HIGHLIGHTED TOPIC | *Hypoxia*

Timing of return from altitude training for optimal sea level performance

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Chapman RF, Laymon Stickford AS, Lundby C, Levine BD. Timing of return from altitude training for optimal sea level performance. *J Appl Physiol* 116: 837–843, 2014. First published December 12, 2013; doi:10.1152/japplphysiol.00663.2013.—While a number of published studies exist to guide endurance athletes with the best practices regarding implementation of altitude training, a key unanswered question concerns the proper timing of return to sea level prior to major competitions. Evidence reviewed here suggests that, altogether, the deacclimatization responses of hematological, ventilatory, and biomechanical factors with return to sea level likely interact to determine the best timing for competitive performance.

hematological acclimatization; ventilatory acclimatization; oxygen cost of breathing; endurance athletes; live high-train low; neocytolysis

FOR NEARLY FIFTY YEARS, endurance athletes have utilized a strategy of moderate altitude (2,000–3,000 m) living and training for the enhancement of sea level racing performance. As practical and scientific knowledge on altitude training has grown, a scientific focus in recent years has been placed on determining the best practice of altitude training for the athlete and coach. Ultimately, several research groups (8, 12, 15, 16, 30, 31, 43, 45, 46, 49) have been able to parse the acclimatization and training response into practical, scientifically based recommendations that can be used to optimize altitude training, such as how high to live, at what altitude to train, how long to stay, the importance of proper iron stores, and how intermittent hypoxic exposure may or may not give the same physiological benefits as true full-time altitude acclimatization. However, there remains a key question, so far unanswered by science, which is of extreme importance to competitive athletes and coaches: When is the best time to return to sea level prior to a major competition to optimize performance?

By no means is the issue of timing an altitude training camp prior to competition a trivial matter. Many endurance competitions, like a marathon or multistage cycling race, are in essence one-shot events, where the athlete can only effectively attempt one or perhaps two races of this nature within a calendar year. Altitude training camps require significant costs for travel and housing and often create logistical challenges with relocating for >4 wk from family, work, coaching, and medical support. In short, a mistimed return to sea level from an altitude camp could result in missing out on professional accomplishment and financial gain for the athlete. In many

ways, scientists have been almost singularly focused on the mechanisms and time course of altitude acclimatization; so much so that there is a paucity of data on the timing of deacclimatization from altitude. The mechanisms and timing of altitude deacclimatization likely do not involve a simple reversal of the well-documented altitude acclimatization response. In the absence of direct scientific data, coaching lore has become full of wide-ranging beliefs on when to return to sea level before competition; however, these beliefs are based largely on anecdotal evidence from a modest number of athlete responses or uncontrolled observations.

Our purpose here is to present select physiological and biomechanical responses associated with "reentry" to sea level, to determine the implications of timing of return from altitude training for endurance sports performance. Although there are many physiological factors involved in the response to hypoxia, we propose that the optimal time to compete after return to sea level after chronic altitude training will be influenced primarily by the interaction of three components: timing of the decay in red blood cell mass, consequences of ventilatory acclimatization, and alterations in biomechanical and neuromuscular factors associated with force production. As research efforts continue in the search to elucidate the best practices and application of altitude training for endurance performance, we believe the issue of timing of return to sea level prior to competition, largely ignored, deserves special focus.

EXISTING DATA AND COMMON BELIEFS ON TIMING OF COMPETITIONS AFTER ALTITUDE TRAINING

Best practices for achieving new limits in human athletic performance rarely begin with science, but instead are largely discovered through the experiences, trials, and errors

