest ('intermittent hypoxic exposure') or during exercise training sessions. Because of reports indicating that 'intermittent hypoxic exposure' alone does not lead to sustained physiological adaptations or improved exercise performance [92, 93], the present review focused on interventions involving physical exercise and hypoxic exposure in combination. Different 'live low-train high' protocols using either systemic ('intermittent hypoxic training' (IHT), 'repeated sprint training in hypoxia' (RSH) and 'resistance training in hypoxia' (RTH) [94]) or local [blood flow restriction (BFR) and ischemic preconditioning (IPC)] hypoxia exist (Fig. 1). The use of a re-breathing mask (carbon dioxide absorption) [95] or voluntary hypoventilation at a low lung volume [96, 97] aims to induce a state of desaturation and hypoxemia similar to altitude exposure, but these are not specifically discussed. They do not provide sufficient hypoxic stimuli to induce the haematological changes associated with chronic hypoxic exposure [93]. Some of the 'live low-train high' methods can improve sprinting performance and, therefore, are discussed below.

6.1 Systemic Hypoxia

6.1.1 Intermittent Hypoxic Training

It has been postulated that IHT may enhance glycolytic enzyme activity [98], muscle buffering capacity [99], muscle mitochondrial and capillary density [100, 101], as well as stimulation of other markers of mitochondrial metabolism and biogenesis [102, 103]. Two studies found that the addition of 10 days of IHT to the normal training programme of well-trained athletes produced worthwhile gains (3–5%) in a 30-s Wingate test [104, 105], while others found no additional benefit of hypoxic exposure above that of training closer to sea level [106–108]. Furthermore, the addition of a NH stimulus equivalent to ~3000 m (FiO_2 14.4%) on anaerobic performance (2-min time trial) or monocarboxylate transporters 1 and 4 protein content after a 3-week training period is ineffective [109]. Finally, a regimen of 1 h day^{-1} (6 × 6-min at FiO_2 10–15% separated by 4 min in normoxia) for 14 consecutive days in rugby players had trivial (<1%) effects on maximum speed and sprint times. During rugby simulation, in the form of seven circuits containing a range of activities characterising a rugby game, hypoxic exposure produced a detrimental (9–15%) effect on peak power in two scrums and also impairments of time in offensive (7%) and tackle (11%) sprints [110].

Regarding RSA, the exact value of IHT continues to be a source of debate [70]. In field hockey and soccer players, daily exposure to hypoxia produced with a rebreathing device for 1 h day^{-1} for 15 days enhanced sprint speed from 1.5% in the first sprint up to 7.0% in the last bout of a series of 6 × 70-m shuttle sprints; substantial effects were still present 12 days after exposure [111]. Similarly, intermittent hypoxic exposure (FiO_2 9–12% using a nitrogen-filtration device) for 15 days over a 3-week period substantially enhanced peak power and repeated-sprint performance (5 × 100 m—15-s recovery) on a kayak ergometer in sub-elite kayakers 3 days after the intervention period [112]. A follow-up study by the same research group aimed to determine whether increasing the number of hypoxic transitions (3 min:3 min vs. 5 min:5 min hypoxia:normoxia alternance) during a 60-min exposure (i.e. ~4350–5000 m or FiO_2 12.0–10.9% at rest for 5 consecutive days per week for 3 weeks) would result in greater enhancement of sprint performance [113]. However, changes in first sprint power were unclear and RSA was impaired by 2% with also no difference between interventions. Reportedly, amateur Australian footballers covered extra distance at low intensity (i.e. improved pacing strategies) during a self-paced team-sport running protocol before and after eight interval treadmill running sessions (4 weeks) performed under hypoxic conditions (FiO_2 15%). In this later study, however, changes in sprinting distance remained unclear in reference to the normoxic training group [114]. Some of the inconclusive IHT findings may be owing to small sample sizes together with methodological differences in (1) test protocols (i.e. sprint length/duration, exercise-to-rest ratio, number of participants), (2) hypoxic methods, (3) details of the intervention (i.e. duration and severity of the hypoxic stimulus), (4) participants training status and (5) timepoints following hypoxic exposure at which performance was assessed. The relative inefficacy of IHT may also be clouded by a reduced training intensity (e.g. −6% in [114]), meaning that IHT participants usually undergo

lower absolute exercise workloads (i.e. not sufficiently intense to stress O_2 delivery and maximise adaptations) than sea-level equivalents.

6.1.2 Repeated-Sprint Training in Hypoxia

To overcome some of the inherent limitations of IHT (e.g. lower training stimulus due to hypoxia), a new hypoxic training method, i.e. the so-called 'repeated-sprint training in hypoxia' or RSH, was developed in Lausanne. This comes from the observation of an up-regulation of circulating microRNA levels, only when exercise was performed at a high intensity and at a high altitude (i.e. and not at lower intensity) [101]. Repeated sprint training in hypoxia is based on the repetition of short (<30 s) 'all-out' sprints with incomplete recovery periods in hypoxia [70]. This model differs from IHT because the intensity of the training stimulus is maximal and therefore would allow one to maintain high fast-twitch muscle fibre recruitment so that positive results can be expected when adding hypoxia to training [72]. Hence, a lower rate of O_2 delivery to the muscles increases the stress on glycolytic flux, which may stimulate the up-regulation of this energy pathway. Faiss et al. [72] were the first to reason that, compared with repeated-sprint training in normoxia, RSH could induce beneficial adaptations at the muscular level, along with improved blood perfusion, which may lead to greater improvements in RSA. To date, the popularity of this method is shown by the publication of approximately ten studies on this topic and its use by many professional sports (e.g. Welsh Rugby Union, Roland Garros training centre, Aspire Football Academy, Australian footballers).

