Chapter 8
Altitude

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The Physiology

Physiological Response to Altitude Stress

Barometric pressure declines with ascent to altitude, and the physical features and subsequent physiological effects that accompany the decline in pressure can have a profound influence on physical performance. Because the percentages of the individual gasses in air remain constant and are independent of altitude, with a given change in the barometric pressure, the inspired partial pressure of oxygen (PIO_2) will change in a proportional manner. The density of air is also affected by changes in pressure and will decrease with the degree of hypobaria (i.e., low ambient air pressure) of altitude. With regard to the specific issue of altitude and heat-related factors, temperature and humidity are both reduced in a hypobaric environment, relative to what they would otherwise be in a normobaric environment. Ultimately, exercise performance at altitude will be affected primarily by the influence of three factors [1] described in Fig. 8.1.

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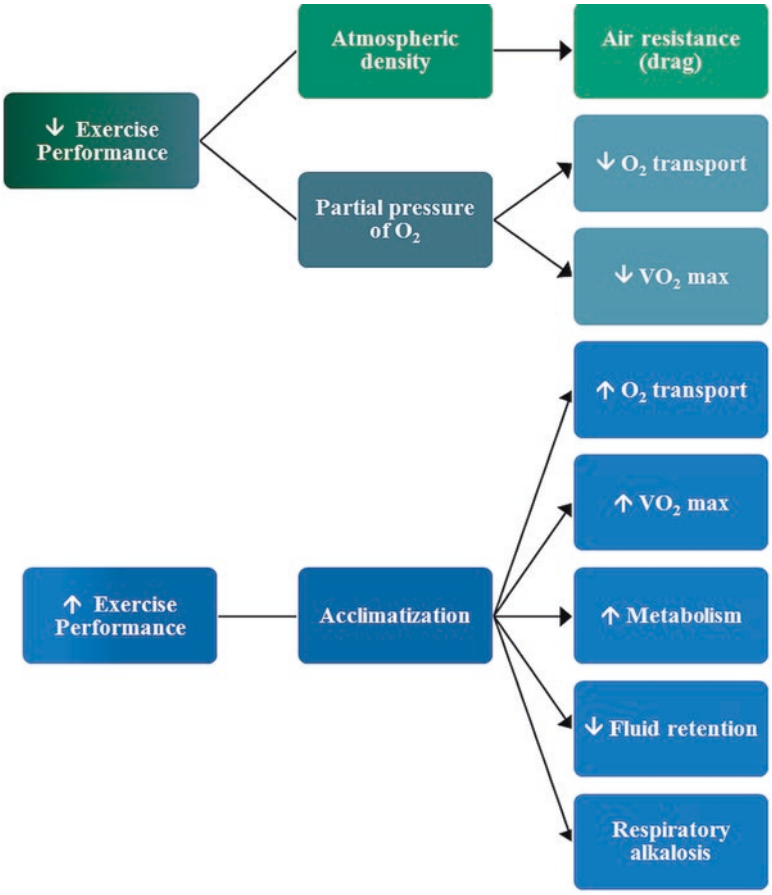

Fig. 8.1 Physiological factors influencing exercise performance at altitude

1. The density of the atmosphere and the resultant effect on air resistance, or drag.
2. The PIO_2 and the resultant effect on oxygen transport and uptake.
3. The process of acclimatization, affecting oxygen transport, metabolism, fluid balance, and acid-base balance.

This chapter will focus on the combined effects of altitude and heat on exercise performance. Therefore, the final two of the above listed factors will be of primary importance.

With acute exposure to altitude, the decline in PIO_2 is translated down each step of the oxygen cascade—that is, altitude exposure results in declines in PO_2 in the alveoli, the arteries, and the capillaries, ultimately impairing oxygen diffusion to the mitochondria in the peripheral tissues. Because of the sigmoidal shape of the oxyhemoglobin dissociation curve, relatively small drops in arterial PO_2 have little effect on arterial oxyhemoglobin saturation (SaO_2) or arterial oxygen content.