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of innovative coaches and athlete practitioners. One U.S. Olympic track and field coach (R. Mann, personal communication) has shared his belief with us that many altitude-based endurance athletes perform best after 10 days of sea level residence. His belief stems from practical experience of >20 yr of coaching at a U.S. university located at 2,100 m. Although anecdotal in nature, the view of this coach is, by our experience, common within the elite track and field coaching community—that it is often best to plan for a period of sea level training prior to a major competition, after returning from an altitude camp. In fact, a technical coaching publication of the International Association of Athletics Federations, the international governing body for track and field, states that, for peak sea level racing performance, "an altitude training camp should only be planned if there is enough time available for a 3 week stay, plus 12–14 days for re-acclimatization to sea level" (6). Other successful altitude training practitioners have published in applied technical journals similar recommendations (see Table 1), and, although the specific timing recommendations vary, they generally advocate ~2–3 wk of sea level training, after return from altitude training, before a prime competition (6, 9, 20, 34, 37, 47). However, these recommendations are largely based on anecdotal evidence and experience, and existing scientific data are quite limited. While there have been a number of recent studies examining performance responses directly after terrestrial live high-train low altitude training (for examples see Refs. 31, 46, and 49), or intermittent hypoxic exposure of varying daily lengths and overall duration (for examples see Refs. 8, 43, and 45), there are very few studies that examine performance serially upon return from altitude training. Most studies that assess performance typically do so prior to departure for altitude, then within a few days upon return, potentially missing out on measures within the 2- to 3-wk "prime window" indicated by coaches. With the original 1997 terrestrial live high-train low study by Levine and Stray-Gundersen (31), no significant changes were observed in postaltitude 5-kilometer performance time, measured weekly for 3 wk. However, it should be noted that it is often difficult to ensure adequate controls on serial measures of performance in the field, particularly in distance runners, because factors such as weather conditions can have a large effect on performance outcomes. Ultimately, looking at the controlled environment of the laboratory, we can ask what the potential physiological mechanisms are that would influence proper timing of competition upon return from altitude training.

DEACCLIMATIZATION RESPONSES WITH RETURN TO SEA LEVEL AFTER ALTITUDE TRAINING

Decay of hematological adaptations. Of the list of acclimatization responses that occur with chronic altitude residence, it is our belief that the altitude-mediated increase in red blood cell and/or hemoglobin mass and oxygen delivery are the most important factors behind any improvement in sea level endurance exercise performance (30, 32, 41). Data have shown that distance runners who substantially improve 5,000-m performance time after 4 wk of altitude training have: *1*) significantly larger erythropoietin levels at 48 h and 14 days after arrival at altitude and *2*) significantly larger increases in red blood cell mass from pre- to postaltitude, compared with athletes who demonstrated no improvement in performance after altitude training (16). Although it is important to note that the relationship between hemoglobin mass increase and maximal oxygen uptake ($\dot{V}O_{2max}$) increase after altitude training may not be as apparent at the individual level (42). Still, the list of studies that have examined the hematological response to acute and chronic hypoxia is quite lengthy. Surprisingly by comparison, data describing the decay and/or normalization of hematological responses with return to normoxia is notably deficient.

In early studies on the topic completed in the 1950s, separate investigations indicated that altitude-acclimatized individuals experience a significant fall in erythrocyte volume within days of return to sea level (25, 35, 36). First, Merino (35) in 1950 noted a reduction in erythrocyte volume ranging between 10% and 18% in chronic altitude residents 1 wk after arrival at sea level; however, these early data derive from a small cohort and some outcome measures require critical appraisal. A year later in 1951, Huff et al. (25) reported a mean reduction of 9% in erythrocyte volume in the first week after return to sea level. Interestingly, the Huff et al. data showed that reticulocyte count did not decline until *Days 8–10* at sea level. Therefore, the authors theorized that the reduction in erythrocyte volume must have come from some physiological mechanism causing hemolysis, and not from an immediate decline in erythropoiesis. Conventional scientific thinking of the time held that red blood cells survive for 90–20 days in the circulation, and no known mechanism could shorten this time frame (39). Perhaps because of this scientific dogma, for over 40 years, this particular hematological normalization response with altitude natives traveling to sea level was largely ignored.

