Altitude Training
for Enhanced Athletic Performance

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Many elite athletes believe that training at altitude improves sea level performance. Yet the scientific evidence, such as it is, would seem to refute this, suggesting that the athlete’s trust may be misplaced. However, these scientific studies do not exclude the possibility that altitude training might produce an effect (<1%) that is too small to be detected by current research methods, but which might be of very real significance to the elite athlete for whom a much smaller effect would be sufficient to ensure an Olympic medal or a new world record. In contrast, living at altitude and training at or near sea level—the process known as “living hi and training low”—may produce larger, more easily measurable effects (>1.25%) in individual athletes. It is speculated that this benefit occurs only in those athletes (i) who mount an appropriate increase in renal erythropoietin (EPO) production with an increase in red blood cell (RBC) mass and (ii) who are able to sustain a high training intensity (running velocity) when training at low altitude. Hence, this effect requires that athletes live at altitudes of at least 2,500 m and perhaps up to 4,000 m, and have adequate iron stores to sustain increased RBC production occasioned by the altitude-induced increase in EPO production. They need also to train at an altitude sufficiently low that they are able to train at velocities equivalent to those achieved during sea level competition. Hence, at present, elite athletes can be neither encouraged to, nor discouraged from, training at altitude. Individual experience, rigorously evaluated, is the sole method by which correct conclusions can be drawn for individual athletes. Even a 1% improvement in performance will not make a sub-elite athlete elite; hence, there is no reason to encourage sub-elite athletes to train at altitude.

Key Words: altitude, training, humans, exercise performance, athletic performance

Key Points:

1. There is no firm evidence that training at altitude improves sea level athletic performance. However, the design of most of the studies of this question was inadequate to detect an effect of less than 2–3%.

2. As elite athletes require a substantially smaller training advantage (~0.2%) to become world champions, these studies do not exclude the possibility that training at altitude is of real value for elite athletes.

3. The advantage of living at altitude and training at a lower altitude (the “Live Hi, Train Lo” method) has been easier to demonstrate. This suggests that athletes wishing to use altitude as a training variant should adopt this technique.

4. The key physiological adaptation would appear to be an increase in the red blood cell (RBC) mass. At present, the optimum altitude to achieve this effect has not been firmly established but could be higher than 3,000 m.
Introduction

The popular Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance (1) first popularized by A.V. Hill and colleagues in the early 1920s (2, 3), predicts that fatigue during high-intensity exercise lasting from 2 to 10 min (and perhaps longer) results from an inadequate oxygen supply to the maximally exercising muscles. As a result, the muscles are forced to work “anaerobically,” producing lactate and hydrogen ions (H⁺). It is held that the accumulation of the latter inhibits a variety of contractile and metabolic processes in the active muscles, leading to impaired contractile function, thereby inducing fatigue and preventing the continuation of exercise at that particular intensity.

Hence, this model predicts that superior athletic performance will be realized by altering those factors that can increase oxygen delivery to the muscles, either by increasing (i) forward blood flow from the heart (cardiac output), (ii) the capacity of the blood to bind oxygen as a result of an increased red blood cell (RBC) mass or concentration, or (iii) the capacity of the muscles to process that oxygen as a result of an increased density of capillaries or an increased mitochondrial volume and enzyme content.

According to this model, one of the most effective forms of training should be to increase the “anaerobic” stress on the body by training at increasing altitude where there is a reduced mass of oxygen in each liter of air inhaled by the athlete. Consequently, the partial pressure of oxygen in the athlete’s body is reduced in proportion to the altitude above sea level at which he lives or trains.

Accordingly, this model predicts that, during maximal exercise, the athlete’s muscles should be working under more anaerobic conditions than at sea level. Hence, the body should adapt by improving its ability to process oxygen, thereby reducing the rate at which lactate and H⁺ are produced by the exercising muscles, thereby delaying fatigue and enhancing performance at altitude. On returning to sea level, these adaptations should increase the athlete’s ability to utilize what is now the usual amount of oxygen in the inspired air. Hence, performance should be enhanced, at least in those sporting activities in which performance is determined by a limiting oxygen delivery to, or use by, the exercising muscles.