Therefore, untrained individuals with normal pulmonary function have the capacity to maintain oxygen delivery and aerobic exercise performance, near to what they would otherwise experience at sea level, at altitudes less than ~1000 m [2]. However, highly trained endurance athletes have been known for some time to be "handicapped to an unusual extent" at altitude [3], and the pulmonary gas exchange limitations common in this population during heavy exercise [4] result in a near-linear fall in maximal oxygen uptake (VO_2max) with ascent from sea level [5]. Contrary to the notion of a "threshold altitude" for aerobic impairment, a statistically significant decline in VO_2max is seen in elite endurance athletes at altitudes as low as 580 m [6]. This is just one of many examples in the literature [7–10] that identifies the ability to maintain delivery oxygen to the periphery as one of paramount importance for endurance exercise capacity at altitude. However, even within the highly trained population, there is substantial individual variability in the response of physiological systems and exercise performance at altitude. For example, distance runners display significant individual variability in the hematological response [11–13], reduction in VO_2max [8], and worsening of exercise performance [9, 13] with acute altitude exposure. With chronic altitude exposure, trained endurance athletes also show substantial variability in the acclimatization response [11], which may be a function of genetic predisposition [14, 15] or the magnitude of the hypoxic stress or living altitude [16, 17]. Ultimately, it is important to keep in mind that the mechanisms by which altitude affects maximal oxygen uptake and exercise performance are *multifactorial* and *variable*.

With arrival at altitude, there are a number of physiological acclimatization responses that take place, all with the goal of trying to maintain tissue oxygenation despite a reduced atmospheric pressure. It is important to note that the severity of these acclimatization responses are often dependent on the nature of specific physiological variable in question and the magnitude of the altitude achieved. To help standardize discussion around altitude nomenclature and the resulting physiological responses, a congress [18] standardized altitude classifications as: near sea level (0–500 m), low altitude (>500–2000 m), moderate altitude (>2000–3000 m), high altitude (>3000–5500 m), and extreme altitude (>5500 m) (Fig. 8.2). Of the multiple physiological systems affected by altitude, the time course of acclimatization responses can range from as short as a few seconds, to time periods covering hours, days, weeks, years, or even generations [1]. For simplicity, and for the purposes of this chapter focusing on altitude and heat, we will confine our review to the physiological responses to terrestrial altitude and the hypobaric hypoxia that exists in that environment.

Perhaps the most immediate response with acute exposure to altitude—one that is typically perceptible by the athlete—is an increase in ventilation, both at rest and during submaximal exercise. Peripheral chemoreceptors, located in the carotid arteries and aortic arch, are sensitive to a drop in the partial pressure of oxygen in the arterial blood. These chemoreceptors respond by stimulating the inspiratory center in the medulla to increase ventilation, and this response helps to defend alveolar PO_2 and SaO_2 with the decline in atmospheric PO_2 at altitude [19]. The sensitivity of the peripheral chemoreceptors is highly individualistic. Interestingly,

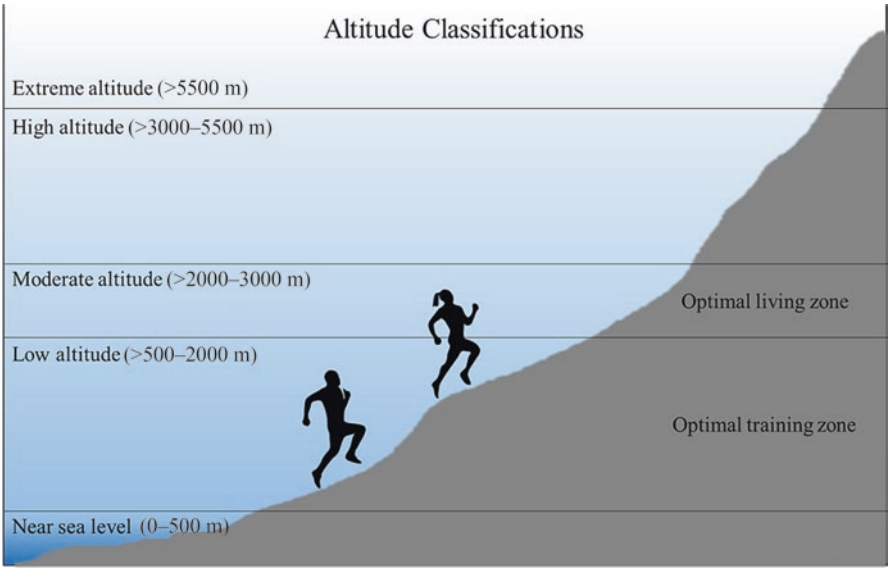

Fig. 8.2 Classifications of altitude exposure as defined in Bärtsch, Dvorak, and Saltin (2008) [18]

endurance trained athletes tend to have a more blunted ventilatory response to hypoxia than lesser trained individuals [20]; however, the sensitivity of the peripheral chemoreceptors to hypoxia appears to be based primarily on hereditary factors [21] and does not appear to be influenced by training [22]. Chronic exposure of 15 days or longer results in an increase in the hypoxic ventilatory response, but this chemoreceptor gain gradually reverts to baseline after return to sea level [23].