The concept of selective red blood cell hemolysis was resurrected in the 1990s through the examination of the 10–15% loss of red blood cell mass that commonly affects astro-

Table 1. *Recommendations of top coaches and applied sport scientists on when to compete after return from altitude training*

Authors (Reference)	Recommended best time to compete after return from altitude training	Recommended times to avoid competition after return from altitude training
Bueno (9)	*Day 17* "prime date for best performances"	
Dick (20)	After *Day 15*	
IAAF technical publication (6)	After *Days 12–14*	
Mann (personal communication)	Within the first 48 h; after 10 days	*Days 3–9*
Popov (37)	*Days 18–21* "80% of best performances"	
Suslov (47)	*Days 3–7; Days 14–25*	*Days 8–13*

References are from applied technical journals.

nauts during spaceflight (2, 3). This physiological process, termed neocytolysis, is hallmarked by the selective hemolysis of the youngest circulating red blood cells when erythropoietin (EPO) levels fall below resting baseline levels (2). While in astronauts EPO reduction is caused by plasma volume shifts with exposure to microgravity, neocytolysis has also been demonstrated to occur in chronic altitude residents with travel to sea level. Rice et al. (40) studied a group of long-term residents of Cerro de Pasco, Peru (4,390 m) before and after descent to sea level and found a 9.6% reduction in red blood cell mass after 3–7 days of descent. Reticulocyte count did not change during the first 6 days at sea level, despite the fall in red blood cell mass, indicating normal release of erythroid precursors that were committed prior to the reduction in EPO levels with return to sea level. Therefore, similar to the early data of Huff et al. (25), the reduction in red blood cell mass in the first week at sea level must come from the selective hemolysis of mature but young red blood cells.

Specifically in endurance athletes, recent data from Prommer et al. (38) have shown that, in a group of elite Kenyan runners descending from moderate altitude (2,090 m) to near sea level (340 m), the total hemoglobin mass began to decrease after 14 days at sea level, reaching a nadir at about 30 days (Fig. 1). Again, this reduction in hemoglobin mass is much faster than the normal 90- to 120-day erythrocyte life span. The controlling factor for neocytolysis appears to be circulating EPO concentration (48). Traditionally, EPO has been thought to act solely in a proliferative nature, allowing early red blood cell progenitors to escape apoptosis, progress to committed precursors, and ultimately develop to mature red blood cells. However, recent data support the theory that EPO also plays a regulatory role in red blood cell mass control outside of the bone marrow, allowing the body to have an improved fine control mechanism in times of red blood cell excess (39). Thus,
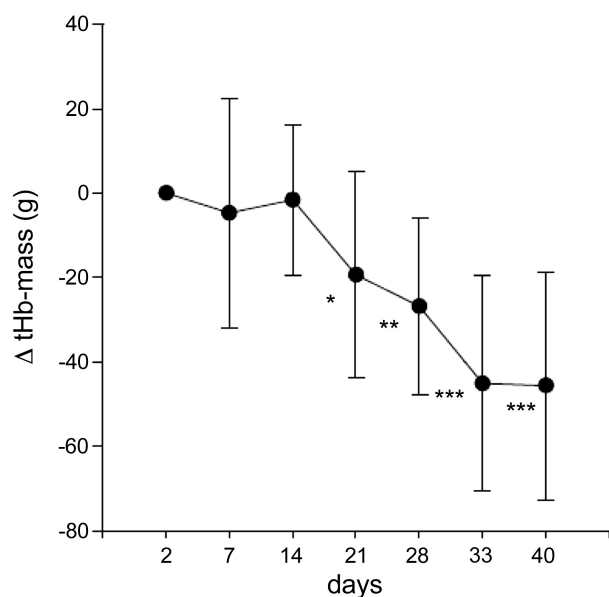
EPO plays a biological role of both positive and negative regulation of red blood cell mass.

