Determinants of team-sport performance: implications for altitude training by team-sport athletes

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ABSTRACT
Team sports are increasingly popular, with millions of participants worldwide. Athletes engaged in these sports are required to repeatedly produce skilful actions and maximal or near-maximal efforts (eg, accelerations, changes in pace and direction, sprints, jumps and kicks), interspersed with brief recovery intervals (consisting of rest or low-intensity to moderate-intensity activity), over an extended period of time (1–2 h). While performance in most team sports is dominated by technical and tactical proficiencies, successful team-sport athletes must also have highly-developed, specific, physical capacities. Much effort goes into designing training programmes to improve these physical capacities, with expected benefits for team-sport performance. Recently, some team sports have introduced altitude training in the belief that it can further enhance team-sport physical performance. Until now, however, there is little published evidence showing improved team-sport performance following altitude training, despite the often considerable expense involved. In the absence of such studies, this review will identify important determinants of team-sport physical performance that may be improved by altitude training, with potential benefits for team-sport performance. These determinants can be broadly described as factors that enhance either sprint performance or the ability to recover from maximal or near-maximal efforts. There is some evidence that some of these physical capacities may be enhanced by altitude training, but further research is required to verify that these adaptations occur, that they are greater than what could be achieved by appropriate sea-level training and that they translate to improved team-sport performance.

INTRODUCTION
Team sports are increasingly popular, with millions of participants worldwide. Athletes engaged in these sports are required to repeatedly produce skilful actions and maximal or near-maximal efforts (eg, accelerations, changes in pace and direction, sprints, jumps and kicks), in a semistochastic fashion, interspersed with brief recovery intervals (consisting of rest or low-intensity to moderate-intensity activity), with and without the ball/puck, over an extended period of time (1–2 h). The physical demands are therefore complex, requiring athletes to have highly-developed speed, agility, muscular strength and power and endurance. Athletes also require the ability to repeatedly execute complex motor skills (eg, passing, defending and tackling) under pressure and while fatigued.1 2

During competitive, field-based team sports, elite athletes may cover 8–14 km at an average intensity of ~85–90% of their maximal heart rate (HRmax) or 75–80% of their maximal oxygen uptake (V̇O₂max), with marked differences related to playing standard and position.3–9 This suggests that a well-developed aerobic energy system is an important physiological determinant of team-sport physical performance. The observation that more than 150 different, brief, intense actions may be performed in a team-sport match, and that athletes may record moderately-large blood (2–14 mM) and muscle lactate values (~15 mmol/kg dry weight) after intense periods of play, indicates that the rate of anaerobic energy turnover is also high during periods of a match.7 10 Much effort goes into designing training programmes to improve these physiological capacities, with expected benefits for team-sport performance.

Recently, some team sports have introduced altitude training (AT) in the belief that it can enhance their sea-level, match-related physical performance. Until now, however, there is scant published evidence showing improved team-sport performance following AT, despite the often considerable expense involved.11 In the absence of such studies, this review will identify important physiological determinants of team-sport physical performance and briefly discuss the evidence that these may be improved by simulated or natural AT. It is beyond the scope of this review to identify technical and tactical abilities that may influence team-sport performance and that might be affected by AT. It is also beyond the scope of this review to discuss how reductions in air density experienced during hypobaric hypoxia may affect factors such as ball flight (air density reduces by about 10% for every 1000 m increase in altitude and will affect flight characteristics).12

Physiological factors determining team-sport physical performance
A better understanding of the physiological factors associated with team-sport physical performance is arguably the first step in order to assess whether AT may play a role in enhancing team-sport performance. As most team sports require athletes to regularly repeat short, high-intensity efforts, interspersed with longer intervals of submaximal exercise, these physiological factors can be broadly described as

For the purpose of this review, altitude training refers to living and/or training at a natural or simulated altitude (eg, live high-train low, live high-train high). It does not include intermittent hypoxic training, which has been the subject of recent reviews.

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Sprint performance
Sprinting, defined as a running velocity above a lower limit ranging from 19 to 25 km/h, amounts to 5–10% of the total distance covered during a match and corresponds to 1–3% of match time in rugby league and soccer (football). The importance of sprint performance for team-sport athletes is highlighted by the observation that straight sprinting is the most frequent action preceding a goal in football (soccer). It has also been estimated that an ~0.8% impairment in sprint speed would have a substantial detrimental effect on the likelihood of a player losing possession of the ball against an opponent, when both players sprint for the ball. In addition, mean sprint speed during a repeated-sprint ability (RSA) test; which is strongly correlated with peak speed and has been correlated with total sprint distance during a professional football match. While further research is required, there is emerging evidence that sprint performance is an important determinant of team-sport performance.