The increase in ventilation at altitude, both at rest and during exercise, may help to defend SaO_2 in the face of a declining PIO_2. However, this increase in ventilation is not without consequences. One is the actual cost of the respiratory effort, both in metabolic terms and perceptual terms. As ventilation increases, the work of breathing increases exponentially [24], which requires a larger fraction of the cardiac output to be delivered to the respiratory musculature. At very heavy work rates where respiratory muscle work nears maximal levels, a sympathetically mediated "metaboreflex" causes vasoconstriction in locomotor muscles [25]. While this response helps to ensure adequate respiratory muscle blood flow and oxygenation [26], high levels of respiratory muscle work has been shown to impair exercise performance [27]. In fact, in cohorts of elite distance runners, 27–37% of the increase in VO_2max observed after 4 weeks of altitude training is estimated to go towards simply fueling the increased metabolic needs of the respiratory musculature, rather than the locomotor muscles [28]. Additionally, often an athlete's internal feedback cues regarding sensation of effort during exercise are closely tied to the discomfort of breathing or dyspnea [29]. When athletes complete high intensity exercise at altitude, data suggest that perception of ventilatory effort may play a significant role in workload selection [30], which may ultimately affect the ability to train at altitude.

Fluid Balance

Perhaps one of the most significant consequences of stimulation of the peripheral chemoreceptors with acute altitude exposure—at least where thermoregulatory capacity is concerned—is a decline in plasma volume and total body water. To a small extent, the increase in ventilation (in the typically dry air of altitude) results in a greater insensible respiratory water loss over time than at sea level [31]. At high and severe altitudes, a reduction in fluid intake [32] and anorexia [33] also contribute incrementally to the altitude-mediated loss in total body water. However, by far the largest portion of the decline in total body water and plasma volume at altitude is due to actions taking place at the renal level.

Hypoxia-induced respiratory alkalosis results in a renal excretion of bicarbonate, which is typically complete after 24 h of exposure to mild and moderate altitudes [34]. Alkalosis, independent of hypoxia, results in an ~15% reduction in plasma volume, due to both the compensatory excretion of bicarbonate [35, 36] and an increase in sodium excretion [37]. Alkalosis also causes a shift in fluid to the extracellular space, likely due to increased capillary hydrostatic pressure [36]. Interestingly, hypoxia itself appears to have a direct stimulating effect on diuresis. While the complete mechanism behind a "hypoxic diuretic response" or HDR is still being elucidated, the HDR begins within hours of exposure to altitude and appears to be biphasic [38]. The early phase involves diuresis without natriuresis [39], possibly due to reduced urine concentrating ability [40] or a decrease in circulating anti-diuretic hormone (ADH) [41]. This early phase is followed by a multifactorial response, resulting from downstream effects of peripheral chemoreceptor stimulation (such as increased atrial natriuretic peptide, increased epinephrine, decreased circulating ADH, and decreased kidney sensitivity to ADH), as well as direct effects of kidney hypoxia (such as increased endothelin-I, increased adrenomedulin, and decreased renin and aldosterone) [38]. Each of these factors leads to inhibition of renal sodium reabsorption and a substantial change in blood buffering capacity. However, what happens to buffering capacity in the muscle is less clear, with some studies reporting a decrease in muscle buffering capacity [42] and others reporting an increase in muscle buffering capacity with altitude acclimatization [43, 44].

The diuresis with acute altitude exposure results in an increase in hemoglobin concentration, hematocrit, and blood viscosity, even before erythropoiesis increases red cell mass. While this short-term adaptation does help to defend arterial oxygen content at altitude, most individuals who train chronically at altitude do so for the benefit of increased total hemoglobin mass [4, 45, 46] Within minutes of exposure to hypoxia or arrival at altitude, hypoxia-inducible factor 1-alpha (HIF-1a) is expressed [47, 48]. Among its many effects, one is to upregulate production of erythropoietin (EPO) in interstitial cells in the peritubular capillary bed of the renal cortex [49], which results in survival and proliferation of erythroid precursors from stem cell to reticulocyte [50]. Typically, with chronic altitude exposure, EPO levels peak after approximately 24–48 h, then gradually decline reaching baseline levels normally observed at sea level after ~14–28 days, depending on individual variation

[11] or altitude of residence [17]. Assuming factors such as adequate iron stores are present prior to departure for altitude and dietary iron is supplemented while at altitude [51, 52], total hemoglobin mass increases on average ~ 1% per week over 3–4 weeks at moderate altitudes [52, 53].