Two forces helped to crystallize these ideas in the thinking of most athletes and many exercise scientists. The first was the holding of the 1968 Olympic Games at altitude (2,200 m) in Mexico City. Athletes native to altitude in East Africa dominated the distance events in those Games. When East African runners subsequently began also to dominate middle- and long-distance running races, also at sea level, the natural conclusion was that athletes who trained at altitude had an unfair advantage during competitions both at altitude and at sea level.

The second factor involved a series of scientific studies of elite U.S. runners, including the mile world record holder, Jim Ryun (5), who trained at altitude before returning to sea level for competition. Three times Jim Ryun trained at altitude and, on returning to compete at sea level, showed improved performance, including establishing a new world record in the mile. Those who wished to believe in the benefits of altitude training on sea level performance needed no additional anecdotes to “prove” the value of high altitude training for performances at sea level according to the predictions of the popular Cardiovascular/Anaerobic Model of Exercise Physiology.

Interestingly, when I personally spoke to Kansas Congressman Jim Ryun 21 years later at a scientific meeting at Stanford University, California, he expressed his uncertainty that altitude training was the sole or even the most important reason for his excellent performances in the summer of 1968. “It was all the rest...”
occasioned by travelling to and from altitude. I had never rested so much before races in my life.” Ryun was, in my opinion, an athletic genius who always trained too hard with intensive interval training, and who did not taper sufficiently before races, a common problem among distance runners in the U.S. in the 1960s.

What then does science currently conclude about the value of training at altitude for improving athletic performance at sea level? There are a number of studies that address this question either directly or indirectly.

Some studies have addressed the physiological and performance effects of acute and chronic exposure to training at medium altitude. These studies show that whereas running performance at altitude improves with continued exposure to living and training at altitude, neither the maximum oxygen consumption (VO$_{2\text{max}}$) nor the maximum heart rate alters with continuing residence at altitude (6). Physiological changes that do occur are a reduction in heart rate and blood lactate concentrations during submaximal exercise (6). Hence, the changes in running performance that occur with residence and training at altitude are accompanied by clear physiological changes that are measurable during submaximal but not during maximal exercise.

However, there is no evidence that these adaptations translate to measurable physiological changes during exercise at sea level. Hence, during exercise at sea level, there are no changes in these physiological measures during either maximal or submaximal exercise, measured before and after altitude exposure (6–11).

Other studies have examined the results of training at altitude on athletic performance at sea level. It must be stressed that these are complex studies because they require that a control group of runners of matched ability remain at sea level and train simultaneously and at the same intensity as the group of athletes training at altitude. Furthermore, it is difficult to control for the natural expectation of both athletes and scientists—that the group of athletes training at altitude will benefit more than will the athletes who continue to train at sea level. If the scientists were not biased to that belief, why would they undertake the research?

In other words, the studies cannot be adequately blinded so that neither the athletes nor the scientists are aware of who trained at altitude and who at sea level. This is an important consideration, as part of any effect might simply be the belief that training at altitude is beneficial, the so-called “placebo effect.”

Since the 1950s, there have been at least 92 studies of this topic. Of these studies, 76 are scientifically unintelligible because they were conducted without an adequate control group of athletes who performed the equivalent training program at sea level at the same time that the intervention group was training at altitude. Of the remaining 16 studies, only 4 found that the performance of the altitude-trained group improved more than did that of the group who trained similarly at sea level (12). An important

Mamo Wolde won a silver medal in the 10,000 m, with Ron Clarke (102) and Ron Hill (404) among the chasing pack. (Giller, N. Marathon Kings. London: Pelham; 1983, p. 83)
finding is that the more carefully controlled the study, the greater the probability that a negative or no effect of altitude training would be found (11, 13). This is a common finding in any study of an intervention, in this case training at altitude, which has little or no effect.