In general, superior performance outcomes including RSA performance in normoxic conditions have been associated with RSH vs. RSN studies. For instance, RSH produces faster mean sprint times and/or smaller speed decrements than RSN [115, 116], likely resulting from an improved fatigue resistance. Following six to eight RSH sessions, the number of sprints before task failure was increased in both well-trained cyclists (+38% [72]) and cross-country skiers (+58% [71]) during an 'open-loop' RSA protocol. In highly trained youth soccer players, the addition of ten repeated-sprint training sessions performed in hypoxia vs. normoxia to their regular soccer practice over a 5-week in-season period was more efficient at enhancing specific RSA (including direction changes), even though there was no additional effect on improvements in maximal sprinting speeds [117]. Conversely, two studies have shown that completion of 12–15 maximal cycling RSH sessions over 4–5 weeks did not lead to further improvement in RSA, compared with similar normoxic training [118, 119]. Arguably, the use of non-specific cycling RSH training for team-sport athletes performing predominantly run-based training [118] and methodological shortcomings (i.e. absence of protective pacing measures, completion of an inappropriately large number of performance tests pre- vs. post-intervention [119, 120]) may potentially explain the absence of the additional effects of RSH vs. RSN.

Practically, Gatterer et al. [121] showed that soccer-specific shuttle-run sprint training is feasible in hypoxic chambers of limited size (4.75 × 2.25 m) and is associated with enhanced running speed maintenance during a RSA test (lower fatigue slope) when compared with normoxic training. We also proposed using a mobile inflatable hypoxic marquee to allow over-ground running during RSH and to increase ecological validity [122] (Fig. 4). Using this unique tool, it was demonstrated that 'innovative' methods (e.g. RSH) combined with 'traditional' simulated altitude methods (e.g. 'live high-train low'), where athletes "live high and train low except for a few intense workouts in altitude" for 14 days, boosts RSA in team sports [85]. When combined with prolonged simulated altitude/hypoxic residence, the superiority of RSH to RSN was demonstrated by twofold immediate RSA performance gains that were only maintained 3 weeks after the intervention in the 'live-high-train low and high' group. A few days after this innovative intervention, changes in hypoxia inducible factor-1α subunit, vascular endothelial growth factor, myoglobin, peroxisome proliferator-activated receptor-gamma coactivator 1α and mitochondrial transcription factor A mRNA levels were generally of larger magnitude than with similar normoxic exercise, yet the decay in molecular adaptations was relatively fast (i.e. benefits already absent 3 weeks post-intervention) [123].

6.1.3 Resistance Training in Hypoxia

While athletic (and clinical) populations have used a variety of resistance training modalities performed under normoxic conditions, there is growing research interest focusing on the relevance of additional hypoxic exposure; the so-called 'resistance training in hypoxia' or RTH [124]. It has been suggested that, in an O_2-deprived environment, the low partial pressure of O_2 would exaggerate metabolite [e.g. blood (La) and anabolic hormones (e.g. growth hormone)] accumulation. This potentially results in an accelerated recruitment of higher threshold motor units, subsequently producing adaptation in the form of hypertrophy of these motor units and eventually greater improvements in muscle strength [125]. Therefore, it has been proposed that RTH may elicit greater muscular adaptations that could be translated into faster sprint times via a better force production/application than the equivalent training in normoxia [124]. After more than a decade of research,

Fig. 4 Mobile inflatable simulated hypoxic equipment [122]. External 45-m running lane (1.8 m width and 2.5 m height) tunnel design (**a**), hypoxic system trailer (including a 55-kW screw compressor) (**b**) and athletes sprinting inside the marquee (**c**, **d**)

however, results of RTH studies are conflicting with some studies finding superiority for RTH in hypertrophic and strength responses and others showing no significant differences. Such divergent findings may relate to alteration in metabolic stress (i.e. level of hypoxia and inter-set rest periods) between studies [124]. Compared with a normoxic equivalent, work-matched (absolute intensity) RTH will provide a higher relative training stimulus; as such, group-wise comparisons must consider whether normoxic resistance training groups were matched for absolute or relative training intensity with the hypoxic group. Regarding sprint performance, the effects of RTH are largely unknown, with only one published study showing beneficial outcomes [126]. We believe it is crucial that future studies continue to include measures of multiple-sprint performance to provide better information regarding the usefulness of RTH for sprinters and/or team- and racquet-sport athletes.

6.2 Local Hypoxia

6.2.1 Blood Flow Restriction

Numerous investigations have reported beneficial morphological and strength responses (albeit small) with the addition of blood flow restriction or BFR (also known as 'Kaatsu training') to low-load resistance exercise [127]. This strategy involves the application of a pressure cuff, which is inflated around a limb (i.e. proximal to the muscles being trained) and produces a hypoxic muscular environment by limiting blood delivery to and from contracting muscles. Recent research has demonstrated maximal and repeated-sprint performance gains [126, 128, 129] after BFR training. In female netball players, for instance, 5 weeks of low-load resistance exercise in conjunction with either BFR or systemic hypoxia increased 5- and 10-m running sprint performance compared with control training (without BFR and hypoxic breathing), the former to a

larger extent than the latter [126]. Larger improvements in performance when exposed to hypoxic conditions may be attributed to a higher relative training intensity. There is currently no clear consensus regarding the best practice for implementing BFR methods. Recently, the efficacy of repeated 30-s 'all-out' cycling bouts combined with post-exercise BFR has been proposed by Taylor et al. [130] as a novel approach to augment maximal O_2 uptake (+4.5%) in trained athletes. However, this did not translate into an enhanced exercise performance (15-km time trial) above that with training with a normal blood perfusion. In this later study, the observation of improved hypoxia inducible factor-1α-mediated cell signalling with the addition of BFR to increase tissue hypoxia might suggest that this form of hypoxic training has the potential to increase the performance of multiple sprints, but this still needs to be verified.