The Evidence

There is some evidence suggesting a potential cross-acclimation effect between different environmental conditions. In fact, some studies have shown that heat acclimation can improve performance in cooler environments [54, 55]. Moreover, some research suggests that altitude acclimatization can be used as an ergogenic aid to improve sea-level performance [56, 57]. This means that certain adaptations to a particular environmental stressor can trigger responses that can enhance tolerance in another environmental stressor, as long as they share a common pathway for adaptation.

Although there have been some studies that investigated the use of heat acclimation as a tool to improve tolerance to hypoxia [58, 59], there has not been any research aimed at understanding the effects of altitude acclimatization on performance in hot environmental conditions. There are some benefits, as described previously, to altitude acclimation on exercise in thermoneutral, sea-level environments. On the other hand, some adaptations to altitude acclimatization may be detrimental to exercise in the heat, such as decreased cardiac output and skeletal muscle blood flow, secondary to a decrease in plasma volume.

Thermoregulation and Thermotolerance

Altitude acclimatization can have an effect on thermoregulation, which can certainly play a major role when exercising in the heat. As discussed previously, there is an increased hemoglobin mass that results from altitude acclimatization [52, 53]. Consequently, the increase in arterial oxygen content may allow a greater fraction of cardiac output to be directed to the cutaneous circulation because the oxygen transport requirements for a given level of submaximal exercise would be accomplished with a lower skeletal muscle perfusion [60]. Thermoregulatory responses via enhanced sweating may also be influenced by the increase in hemoglobin mass, which is observed after altitude acclimatization. One study suggested that increasing hematocrit (by 400 ml infusion of whole blood) resulted in an enhanced thermoregulatory response, which was due to enhanced sweating that decreased core temperature and reduced the skin blood flow response [61]. Finally, the study illustrated that exposures to a hypobaric hypoxic and warm environment (i.e., ten sessions over a 2-week period) enhanced the thermoregulatory response,

indicated by an increase in the forearm vascular conductance (FVC) in response to changes in esophageal temperature [62]. However, this FVC response could be an adaptation from the atmospheric pressure or temperature during the intervention exposures. Finally, it's important to keep in mind that the loss in plasma volume that accompanies altitude exposure [63, 64] may also impair the ability to thermoregulate by attenuating skin blood flow responses, particularly when exercising in hot environments.

Heat Shock Proteins

Heat shock proteins (HSPs), particularly HSP-72 and HSP-90, play key regulatory roles in protein transport across cell membranes, refolding denatured proteins, and preventing cell death in response to stressors [65]. The increased in HSP expression have been associated with enhanced cytoprotection, such that cells survive longer when exposed to heat stress [66]. There is a potential for altitude acclimatization and cross-tolerance to hot environments via enhanced HSP expression from studies done in humans and animal models [67, 68]. Taylor et al. [68] reported that 10 days of intermittent hypoxic exposure (75 min at ~3000 m of altitude) increased basal levels of HSP-72 in cells of eight healthy males. Likewise, a study done with animals (yak and rabbits) indicated that an enhanced HSP response to hypoxia may occur in altitude-acclimatized mammal [67].

The enhanced HSP-90 expression has been linked, at least in part, to the sodium-reabsorbing actions of aldosterone in the kidneys and sweat glands [69] that may lead to plasma volume expansion and reduced sodium content in sweat. HSP-90 regulates steroid hormone receptors and could therefore affect mineralcorticoid receptors in the sweat glands and kidneys to reduce sodium losses, leading to more dilute sweat and plasma volume expansion [65]. While there are no studies that firmly establish the relationship between HSP expression and systemic altitude acclimatization adaptations, these thermoregulatory adaptations via increased HSP-mediated pathways can potentially enhance exercise performance in hot environments.