In fact, one of the most carefully conducted studies (11) found that the running performance of middle-distance athletes was impaired immediately after they returned from altitude to sea level after a period of training at altitude. The authors concluded that this was the result of the much higher rate of infections at altitude, and the inability of the athletes to run as fast at altitude as at sea level.

There is other indirect evidence that high altitude training may be of little value for improving the performances of runners competing in the range of running distances from 1,500 to 10,000 m. Péronnet (14) has reported that the rate of progression of world running records at those distances did not increase after 1968 when altitude training first began to be practiced by the world’s best athletes. In fact, no world records were set at those distances from 1968–1971. This was an unusual event that had not previously occurred in the 36-year period between 1930 and 1968. It was as if this were a direct result of the world’s best athletes suddenly beginning to train at altitude.

Furthermore, the rate of progression of the men’s world records for those distances was slower between 1968 and 1994 than between 1950 and 1968, the opposite of what would be expected if altitude training enhanced running performance at sea level.

Péronnet concluded that “these observations fail to show any overall beneficial effect of the introduction of altitude training on sea level performance for elite male runners. . . . Accordingly, the beneficial effects of altitude training for sea level performance remain to be convincingly demonstrated and should not be taken for granted” (14).

Similarly, Professor Bengt Saltin (6) concluded: “It is clear that the available scientific data do not support the ‘myth’ that training at medium altitude adds something significant to an athlete’s maximal aerobic power.”

However, Professor Saltin’s conclusion needs to be interpreted correctly. The fact that altitude training does not improve sea level running performance in certain events, or increase the maximal aerobic power (VO2max), is certainly quite damaging for the Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance. Nevertheless, it does not exclude the possibility that athletic performance in some events at other running distances could still be enhanced. This would occur, for example, if skeletal muscle oxygen deficiency does not limit performance in those events. In other words, altitude training could still be valuable if it alters, more effectively than an equivalent amount of sea level training, those physiological factors, which determine running performance at those specific running distances that are not limited by a putative oxygen deficiency in the active muscles. Such events could be marathon and ultramarathon foot races, and long-distance cycling and triathlon events, for example. These events have not been studied; thus, the potential of any altitude-related training benefit for athletes involved in those disciplines cannot be discounted on current evidence.

Because of this generally negative attitude, at least amongst scientists, of the (unproven) benefits of training at altitude, alternative options have also been evaluated. These include the use of red blood cell infusions or of the drug, erythropoeitin. Both of these techniques aim to increase oxygen delivery to the exercising muscle (and heart) by increasing the mass of circulating red blood cells. Although presently undetectable, both techniques are currently banned by the International Olympic Committee.

The third alternative technique, which aims legally to achieve the same effect of increasing exercise performance, in part by increasing the red cell mass, has become known as the “Live Hi, Train Lo” model. In this model, athletes live at altitude but train at a lower or sea level altitude especially when performing high-intensity training (15). The proposed benefit is that training at sea level allows the athlete to train at the necessary competitive speeds; while the established negative effects of living continuously at altitude, including the increased risk of infections at altitude (11), is reduced, whereas the beneficial adaptations of the increase in the red blood cell mass are maintained.

Thus, two studies (16, 17), carefully controlled and meticulously conducted, have found that subelite middle-distance runners, who lived at 2,500 m but trained daily for 4 weeks at 1,300 m, showed a 1.4% (14 s) improvement in 5,000-m time trial performance for up to 3 weeks after returning to sea level (16).
In contrast, athletes who trained either at sea level for the same period or, equally importantly, who lived and trained at high altitude (2,500 m) did not show any improvement in sea level running performance. The latter finding is again consistent with the conclusion that high altitude training, by itself, does not improve running performance at sea level (6, 11–13).

