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Authors: David Bishop and Olivier Girard

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Address for Correspondence:
Prof. David Bishop
Institute of Sport, Exercise and Active Living (ISEAL),
College of Sport and Exercise Science,
Victoria University, Melbourne, Australia.
Phone: (03) 9919-4499 International +61-3-9919-4499
Fax: (03) 9919-4891 International +61-3-9919-4891
E-mail: David.Bishop@vu.edu.au

Contribution Statement

David Bishop and Olivier Girard both contributed to the conception of this review, initial drafts, critically revising the article for important intellectual content and final approval

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 (*e.g.* accelerations, changes in pace and direction, sprints, jumps and kicks), interspersed with brief recovery intervals (consisting of rest or low- to moderate-intensity activity), over an extended period of time (1 to 2 hours). 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 programs 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. To date 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 these adaptations translate to improved team-sport performance.

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.

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 (*e.g.* accelerations, changes in pace and direction, sprints, jumps and kicks), in a semi-stochastic fashion, interspersed with brief recovery intervals (consisting of rest or low- to moderate-intensity activity), with and without the ball/puck, over an extended period of time (1 to 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 (*e.g.* passing, defending and tackling) under pressure and while fatigued [1 2].

During competitive, field-based, team sports, elite athletes may cover 8 to 14 km at an average intensity of ~ 85-90% of their maximal heart rate (HR_{max}) or 75-80% of their maximal oxygen uptake (VO_{2max}), 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 $mmol \cdot kg^{-1}$ d.w.) 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 programs to improve these physiological capacities, with expected benefits for team-sport performance.

Recently, some team sports have introduced altitude training[1] (AT) in the belief that it can enhance their sea-level, match-related, physical performance. To date, 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 which 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].

2. 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 sub-maximal exercise, these physiological factors can be broadly described as factors that affect either sprint performance, or the ability to recover from maximal or near-maximal efforts (Figure 1). It should be noted, however, that many of these factors may also influence other aspects of team-sport physical performance (e.g., explosive power may also influence jump performance).

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

INSERT FIGURE 1 ABOUT HERE

2.1. Sprint performance

Sprinting, defined as a running velocity above a lower limit ranging from 19 to 25 km·h^{-1}, 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) [13 14]. 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)[15]. 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 [16]. In addition, mean sprint speed during a repeated-sprint ability (RSA test) (which is strongly correlated with peak speed [17-20]) has been correlated with total sprint distance during a professional football match [21]. 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 [11], 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 both 150-m [22] and 400-m [23] 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 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) [24]. Power has also been related to the ability to supply ATP at a fast rate, and to the percentage of fast-twitch fibres [25]. Sprint performance is also determined by stride frequency, which is related to factors such as intra-muscular coordination. Below we summarise 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 [26]. 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 (i.e., their anaerobic capacity)[27]. Anaerobic performance, lasting 30 s or less, on either a cycle ergometer (Wingate test: [28-30]) or a non-motorized treadmill [31 32], is generally not adversely affected at altitude due to enhanced anaerobic energy release (*i.e.*, higher oxygen deficit or muscular lactate concentration; [28 30] 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 [33]. 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%; [34]) or after 14 nights spent at 2100 m and training at 2700 m (29%; [35]). As training has not been reported to increase PCr breakdown during high-intensity exercise [36-38], these increases in maximal accumulated oxygen deficit (an indirect measure of anaerobic capacity; [39]) 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 PFK activity have been reported when sprint interval training is performed in normobaric hypoxia (~3200 m), compared to normoxia [40]. In contrast, research involving endurance athletes has reported a decrease in phosphofructokinase (PFK) activity after either a "live high-train low" intervention (2 x 8 h/wk for 3 wk; hypoxic dose < 50 h) [41] or training in a hypobaric chamber (4–5 sessions/wk for 3–4 weeks at ~2300 m) [42]. 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 these studies (< 2500 m) may not have been sufficient to elicit an additional activation of anaerobic pathways beyond that observed in normoxia [43]. 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 [44]. It appears that hypoxia alone is insufficient to induce muscle hypertrophy, increase muscle strength (1RM), or improve sea-level (repeated) sprint performance [45]. However, it has been hypothesized that resistance training combined with systemic hypoxia may lead to greater improvements in muscle strength [45 46]. 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 (e.g., growth hormone) [47]. 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 [48 49].