6.2.2 *Ischaemic Preconditioning*

Ischaemic preconditioning (IPC) is defined as the exposure to brief periods of circulatory occlusion and reperfusion [131] completed several minutes prior to a given exercise. While IPC was originally developed to locally or systemically (remote IPC) protect organs against subsequent bouts of ischemia, it has also been proposed as an attractive ergogenic aid for athletes to improve exercise capacity. Research into the effects of IPC on the performance of multiple sprints is relatively recent and remains equivocal. In two studies, IPC likely increased mechanical power during repeated cycling sprints in comparison to a placebo [132, 133]. Other studies, however, reported similar performance outcomes with or without IPC [134, 135]. Another study even showed a detrimental effect on a series of Wingate tests [136]. Furthermore, IPC has been shown to be beneficial to swim time completion of single 100-m [137] and repeated 50-m 'all-out' efforts [138], whereas, it had no effect during 3 × 30-m runs repeated every minute [139]. The most common IPC protocol, as adopted in all the studies reviewed here, involves three or four cycles of 5 min of circulatory occlusion and reperfusion [131]. However, the large variability in participant characteristics (e.g. IPC responsiveness, training status), exercise modes (e.g. cycling, running, swimming) and study methods [e.g. size (muscle mass) of the occluded limb, unilateral or bilateral occlusion, and time lag between IPC and exercise start, cuff inflation pressure] across studies makes it difficult to compare them directly and could potentially explain discrepant findings. Reportedly, IPC has been observed to increase SpO_2 during successive 'all-out' efforts [133], whereas, the level of perceived effort [133, 134] and [La] [133, 138] remained changed. The precise mechanisms involved in mediating the aforementioned performance changes are incompletely understood. Pending confirmatory research, it is thought that IPC may enhance muscle activation as well as muscle efficiency in ATP use via ATP sparing, augment electron mitochondrial flux, and/or improve efficiency of excitation-contraction coupling [131].

7 Future Research Directions

Some of the key research gaps in the field of altitude/hypoxic training relevant for athletes engaged in sprint-based disciplines can be addressed by considering the following unresolved performance-orientated and mechanistic issues:

- Most of our knowledge about altitude/hypoxia-mediated changes in RSA has been sourced from laboratory findings in NH. Mounting evidence, however, indicates that HH likely induces more severe physiological responses (SpO_2 and heart rate) than NH [68]. This would suggest that performance, as well as physiological and biomechanical alterations, may be different when sprinting repeatedly at terrestrial altitude. Direct comparisons of repeated-sprint exercises between NH and HH are required.
- Repeated sprints can induce exercise-induced hypoxemia (i.e., a 3% reduction in SpO_2 compared with sea-level or pre-exercise values) that in turn has been correlated with the attenuation of the EMG activity of the active musculature, and the reduction in mechanical work during 20 × 5-s cycles sprints [59]. It is plausible that reduced SpO_2 can contribute to fatigue during repeated sprinting, given that phosphocreatine resynthesis and H^+ removal are O_2-dependent processes [140]. Individuals with higher aerobic capacity often, during intense exercise, exhibit the lowest SpO_2 values and are thus more prone to exercise-induced hypoxemia [141, 142]. Whether performance level and/or sex differences exist when completing RSA at different altitude/hypoxic levels warrants further investigation.
- The ability to apply mechanical horizontal forces is particularly vulnerable in O_2-deprived environments [14]. Individuals willing to improve their acceleration might need to put a greater emphasis on developing/maintaining maximal horizontal force production capacity, potentially by implementing horizontally oriented resistance exercises. Taking into consideration some of the individual differences in performance, with respect to acceleration and maximal sprinting speed but also the effect of a reduced air density at terrestrial altitude, would allow more individualised and accurate evaluation, monitoring and training practices based on athletes' mechanical characteristics when training and/

or competing at altitude. Here, a promising modelling approach, based on data that are now rather simple to obtain in field conditions (e.g. body mass, jump height, sprint times or speed), can be useful to investigate if altitude modifies the power-force-speed sprint profiling of athletes [143].

- Absolute acute or chronic hypoxia-mediated changes in athlete physiology do not necessarily translate into proportional changes in multiple-sprint performance, owing to greater variability in the latter. Consideration of the current training phase, the long-term consequences (longer than several days post-intervention) and the influence of training load changes (as the result of intensified training) on these responses is required when implementing an altitude/hypoxic training programme [144]. The magnitude of any hypoxic-mediated effect on performance of multiple sprints will also depend on the duration of the sprint and the exercise-to-rest ratio [145]. For example, Brosnan et al. [146] revealed no difference in peak power over the course of 6 × 15-s cycling sprints between simulated altitudes of 585 and 2100 m (FiO_2 19.5 and 16.2%, respectively), provided the exercise-to-rest ratio was 1:3. For shorter recovery periods (exercise-to-rest ratios of 1:2 and 1:1), there was a small effect of altitude (~2.5% decrease per 1000 m), induced by the predominant effect of fatigue occurring from the repetitive sprint itself. Further work should therefore establish the optimal period of a competitive season, hypoxic dose and exercise-to-rest ratio for optimal altitude training benefits.

8 Conclusion

Performance of a single sprint is generally not negatively affected by acute exposure to NH because an enhanced anaerobic energy release can compensate for the reduced aerobic ATP production. Conversely, the reduction in air density upon ascent to terrestrial altitude (i.e. HH) means that performance may be improved in sprinting events where air resistance is a limiting factor. With the repetition of maximal efforts, however, RSA is altered more so (i.e. with earlier and larger performance decrements) at high altitudes (>3000–3600 m or FiO_2 <14.4–13.3%), be it HH or NH, compared with either normoxia or low-to-moderate altitudes (<3000 m or FiO_2 >14.4%). Not only does acute hypoxic exposure decrease convective O_2 transport (i.e. reduction in arterial O_2 saturation values), but also challenges multiple regulatory systems by increasing cardiorespiratory (i.e. higher heart rate, minute ventilation), metabolic (i.e. slower muscle re-oxygenation responses) and/or neuromuscular (i.e. incomplete muscle activation) requirements during sprinting and subsequent recovery periods. Furthermore, beneficial effects of altitude/hypoxic training using traditional methods (i.e. residence at altitude) on sprint performance are still debated. This is perhaps because haematological adaptations are not central to sprint-based disciplines. Recently, innovative 'live low-train high' methods, in isolation or in combination with chronic HH/NH exposure, have emerged as 'resistance training in hypoxia' or 'repeated-sprint training in hypoxia' with the belief that up-regulated non-haematological peripheral adaptations (i.e. additional activation of anaerobic and neuromuscular pathways) may further improve performance of multiple sprints compared with similar normoxic interventions. These claims, however, must be further substantiated in relation to competitive sporting events.