The authors concluded that two adaptations are necessary to achieve a beneficial effect from living at high altitude and training at sea level. The first is the increase in RBC mass that increases the potential oxygen delivery to the skeletal muscles (and heart). But as this occurred equally in both groups living at altitude while only the group that trained at a lower altitude improved their sea level running performance, this adaptation alone is insufficient. Second, the authors concluded that it was the inability of the group training at high altitude to train sufficiently fast that explains why they failed also to benefit from this adaptation. Thus, the athlete’s muscles must continue to be trained at speeds achieved in competition if the benefits of living at high altitude are to be realized.

The major physiological adaptations measured in the group that lived high and trained low was an increased VO$_{2\text{max}}$, accompanied by a reduced cardiac output during submaximal exercise at sea level. Surprising to these authors, but not to those who argue that increased oxygen delivery to the heart and not to skeletal muscle is the crucial factor explaining how an increased red cell mass enhances athletic performance (1, 18), neither skeletal muscle capillarisation nor enzyme content was altered with any form of training (19).

The authors also noted substantial individual variation in the response to altitude training. Hence they concluded that those who responded to altitude exposure were those who increased their kidney erythropoietin production and who, consequently, demonstrated an increase in RBC mass and subsequently in VO$_{2\text{max}}$ (17).

In summary, these authors suggest that for athletes to benefit from training or living at altitude, they must fulfil three criteria. First, they must live at an altitude that will produce in them an increase in EPO production adequate to stimulate an increase in RBC mass. Second, they must either have adequate whole body iron reserves or must receive adequate amounts of iron so that they respond appropriately to increased EPO production by increasing their RBC mass appropriately. Third, they must be able to maintain the running velocity as close as possible to the speeds they normally achieve at sea level. This requires that they train at altitudes as close to sea level as possible.

Altitude Training for Improved Performance at Sea Level: The Main Conclusions

Why is it so difficult to find convincing evidence that training at altitude definitely improves running performance or alternatively that living at altitude and training at a lower altitude definitely improves running performance in everyone?

The first answer may be that the effect is quite small and that the cost of proving conclusively such a small effect may be prohibitive. For example, the smaller the effect, the more athletes are required to participate in the trial. Most of these studies typically include a total of less than 20 runners in the two study groups: control and intervention. However, statistical analysis shows that a study using this number of subjects can only detect an improvement in performance of greater than 3%. Yet, the difference between first and fourth place in Olympic competition is usually only a fraction of 1%. Nevertheless, to detect an effect as large as 1% would require the testing of more than 100 athletes, a logistical impossibility at present.

Thus, most of the studies that have been undertaken were not designed to identify an effect of a size (less than 1%) that would still be perfectly acceptable to an elite Olympic athlete whose goal is to improve his or her performance sufficiently to become a medal contender, not by the 3% that would take them to another, quite unattainable and unrealistic level of performance.

Conversely, one might conclude that even if it does indeed improve running performance, the effect of altitude training must be small enough (perhaps less than about 1%), compared to the benefit of the same training undertaken at sea level. The size of this effect is perhaps sufficiently small that other effects of training away from home for prolonged periods probably quite easily negates it. On the other hand, this
small effect might be all that the elite Olympic athlete is looking for in his/her quest to become a gold medallist.

The second reason why altitude training may be of limited or no value could be because the physiological model, which gives it legitimacy, may be incorrect. As detailed elsewhere (1, 18) there is an argument that the heart, not skeletal muscle, may be the organ at greatest risk for developing an oxygen deficiency during maximal exercise both at sea level and at altitude.

The basis for this theory is the clear evidence that, at altitude, a mechanism exists, which constrains the exercising muscles (and heart) from working so hard that they contract anaerobically—the so-called “lactate paradox.” Furthermore, perhaps the same mechanism constrains the heart from working as hard at altitude as it can at sea level. The sole logical explanation for both these findings is the existence of a “governor” in the brain that prevents the muscles from working sufficiently hard that they force the heart to contract ischaemically, which would then cause the development of heart failure and skeletal muscle anaerobiosis. In this model, the heart activates the “governor” shortly before an exercise intensity is reached at which the heart’s blood supply becomes inadequate. The “governor” then either prevents the brain from recruiting additional muscle to increase the exercise intensity, or it actively reduces the amount of active muscle that is being recruited, thereby forcing the athlete to slow down.