To date, 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 per week for 4 weeks) combined with hypoxia (FiO_2 = 0.12, ~ 4000 m) had no additional effect on maximal strength compared to identical exercise completed under normoxic conditions [50]. In contrast, another research group has reported larger increases in strength following resistance training performed in hypoxia *versus* normoxia [46 51]. In the one available study involving a team-sport population (*i.e.*, 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 one repetition maximum) was combined with hypoxic air to generate blood oxy-haemoglobin 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 [51]. 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 which form of resistance training (maximal *versus* explosive muscle strength), and which hypoxic, dose is best for maximizing improvements in muscle strength. As the orientation of the total force applied to the supporting ground during a sprint acceleration is more important to performance than its amount [52], future AT 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 [53]; elastic strength has been shown to be an important determinant of sprint performance [54]. 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 intra-muscular coordination, leading to increases in stride frequency, should theoretically improve sprint performance [24]. 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 [55], mathematical models [56 57] 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 [12]. This raises the intriguing possibility of developing over-speed routines when training at natural altitude to improve intra-muscular coordination and stride frequency. In support of this, two weeks of strength and speed training at a 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 [22]. 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 [58 59]. 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 [60 61]; this is presumably the result of hypoxia-induced increased levels of intramuscular metabolites known to stimulate group III-IV muscle afferents (*i.e.* accelerated development of peripheral fatigue) at moderate to high hypoxic levels (simulated altitudes < 4000 m) [62]. At higher altitudes, the exaggerated development of central fatigue is primarily determined by a stronger reflex inhibition due to brain hypoxia [63]. These heights, however, are clearly not relevant for team-sport altitude training purposes; *i.e.* 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 5260 m) has the potential to attenuate the development of central fatigue during continuous, whole-body exercise [64], whether comparable response of the central nervous system 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 an hypoxia-induced improvement in central motor drive resulting from AT may improve musculo-skeletal stiffness regulation (*i.e.*, less energy wasted on braking forces and minimal vertical oscillation of the center of mass), leading to a faster stride frequency and thereby improved sea-level repeated-sprint performance.

2.2. Recovery between efforts

VO_{2max}

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 a high aerobic fitness is important for team-sport success. The most widely-accepted measure of aerobic fitness is the VO_{2max}, which represents the maximum rate at which aerobic metabolism can supply energy [65]. In support of the importance of VO_{2max}, studies have reported a correlation between VO_{2max} and distance covered during team sports [66-68]. It has also been reported that participants with a greater VO_{2max} are better able to maintain power outputs/sprint times during repeated-sprint exercise, and that there are moderate correlations ($r = -0.20$ to -0.75), not always significant, between VO_{2max} and performance drop-off indices [69-77]. While some studies have reported increases in VO_{2max} following AT [78-80], this is not a universal finding - especially in well-trained athletes [34 81].

As indicated by the Fick equation, VO_{2max} is determined by both central and peripheral factors. To date, however, there has been limited research investigating the relationship between the central and peripheral determinants of VO_{2max} and team-sport physical performance. In one of the few studies, McMahon [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_{2max} and team-sport physical performance appear to be peripherally located [82]. In particular, the importance of the peripheral component of VO_{2max} is highlighted by the similar relationship between both the arteriovenous oxygen difference (a-VO_2 diff) and VO_{2max}, and the ability to maintain power output during brief, intermittent sprints [82]. This suggests that adaptations at the tissue level (e.g., 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 (EPO) resulted 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 [78]. In contrast, research suggests that short-duration (< 4 wk), “live high-train low” AT protocols do not increase capillarisation [87-89]. However, training under normobaric hypoxic compared 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 wk), but not short-term (< 4 wk) [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 to 4000 m) has been reported to result in greater increases in citrate synthase activity [91 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 (Hbmass) 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 Hbmass [102], team-sport athletes may be more likely to present increased haemoglobin mass in response to altitude training than elite cyclists. Even though increases in Hbmass do not necessarily lead to improvements in VO_{2max} [99], there may be benefits for aerobic metabolism via the compensatory decrease in blood flow which may slow 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_{2max} 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 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 both single 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 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 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 PCr resynthesis rate following AT are likely to closely reflect mitochondrial adaptations (which have been equivocal to date and also require further research).

Buffer capacity

In contrast to the good evidence that both VO_{2max} and PCr resynthesis rate are important determinants of team-sport physical performance, the importance of hydrogen ion (H^+) buffering is more controversial. A number of studies [118-123], but not all [124 125], have reported that increasing blood buffer capacity is likely to improve both repeated and intermittent sprint performance. However, the importance of muscle buffer capacity (βm) is less convincing. Despite a persistent low muscle pH, sprint power output has been reported to partially recover six minutes 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 [104]. Similarly, previous studies have shown that sprinting abilities were restored faster than muscle pH [106 126], and that the decline in sprint performance during a football (soccer) match was not correlated with muscle pH [10]. There has been one study that has reported a moderate correlation between muscle buffer capacity and RSA [71], but no studies to our knowledge that have correlated βm with team-sport physical performance.

Another way to assess the importance of muscle buffer capacity 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) [127], an important muscle buffer that has been estimated to account for ~ 10% of the total buffering capacity in the human vastus lateralis muscle [128]. β-alanine supplementation has been reported to improve yo-yo test performance [129] (a test that correlates well with match physical performance in soccer players [130 131]), but not intermittent sprint performance [132]. Thus, while there is some evidence that βm may influence team-sport performance, more research is required.

To date, five studies have investigated changes in βm in response to various forms of AT (with an average increase of ~ 7%; range = 0-18%) [35 41 87 133-135]. However, the response is quite variable with both the smallest and the largest changes in βm reported following very similar altitude-training protocols by the same research group [133 134]. 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 [136 137]. It is therefore difficult, based on current evidence, to justify the expenses associated with AT if the goal is to maximize improvements in βm.

3.0 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 and 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 physical 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 (e.g., “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 both match-related physical performance and the different physiological determinants of team-sport physical performance identified in this review.

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.