Despite its importance, and possibly due to an emphasis on the effects of AT on endurance performance, there has been scant research into the effects of AT on sprint performance. In the two published studies that we are aware of, AT was reported to result in a greater improvement in 150 m and 400 m sea-level running performance compared to sea-level training. However, as the physiological and metabolic demands of these running distances will differ from the types of sprints typically performed by team-sport athletes (<6 s), further research is clearly required to investigate the effects of AT on brief sprint performance and its determinants.

Determinants of sprint performance
In simple terms, sprint performance is determined by stride length and stride frequency (figure 1). To improve speed, an increase in one or both of these parameters must occur within the context of sound technique. Improvements in stride length, and hence speed, are intimately linked to improvements in power—which is directly related to strength, elastic strength and dynamic flexibility (the ability to move the appropriate joints through a large range of motion at high speeds). Power has also been related to the ability to supply ATP at a fast rate and to the percentage of fast-twitch fibres. Sprint performance is also determined by stride frequency, which is related to factors such as intramuscular coordination. We summarise below the research investigating the effects of AT on these determinants of sprint performance.

ATP supply
Maximal sprint efforts rely on a fast and constant turnover of ATP powered by phosphocreatine (PCr) breakdown and anaerobic glycolysis. As such, team-sport athletes may be able to improve their sprint performance if they are able to enhance their ability to deplete large amounts of high-energy phosphates at a fast rate (ie, their anaerobic capacity). Anaerobic performance lasting 30 s or less on either a cycle ergometer (Wingate test) or a non-motorised treadmill is generally not adversely affected at altitude due to enhanced anaerobic energy release (ie, higher oxygen deficit or muscular lactate concentration), to compensate for the reduced aerobic ATP production. A high rate of anaerobic energy release during exercise has been proposed to be an important stimulus to increase anaerobic capacity. It could therefore be hypothesised that this lower rate of oxygen delivery to muscles when training at altitude would increase the flux through the anaerobic energy systems and lead to greater improvements in anaerobic capacity. In support of this assumption, increases in maximal accumulated oxygen deficit have been reported either after 15 days spent at 2650 m and training at 610 m (10%) or after 14 nights spent at 2100 m and training at 2700 m (29%). As training has not been reported to increase PCr breakdown during high-intensity exercise, these increases in maximal accumulated oxygen deficit (an indirect measure of anaerobic capacity) can most likely be attributed to increases in the rate of anaerobic glycolysis.

There are conflicting results concerning the effects of AT on glycolytic adaptations. For example, greater increases in phosphofructokinase (PFK) activity have been reported when sprint

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Figure 1: A summary of the main physiological factors that affect team-sport physical performance; these can be broadly described as factors that affect either sprint performance or the ability to recover from maximal or near-maximal efforts.
interval training is performed in normobaric hypoxia (~3200 m), compared to normoxia. In contrast, research involving endurance athletes has reported a decrease in PFK activity after either a ‘live high-train low’ intervention (2×8 h/week for 3 weeks; hypoxic dose <50 h) or training in a hypobaric chamber (4–5 sessions/week for 3–4 weeks at ~2300 m). These negative findings can probably be attributed to the study design whereby endurance athletes performed training at altitude that consisted primarily of aerobic workouts. In addition, the low level of hypoxia used in some of these studies (<2500 m) may not have been sufficient to elicit an additional activation of anaerobic pathways beyond that observed in normoxia. While further research is required, it appears that team-sport athletes may be able achieve greater increases in anaerobic capacity, and possibly sprint performance, by performing sprint training at altitude.

**Strength**

Maximal muscle strength can be defined as the maximal force a muscle or muscle group can generate at a specific velocity. It appears that hypoxia alone is insufficient to induce muscle hyper trophy, increase muscle strength (one repetition maximum, 1RM) or improve sea-level (repeated) sprint performance. However, it has been hypothesised that resistance training combined with systemic hypoxia may lead to greater improvements in muscle strength. Resistance training with systemic hypoxia causes a reduction in the concentration of oxygen in the blood and tissue, inducing greater accumulation of metabolites (blood lactate) and anabolic hormones (eg, growth hormone). Training under these circumstances would also result in an accelerated recruitment of type II motor units, potentially increasing the stress on these units and subsequently producing adaptation in the form of hypertrophy of these motor units.