For the endurance athlete, any loss of red blood cell mass and oxygen-carrying capacity over time at sea level would clearly have a negative impact on performance. However, most coaches recommend a sea level training period after chronic altitude training for the anecdotal positive performance benefits it appears to provide. This recommendation would appear to run counter to the physiological off-response of neocytolysis and the documented timing of red blood cell and hemoglobin mass decline over time at sea level. This contradiction begs the question, Are there other physiological off-responses with prolonged time at sea level that could be positive for performance; responses which are theoretically large enough to counteract the negative effects of a loss of red blood cell mass and oxygen-carrying capacity over time at sea level?

Off-response of ventilatory acclimatization. With chronic residence at altitude, ventilatory acclimatization results in an increase in ventilation both at rest and at all exercise workloads. This increase in ventilation is commonly viewed as a positive adaptive benefit for altitude residence, one that helps to defend alveolar oxygen partial pressure (P_{O_2}) and creates a higher pressure head for diffusion of O_2 into the arterial blood. Particularly for the large portion of elite endurance athletes who already display some degree of pulmonary gas exchange limitations at sea level (19), increasing alveolar ventilation at altitude may be the only practical strategy for mitigating the decline in arterial P_{O_2} at altitude (13).

When the athlete ultimately returns to sea level, the gain in the ventilatory response to exercise that developed with acclimatization to altitude often persists as an elevated exercise minute ventilation ($\dot{V}_E$), both at submaximal and maximal workloads (11, 18, 21, 22, 31, 46). In a group of 22 elite distance runners who completed 27 days of residence at 2,500 m following the live high-train low paradigm (46), $\dot{V}_E$ at $\dot{V}_{O_{2max}}$, measured within the first 48 h after return to sea level, was 6% higher than prealtitude camp measures. $\dot{V}_{O_{2max}}$ was significantly increased by 3% after altitude training, and a significant correlation ($r = 0.67$) existed between the increase in maximal $\dot{V}_E$ and the increase in $\dot{V}_{O_{2max}}$ after altitude training. However, despite data that indicate a reduction in dyspnea and perception of ventilatory effort during sea level exercise after return from altitude training (54), a ventilatory acclimatization-mediated increase in maximal $\dot{V}_E$ will increase the work of breathing and thus the oxygen consumed by the respiratory musculature. Therefore, the question still remains: Is the observed increase in ventilation with chronic altitude training a positive physiological adaptation which enhances sea level performance? The answer may depend on the magnitude of the metabolic cost of the added ventilation and other potential consequences of increased ventilatory work.

As ventilation increases with progressive exercise, the muscular work necessary to ventilate the lungs increases. As the exercise workload approaches $\dot{V}_{O_{2max}}$, the increase in $\dot{V}_E$ causes a disproportionate increase in both the work and oxygen cost of breathing (1, 4, 26, 33). In moderately trained individuals, the oxygen cost of breathing during maximal exercise has been shown to account for ~10% of whole body $\dot{V}_{O_2}$ (1); however, ventilatory volumes in moderately trained individuals during maximal exercise are typically lower than in elite endurance athletes (26). Additionally, within the elite endur-

Fig. 1. Values are means ± SD. Changes in absolute total hemoglobin mass (Δ tHb-mass) of Kenyan runners ($n = 10$), permanent residents of 2,090 m, over the course of a 6-wk stay near sea level (340 m). Significant differences against *Day 2* are indicated by *$P < 0.05$, **$P < 0.01$, and ***$P < 0.001$. [From Prommer et al. (38). Reprinted with permission from Wolter Kluwers Health.]

ance athlete population, exercise at high work rates often results in approaching or achieving expiratory flow limitation (14, 26), where the only strategies these flow-limited athletes can employ to increase $\dot{V}E$ is to either *1*) increase expiratory flow and pressure rates early in expiration before tidal volume reaches the maximal flow-volume envelope, or *2*) hyperinflate the lung, increasing the end-inspiratory and end-expiratory lung volumes in an attempt to move tidal volume away from the flow-limiting portion of the maximal flow-volume envelope (1, 5, 17, 26). Both strategies result in a substantial increase in the work and cost of breathing. Taken together, the higher exercise ventilatory volumes and greater percentage of expiratory flow limitation in elite endurance athletes likely results in a higher percentage of whole body $\dot{V}O_2$ going to fuel the metabolic cost of breathing—perhaps as high as 15–20% during maximal exercise (1).