References

1. Rampinini E, Impellizzeri FM, Castagna C, et al. Effect of match-related fatigue on short-passing ability in young soccer players. Med. Sci. Sports Exerc. 2008;**40**(5):934-42 doi: 10.1249/MSS.0b013e3181666eb8[published Online First: Epub Date]|.
2. Rampinini E, Impellizzeri FM, Castagna C, et al. Technical performance during soccer matches of the Italian Serie A league: effect of fatigue and competitive level. J. Sci. Med. Sport 2009;**12**(1):227-33 doi: 10.1016/j.jsams.2007.10.002[published Online First: Epub Date]|.
3. Spencer M, Bishop D, Dawson B, et al. Physiological and metabolic responses of repeated-sprint activities: specific to field-based team sports. Sports Med. 2005;**35**:1025-44
4. Spencer M, Lawrence S, Rechichi C, et al. Time-motion analysis of elite field hockey, with special reference to repeated-sprint activity. Journal of sports science 2004;**22**(9):843-50
5. Spencer M, Rechichi C, Lawrence S, et al. Time-motion analysis of elite field hockey during several games in succession: A tournament scenario. J. Sci. Med. Sport 2005;**8**(4):382-91
6. Gabbett TJ. Science of rugby league football: a review. J. Sports Sci. 2005;**23**(9):961-76 doi: 10.1080/02640410400023381[published Online First: Epub Date]|.
7. Bangsbo J, Mohr M, Krustrup P. Physical and metabolic demands of training and match-play in the elite football player. J. Sports Sci. 2006;**24**(7):665-74
8. Aughey RJ. Increased high-intensity activity in elite Australian football finals matches. Int J Sports Physiol Perform 2011;**6**(3):367-79
9. Aughey RJ. Widening margin in activity profile between elite and sub-elite Australian football: A case study. J. Sci. Med. Sport 2013;**16**(4):382-6 doi: 10.1016/j.jsams.2012.10.003[published Online First: Epub Date]|.
10. Krustrup P, Mohr M, Steensberg A, et al. Muscle and Blood Metabolites during a Soccer Game: Implications for Sprint Performance. Med. Sci. Sports Exerc. 2006;**38**(6):1165-74
11. Billaut F, Gore CJ, Aughey RJ. Enhancing team-sport athlete performance: is altitude training relevant? Sports Med. 2012;**42**(9):751-67 doi: 10.2165/11634050-000000000-00000[published Online First: Epub Date]|.
12. Levine BD, Stray-Gundersen J, Mehta RD. Effect of altitude on football performance. Scand. J. Med. Sci. Sports 2008;**18 Suppl 1**:76-84 doi: 10.1111/j.1600-0838.2008.00835.x[published Online First: Epub Date]|.
13. Gabbett T, King T, Jenkins D. Applied physiology of rugby league. Sports Med. 2008;**38**(2):119-38 doi: Doi 10.2165/00007256-200838020-00003[published Online First: Epub Date]|.
14. Nedelec M, McCall A, Carling C, et al. Recovery in Soccer Part I - Post-Match Fatigue and Time Course of Recovery. Sports Med. 2012;**42**(12):997-1015
15. Faude O, Koch T, Meyer T. Straight sprinting is the most frequent action in goal situations in professional football. J. Sports Sci. 2012;**30**(7):625-31 doi: 10.1080/02640414.2012.665940[published Online First: Epub Date]|.
16. Paton CD, Hopkins WG, Vollebregt L. Little effect of caffeine ingestion on repeated sprints in team-sport athletes. Med. Sci. Sports Exerc. 2001;**33**(5):822-5
17. Mendez-Villanueva A, Hamer P, Bishop D. Fatigue responses during repeated sprints matched for initial mechanical output. Med. Sci. Sports Exerc. 2007;**39**(12):2219-25
18. Mendez-Villanueva A, Hamer P, Bishop D. Fatigue in repeated-sprint exercise is related to muscle power factors and reduced neuromuscular activity. Eur. J. Appl. Physiol. 2008;**103**:411–19
19. Bishop DJ. Fatigue during intermittent-sprint exercise. Clin. Exp. Pharmacol. Physiol. 2012;**39**(9):836-41 doi: 10.1111/j.1440-1681.2012.05735.x[published Online First: Epub Date]|.