Until now, only a few studies have investigated whether resistance training performed in hypoxia is more efficient at improving maximal strength and eventually single-sprint performance than similar training in normoxia. In one study, low-resistance exercise (6 sets of 25 repetitions at 30% 1RM, 3 times/week for 4 weeks) combined with hypoxia (fractional inspired oxygen, FiO₂ = 0.12, ~4000 m) had no additional effect on maximal strength compared to identical exercise completed under normoxic conditions. In contrast, another research group has reported larger increases in strength following resistance training performed in hypoxia versus normoxia. In the one available study involving a team-sport population (ie, female netball athletes), resistance training under hypoxic conditions (5 weeks of training of the knee flexor and extensor muscles in which low-load resistance exercise (20% of 1RM) was combined with hypoxic air to generate blood oxyhaemoglobin levels of approximately 80%) not only improved muscle strength (15%) and muscle hypertrophy (6%), but also induced faster (4%) 5 and 10 m sprint times. Thus, while further research is required, especially incorporating resistance-training protocols more specific to those used by team-sport athletes, there is emerging evidence that resistance training at altitude may lead to greater improvements in muscle strength. Future studies should also determine whether any enhancements in maximal strength translate into a better force application technique and better sprint performance.

**Elastic strength**

Elastic strength, or reactive strength, is dependent on the stretch-shortening cycle and is the ability to exert maximal force during a high-speed movement; elastic strength has been shown to be an important determinant of sprint performance. To our knowledge, however, there is no published research that has directly investigated the effects of AT on elastic strength. Future AT studies, incorporating team-sport-specific speed, strength, and power training, performed in hypoxia, should consider including measures of elastic strength to address this knowledge gap.

**Neural drive/coordination**

Improved intramuscular coordination, leading to increases in stride frequency, should theoretically improve sprint performance. The question of whether training at altitude can lead to greater improvements in stride frequency during sprinting has not been specifically addressed. However, the scientific literature, mathematical models, and performance results (1968 Olympic Games in Mexico) all suggest that sprint performance is enhanced during acute exposure to natural altitude, which has been attributed to the lower air density at altitude. This raises the intriguing possibility of developing over-speed routines when training at natural altitude to improve intramuscular coordination and stride frequency. In support of this, 2 weeks of strength and speed training at natural altitude of 1860 m significantly improved 150 m sprint performance in five-national level sprinters, compared to a control group that trained simultaneously according to a similar programme at sea level. However, as this study did not specifically measure changes in stride frequency, or recruit team-sport athletes, more research is required.

Another important consideration for team-sport athletes is that the ability to repeat sprint performance has been associated with the ability to maintain faster stride frequencies, through retaining higher vertical stiffness. Mounting evidence, gathered from laboratory-based studies, suggests that biomechanical manifestations of fatigue are likely to be driven, at least partially, by hypoxia severity-dependent reductions in neural drive to the active musculature; this is presumably the result of hypoxia-induced increased levels of intramuscular metabolites known to stimulate group III–IV muscle afferents (ie, accelerated development of peripheral fatigue) at moderate-to-high hypoxic levels (simulated altitudes <4000 m). At higher altitudes, the exaggerated development of central fatigue is primarily determined by a stronger reflex inhibition due to brain hypoxia. These heights, however, are clearly not relevant for team-sport AT purposes; that is, if too severe, hypoxia compromises training quality and hence counteracts the possible benefits to be derived from the greater stimuli to adapt. Although chronic altitude exposure (a 14-day exposure at 3260 m) has the potential to attenuate the development of central fatigue during continuous, whole body exercise, whether a comparable response of the central nervous system can occur during high-intensity intermittent exercises after training at heights similar to those commonly used by team-sport players (1500–3600 m) is currently unknown. Although scientific support is currently lacking, it could also be that a hypoxia-induced improvement in the central motor drive resulting from AT may improve musculoskeletal stiffness regulation (ie, less energy wasted on braking forces and minimal vertical oscillation of the centre of mass), leading to a faster stride frequency and thereby improved sea-level repeated-sprint performance.
Recovery between efforts

**Maximal oxygen uptake**

Given the total distance travelled in a match, the relatively high average match intensity and the necessity to recover from brief, high-intensity activities, it is generally believed that high aerobic fitness is important for team-sport success. The most widely accepted measure of aerobic fitness is the VO$_{2\text{max}}$, which represents the maximum rate at which aerobic metabolism can supply energy.$^{65}$ In support of the importance of VO$_{2\text{max}}$ studies have reported a correlation between VO$_{2\text{max}}$ and the distance covered during team sports.$^{66–68}$ It has also been reported that participants with a greater VO$_{2\text{max}}$ are better able to maintain power outputs/sprint times during a repeated-sprint exercise and that there are moderate correlations ($r=−0.20$ to $−0.75$), not always significant, between VO$_{2\text{max}}$ and performance drop-off indices.$^{69–77}$ While some studies have reported increases in VO$_{2\text{max}}$ following AT,$^{78–80}$ this is not a universal finding, especially in well-trained athletes.$^{81}$