Hypoxia-Inducible Factors

Altitude acclimatization increases hypoxia-inducible factors HIF-1α and HIF-1β. The enhanced HIF-1 pathways in various tissues in response to altitude exposures may initiate several of the altitude acclimatization responses such as an increased ventilatory response [70], increased erythrocyte production [48], enhanced angiogenesis via vascular endothelial growth factor (VEGF) [71], increased glycolytic enzyme expression [72], and enhanced vasodilatory mechanisms via inducible nitric oxide synthase (iNOS) to improve tissue perfusion [73]. Although the

relationship between the HIF-1 pathway and systemic responses in humans during exercise heat stress has not been explored, there is a potential for HIF-1 mediated changes to modulate some of the heat acclimation responses. HIF-1 induces angiogenesis (via VEGF) [71] and vasodilation (via iNOS) [73], which could increase maximal skin blood flow and sweating. These enhanced thermoregulatory responses can improve performance in the heat. Likewise, the HIF-1 mediated increased capillarization, glycolytic enzyme activity, and altered skeletal muscle metabolic rate could potentially be linked to the increased exercise performance and lactate threshold seen after heat acclimation [55].

HSPs and HIF-1 Cross-Regulation Pathways

There is evidence suggesting that HIF-1 can directly upregulate heat shock factor during hypoxia exposures, which leads to a further up-regulation of HSPs [74]. The cross-regulation between the HIF-1 and HSP pathways is perhaps the strongest piece of evidence that may suggest a potential for some physiological and cellular responses to chronic hypoxia that may carry over to improve tolerance and performance in hot environmental conditions. The cross-tolerance between hypoxic and heat acclimation via HIF-1 and HSP interactions have resulted in cytoprotective adaptations that can potentially lead to enhanced performance in hot environmental conditions [65].

Fluid Balance

Perhaps, one of the biggest challenges of the use of altitude acclimatization as a cross-tolerance model to enhance performance in the heat is the loss of plasma volume and total body water. This loss of plasma volume can have negative effects on overall cardiac function (leading to reduced muscle blood flow), as well as impaired thermoregulation (potential lower skin blood flow) during exercise heat stress. The fluid volume dynamics may depend on the altitude and duration of the acclimatization [63]. This loss in plasma volume takes place within 24 h of the hypoxic stimulus [64]. Although hemoglobin concentration and hematocrit increase, the total number or circulating red blood cells does not increase. In fact, a real increase in circulating number of red blood cells does not occur until 3–4 days. The loss of plasma volume can have a significant impact on cardiovascular function during exercise in hot environments. With a loss of plasma volume from altitude acclimatization, exercise in hot environmental conditions can further challenge the cardiovascular system because of the enhanced thermoregulatory demands for skin blood flow coupled with hyperthermia and dehydration, which may impair the adequate delivery of oxygen to working muscles [75].

Cardiovascular Function

It is clear that exercise in the heat imposes a greater cardiovascular and thermoregulatory strain compared with the same absolute exercise intensity in temperate environments. As previously mentioned, a decrease in plasma volume from altitude acclimatization could further increase this strain in hot environmental conditions. The cardiovascular adaptations from altitude acclimatization may vary with the degree and duration of the altitude exposure. Although a study reported no differences in submaximal and maximal cardiac output following 28 days of altitude acclimatization at moderate altitudes (2500 m) [56, 57], other studies have shown that following acclimatization at high altitude (3000–4500 m) there is a decline in cardiac output at rest and during submaximal and maximal exercise, secondary to a reduction in stroke volume [76–78]. Although these responses are multifactorial and variable, the reduction in stroke volume could be largely attributed to the reduction in plasma volume, although a study also suggested a diminished myocardial contractility [79]. The time course for recovery of stroke volume and cardiac output upon the return to sea level is not well understood although it seems that the restoration is not immediate [77]. The decreased cardiac output, stroke volume, and muscle blood flow can be accompanied by a higher arteriovenous oxygen difference (a-vDO_2), thus maintaining VO_2 despite the lower oxygen delivery [57]. Although these cardiovascular adaptations could be beneficial to exercise at altitude (reduced work of the heart, given the increase in blood viscosity due to elevated red blood cells and reduced plasma volume), it is not clear that they would transfer to exercise in hot environments.

Oxygen Transport and Delivery

As previously described, within 24 h of altitude exposure there is a decrease in plasma volume, which results in an increase in hemoglobin concentration and hematocrit [64]. The increase in total number of circulating red blood cells occurs within 3–4 days of altitude exposure. Hemoconcentration due to altitude exposure leads to an increase in arterial oxygen carrying capacity, so more oxygen can be delivered to the tissues at a given level of cardiac output. Although hypothetical, this adaptation may allow a greater fraction of cardiac output to be directed to the skin for thermoregulatory purposes during exercise in the heat.