20. Billaut F, Bishop DJ. Mechanical work accounts for sex differences in fatigue during repeated sprints. Eur. J. Appl. Physiol. 2012;**112**(4):1429-36 doi: 10.1007/s00421-011-2110-1[published Online First: Epub Date]|.
21. Rampinini E, Bishop D, Marcora SM, et al. Validity of simple field tests as indicators of match-related physical performance in top-level professional soccer players. Int. J. Sports Med. 2007;**28**(3):228-35
22. Karvonen J, Peltola E, Saarela J, et al. Changes in Running Speed, Blood Lactic-Acid Concentration and Hormone Balance during Sprint Training Performed at an Altitude of 1860 Meters. J. Sports Med. Phys. Fitness 1990;**30**(2):122-26
23. Nummela A, Rusko H. Acclimatization to altitude and normoxic training improve 400-m running performance at sea level. J. Sports Sci. 2000;**18**(6):411-9 doi: 10.1080/02640410050074340[published Online First: Epub Date]|.
24. Ross A, Leveritt M, Riek S. Neural influences on sprint running: training adaptations and acute responses. Sports Med. 2001;**31**(6):409-25
25. Pereira J, Sargeant AJ, Rademaker A, et al. Myosin heavy chain isoform expression and high energy phosphate content in human muscle fibres at rest and post-exercise. J. Physiol. 996;**496(2)**:583-88
26. Gaitanos GC, Williams C, Boobis LH, et al. Human muscle metabolism during intermittent maximal exercise. J. Appl. Physiol. 1993;**75(2)**:712-19
27. Hirvonen J, Rehunen S, Rusko H, et al. Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. Eur. J. Appl. Physiol. 1987;**56**:253-59
28. McLellan TM, Kavanagh MF, Jacobs I. The effect of hypoxia on performance during 30 s or 45 s of supramaximal exercise. Eur J Appl Physiol Occup Physiol 1990;**60**(2):155-61
29. Grassi B, Mognoni P, Marzorati M, et al. Power and peak blood lactate at 5050 m with 10 and 30 s 'all out' cycling. Acta Physiol. Scand. 2001;**172**(3):189-94 doi: 10.1046/j.1365-201x.2001.00857.x[published Online First: Epub Date]|.
30. Calbet JAL, De Paz JA, Garatachea N, et al. Anaerobic energy provision does not limit Wingate exercise performance in endurance-trained subjects. J. Appl. Physiol. 2003;**94**:668-76
31. Weyand PG, Lee CS, Martinez-Ruiz R, et al. High-speed running performance is largely unaffected by hypoxic reductions in aerobic power. J. Appl. Physiol. 1999;**86**(6):2059-64
32. Ogawa T, Ohba K, Nabekura Y, et al. Intermittent short-term graded running performance in middle-distance runners in hypobaric hypoxia. European Journal of Applied Physiology. 2005;**94**(3):254-61 doi: DOI 10.1007/s00421-005-1322-7[published Online First: Epub Date]|.
33. Medbo JI, Burgers S. Effect of training on the anaerobic capacity. Med. Sci. Sports Exerc. 1990;**22**(4):501-07
34. Roberts AD, Clark SA, Townsend NE, et al. Changes in performance, maximal oxygen uptake and maximal accumulated oxygen deficit after 5, 10 and 15 days of live high:train low altitude exposure. Eur. J. Appl. Physiol. 2003;**88**(4-5):390-5 doi: 10.1007/s00421-002-0720-3[published Online First: Epub Date]|.
35. Mizuno M, Juel C, Bro-Rasmussen T, et al. Limb skeletal muscle adaptation in athletes after training at altitude. J. Appl. Physiol. 1990;**68(2)**:496-502
36. Harmer AR, McKenna MJ, Sutton JR, et al. Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. J. Appl. Physiol. 2000;**89**:1793-803
37. Bishop D, Edge J, Thomas C, et al. Effects of high-intensity training on muscle lactate transporters and postexercise recovery of muscle lactate and hydrogen ions in women. Am J Physiol Regul Integr Comp Physiol 2008;**295**(6):R1991-98

38. Mohr M, Krustrup P, Nielsen JJ, et al. Effect of two different intense training regimens on skeletal muscle ion transport proteins and fatigue development. Am J Physiol Regul Integr Comp Physiol 2007;**292**(4):R1594-602 doi: 10.1152/ajpregu.00251.2006[published Online First: Epub Date]|.
39. Medbo JI, Mohn AC, Tabata I, et al. Anaerobic capacity determined by maximal accumulated O2 deficit. J. Appl. Physiol. 1988;**64**(1):50-60
40. Puype J, Van Proeyen K, Raymackers J, et al. Sprint interval training in hypoxia stimulates glycolytic enzyme activity. Medicine and science in sport and exercise 2013;**In press** doi: 10.1249/MSS.obo13e31829734ae[published Online First: Epub Date]|.
41. Basset FA, Joanisse DR, Boivin F, et al. Effects of short-term normobaric hypoxia on haematology, muscle phenotypes and physical performance in highly trained athletes. Exp. Physiol. 2006;**91**(2):391-402 doi: 10.1113/expphysiol.2005.031682[published Online First: Epub Date]|.
42. Terrados N, Melichna J, Sylven C, et al. Effects of training at simulated altitude on performance and muscle metabolic capacity in competitive road cyclists. Eur J Appl Physiol Occup Physiol 1988;**57**(2):203-9
43. Ogura Y, Katamoto S, Uchimaru J, et al. Effects of low and high levels of moderate hypoxia on anaerobic energy release during supramaximal cycle exercise. In addition to a high aerobic fitness, the ability to buffer hydrogen ions (H+) may also be important for repeated-sprint ability (RSA). We therefore investigated the relationship between muscle buffer capacity (be 2006;**98**(1):41-47 doi: DOI 10.1007/s00421-006-0214-9[published Online First: Epub Date]|.
44. Rhea MR. Determining the magnitude of treatment effects in strength training research through the use of the effect size. Castagna, C., V. Manzi, S. D'Ottavio, G. Annino,E. Padua, and D. Bishop. Relation between maximal aerobic power and the ability to repeat sprints in young basketball players. J. Strength Cond. Res. 21(4):117 2004;**18(4)**:918-20
45. Nishimura A, Sugita M, Kato K, et al. Hypoxia Increases Muscle Hypertrophy Induced by Resistance Training. Invited CommentaryAs sports scientists, we claim to make a significant contribution to the body of knowledge that influences athletic practice and performance. Is this the reality? At the inaugu 2010;**5**(4):497-508
46. Manimmanakorn A, Manimmanakorn N, Taylor R, et al. Effects of resistance training combined with vascular occlusion or hypoxia on neuromuscular function in athletes. European Journal of Applied Physiology. 2013;**113**(7):1767-74 doi: DOI 10.1007/s00421-013-2605-z[published Online First: Epub Date]|.
47. Kon M, Ikeda T, Homma T, et al. Effects of acute hypoxia on metabolic and hormonal responses to resistance exercise. Med. Sci. Sports Exerc. 2010;**42**(7):1279-85 doi: 10.1249/MSS.0b013e3181ce61a5[published Online First: Epub Date]|.
48. Melissa L, MacDougall JD, Tarnopolsky MA, et al. Skeletal muscle adaptations to training under normobaric hypoxic versus normoxic conditions. Med. Sci. Sports Exerc. 1997;**29**(2):238-43
49. Kawada S, Ishii N. Skeletal muscle hypertrophy after chronic restriction of venous blood flow in rats. Med. Sci. Sports Exerc. 2005;**37**(7):1144-50
50. Friedmann B, Kinscherf R, Borisch S, et al. Effects of low-resistance/high-repetition strength training in hypoxia on muscle structure and gene expression. Pflug Arch Eur J Phy 2003;**446**(6):742-51 doi: DOI 10.1007/s00424-003-1133-9[published Online First: Epub Date]|.
51. Manimmanakorn A, Hamlin MJ, Ross JJ, et al. Effects of low-load resistance training combined with blood flow restriction or hypoxia on muscle function and performance in netball athletes. J. Sci. Med. Sport 2013;**16**(4):337-42 doi: 10.1016/j.jsams.2012.08.009[published Online First: Epub Date]|.
52. Morin JB, Edouard P, Samozino P. Technical ability of force application as a determinant factor of sprint performance. Med. Sci. Sports Exerc. 2011;**43**(9):1680-8 doi: 10.1249/MSS.0b013e318216ea37[published Online First: Epub Date]|.