As indicated by the Fick equation, VO$_{2\text{max}}$ is determined by central and peripheral factors. Until now, however, there has been limited research investigating the relationship between the central and peripheral determinants of VO$_{2\text{max}}$ and team-sport physical performance. In one of the few studies, McMahon and Wenger$^{82}$ reported a weak correlation between cardiac output and the maintenance of power output during intermittent sprint exercise. While further research is required, it seems unlikely that increases in cardiac output will contribute to improvements in team-sport physical performance following AT.

The dominant factors explaining the association between VO$_{2\text{max}}$ and team-sport physical performance appear to be peripherally located.$^{82}$ In particular, the importance of the peripheral component of VO$_{2\text{max}}$ is highlighted by the similar relationship between the arteriovenous oxygen difference (a-VO$_2$ diff) and VO$_{2\text{max}}$ and the ability to maintain power output during brief, intermittent sprints.$^{82}$ This suggests that adaptations at the tissue level (eg, muscle oxidative capacity, capillarisation, haemoglobin mass) may be important determinants of the ability to frequently perform high-intensity activities during a team sport.$^{83}$ In support of this, it has been reported that the fatigue index during repeated-sprint exercise was inversely correlated with maximal ADP-stimulated mitochondrial respiration measured directly on muscle fibres,$^{84}$ that capillary density was significantly related to recovery following a bout of maximal knee extensions,$^{85}$ and that giving erythropoietin results in a reduced accumulation of anaerobic metabolites in the blood following an intermittent sprint task.$^{86}$ Further research is required, however, to establish the relationship between these peripheral factors and actual team-sport physical performance.

Despite the need for further team-sport-specific research, there is evidence that some of these peripheral factors can be improved by AT. Compared to sea-level training, ‘live high-train low’ AT has been reported to increase the a-VO$_2$ diff.$^{87}$ In contrast, research suggests that short-duration (<4 week) ‘live high-train low’ AT protocols do not increase capillarisation.$^{87–89}$ However, training under normobaric hypoxic conditions to normoxic conditions has been reported to result in greater increases in capillary density in one study,$^{90}$ but not another.$^{91}$ The effects of AT on mitochondrial adaptations remain unresolved. Mitochondrial respiration has been reported to diminish following 28 days of exposure to ∼3500 m,$^{92}$ to remain unchanged following 9–11 days of exposure to ∼4500 m$^{93}$ or to increase following 19 days of exposure to ∼3200 m (Bishop et al unpublished research). It is now established that long-term (>4 week), but not short-term (<4 week),$^{94}$ exposure to extreme (>5500 m) environmental hypoxia decreases the mitochondrial content of muscle fibres.$^{95}$ However, compared to normoxic training, training under hypoxic conditions (∼2000–4000 m) has been reported to result in greater increases in citrate synthase activity,$^{96}$ (citrate synthase is an enzyme that is exclusively located in the mitochondria$^{97}$ and is strongly correlated with mitochondrial content$^{98}$).

While there is some controversy,$^{99}$ 100 increases in haemoglobin mass (Hb$_{\text{mass}}$) are often reported following different types of AT, assuming an appropriate ‘hypoxic dose’ (∼300 h).$^{78}$ 79 101 Also, as the magnitude of haemoglobin (Hb) increase has been suggested to be related to baseline Hb$_{\text{mass}}$,$^{102}$ team-sport athletes may be more likely to present increased Hb$_{\text{mass}}$ in response to AT than elite cyclists. Even though increases in Hb$_{\text{mass}}$ do not necessarily lead to improvements in VO$_{2\text{max}}$,$^{99}$ there may be benefits for aerobic metabolism through the compensatory decrease in blood flow which may slow the mean blood transit time and improve the exchange of gases, substrates and metabolites.$^{103}$ Thus, while there is emerging evidence that many of the peripheral determinants of VO$_{2\text{max}}$ can be improved by either living and/or training under hypoxic conditions, further research is required to optimise the hypoxic stimulus and to investigate the effects of these changes on subsequent team-sport-related physical performance.