Within a few hours of altitude exposure there is an increase in the concentration of 2,3-diphosphoglycerate (2,3-DPG), secondary to a rise in intracellular pH. The concomitant rightward shift in the oxygen-hemoglobin dissociation curve enhances oxygen release at the tissue level. This adaptation is observed after 3 days of living in a simulated altitude environment and training near sea level. However, 2,3-DPG returned to baseline values 1 day after elimination of the hypoxic stimulus [80]. Therefore, any potential for enhanced exercise performance in the heat is unclear.

Muscle Function

There are skeletal muscle adaptations following hypoxia that may affect muscle function during exercise heat stress. These include an increase in myoglobin content, a reduced diffusion distance between skeletal muscle fibers and capillaries (due to possible loss in muscle mass), decreased lactate production and enhanced lactate clearance, improved mechanical efficiency, and enhanced skeletal muscle buffering capacity [81]. The altitude acclimatization effects of skeletal muscle and its adaptations on exercise heat stress remains to be elucidated.

Performance

There have not been any studies that investigated the use of altitude acclimatization as a cross-tolerance model to improve exercise performance in the heat. Given the wide variety of adaptations to altitude acclimatization, some may be beneficial when exercising in the heat, whereas others may not. Potential benefits of altitude acclimatization include increases in blood hemoglobin concentration, increased hematocrit, elevated buffering capacity, and improvements in the structural and biochemical properties in skeletal muscle. Some other adaptations that may negatively impact exercise in the heat include decreased cardiac output and skeletal muscle blood flow, secondary to a decrease in plasma volume. Clearly, studies are warranted to elucidate the potential use of altitude acclimation as an ergogenic tool to improve exercise in hot environmental conditions.

The Application

The response of an athlete to altitude training is variable in both how acclimatization is achieved, as well as the impact on athletic performance. The extent of performance change and altitude adaptation is ultimately individualistic for each athlete; thus, training decisions may vary even within a team. To gain these adaptations as a mechanism to maximize performance and safety, altitude training models have been developed and researched with a wide variety of athletes (i.e., runners, cyclists, team-sport athletes.)

Other considerations for altitude training, such as time required at specific altitude, hydration planning, and timing of return for event performance must be considered to gain maximum benefit. The following discussion of these aspects will provide some of the many considerations for an athlete aiming to enhance both performance and safety while exercising in the heat. The information provided in Table 8.1 may be useful for maximizing performance and safety while exercising in the heat via altitude training.

Table 8.1 Tips for successful implementation of altitude training for heat safety

1. Utilize the live high, train-low model
2. Perform easy-moderate intensity exercise at moderate altitude
3. Perform moderate-high intensity exercise at low altitude
4. Heat acclimatize during training, either in a naturally warm environment or a simulated warm environment
5. If heat acclimatization is not possible during training, leave altitude with enough time to heat acclimatize closer to sea level
6. Hydrate appropriately before, during, and after exercise
7. Time the return to sea level either in the first few days, or more than 10 days prior to competition

Training Models

The three most common paradigms for altitude training include altitude exposure in daily living, exercise, or in combination. The live high, train-low (LHTL) model is the most commonly adopted altitude training model worldwide. A review of the LHTL model by Levine and Stray-Gundersen in 2006 describes two essential features for success with this model. A large number of these studies suffer from experimental design issues, most of which are in the area of adequate pre-altitude/hypoxia controls, such as group training effects, training camp effects, training levels prior to the altitude camp, and iron storage levels and supplementation [82]. Without these controls, it becomes difficult to partition out the variance in outcomes that is truly due to the effect of altitude training or hypoxic exposure, and what is due to some other factor.