53. Komi PV. Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. J. Biomech. 2000;**33**(10):1197-206
54. Young W, McLean B, Ardagna J. Relationship between strength qualities and sprinting performance. Sports Med. Phys. Fitness 1995;**35(1)**:13-19
55. Hamlin MJ, Hinckson EA, Wood MR, et al. Simulated rugby performance at 1550-m altitude following adaptation to intermittent normobaric hypoxia. J. Sci. Med. Sport 2008;**11**(6):593-9 doi: 10.1016/j.jsams.2007.07.005[published Online First: Epub Date]|.
56. Arsac LM. Effects of altitude on the energetics of human best performances in 100 m running: a theoretical analysis. Eur. J. Appl. Physiol. 2002;**87**(1):78-84 doi: 10.1007/s00421-002-0587-3[published Online First: Epub Date]|.
57. Peronnet F, Thibault G, Cousineau DL. A theoretical analysis of the effect of altitude on running performance. J. Appl. Physiol. 1991;**70**(1):399-404
58. Girard O, Micallef JP, Millet GP. Changes in spring-mass model characteristics during repeated running sprints. European Journal of Applied Physiology. 2011;**111**(1):125-34 doi: DOI 10.1007/s00421-010-1638-9[published Online First: Epub Date]|.
59. Girard O, Racinais S, Kelly L, et al. Repeated sprinting on natural grass impairs vertical stiffness but does not alter plantar loading in soccer players. European Journal of Applied Physiology. 2011;**111**(10):2547-55 doi: DOI 10.1007/s00421-011-1884-5[published Online First: Epub Date]|.
60. Smith KJ, Billaut F. Influence of cerebral and muscle oxygenation on repeated-sprint ability. Eur. J. Appl. Physiol. 2010;**109**(5):989-99 doi: 10.1007/s00421-010-1444-4[published Online First: Epub Date]|.
61. Bowtell JL, Cooke K, R T. Acute physiological and performance responses to repeated sprints in varying degrees of hypoxia. J. Sci. Med. Sport 2013;**In Press**
62. Amann M, Romer LM, Subudhi AW, et al. Severity of arterial hypoxaemia affects the relative contributions of peripheral muscle fatigue to exercise performance in healthy humans. J Physiol-London 2007;**581**(1):389-403 doi: DOI 10.1113/j.physiol.2007.129700[published Online First: Epub Date]|.
63. Hogan MC, Richardson RS, Haseler LJ. Human muscle performance and PCr hydrolysis with varied inspired oxygen fractions: a 31P-MRS study. J. Appl. Physiol. 1999;**86**(4):1367-73
64. Amann M, Goodall S, Twomey R, et al. Altitudeomics: On the Consequences of High Altitude Acclimatization for the Development of Fatigue during Locomotor Exercise in Humans. J. Appl. Physiol. 2013 doi: 10.1152/japplphysiol.00606.2013[published Online First: Epub Date]|.
65. Tomlin DL, Wenger HA. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. Sports Med. 2001;**31(1)**:1-11
66. Bangsbo J, Lindquist F. Comparison of various exercise tests with endurance performance during soccer in professional players. Int. J. Sports Med. 1992;**13**(2):125-32 doi: 10.1055/s-2007-1021243[published Online First: Epub Date]|.
67. Castagna C, D'Ottavio S. Effect of maximal aerobic power on match performance in elite soccer referees. J Strength Cond Res 2001;**15**(4):420-5
68. Bangsbo J. Energy demands in competitive soccer. J. Sports. Sci. 1994;**12**:S5-S12
69. Aziz AR, Chia M, Teh KC. The relationship between maximal oxygen uptake and repeated sprint performance indices in field hockey and soccer players. Sports Med. Phys. Fitness 2000;**40**:195-200
70. Aziz AR, Mukherjee S, Chia MY, et al. Relationship between measured maximal oxygen uptake and aerobic endurance performance with running repeated sprint ability in young elite soccer players. J. Sports Med. Phys. Fitness 2007;**47**(4):401-7