For the athlete who acquires an increase in $\dot{V}O_{2max}$ after altitude training, how much of the increase in $\dot{V}O_{2max}$ goes simply to fuel the extra ventilation? Although this has not been directly measured, we can make some estimates using published data (1) and known responses of athletes returning from altitude training sojourns (46, 53). In the Stray-Gundersen et al. cohort (46), of a mean 201 ml/min increase in $\dot{V}O_{2max}$ after altitude training, an estimated 54 ml/min, or 27% of the $\dot{V}O_{2max}$ increase went to fuel the added respiratory muscle work required by a mean maximal $\dot{V}E$ increase of 9 l/min. In a recent study of seven elite distance runners (53), of a 196 ml/min mean increase in $\dot{V}O_{2max}$ after 28 days of high-low altitude training, an estimated 72 ml/min or a 37% of the whole body increase in $\dot{V}O_{2max}$ came from the increased metabolic cost of ventilation ($\dot{V}O_2RM$; Fig. 2). When utilizing the relationship between exercise $\dot{V}E$ and metabolic cost of ventilation published by Aaron et al. (1) to make these estimates, it is important to note that their athlete cohort can be classified as moderately trained ($\dot{V}O_{2max} = 51.1 \pm 2.6$ ml·kg^{-1}·min^{-1}) with a maximal $\dot{V}E$ of 152.5 ± 9.1 l/min. By comparison, the above-referenced elite athlete cohorts (46, 53) had significantly higher $\dot{V}O_{2max}$ and maximal $\dot{V}E$ responses, the latter likely

requiring an even higher percentage of whole body $\dot{V}O_2$ to fuel the respiratory musculature.

An interesting potential consequence of added respiratory muscle work with ventilatory acclimatization is the resulting competition for a limited cardiac output during maximal exercise (29). With added work of breathing during very high exercise workloads, sympathetic activation from either baroreceptor unloading or direct diaphragmatic afferent signaling diverts blood flow away from the locomotor muscles to the respiratory musculature, similar to what happens when arm exercise is added to leg exercise (44). In an elegant study by Harms et al. (23), the authors measured blood flow to the leg muscles via cold saline thermodilution in cyclists exercising at or very near $\dot{V}O_{2max}$. In trials where resistance to inspiratory flow was added, increasing the work of breathing, blood flow to the legs decreased. Similarly, when ventilatory work was unloaded by ~60% via a pulmonary assist ventilator, blood flow to the legs increased. Specific to our question, whether and how any increase in the work of breathing after altitude training affects locomotor blood flow has not been investigated. However, we do know that over time after return to sea level, the effects of ventilatory acclimatization decay, probably more rapidly than the hematological adaptation, and $\dot{V}E$ at any given workload returns to (or with increased fitness, perhaps is reduced below) prealtitude training camp levels. In theory, the reduction in ventilatory cost at any workload with ventilatory deacclimatization could have the positive effect of freeing additional cardiac output for the locomotor muscles, thereby increasing the useful locomotor $\dot{V}O_2$ and positively impacting performance (24). In 2.5 min cycling trials at $\dot{V}O_{2max}$, Harms et al. (23) found that $\dot{V}O_2$ of the legs was 80.6% of whole body $\dot{V}O_2$, but rose to 88.6% when the work of breathing was reduced to ~35% of control levels (23). Similarly, time to exhaustion at 90% of $\dot{V}O_{2max}$ increased by 14.4% when ventilatory work was decreased 37–45% from control levels (24). It is also important to note that while the decrease in leg blood flow with increased work of breathing is significant during maximal exercise, during submaximal exercise at 50% and 75% of $\dot{V}O_{2max}$, increasing the work of breathing does not cause a change in leg blood flow (50). For the altitude-trained athlete returning to sea level, this may suggest that any negative effects of ventilatory acclimatization on locomotor blood flow may be minimal in events requiring prolonged submaximal exercise; that is, athletes competing in longer distance events requiring a lower fractional utilization of $\dot{V}O_{2max}$, such as the marathon, may experience fewer negative consequences of ventilatory acclimatization and may find the ideal time to compete is within a short time window upon return from altitude (when red blood cell mass is the highest). The corollary would be that the negative consequences of a higher work of breathing postaltitude may primarily manifest itself in events approaching or exceeding $\dot{V}O_{2max}$ velocity, for example, events of 5,000 m and shorter in track and field (7).