One central issue to the concept of altitude training for sea-level performance is the ultimate effect of chronic training in hypoxia. For years, coaches, athletes, and many sport scientists have operated under the assumption that training in a hypoxic environment will augment the training response, particularly in muscle structure, vascular development, or metabolic pathways. Part of this belief stemmed from early reports (i.e., 1962) on miners who were permanent residents at high altitude had significantly greater cytochrome c reductase activity and myoglobin content compared to lowland residents [83]. Indeed, in animal models, hypoxic training has been shown to increase Type IIa fibers, as well as increase citrate synthase, hexokinase, and fatty acid β oxidation enzymatic activity [84]. In humans, a number of studies have shown similar improvements in Krebs cycle enzymatic activity with hypoxic training [85], and mitochondrial and capillary densities have been reported to be significantly increased after chronic training in hypoxia [86]. However, in most human studies that have shown significant skeletal muscle changes with hypoxic exercise, most have utilized unique protocols using a smaller muscle mass or training one leg in hypoxia and the other leg in normoxia, and often the same absolute workloads are used in hypoxia [1, 87]. During whole body dynamic exercise, like endurance running, training at altitude has been shown to limit exercise intensity and oxygen flux, which may actually promote deconditioning over time

[88–91]. Current evidence suggests that HIF-1a signaling in skeletal muscle displays only a modest response to hypoxia [92], and structural changes within skeletal muscle—over and above those that occur with training in normoxia—are less likely to occur when hypoxia is added to chronic exercise [87]. Therefore, athletes gain hematological benefits from chronic altitude exposure, but minimizing negative effects from hypoxic training is questionable.

The concept of LHTL altitude training, as proposed by Levine and Stray-Gundersen [57] theorizes that by chronic living at a moderate altitude, a significant increase in total hemoglobin mass will significantly increase maximal oxygen uptake. Moreover, by completing training at a lower altitude, training speeds and muscle capillary oxygen flux will be maintained near what they are at sea level, preserving the positive training response in the periphery to exercise [91].

In an elegant, well-controlled study, athletes who lived high and trained low daily for 4 weeks showed an improvement in VO_2max, maximal steady state, and 5000 m run time at sea level, where groups who lived and trained at moderate altitude (and a sea-level control), showed no change in 5000 m run time [57]. Subsequent iterations to the LHTL model have shown that similar performance improvements can be seen with going to low altitude only for high intensity workouts (i.e., workouts that are performed at a workload faster than the maximal steady state) [91, 93]. This finding makes completing the LHTL model of altitude training much more logistically convenient, as travel to low altitude only needs to be undertaken two to three times per week. Still, there are only a handful of locations where it is convenient to live above 2000 m, but be able to travel to near sea level in a short period of time. Since these initial studies, a number of laboratories from multiple countries have independently shown the advantage of the LHTL altitude training model, compared to traditional "live high—train high" altitude training (LHTH) [94–99]. A recent meta-analysis using terrestrial altitude studies has shown increases in maximal power of 4.0 ± 3.7% in elite athletes and 4.2 ± 2.9% in sub-elite athletes completing LHTL protocols, but a maximal power improvement only 1.6 ± 2.7% in elites and 0.9 ± 2.9% in sub-elites completing LHTH protocols [100].

Evidence on the live low, train high (LLTH) model describes minimal benefit to athletic performance. The LLTH model uses intermittent exposure to hypoxic environments, typically using artificial hypoxia, and rarely daily ascent. Either all or some of the athlete's training takes place at moderate or high altitude. A common assumption is that increased physiological stress from training at altitude would elicit a greater adaptation, thereby enhancing athletic performance. However, the literature on the LLTH model does not support its use for improving endurance performance [101]. Theories to describe this include: (1) artificially induced hypoxia is likely not as potent as a field environment; (2) brief exposure time does not meet the threshold for adaptation; (3) overall exercise intensity is lower at altitude, thus creating less of a training stimulus. The artificial intermittent exposure may be a primary factor in the limited exercise enhancement that comes from these modalities. As described previously in this chapter, the increased physiological and perceptual strain results in the inability to sustain high intensity exercise. Thus, if exercise intensity cannot be maintained, some detraining may occur, although the body may

overcome the short-term environmental stress without long-term adaptation in the LLTH model. In addition, LLTH models are typically carried out in laboratory settings, which may be impractical for athletes not near an environmental laboratory. Either all or some of the athlete's training may occur at altitude, but studies of both full and partial training at altitude have shown inconsistent performance benefit [101, 102].