71. Bishop D, Edge J, Goodman C. Muscle buffer capacity and aerobic fitness are associated with repeated-sprint ability in women. Eur. J. Appl. Physiol. 2004;**92**:540-47
72. Bishop D, Spencer M. Determinants of repeated-sprint ability in well-trained team-sport athletes and endurance-trained athletes. J. Sports Med. Phys. Fitness 2004;**44**(1):1-7
73. Bishop D, Edge J. Determinants of repeated-sprint ability in females matched for single-sprint performance. European Journal of Applied Physiology. 2006;**97**(4):373-79
74. Dawson B, FitzSimons M, Ward D. The relationship of repeated sprint ability to aerobic power and performance measures of anaerobic work capacity and power. Aust. J. Sci. Med. Sport 1993;**25(4)**:88-93
75. Bishop D, Girard O, Mendez-Villanueva A. Repeated-sprint ability - part II: recommendations for training. Sports Med. 2011;**41**(9):741-56 doi: 10.2165/11590560-000000000-000004 [pii][published Online First: Epub Date]|.
76. Girard O, Mendez-Villanueva A, Bishop D. Repeated-sprint ability - part I: factors contributing to fatigue. Sports Med. 2011;**41**(8):673-94 doi: 10.2165/11590550-000000000-000004 [pii][published Online First: Epub Date]|.
77. Fernandes da Silva J, Guglielmo LGA, Bishop D. Relationship between different measures of aerobic fitness and repeated-sprint ability in elite soccer players. Castagna, C., V. Manzi, S. D'Ottavio, G. Annino,E. Padua, and D. Bishop. European Journal of Applied Physiology. 2010;**24**(8):2115-21
78. Levine BD, Stray-Gundersen J. "Living high-training low": effect of moderate-altitude acclimatization with low-altitude training on performance. J. Appl. Physiol. 1997;**83**(1):102-12
79. Stray-Gundersen J, Chapman RF, Levine BD. "Living high-training low" altitude training improves sea level performance in male and female elite runners. J. Appl. Physiol. 2001;**91**(3):1113-20
80. Clark SA, Quod MJ, Clark MA, et al. Time course of haemoglobin mass during 21 days live high:train low simulated altitude. Eur. J. Appl. Physiol. 2009;**106**(3):399-406 doi: 10.1007/s00421-009-1027-4[published Online First: Epub Date]|.
81. Gore CJ, Hahn A, Rice A, et al. Altitude training at 2690m does not increase total haemoglobin mass or sea level VO2max in world champion track cyclists. J. Sci. Med. Sport 1998;**1**(3):156-70
82. McMahon S, Wenger HA. The relationship between aerobic fitness and both power output and subsequent recovery during maximal intermittent exercise. J. Sci. Med. Sport 1998;**1(4)**:219-27
83. Faiss R, Leger B, Vesin JM, et al. Significant Molecular and Systemic Adaptations after Repeated Sprint Training in Hypoxia. Plos One 2013;**8**(2) doi: ARTN e56522DOI 10.1371/journal.pone.0056522[published Online First: Epub Date]|.
84. Thomas C, Sirvent P, Perrey S, et al. Relationships between maximal muscle oxidative capacity and blood lactate removal after supramaximal exercise and fatigue indexes in humans. J. Appl. Physiol. 2004;**97**(6):2132-38 doi: 10.1152/japplphysiol.00387.2004[published Online First: Epub Date]|.
85. Tesch PA, Wright JE. Recovery from short-term exercise: its relation to capillary supply and blood lactate concentrations. Eur. J. Appl. Physiol. 1983;**53**:98-103
86. Balsom P, Ekblom B, Sjodin B. Enhanced oxygen availablility during high intensity intermittent exercise decreases anaerobic metabolite concentration in blood. Acta Physiol. Scand. 1994;**150**:455-56
87. Mizuno M, Savard GK, Areskog NH, et al. Skeletal muscle adaptations to prolonged exposure to extreme altitude: a role of physical activity? High altitude medicine & biology 2008;**9**(4):311-7 doi: 10.1089/ham.2008.1009[published Online First: Epub Date]|.
88. Green HJ. Muscular adaptations at extreme altitude: metabolic implications during exercise. Int. J. Sports Med. 1992;**13 Suppl 1**:S163-5 doi: 10.1055/s-2007-1024627[published Online First: Epub Date]|.