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References

1. Schimpchen J, Skorski S, Nopp S, Meyer T. Are, "classical" tests of repeated-sprint ability in football externally valid? A new approach to determine in-game sprinting behaviour in elite football players. J Sports Sci. 2016;34(6):519–26.
2. Girard O, Mendez-Villanueva A, Bishop D. Repeated-sprint ability. Part I: factors contributing to fatigue. Sports Med. 2011;41(8):673–94.
3. Jokl E, Jokl P, Seaton DC. Effect of altitude upon 1968 Olympic Games running performances. Int J Biometeorol. 1969;13(3):309–11.
4. Nassis GP. Effect of altitude on football performance: analysis of the 2010 FIFA World Cup Data. J Strength Cond Res. 2013;27(3):703–7.
5. McSharry PE. Effect of altitude on physiological performance: a statistical analysis using results of international football games. BMJ. 2007;335(7633):1278–81.
6. George TM, Olsen PD, Kimber NE, et al. The effect of altitude and travel on rugby union performance: analysis of the 2012 Super Rugby competition. J Strength Cond Res. 2015;29(12):3360–6.
7. Wilber RL, Stray-Gundersen J, Levine BD. Effect of hypoxic "dose" on physiological responses and sea-level performance. Med Sci Sports Exerc. 2007;39(9):1590–9.
8. Millet GP, Roels B, Schmitt L, et al. Combining hypoxic methods for peak performance. Sports Med. 2010;40(1):1–25.
9. Girard O, Amann M, Aughey R, et al. Position statement: altitude training for improving team-sport players' performance:

current knowledge and unresolved issues. Br J Sports Med. 2013;47(Suppl. 1):i8–16.
10. Gore CJ, Clark SA, Saunders PU. Nonhematological mechanisms of improved sea-level performance after hypoxic exposure. Med Sci Sports Exerc. 2007;39(9):1600–9.
11. McLellan TM, Kavanagh MF, Jacobs I. The effect of hypoxia on performance during 30 s or 45 s of supramaximal exercise. Eur J Appl Physiol Occup Physiol. 1990;60(2):155–61.
12. Calbet JA, De Paz JA, Garatachea N, et al. Anaerobic energy provision does not limit Wingate exercise performance in endurance-trained cyclists. J Appl Physiol. 2003;94(2):668–76.
13. Bowtell JL, Cooke K, Turner R, et al. Acute physiological and performance responses to repeated sprints in varying degrees of hypoxia. J Sci Med Sport. 2014;17(4):399–403.
14. Brocherie F, Millet GP, Morin JB, Girard O. Mechanical alterations to repeated treadmill sprints in normobaric hypoxia. Med Sci Sports Exerc. 2016;48(8):1570–9.
15. Balsom PD, Gaitanos GC, Ekblom B, Sjodin B. Reduced oxygen availability during high intensity intermittent exercise impairs performance. Acta Physiol Scand. 1994;152(3):279–85.
16. Smith KJ, Billaut F. Influence of cerebral and muscle oxygenation on repeated-sprint ability. Eur J Appl Physiol. 2010;109(5):989–99.
17. Billaut F, Buchheit M. Repeated-sprint performance and vastus lateralis oxygenation: effect of limited O(2) availability. Scand J Med Sci Sports. 2013;23(3):e185–93.
18. Ogura Y, Katamoto S, Uchimaru J, et al. Effects of low and high levels of moderate hypoxia on anaerobic energy release during supramaximal cycle exercise. Eur J Appl Physiol. 2006;98(1):41–7.
19. Weyand PG, Lee CS, Martinez-Ruiz R, et al. High-speed running performance is largely unaffected by hypoxic reductions in aerobic power. J Appl Physiol. 1999;86(6):2059–64.
20. Ogawa T, Ohba K, Nabekura Y, et al. Intermittent short-term graded running performance in middle-distance runners in hypobaric hypoxia. Eur J Appl Physiol. 2005;94(3):254–61.
21. Morales-Alamo D, Ponce-Gonzalez JG, Guadalupe-Grau A, et al. Increased oxidative stress and anaerobic energy release, but blunted Thr172-AMPKalpha phosphorylation, in response to sprint exercise in severe acute hypoxia in humans. J Appl Physiol. 2012;113(6):917–28.
22. Wehrlin JP, Hallen J. Linear decrease in VO_{2max} and performance with increasing altitude in endurance athletes. Eur J Appl Physiol. 2006;96(4):404–12.
23. Hollings SC, Hopkins WG, Hume PA. Environmental and venue-related factors affecting the performance of elite male track athletes. Eur J Sport Sci. 2012;12(3):201–6.
24. Hamlin MJ, Hopkins WG, Hollings SC. Effects of altitude on performance of elite track-and-field athletes. Int J Sports Physiol Perform. 2015;10(7):881–7.
25. Gastin PB. Energy system interaction and relative contribution during maximal exercise. Sports Med. 2001;31(10):725–41.
26. Burtscher M, Faulhaber M, Flatz M, et al. Effects of short-term acclimatization to altitude (3200 m) on aerobic and anaerobic exercise performance. Int J Sports Med. 2006;27(8):629–35.
27. Levine BD, Stray-Gundersen J, Mehta RD. Effect of altitude on football performance. Scand J Med Sci Sports. 2008;18(Suppl. 1):76–84.
28. Noordhof DA, Mulder RC, de Koning JJ, Hopkins WG. Race factors affecting performance times in elite long-track speed skating. Int J Sports Physiol Perform. 2016;11(4):535–42.
29. Peronnet F, Thibault G, Cousineau DL. A theoretical analysis of the effect of altitude on running performance. J Appl Physiol. 1991;70(1):399–404.
30. Feriche B, Garcia-Ramos A, Calderon-Soto C, et al. Effect of acute exposure to moderate altitude on muscle power: hypobaric hypoxia vs. normobaric hypoxia. PLoS One. 2014;9(12):e114072.
31. Ward-Smith AJ. Air resistance and its influence on the biomechanics and energetics of sprinting at sea level and at altitude. J Biomech. 1984;17(5):339–47.
32. Arsac LM. Effects of altitude on the energetics of human best performances in 100 m running: a theoretical analysis. Eur J Appl Physiol. 2002;87(1):78–84.
33. Mureika JR. The legality of wind and altitude assisted performances in the sprints. New Studies Athletics. 2000;15(3/4):53–60.
34. Quinn MD. The effects of wind and altitude in the 200-m sprint. J Appl Biomech. 2003;19:49–59.
35. Quinn MD. The effects of wind and altitude in the 400-m sprint. J Sports Sci. 2004;22(11–12):1073–81.
36. Frohlich C. Effect of wind and altitude on record performance in foot races, pole vault, and long jump. Am J Phys. 1985;53:726.
37. Billaut F, Kerris JP, Rodriguez RF, et al. Interaction of central and peripheral factors during repeated sprints at different levels of arterial O_2 saturation. PLoS One. 2013;8(10):e77297.
38. Girard O, Brocherie F, Morin JB, Millet GP. Running mechanical alterations during repeated treadmill sprints in hot versus hypoxic environments: a pilot study. J Sports Sci. 2016;34(12):1190–8.
39. Hamlin MJ, Hinckson EA, Wood MR, Hopkins WG. Simulated rugby performance at 1550-m altitude following adaptation to intermittent normobaric hypoxia. J Sci Med Sport. 2008;11(6):593–9.
40. Goods PS, Dawson BT, Landers GJ, et al. Effect of different simulated altitudes on repeat-sprint performance in team-sport athletes. Int J Sports Physiol Perform. 2014;9(5):857–62.
41. Amann M, Calbet JA. Convective oxygen transport and fatigue. J Appl Physiol. 2008;104(3):861–70.
42. Morrison J, McLellan C, Minahan C. A clustered repeated-sprint running protocol for team-sport athletes performed in normobaric hypoxia. J Sports Sci Med. 2015;14(4):857–63.
43. Morrison JD, Lovell D, McLellan C, Minahan C. Performance and metabolic responses of highly-trained team-sport athletes during repeated sprinting in hypoxia. J Athletic Enhance. 2015;4(2):1–5.
44. Kon M, Nakagaki K, Ebi Y, et al. Hormonal and metabolic responses to repeated cycling sprints under different hypoxic conditions. Growth Horm IGF Res. 2015;25(3):121–6.
45. Feriche B, Delgado M, Calderon C, et al. The effect of acute moderate hypoxia on accumulated oxygen deficit during intermittent exercise in nonacclimatized men. J Strength Cond Res. 2007;21(2):413–8.
46. Robach P, Biou D, Herry JP, et al. Recovery processes after repeated supramaximal exercise at the altitude of 4,350 m. J Appl Physiol. 1997;82(6):1897–904.
47. Bishop DJ, Girard O. Determinants of team-sport performance: implications for altitude training by team-sport athletes. Br J Sports Med. 2013;47(Suppl. 1):i17–21.
48. Faude O, Koch T, Meyer T. Straight sprinting is the most frequent action in goal situations in professional football. J Sports Sci. 2012;30(7):625–31.
49. Gregson W, Drust B, Atkinson G, Salvo VD. Match-to-match variability of high-speed activities in premier league soccer. Int J Sports Med. 2010;31(4):237–42.
50. Turner G, Gibson OR, Maxwell NS. Simulated moderate hypoxia reduces intermittent sprint performance in games players. J Sports Med Phys Fitness. 2014;54(5):566–74.
51. Aldous JW, Chrismas BC, Akubat I, et al. Hot and hypoxic environments inhibit simulated soccer performance and exacerbate performance decrements when combined. Front Physiol. 2016;6:421.