**Phosphocreatine resynthesis rate**

We are unaware of studies directly investigating the influence of the PCr resynthesis rate on team-sport physical performance. Nonetheless, there is good evidence that PCr resynthesis is an important determinant of the ability to recover single-sprint and repeated-sprint performance.$^{104–108}$ This is supported by the observation that occlusion of the circulation to one leg prevents PCr resynthesis and reduces total work in subsequent sprints.$^{109}$ The importance of PCr resynthesis for intermittent sprint performance is further supported by research demonstrating that creatine supplementation (which increases the PCr resynthesis rate$^{110}$) improves multiple-sprint performance, especially when the recovery between sprints ranges from 50–120 s$^{111–114}$ and also improves some 20 m sprints and agility tasks during an exercise protocol designed to simulate match play in female football (soccer) players.$^{115}$

The importance of the PCr resynthesis rate for the ability to recover from high-intensity exercise suggests that future studies should investigate the influence on AT on the rate of PCr resynthesis in team-sport athletes. It has been reported that the PCr resynthesis rate is positively correlated with citrate synthase activity$^{116}$ and is reduced in patients with mitochondrial myopathies.$^{117}$ Therefore, changes in the PCr resynthesis rate following AT are quite likely to closely reflect mitochondrial adaptations (which have been equivocal until now) and also require further research.

**Buffer capacity**

In contrast to the good evidence that VO$_{2\text{max}}$ and the PCr resynthesis rate are important determinants of team-sport physical performance, the importance of the hydrogen ion (H$^+$) buffering is more controversial. A number of studies,$^{118–123}$ but not all,$^{124}$ 125 have reported that increasing the blood buffer capacity is quite likely to improve repeated and intermittent sprint performance. However, the importance of muscle buffer capacity (Hb) is less convincing. Despite a persistent low muscle pH, sprint power output has been reported to partially recover 6 min after a repeated-sprint test.$^{104}$ Moreover, no significant
correlations were noted between the recovery of pH and the recovery of power output during single or repeated sprints. Similarly, previous studies have shown that sprinting abilities were restored faster than muscle pH and that the decline in sprint performance during a football (soccer) match was not correlated with muscle pH. There has been one study that has reported a moderate correlation between βm and RSA, but to our knowledge no studies have correlated βm with team-sport physical performance.

Another way to assess the importance of βm is to assess team-sport-related physical performance before and after β-alanine supplementation. β-alanine is an important precursor of carnosine (β-alanyl-L-histidine), an important muscle buffer that has been estimated to account for ~10% of the total buffering capacity in the human vastus lateralis muscle. β-alanine supplementation has been reported to improve Yo-Yo test performance (a test that correlates well with match physical performance in soccer players), but not intermittent sprint performance. Thus, while there is some evidence that βm may influence team-sport performance, more research is required.

Until now, five studies have investigated changes in βm in response to various forms of AT (with an average increase of ~7%); range=0–18%). However, the response is quite variable with the smallest and the largest changes in βm reported following very similar altitude-training protocols by the same research group. Nonetheless, while this research suggests a possible benefit of AT on βm, and therefore potentially team-sport physical performance, greater gains in βm have typically been reported in response to interval training. It is therefore difficult, based on current evidence, to justify the expenses associated with AT if the goal is to maximise improvements in βm.

CONCLUSIONS AND FUTURE DIRECTIONS

There are many physiological qualities, important for team-sport performance, that could theoretically be improved by AT. However, much of this information is derived from studies conducted with endurance (individual) athletes. Further research is required to verify that these adaptations occur in team-sport athletes after AT and that these adaptations translate to improved team-sport performance. It will also be important to determine whether these adaptations are greater than what can be achieved by regular sea-level training. Given the many ways in which AT may be performed (eg, ‘live high-train low’, ‘live high-train high’, ‘live low-train high’) and the different levels or conditions of hypoxic exposure possible, more research is required to optimise the AT stimulus to improve match-related physical performance and the different physiological determinants of team-sport physical performance identified in this review.

What are the new findings?

- This review summarises the physiological determinants of team-sport physical performance that could potentially be improved by altitude training.
- While the theoretical rationale is quite strong, there are very few published studies that have investigated changes in team-sport physical performance, or its determinants, in response to altitude training.
- There are many conflicting findings in the literature, which indicates the need to better control for diet, training and the altitude dose.

How might it impact on clinical practice in the near future?

This review highlights that there is theoretical support for the use of altitude training by team-sport athletes. However, it also highlights the need for further research to verify that altitude training can promote greater physiological adaptations than appropriate sea-level training and that these greater physiological adaptations translate to improved team-sport physical performance.

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