Timing

Once a training model is selected, several other considerations for exercise training utilizing altitude must be made. Timing of the program, including time living at altitude, as well as return to sea level prior to competition may impact performance in the heat. The long-duration exposure of the LHTL model can be as brief as 4 weeks, training at low altitude only two to three times per week, can be used to gain performance benefit [57]. Timing of return to sea level should be planned to benefit from both improved performance and enhanced safety while exercising in the heat. Anecdotal evidence suggests that some period of maximum benefit for performance exists upon return from training or living at altitude for an event occurring at sea level. A review by Chapman et al. [103] used anecdotal evidence from elite endurance coaches that use altitude training regularly to describe this period of time. Generally, three periods of time were described to impact performance: first 48 h, 3–14 days, and 14 or more days. Athletes were permitted to perform either in the first 48 h, or 14 or more days following return from moderate altitude [103]. These periods of time were viewed as permissible due to the perceived performance benefits of the altitude training, but providing additional time to adapt to the sea-level environment. Also, some hematologic adaptations may remain elevated for 2–4 weeks post-return [4]. This may be of benefit to athletes aiming to improve performance in warm environments because heat acclimatization and proper hydration may be established in this time at sea level.

Fluid Balance

Fluid balance is commonly compromised during training at altitude due to the mechanisms described previously in this chapter. Maintenance of adequate hydration should be considered during training at moderate and low altitude. Hypoxia and hypohydration have been shown to independently cause reduction in aerobic time-trial performance, with an even greater reduction in performance in combination [104]. Thus, hypohydration may further limit altitude exercise performance described earlier in this chapter [88]. Rudimentary hydration indicators may be used over time to maximize safety and performance both at altitude and sea level. This may include estimation of sweat rate at a given work intensity, analysis of urine

specific gravity, volume, and frequency, and body mass change over time. Exercise at altitude, whether in a warm or cold environment, results in a similar sweat rate as the same exercise intensity, with additional respiratory water loss [104, 105]. Additional information about hydration assessment and fluid replacement can be found in Chap. 5.

To our knowledge, only one study has been done to specifically elucidate the impact of altitude acclimatization and training on exercise performance in a hot (presumably sea level) environment. A comparison of aerobic training in either hot or hot and hypoxic environments revealed that a performance benefit was only evident from training in a hot environment, without changes in hemoglobin mass, or perceptual measures of intensity [106]. Although theoretical links in physiological crosstalk theory are compelling, recommendations for maximal training benefit should be taken as such, theoretical. Based on expert opinion and physiological rationale, living or training at altitude may provide some benefit to thermoregulation and exercise performance, if fluid balance is well controlled.

Case Example *Perhaps one of the most well-known and commonly cited examples of altitude training for performance in the heat is from Deena Kastor (D.K.) in the 2004 Olympic marathon in Athens, Greece. Joe Virgil, a coach for D.K., described her training leading up to the 2004 marathon, including the use of altitude training to enhance physiological function in the hot, humid environment in Athens* [107].

Training for the event included typical race preparation training, and incorporation of a LHTH model, with only a small portion of training occurring at low or sea-level altitude. D.K. moved to Mammoth Lakes, CA (2400–3100 m) to set up training camp, and to focus the training program on altitude adaptation. Over a period of years, D.K. performed the majority of training at moderate altitude, and incorporated high altitude (~3000 m) training at frequent intervals. To practice the race course, D.K. and coaches utilized a course in Mammoth Lakes that was nearly identical in terrain to the Athens course, but at moderate altitude (2100–2400 m) instead.

Mammoth Lakes, CA is not a warm or humid environment like Athens, thus D.K. used other means to prepare for the heat in addition to her aerobic training. They anticipated that D.K. would need to prepare by acclimating to the heat, and hydrating appropriately. To provide some heat stimulus without a naturally warm environment, she created artificial warmth by wearing extra clothing during training to prevent some heat loss to the environment. To prepare for hydration maintenance in the warm environment, they utilized gut training and trained D.K. to take in fluid every 15 min to make hydration more regular and comfortable. Lastly, they anticipated that heat acclimatization would be necessary. They went to Crete, the largest Greek island, 2 weeks prior to competition to prepare. Crete provided two very necessary environmental components: altitude among several mountains and a hot-humid environment. These 2 weeks were likely enough to provide D.K. with appropriate heat acclimatization prior to the race without sacrificing her altitude adaptations.

The temperature on race day was 102 °F/38.9 °C, and 54% relative humidity at the start of the race; within the expected range. The race course had one 8-mile incline section in the second half; the peak at approximately 70 m, considered near sea level altitude. Overall, D.K. finished the race with a Bronze medal, and only 1:16 min away from her target time. The intense altitude training D.K. did leading up to the event was likely a major component to her Olympic success in 2004 [107]. *This case exemplifies the potential impact and benefit of utilizing altitude training to enhance performance in warm environments. When utilized with other elements, such as hydration and exercise heat exposure, altitude training can benefit performance in warm environments.*

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