52. Billaut F, Aughey RJ. Update in the understanding of altitude-induced limitations to performance in team-sport athletes. Br J Sports Med. 2013;47(Suppl. 1):i22–5.
53. Garvican LA, Hammond K, Varley MC, et al. Lower running performance and exacerbated fatigue in soccer played at 1600 m. Int J Sports Physiol Perform. 2014;9(3):397–404.
54. Aughey RJ, Hammond K, Varley MC, et al. Soccer activity profile of altitude versus sea-level natives during acclimatisation to 3600 m (ISA3600). Br J Sports Med. 2013;47(Suppl. 1):i107–13.
55. Brocherie F, Millet GP, Girard O. Psycho-physiological responses to repeated-sprint training in normobaric hypoxia and normoxia. Int J Sports Physiol Perform. 2017;12(1):115–23.
56. Tucker R, Rauch L, Harley YX, Noakes TD. Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. Pflugers Arch. 2004;448(4):422–30.
57. Mohr M, Nybo L, Grantham J, Racinais S. Physiological responses and physical performance during football in the heat. PLoS One. 2012;7(6):e39202.
58. Ogawa T, Hayashi K, Ichinose M, et al. Metabolic response during intermittent graded sprint running in moderate hypobaric hypoxia in competitive middle-distance runners. Eur J Appl Physiol. 2007;99(1):39–46.
59. Billaut F, Smith K. Prolonged repeated-sprint ability is related to arterial O_2 desaturation in men. Int J Sports Physiol Perform. 2010;5(2):197–209.
60. Monroe DC, Gist NH, Freese EC, et al. Effects of sprint interval cycling on fatigue, energy, and cerebral oxygenation. Med Sci Sports Exerc. 2016;48(4):615–24.
61. Girard O, Brocherie F, Millet GP. High altitude increases alteration in maximal torque but not in rapid torque development in knee extensors after repeated treadmill sprinting. Front Physiol. 2016;7:97.
62. Smith KJ, Billaut F. Tissue oxygenation in men and women during repeated-sprint exercise. Int J Sports Physiol Perform. 2012;7(1):59–67.
63. Girard O, Brocherie F, Morin JB, Millet GP. Neuro-mechanical determinants of repeated treadmill sprints: usefulness of an "hypoxic to normoxic recovery" approach. Front Physiol. 2015;6:260.
64. Favret F, Richalet JP. Exercise and hypoxia: the role of the autonomic nervous system. Respir Physiol Neurobiol. 2007;158(2–3):280–6.
65. Hogan MC, Richardson RS, Haseler LJ. Human muscle performance and PCr hydrolysis with varied inspired oxygen fractions: a 31P-MRS study. J Appl Physiol. 1999;86(4):1367–73.
66. Goodall S, Twomey R, Amann M. Acute and chronic hypoxia: implications for cerebral function and exercise tolerance. Fatigue. 2014;2(2):73–92.
67. Ainslie PN, Ogoh S. Regulation of cerebral blood flow in mammals during chronic hypoxia: a matter of balance. Exp Physiol. 2010;95(2):251–62.
68. Millet GP, Faiss R, Pialoux V. Point: hypobaric hypoxia induces different physiological responses from normobaric hypoxia. J Appl Physiol. 2012;112(10):1783–4.
69. Amann M, Goodall S, Twomey R, et al. AltitudeOmics: on the consequences of high-altitude acclimatization for the development of fatigue during locomotor exercise in humans. J Appl Physiol. 2013;115(5):634–42.
70. Faiss R, Girard O, Millet GP. Advancing hypoxic training in team sports: from intermittent hypoxic training to repeated sprint training in hypoxia. Br J Sports Med. 2013;47(Suppl. 1):i45–50.
71. Faiss R, Willis S, Born DP, et al. Repeated double-poling sprint training in hypoxia by competitive cross-country skiers. Med Sci Sports Exerc. 2015;47(4):809–17.
72. Faiss R, Leger B, Vesin JM, et al. Significant molecular and systemic adaptations after repeated sprint training in hypoxia. PLoS One. 2013;8(2):e56522.
73. McCully KK, Iotti S, Kendrick K, et al. Simultaneous in vivo measurements of HbO_2 saturation and PCr kinetics after exercise in normal humans. J Appl Physiol. 1994;77(1):5–10.
74. McMahon S, Jenkins D. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. Sports Med. 2002;32(12):761–84.
75. Morin JB, Edouard P, Samozino P. Technical ability of force application as a determinant factor of sprint performance. Med Sci Sports Exerc. 2011;43(9):1680–8.
76. Girard O, Micallef JP, Millet GP. Changes in spring-mass model characteristics during repeated running sprints. Eur J Appl Physiol. 2011;111(1):125–34.
77. Morin JB, Samozino P, Bonnefoy R, et al. Direct measurement of power during one single sprint on treadmill. J Biomech. 2010;43(10):1970–5.
78. Girard O, Brocherie F, Morin JB, Millet GP. Intrasession and intersession reliability of running mechanics during treadmill sprints. Int J Sports Physiol Perform. 2016;11(4):432–9.
79. Alvarez-Herms J, Julia-Sanchez S, Hamlin MJ, et al. Popularity of hypoxic training methods for endurance-based professional and amateur athletes. Physiol Behav. 2015;143:35–8.
80. Karvonen J, Peltola E, Saarela J, Nieminen MM. Changes in running speed, blood lactic acid concentration and hormone balance during sprint training performed at an altitude of 1860 metres. J Sports Med Phys Fitness. 1990;30(2):122–6.
81. Rega C, Stephan H, Brue F. Les effets physiques et physiologiques d'un entrainement en moyenne altitude chez le coureur de 400 mètres. Sci Mot. 1989;8:3–12.
82. Nummela A, Rusko H. Acclimatization to altitude and normoxic training improve 400-m running performance at sea level. J Sports Sci. 2000;18(6):411–9.
83. Fornasiero D, Martin DT, Brosnan MJ, et al. Effects of altitude training on repeat sprint and graded exercise test performance in female road cyclists (abstract). Canberra: Sports Medicine Australia; 1999. p. 90.
84. Gore CJ, Sharpe K, Garvican-Lewis LA, et al. Altitude training and haemoglobin mass from the optimised carbon monoxide rebreathing method determined by a meta-analysis. Br J Sports Med. 2013;47(Suppl. 1):i31–9.
85. Brocherie F, Millet GP, Hauser A, et al. "Live high-train low and high" hypoxic training improves team-sport performance. Med Sci Sports Exerc. 2015;47(10):2140–9.
86. Svedenhag J, Saltin B, Johonson C, Kaijeser L. Aerobic and anaerobic exercise capacities of elite middle-distance runners after two weeks of training at moderate altitude. J Med Sci Sports. 1991;11:205–14.
87. Roberts AD, Clark SA, Townsend NE, et al. Changes in performance, maximal oxygen uptake and maximal accumulated oxygen deficit after 5, 10 and 15 days of live high:train low altitude exposure. Eur J Appl Physiol. 2003;88(4–5):390–5.
88. Mizuno M, Juel C, Bro-Rasmussen T, et al. Limb skeletal muscle adaptation in athletes after training at altitude. J Appl Physiol. 1990;68(2):496–502.
89. Nordsborg NB, Siebenmann C, Jacobs RA, et al. Four weeks of normobaric "live high-train low" do not alter muscular or systemic capacity for maintaining pH and K(+) homeostasis during intense exercise. J Appl Physiol. 2012;112(12):2027–36.
90. Di Prampero PE, Mognoni P, Veicsteinas A. The effects of hypoxia on maximal anaerobic alactic power in man. In:

Brendel W, Zink RA, editors. High altitude physiology and medicine. Berlin: Springer; 1982. p. 88–93.
91. Grassi B, Mognoni P, Marzorati M, et al. Power and peak blood lactate at 5050 m with 10 and 30 s 'all out' cycling. Acta Physiol Scand. 2001;172(3):189–94.
92. Bartsch P, Dehnert C, Friedmann-Bette B, Tadibi V. Intermittent hypoxia at rest for improvement of athletic performance. Scand J Med Sci Sports. 2008;18(Suppl. 1):50–6.
93. Lundby C, Millet GP, Calbet JA, et al. Does 'altitude training' increase exercise performance in elite athletes? Br J Sports Med. 2012;46(11):792–5.
94. McLean BD, Gore CJ, Kemp J. Application of 'live low-train high' for enhancing normoxic exercise performance in team sport athletes. Sports Med. 2014;44(9):1275–87.
95. Porcari JP, Probst L, Forrester K, et al. Effect of wearing the elevation training mask on aerobic capacity, lung function, and hematological variables. J Sports Sci Med. 2016;15(2):379–86.
96. Woorons X, Gamelin FX, Lamberto C, et al. Swimmers can train in hypoxia at sea level through voluntary hypoventilation. Respir Physiol Neurobiol. 2014;190:33–9.
97. Trincat L, Woorons X, Millet GP. Repeated sprint training in hypoxia induced by voluntary hypoventilation in swimming. Int J Sports Physiol Perform. 2016;24:1–24.
98. Katayama K, Sato K, Matsuo H, et al. Effect of intermittent hypoxia on oxygen uptake during submaximal exercise in endurance athletes. Eur J Appl Physiol. 2004;92(1–2):75–83.
99. Gore CJ, Hahn AG, Aughey RJ, et al. Live high:train low increases muscle buffer capacity and submaximal cycling efficiency. Acta Physiol Scand. 2001;173(3):275–86.
100. Geiser J, Vogt M, Billeter R, et al. Training high–living low: changes of aerobic performance and muscle structure with training at simulated altitude. Int J Sports Med. 2001;22(8):579–85.
101. Vogt M, Puntschart A, Geiser J, et al. Molecular adaptations in human skeletal muscle to endurance training under simulated hypoxic conditions. J Appl Physiol. 2001;91(1):173–82.
102. Terrados N, Jansson E, Sylven C, Kaijser L. Is hypoxia a stimulus for synthesis of oxidative enzymes and myoglobin? J Appl Physiol. 1990;68(6):2369–72.
103. Zoll J, Ponsot E, Dufour S, et al. Exercise training in normobaric hypoxia in endurance runners. III: Muscular adjustments of selected gene transcripts. J Appl Physiol. 2006;100(4):1258–66.
104. Hamlin MJ, Marshall HC, Hellemans J, et al. Effect of intermittent hypoxic training on 20 km time trial and 30 s anaerobic performance. Scand J Med Sci Sports. 2010;20(4):651–61.
105. Hendriksen IJ, Meeuwsen T. The effect of intermittent training in hypobaric hypoxia on sea-level exercise: a cross-over study in humans. Eur J Appl Physiol. 2003;88(4–5):396–403.
106. Truijens MJ, Toussaint HM, Dow J, Levine BD. Effect of high-intensity hypoxic training on sea-level swimming performances. J Appl Physiol. 2003;94(2):733–43.
107. Morton JP, Cable NT. Effects of intermittent hypoxic training on aerobic and anaerobic performance. Ergonomics. 2005;48(11–14):1535–46.
108. De Smet S, Van Thienen R, Deldicque L, et al. Nitrate intake promotes shift in muscle fiber type composition during sprint interval training in hypoxia. Front Physiol. 2016;7:233.
109. Millet G, Bentley DJ, Roels B, et al. Effects of intermittent training on anaerobic performance and MCT transporters in athletes. PLoS One. 2014;9(5):e95092.
110. Hinckson EA, Hamlin MJ, Wood MR, Hopkins WG. Game performance and intermittent hypoxic training. Br J Sports Med. 2007;41(8):537–9.
111. Wood MR, Dowson MN, Hopkins WG. Running performance after adaptation to acutely intermittent hypoxia. Eur J Sport Sci. 2006;6(3):163–72.
112. Bonetti DL, Hopkins WG, Kilding AE. High-intensity kayak performance after adaptation to intermittent hypoxia. Int J Sports Physiol Perform. 2006;1(3):246–60.
113. Bonetti DL, Hopkins WG. Sea-level exercise performance following adaptation to hypoxia: a meta-analysis. Sports Med. 2009;39(2):107–27.
114. McLean BD, Tofari PJ, Gore CJ, Kemp JG. Changes in running performance after four weeks of interval hypoxic training in Australian footballers: a single-blind placebo-controlled study. J Strength Cond Res. 2015;29(11):3206–15.
115. Galvin HM, Cooke K, Sumners DP, et al. Repeated sprint training in normobaric hypoxia. Br J Sports Med. 2013;47(Suppl. 1):i74–9.
116. Kasai N, Mizuno S, Ishimoto S, et al. Effect of training in hypoxia on repeated sprint performance in female athletes. Springerplus. 2015;4:310.
117. Brocherie F, Girard O, Faiss R, Millet GP. High-intensity intermittent training in hypoxia: a double-blinded, placebo-controlled field study in youth football players. J Strength Cond Res. 2015;29(1):226–37.
118. Goods PS, Dawson B, Landers GJ, et al. No additional benefit of repeat-sprint training in hypoxia than in normoxia on sea-level repeat-sprint ability. J Sports Sci Med. 2015;14(3):681–8.
119. Montero D, Lundby C. No improved performance with repeated sprint training in hypoxia versus normoxia: a double-blind and cross-over study. Int J Sports Physiol Perform. 2017;12(2):161–7.
120. Millet GP, Brocherie F, Faiss R, Girard O. Clarification on altitude training. Exp Physiol. 2017;102(1):130–1.
121. Gatterer H, Philippe M, Menz V, et al. Shuttle-run sprint training in hypoxia for youth elite soccer players: a pilot study. J Sports Sci Med. 2014;13(4):731–5.
122. Girard O, Brocherie F, Millet GP. On the use of mobile inflatable hypoxic marquees for sport-specific altitude training in team sports. Br J Sports Med. 2013;47(Suppl. 1):i121–3.
123. Brocherie F, Millet GP, D'Hulst G, et al. Repeated maximal-intensity hypoxic exercise superimposed to hypoxic residence boosts skeletal muscle transcriptional response in elite team-sport athletes. Acta Physiol (Oxf). 2017. doi:10.1111/apha.12851 **(Epub ahead of print)**.
124. Scott BR, Slattery KM, Sculley DV, Dascombe BJ. Hypoxia and resistance exercise: a comparison of localized and systemic methods. Sports Med. 2014;44(8):1037–54.
125. Scott BR, Slattery KM, Dascombe BJ. Intermittent hypoxic resistance training: is metabolic stress the key moderator? Med Hypotheses. 2015;84(2):145–9.
126. Manimmanakorn A, Manimmanakorn N, Taylor R, et al. Effects of resistance training combined with vascular occlusion or hypoxia on neuromuscular function in athletes. Eur J Appl Physiol. 2013;113(7):1767–74.
127. Scott BR, Loenneke JP, Slattery KM, Dascombe BJ. Exercise with blood flow restriction: an updated evidence-based approach for enhanced muscular development. Sports Med. 2015;45(3):313–25.
128. Abe T, Yasuda T, Midorikawa T, et al. Skeletal muscle size and circulating IGF-1 are increased after two weeks of twice daily KAATSU resistance training. Int J Kaatsu Tran Res. 2005;1:6–12.
129. Cook CJ, Kilduff LP, Beaven CM. Improving strength and power in trained athletes with 3 weeks of occlusion training. Int J Sports Physiol Perform. 2014;9(1):166–72.
130. Taylor CW, Ingham SA, Ferguson RA. Acute and chronic effect of sprint interval training combined with postexercise blood-flow restriction in trained individuals. Exp Physiol. 2016;101(1):143–54.