Ultimately, in combining the hematological and ventilatory deacclimatization responses with return to sea level, several issues remain for the athlete: *1*) we do not know the exact time course of the decay in red blood cell mass and oxygen delivery, *2*) we do not know the exact time course of the decay in ventilatory acclimatization with return to sea level in athletes, and *3*) we do not know whether the magnitude of increase in ventilatory work after altitude training is large enough to

Prospective Cohort   **Retrospective Cohort Stray-Gundersen et al. (2001)**

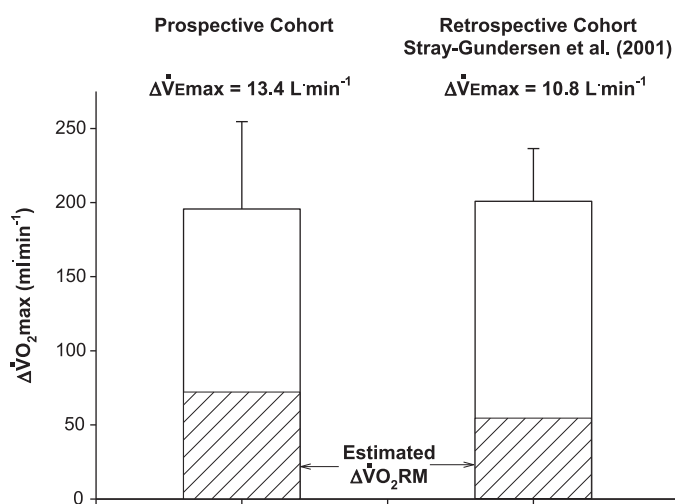$\Delta\dot{V}Emax = 13.4$ L·min^{-1} $\Delta\dot{V}Emax = 10.8$ L·min^{-1}

Fig. 2. Values are means $\pm$ SE. The change in maximal oxygen uptake ($\dot{V}O_{2max}$; open bars) and the change in estimated respiratory muscle oxygen uptake ($\dot{V}O_2$ RM; hatched bars) after return from 4 wk of live high-train low altitude training in 2 separate cohorts. $\dot{V}Emax$, maximal exercise ventilation. [From Wilhite et al. (53). Reprinted with permission from Springer.]

impair blood flow to locomotor muscles at high work rates. The interaction between these factors, and possibly others, may strongly influence the optimal time to compete upon return to sea level.

Biomechanical and neuromuscular factors. Upon return to sea level from an altitude training camp, many distance runners anecdotally report that they feel like they have lost turnover, that is, the sensation of feeling coordinated at fast running speeds (34, 51, 52). It is not known whether this sensation is psychological in origin, due to some frank change in neuromuscular function as a result of altitude acclimatization, or is an adaptation in motor control, due to the chronic daily training at slower speeds at altitude. One of the reasons given by coaches and athletes for planning a postaltitude sea level training period, where faster speeds can be maintained for longer periods of time during workouts, is to help reestablish the neuromuscular sensation of having coordinated running mechanics at fast speeds mirroring competitive paces (20).