This model predicts that altitude training will be of value only if it induces adaptations that increase the amount of blood reaching the heart during maximal exercise, or improves the heart’s efficiency so that it can produce a higher (cardiac) output at the same oxygen demand.

The most likely beneficial adaptation to achieve these benefits would be any increase in the amount or concentration of the circulating red blood cells, the exact adaptation identified by Chapman et al. (17) as crucial for any beneficial performance effect of training at altitude.

However, this is an extremely difficult adaptation to achieve (11). Recent studies, for example, show that residence and training at an altitude of 1,800 m for 18 days failed to increase red cell mass (20). Similarly, elite Australian athletes who slept in a “high altitude” or “nitrogen” house for either 23 days at 3,000 m (21) or for 12 days at 2,650 m (22) failed to show any increase in red blood cell production. This suggests that despite the findings of the “Live Hi, Train Lo” studies, the optimum altitude for altitude residence has yet to be established and might be as high as 4,000 m.

This failure of altitude exposure to increase RBC mass would then explain why the VO\textsubscript{2max} does not increase during continuing residence at altitudes less than perhaps 2,500 m. Without any increase in oxygen supply, this model predicts that there will not be any increase in the maximum cardiac output necessary for any increase in VO\textsubscript{2max}. Hence it is clear that exposure must be to altitudes equal to or greater than 2,500 m in order to achieve an increased red cell mass (16).

In contrast, the presence of the governor reducing the intensity of physical activity that can be undertaken at altitude must also cause some processes of skeletal muscle de-adaptation. At extreme altitude, this process of de-adaptation causes a 20–25% reduction in muscle cell size and a reduced concentration of mitochondrial enzymes, for example, in climbers exposed to a simulated 40-day climb to an altitude equivalent to the summit of Mount Everest (23). It is difficult to believe that these adaptations are beneficial for running performance at sea level. However, studies at lower altitudes show that mitochondrial enzyme concentrations do not change more with training at altitude than they would at sea level (6).

Interestingly, this model of the central governor limiting exercise at high altitude can also explain why, despite their markedly superior performance at extreme altitudes, the leg muscles of Sherpas have lower mitochondrial enzyme concentrations than sedentary Caucasians living at sea level (24). Because it is not the capacity of their leg muscles to use oxygen this would explain the Sherpa’s superior performance at altitude. Rather, this model predicts that it is the ability of their coronary blood vessels to provide their heart with a greater oxygen supply during maximal exercise at extreme altitude that would explain the Sherpa’s superiority. This superiority would result from (i) a higher blood RBC concentration, (ii) a superior respiratory capacity able to maintain a higher arterial partial pressure of oxygen, (iii) a more extensive network of blood vessels in their hearts that are able to accommodate a larger coronary blood flow, (iv) an increased volume of mitochondria in their hearts, and (v) perhaps, as a result, superior
myocardial efficiency so that an even larger cardiac output can be achieved at these higher levels of blood and oxygen supply to the heart.

That high altitude residents and those living at sea level are fundamentally different is shown by the smaller difference in sea level and altitude VO2max in persons native to altitude compared to those who have always lived at sea level. Hence, altitude natives with the same sea level VO2max as persons living at sea level have substantially higher VO2max at altitude, presumably because they are better able to supply their hearts with oxygen during maximal exercise at altitude (23).

The final reason why altitude training may prove less effective than expected may be because the risk of infections of either the upper respiratory or gastrointestinal tracts is increased at altitude (11). Thus the detrimental effects of illness would cancel out any potential benefits of altitude training.

References