131. Incognito AV, Burr JF, Millar PJ. The effects of ischemic preconditioning on human exercise performance. Sports Med. 2016;46(4):531–44.
132. Kraus AS, Pasha EP, Machin DR, et al. Bilateral upper limb remote ischemic preconditioning improves anaerobic power. Open Sports Med J. 2015;9:1–6.
133. Patterson SD, Bezodis NE, Glaister M, Pattison JR. The effect of ischemic preconditioning on repeated sprint cycling performance. Med Sci Sports Exerc. 2015;47(8):1652–8.
134. Gibson N, Mahony B, Tracey C, et al. Effect of ischemic preconditioning on repeated sprint ability in team sport athletes. J Sports Sci. 2015;33(11):1182–8.
135. Lalonde F, Curnier DY. Can anaerobic performance be improved by remote ischemic preconditioning? J Strength Cond Res. 2015;29(1):80–5.
136. Paixao RC, da Mota GR, Marocolo M. Acute effect of ischemic preconditioning is detrimental to anaerobic performance in cyclists. Int J Sports Med. 2014;35(11):912–5.
137. Jean-St-Michel E, Manlhiot C, Li J, et al. Remote preconditioning improves maximal performance in highly trained athletes. Med Sci Sports Exerc. 2011;43(7):1280–6.
138. Ferreira TN, Sabino-Carvalho JL, Lopes TR, et al. Ischemic preconditioning and repeated sprint swimming: a placebo and nocebo study. Med Sci Sports Exerc. 2016;48(10):1967–75.
139. Gibson N, White J, Neish M, Murray A. Effect of ischemic preconditioning on land-based sprinting in team-sport athletes. Int J Sports Physiol Perform. 2013;8(6):671–6.
140. Tomlin DL, Wenger HA. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. Sports Med. 2001;31(1):1–11.
141. Chapman RF, Emery M, Stager JM. Degree of arterial desaturation in normoxia influences VO_{2max} decline in mild hypoxia. Med Sci Sports Exerc. 1999;31(5):658–63.
142. Dempsey JA, Wagner PD. Exercise-induced arterial hypoxemia. J Appl Physiol. 1999;87(6):1997–2006.
143. Morin JB, Samozino P. Interpreting power-force-velocity profiles for individualized and specific training. Int J Sports Physiol Perform. 2016;11(2):267–72.
144. Billaut F, Gore CJ, Aughey RJ. Enhancing team-sport athlete performance: is altitude training relevant? Sports Med. 2012;42(9):751–67.
145. Millet GP, Faiss R. Hypoxic conditions and exercise-to-rest ratio are likely paramount. Sports Med. 2012;42(12):1081–3 **(author reply 3–5)**.
146. Brosnan MJ, Martin DT, Hahn AG, et al. Impaired interval exercise responses in elite female cyclists at moderate simulated altitude. J Appl Physiol. 2000;89(5):1819–24.
147. Millet GP, Faiss R, Brocherie F, Girard O. Hypoxic training and team sports: a challenge to traditional methods? Br J Sports Med. 2013;47(Suppl. 1):i6–7.
148. Dill DB, Adams WC. Maximal oxygen uptake at sea level and at 3,090-m altitude in high school champion runners. J Appl Physiol. 1971;30(6):854–9.
149. Roskamm H, Landry F, Samek L, et al. Effects of a standardized ergometer training program at three different altitudes. J Appl Physiol. 1969;27(6):840–7.
150. Levine BD, Stray-Gundersen J. "Living high-training low": effect of moderate-altitude acclimatization with low-altitude training on performance. J Appl Physiol. 1997;83(1):102–12.
151. Friedmann B, Kinscherf R, Borisch S, et al. Effects of low-resistance/high-repetition strength training in hypoxia on muscle structure and gene expression. Pflugers Arch. 2003;446(6):742–51.
152. Rodriguez FA, Iglesias X, Feriche B, et al. Altitude training in elite swimmers for sea level performance (Altitude Project). Med Sci Sports Exerc. 2015;47(9):1965–78.