It has been suggested that exposure to hypoxia could alter neural activation and muscle recruitment. For example, with acclimatization to extreme altitude (5,050 m), Kayser et al. (27) have demonstrated no increase in vastus electromyography during constant load maximal work rate cycle ergometer exercise. This is in contrast to a 66% increase in vastus electromyography during maximal work rate exercise in the same subjects at sea level. However, any conclusions of altered neural activation or muscle recruitment patterns from this extreme altitude may not apply to individuals training at the more moderate altitudes (<3,000 m) typically utilized by endurance athletes. From a biomechanical standpoint, data from seven elite male distance runners showed no change after altitude training (28 days at 2,150 m) in stride length, stride frequency, ground contact time, or aerial time at running speeds ranging from 5 to 7 m/s (28). These are speeds typically achieved by elite male distance runners during competition in events ranging from the 1,500 m through the marathon. Additionally, there was no difference in H-reflex measures from pre- to postaltitude. These data would suggest that neuromuscular function and running mechanics are maintained after altitude training in elite distance runners. However, it should be noted that this elite athlete cohort followed the live high-train low paradigm, where out of as many as 40 total training sessions, 7 training sessions classified as higher intensity were performed at lower altitudes (365 m to 1,150 m). One of the primary premises behind the live high-train low training model argues that low-altitude training allows the athlete to maintain faster training paces, creating a training stimulus different from the same workout performed at high altitude (31). While no definitive conclusions can be drawn from this set of data, it can be suggested that, in distance runners completing live high-train low altitude training, there are no significant changes in running mechanics or which would require a corrective period of training at sea level; however, athletes who are competing a classic live high-train high altitude training model may show a different biomechanical response as a result of chronic slower training at altitude.

RECOMMENDATIONS FOR THE ENDURANCE ATHLETE AND COACH AND FUTURE RESEARCH DIRECTION

The recommendation of when to compete after completing an altitude training sojourn may ultimately be dependent on the individual responses to altitude training and acclimatization, altitude deacclimatization, as well as the training response that occurs within the first several days postaltitude. For example, we have seen that both the acclimatization response to altitude and the training response in hypoxia show substantial interindividual variability (16). Our opinion, based on the literature at hand, is that there is no reason to think that the deacclimatization response with return to sea level would show any less individual variability. From an applied standpoint, athletes who experience a high level of ventilatory acclimatization or mechanical limitations to ventilatory flow may be better off with a period of time at sea level before competing. This time may be less for the sea level training period as espoused by many coaches, but more simply as a time to allow for the ventilatory acclimatization to altitude to decay. However, for an athlete with a faster than normal decline in red blood cell mass with time at sea level [perhaps compared with the mean response of the Prommer et al. (38) cohort], competing as soon as possible upon return to sea level may be the most beneficial. In this way, the athlete can take full advantage of the increased oxygen-carrying capacity achieved with altitude acclimatization; however, performance may be mitigated somewhat by the increased metabolic cost and blood flow demands of the respiratory musculature.

Although not the same as the red blood cell mass-mediated ergogenic effects after altitude training, practical performance and physiological information can also be obtained from studies examining the effects of blood doping. In a classic study by Buick et al. (10), the authors demonstrated that, despite a normalization of hemoglobin concentration 16 wk after infusion of two units of blood, $\dot{V}_{O_{2max}}$ was still significantly improved (~5%) over preinfusion levels. The argument typically given to support these data is that the blood-doped athletes could now train at a higher, faster level than they did preinfusion. As a result, the increased training loads over time while in the blood-doped state may have created a milieu for other physiological adaptations which maintained performance, despite the gradual normalization of red blood cell mass. This concept is also related to a key premise behind the low altitude training portion of the live high-train low altitude training paradigm. By training at low altitude, the athlete is able to train at faster speeds and achieve a higher oxygen flux between the muscle capillary and mitochondria compared with training at higher altitudes (16, 31). This higher oxygen flux with low altitude training is believed to be a primary stimulus behind the improved mean steady-state workload improvement in athletes who live high and train low (31). Taken together, these data would suggest that a sea level training period after altitude training, as recommended by many coaches, may in fact be beneficial, if the athlete can gain an additional positive training response due to the added red blood cell and/or hemoglobin mass from altitude acclimatization. Again, athletes who complete the live high-train low form of altitude training may not need a period of sea level training time to achieve peak performance, due to the regular high-intensity workouts that can be achieved while training low and a lack of changes in

biomechanical variables during running at competitive velocities (28).