89. Green H, Roy B, Grant S, et al. Human skeletal muscle exercise metabolism following an expedition to mount denali. American Journal of Physiology - Regulatory Integrative & Comparative Physiology. 2000;**279**(5):R1872-9
90. Geiser J, Vogt M, Billeter R, et al. Training high--living low: changes of aerobic performance and muscle structure with training at simulated altitude. Int. J. Sports Med. 2001;**22**(8):579-85 doi: 10.1055/s-2001-18521[published Online First: Epub Date]|.
91. Melissa L, MacDougall JD, Tarnopolsky MA, et al. Skeletal muscle adaptations to training under normobaric hypoxic versus normoxic conditions. Medicine & Science in Sports & Exercise. 1997;**29**(2):238-43
92. Jacobs RA, Siebenmann C, Hug M, et al. Twenty-eight days at 3454-m altitude diminishes respiratory capacity but enhances efficiency in human skeletal muscle mitochondria. FASEB J. 2012;**26**(12):5192-200 doi: 10.1096/fj.12-218206[published Online First: Epub Date]|.
93. Jacobs RA, Boushel R, Wright-Paradis C, et al. Mitochondrial function in human skeletal muscle following high-altitude exposure. Exp. Physiol. 2013;**98**(1):245-55 doi: 10.1113/expphysiol.2012.066092[published Online First: Epub Date]|.
94. Levett DZ, Radford EJ, Menassa DA, et al. Acclimatization of skeletal muscle mitochondria to high-altitude hypoxia during an ascent of Everest. FASEB J. 2012;**26**(4):1431-41 doi: 10.1096/fj.11-197772[published Online First: Epub Date]|.
95. Hoppeler H, Vogt M, Weibel ER, et al. Response of skeletal muscle mitochonrial to hypoxia. Exp. Physiol. 2003;**88**(1):109-19
96. Terrados N, Jansson E, Sylven C, et al. Is hypoxia a stimulus for synthesis of oxidative enzymes and myoglobin? J. Appl. Physiol. 1990;**68**(6):2369-72
97. Tonkonogi M, Sahlin K. Rate of oxidative phosphorylation in isolated mitochondria from human skeletal muscle: effect of training status. Acta Physiol. Scand. 1997;**161**(3):345-53
98. Larsen S, Nielsen J, Hansen CN, et al. Biomarkers of mitochondrial content in skeletal muscle of healthy young human subjects. J Physiol 2012;**590**(Pt 14):3349-60 doi: 10.1113/jphysiol.2012.230185[published Online First: Epub Date]|.
99. Gore CJ, Clark SA, Saunders PU. Nonhematological mechanisms of improved sea-level performance after hypoxic exposure. Med. Sci. Sports Exerc. 2007;**39**(9):1600-9 doi: 10.1249/mss.0b013e3180de49d3[published Online First: Epub Date]|.
100. Siebenmann C, Robach P, Jacobs RA, et al. "Live high-train low" using normobaric hypoxia: a double-blinded, placebo-controlled study. J. Appl. Physiol. 2012;**112**(1):106-17 doi: 10.1152/japplphysiol.00388.2011[published Online First: Epub Date]|.
101. Green HJ. Altitude acclimatization, training and performance. J. Sci. Med. Sport 2000;**3**(3):299-312
102. Robach P, Siebenmann C, Jacobs RA, et al. The role of haemoglobin mass on VO(2)max following normobaric 'live high-train low' in endurance-trained athletes. Br. J. Sports Med. 2012;**46**(11):822-7 doi: 10.1136/bjsports-2012-091078[published Online First: Epub Date]|.
103. Saltin B, Kiens B, Savard G, et al. Role of hemoglobin and capillarization for oxygen delivery and extraction in muscular exercise. Acta Physiol. Scand. Suppl. 1986;**556**:21-32
104. Mendez-Villanueva A, Edge J, Suriano R, et al. The recovery of repeated-sprint exercise is associated with PCr resynthesis, while muscle pH and EMG amplitude remain depressed. PLoS One 2012;**7**(12):e51977 doi: 10.1371/journal.pone.0051977[published Online First: Epub Date]|.
105. Bogdanis GC, Nevill ME, Boobis LH, et al. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. J. Appl. Physiol. 1996;**80**(3):876-84
106. Bogdanis GC, Nevill ME, Boobis LH, et al. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. J. Physiol. (Lond.) 1995;**482**:467-80
107. Spencer M, Bishop D, Dawson B, et al. Metabolism and performance in repeated cycle sprints: active versus passive recovery. Med. Sci. Sports Exerc. 2006;**38**(8):1492-9