For the coach and athlete, there are several challenges with regard to implementation of the best practice for altitude training for each individual, as well as determining the individual rates of decay in hematological, biomechanical, and ventilatory adaptations with return to sea level. In recent years, many coaches and athletes have been utilizing the logistical convenience of intermittent exposure to normobaric hypoxia (51), which may or may not provide the same hypoxic dose as > 20 h per day exposure to terrestrial altitude (30). As a result, the decay of physiological and biomechanical responses with full time return to normoxia may not follow the same time course and deacclimatization responses as return to sea level from altitude residence. Similarly, many coaches lack the anecdotal experience of competition timing after simulated, intermittent altitude exposure. After chronic altitude or intermittent hypoxic exposure, serial laboratory testing to determine physiological deacclimatization rates may not be practical or accessible for the typical athlete or coach. Low-cost measures of hemoglobin concentration or hematocrit are not robust enough to provide meaningful information. A simple, useful measure might be to perform serial bouts of steady rate exercise under well-controlled conditions after return to sea level, with a focus on monitoring heart rate—a variable which has been shown previously to be reduced from prealtitude levels at *Days 3* and *15* after return to sea level (8). Data from Brugniaux et al. (8) suggest that measuring heart rate after 10 min of steady-state running at a pace equivalent to ~3,000 m race velocity would serve as a convenient monitor of aerobic fitness change over time.

Finally, it has been recommended (30, 32) that athletes live at altitude for a minimum of 28 days to maximize hematological acclimatization—it may be that shorter altitude sojourns are beneficial in the unique situation when they follow a combined altitude training camp and a sea level competition phase. For example, if an athlete completes a 4-wk altitude training camp, followed by a short time (~7–14 days) at sea level to compete, returning to altitude even for a short time may mitigate or delay the effects of neocytolysis by reestablishing EPO levels (18)—again, not necessarily for added erythropoiesis as is typically done with altitude residence, but more to delay the selective destruction of neocytes due to lower than baseline EPO concentrations if the athlete remained at sea level. It has been shown that relatively brief periods (3 h) of severe hypoxia is sufficient to significantly increase EPO concentrations, though it is not sufficient to accelerate erythropoiesis by itself (30). Perhaps periods of intermittent hypoxia, either at night while sleeping, during the day, or even with the hypobaria of airline travel could result in enough EPO release to prevent neocytolysis and preserve the hematological acclimatization response for a longer time, thereby expanding the window for optimal competition; however, this would need to be examined directly.

Ultimately, the best time to return from altitude training prior to a major competition for peak performance remains undocumented from a physiological standpoint. Future research should simultaneously explore in detail the time course of the changes in red blood cell mass, ventilatory acclimatization, biomechanical and/or neuromuscular factors, and performance in elite endurance athletes after return to sea level. We believe the interaction between these variables will strongly influence the ideal time to compete after altitude training. Each athlete may display his or her own signature of deacclimatization with sea level residence, and knowledge of personal decay rates may allow for individualized prescriptions of when best to complete postaltitude camp.

DISCLOSURES

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AUTHOR CONTRIBUTIONS

Author contributions: R.F.C. prepared figures; R.F.C. drafted manuscript; R.F.C., A.S.L.S., C.L., and B.D.L. edited and revised manuscript; R.F.C., A.S.L.S., C.L., and B.D.L. approved final version of manuscript.

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