108. Spencer M, Dawson B, Goodman C, et al. Performance and metabolism in repeated sprint exercise: effect of recovery intensity. Eur. J. Appl. Physiol. 2008;**103**(5):545-52
109. Trump ME, Heigenhauser GJF, Putman CT, et al. Importance of muscle phosphocreatine during intermittent maximal cycling. J. Appl. Physiol. 1996;**80(5)**:1574-80
110. Greenhaff PL, Bodin K, Soderlund K, et al. Effect of oral creatine supplementation on skeletal muscle phosphocreatine resynthesis. Am. J. Physiol. 1994;**266**:E725-E30
111. Skare OC, Skadberg O, Wisnes AR. Creatine supplementation improves sprint performance in male sprinters. Scand. J. Med. Sci. Sports 2001;**11**:96-102
112. Preen D, Dawson B, Goodman C, et al. The effect of oral creatine supplementation on 80 minutes of repeated-sprint exercise. Med. Sci. Sports Exerc. 2001;**33**(5):814-25
113. Wiroth J, Bermon S, Andrei S, et al. Effects of oral creatine supplementation on maximal pedalling performance in older adults. Eur. J. Appl. Physiol. 2001;**84**:533-39
114. van Loon LJC, Oosterlaar AM, Hartgens F, et al. Effects of creatine loading and prolonged creatine supplementation on body composition, fuel selection, sprint and endurance performance in humans. Clin. Sci. 2003;**104**(2):153-62
115. Cox G, Mujika I, Tumilty D, et al. Acute creatine supplementation and performance during a field test simulating match play in elite female soccer players. Int J Sport Nutr Exerc Metab 2002;**12**(1):33-46
116. McCully KK, Fielding RA, Evans WJ, et al. Relationships between in vivo and in vitro measurements of metabolism in young and old human calf muscles. J. Appl. Physiol. 1993;**75**:813-19
117. Radda GK, Bore PJ, Gadian DG, et al. 31P NMR examination of two patients with NADH-CoQ reductase deficiency. Nature 1982;**295**(5850):608-9
118. Bishop D, Claudius B. Effects of induced metabolic alkalosis on prolonged intermittent-sprint performance. Med. Sci. Sports Exerc. 2005;**37**(5):759-67
119. Bishop D, Edge J, Davis C, et al. Induced metabolic alkalosis affects muscle metabolism and repeated-sprint ability. Med. Sci. Sports Exerc. 2004;**36**(5):807-13
120. Lavender G, Bird SR. Effect of sodium bicarbonate ingestion upon repeated sprints. Br. J. Sports Med. 1989;**23(1)**:41-45
121. Price M, Moss P, Rance S. Effects of sodium bicarbonate ingestion on prolonged intermittent exercise. Med. Sci. Sports Exerc. 2003;**35(8)**:1303-08
122. Bishop D. Dietary supplements and team-sport performance. Sports Med. 2010;**40**(12):995-1017 doi: 10.2165/11536870-000000000-000002 [pii][published Online First: Epub Date]|.
123. Edge J, Bishop D, Hill-Haas S, et al. Comparison of muscle buffer capacity and repeated-sprint ability of untrained, endurance-trained and team-sport athletes. Eur. J. Appl. Physiol. 2006;**96**(3):225-34
124. Gaitanos GC, Nevill ME, Brooks S, et al. Repeated bouts of sprint running after induced alkalosis. J. Sports Sci. 1991;**9**:355-69
125. Tan F, Polglaze T, Cox G, et al. Effects of induced alkalosis on simulated match performance in elite female water polo players. Int J Sport Nutr Exerc Metab 2010;**20**(3):198-205
126. Bogdanis GC, Nevill ME, Lakomy HKA, et al. Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. Acta Physiol. Scand. 1998;**163**:261-72
127. Harris RC, Tallon MJ, Dunnett M, et al. The absorption of orally supplied β-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. Amino Acids 2006;**30**(3):279-89
128. Hill CA, Harris RC, Kim HJ, et al. Influence of β-alanine supplementation on skeletal muscle carnosine concentrations and high intensity cycling capacity. Amino Acids 2007;**32**(2):225-33

129. Saunders B, Sunderland C, Harris RC, et al. beta-alanine supplementation improves YoYo intermittent recovery test performance. J Int Soc Sports Nutr 2012;**9**(1):39 doi: 10.1186/1550-2783-9-39[published Online First: Epub Date]|.
130. Krustrup P, Mohr M, Amstrup T, et al. The yo-yo intermittent recovery test: physiological response, reliability, and validity.[see comment]. Medicine & Science in Sports & Exercise. 2003;**35**(4):697-705
131. Krustrup P, Mohr M, Ellingsgaard H, et al. Physical demands during an elite female soccer game: Importance of training status. Med. Sci. Sports Exerc. 2005;**37(7)**:1242-48
132. Sweeney KM, Wright GA, Glenn Brice A, et al. The effect of beta-alanine supplementation on power performance during repeated sprint activity. J Strength Cond Res 2010;**24**(1):79-87 doi: 10.1519/JSC.0b013e3181c63bd5[published Online First: Epub Date]|.
133. Gore CJ, Hahn AG, Aughey RJ, et al. Live high:train low increases muscle buffer capacity and submaximal cycling efficiency. Acta Physiol. Scand. 2001;**173**:275-86
134. Clark S, Aughey R, Gore CJ, et al. Effects of live high, train low hypoxic exposure on lactate metabolism in trained humans. J. Appl. Physiol. 2004;**96**:517-25
135. Saltin B, Kim CK, Terrados N, et al. Morphology, enzyme activities and buffer capacity in leg muscles of Kenyan and Scandinavian runners. Scand. J. Med. Sci. Sports 1995;**5**:222-30
136. Edge J, Bishop D, Goodman C. The effects of training intensity on muscle buffer capacity in females. European Journal of Applied Physiology. 2006;**96**(1):97 - 105
137. Edge J, Bishop D, Goodman C. Effects of chronic NaHCO3 ingestion during interval training on changes to muscle buffer capacity, metabolism, and short-term endurance performance. J. Appl. Physiol. 2006;**101**(3):918-25