Downloaded from bjsm.bmj.com on November 28, 2013 - Published by group.bmj.com

OPEN ACCESS

Update in the understanding of altitude-induced limitations to performance in team-sport athletes

François Billaut,[1] Robert J Aughey[2,3]

[1]Institut national du sport du Québec, Montréal, Québec, Canada
[2]Institute of Sport, Exercise and Active Living, Victoria University, Melbourne, Victoria, Australia
[3]Western Bulldogs Football Club, Melbourne, Victoria, Australia

Correspondence to
Dr François Billaut, Institut national du sport du Québec, 1000, avenue Émile-Journault —bureau 1.72, Montréal, QC, Canada H2M 2E7; fbillaut@insquebec.org

Received 29 June 2013
Revised 8 September 2013
Accepted 22 September 2013

ABSTRACT

The internationalism of field-based team sports (TS) such as football and rugby requires teams to compete in tournaments held at low to moderate altitude (~1200–2500 m). In TS, acceleration, speed and aerobic endurance are physical characteristics associated with ball possession and, ultimately, scoring. While these qualities are affected by the development of neuromuscular fatigue at sea level, arterial hypoxaemia induced by exposure to altitude may further hinder the capacity to perform consecutive accelerations (CAC) or sprint endurance and thereby change the outcome of a match. The higher the altitude, the more severe the hypoxaemia, and thus, the larger the expected decline in aerobic endurance, CAC and match running performance. Therefore, it is critical for athletes and coaches to understand how arterial hypoxaemia affects aerobic endurance and CAC and the magnitude of decline they may face at altitude for optimal preparation and increased chances of success. This mini review summarises the effects of acute altitude/hypoxia exposure on aerobic endurance, CAC and activity profiles of TS athletes performing in the laboratory and during matches at natural altitude, and analyses the latest findings about the consequences of arterial hypoxaemia on the relationship between peripheral perturbations, neural adjustments and performance during repeated sprints or CAC. Finally, we briefly discuss how altitude training can potentially help athletes prepare for competition at altitude.

INTRODUCTION

The internationalism of field-based team sports (TS) such as football and rugby means it is increasingly common for athletes to compete in tournaments that are held at low to moderate altitude (~1200–2500 m). For example, much of the 2010 FIFA World Cup host country, South Africa, is located at 1200 m with Johannesburg—the host city of the opening and final games—located at 1740 m. Furthermore, of the last 19 football World Cup tournaments since 1930, eight hosting countries were located at altitude, and the recent youth World Cup tournament for the under 17 (U17) as well as U20 age groups were held in moderate altitude venues (Mexico City, Mexico and Manizales, Colombia).

Field-based TS are largely endurance-based,[1–3] albeit interspersed with bouts of high-intensity activity and numerous explosive bursts requiring high speed and power.[4 5] Although the number of sprints (where players reach a high velocity, eg, greater than 6.94 m/s)[6] performed in matches is relatively low,[7 8] speed and acceleration qualities are associated with ball possession and, ultimately, scoring.[9] Laboratory studies have shown that when arterial hypoxaemia—defined as a 3% reduction in arterial blood O_2 saturation (SaO_2) below pre-exercise level[10] develops, several physiological responses are affected (eg, phosphocreatine (PCr) resynthesis, Na^+, K^+–ATPase enzyme activity, cerebral oxygenation and neural drive) that hinder the capacity to perform consecutive accelerations (CAC) and work at high intensity.[11–13] Although current field evidence is scarce,[14] this hypoxaemia-induced impairment in CAC is quite likely to limit TS match running performance at altitude. This presents a marked disadvantage for the 'away' team, especially if their normal place of residence is at sea level.[15]

Quantifying the extent of running performance decrement at low to moderate altitude would provide coaches with valuable knowledge to prepare TS athletes to better compete under hypoxic conditions. Therefore, the aims of this mini review are to (1) provide updates about the effects of acute altitude/hypoxia exposure on aerobic endurance, CAC and activity profiles of TS athletes performing in the laboratory and during matches, (2) analyse the latest findings about the consequences of arterial hypoxaemia on the complex relationship between peripheral perturbations, neural adjustments and performance regulation during repeated sprints, and (3) highlight the altitude-training methods that may help attenuate some of these decrements when competing at altitude. Such a review could be useful to guide the practice of many athletes around the world in maximising their preparation to cope with running performance decrement at altitude.

ACTIVITY PROFILES OF TS

Understanding the activity profiles of TS athletes and the bioenergetics systems that contribute to energy production during matches is necessary to comprehend the potentially negative effects of altitude on athletes' performances. This section only provides brief synopses of the key performance determinants during TS matches, which have been dealt with elsewhere in this supplement[16] and in previous reviews.[14 17]

Typical match movement characteristics

Movements in TS matches can be measured through Global Positioning System (GPS),[4] or other systems[2] and activity profile variables, expressed in metres per minute of match time (m/min), which typically include total distance, standing, walking, jogging, striding and sprinting.[2 4] Striding and sprinting are categorised as 'high-intensity activities', while standing, walking and jogging, which occur in between high-intensity bouts to recover from these bouts, are described as 'low-intensity

Open Access
Scan to access more free content

To cite: Billaut F, Aughey RJ. *Br J Sports Med* 2013;**47**:i22–i25.

Downloaded from bjsm.bmj.com on November 28, 2013 - Published by group.bmj.com

activities'.[7 18 19] Intensity, however, is dependent on an individual's perception of effort,[20 21] and 'activity' can include movements such as accelerating or leaping for ball possession. As this review focuses on movement characteristics, walking and jogging will be referred to as low-velocity running (LVR, 0.01–4.16 m/s), and striding and sprinting as high-velocity running (HiVR, 4.17–10 m/s). In addition, the frequency of maximal accelerations per minute (≥2.78 m/s^2) can be quantified.[8 22]

Soccer athletes, for example, regularly repeat short, high-intensity efforts, interspersed with longer intervals of rest or submaximal exercise over ≥30 min.[2] The typical distance covered by top-class centre-midfielders is ~9–13 km/match,[1 2] which is comprised of 70% low-intensity activity and ~150–250 brief intense actions (average sprint distances of ~10–20 m).[7 23 24] Australian footballers cover the highest distances in field sports,[4] up to 15 km in finals matches,[1] and undertake up to 30% of this distance as HiVR.[1 23] In addition to these locomotive activities, athletes must perform other energy-demanding activities (eg, jumping, moving while controlling the ball and tackling) during matches that require them to accelerate.[7 23 25 26] These high-intensity movements and accelerations often occur in response to cues such as ball movement or opponents' actions. Such movements are typically short and, when started from a low velocity, have not been traditionally accounted for in match analysis research.[8] In fact, analysis from elite soccer athletes reveals that 85% of accelerations (≥2.78 m/s^2) do not exceed the determined HiVR threshold.[8] Thus, in match analysis studies that only report movements occurring at high velocities, accelerations are often excluded. A recent study demonstrated a 13-fold increase in high-intensity actions over the course of a match when combining sprint bouts and accelerations in the analysis.[8] It is imperative to consider these short actions, as they are energetically the most challenging during a match[5] and thus contribute greatly to total energy expenditure in a match.[5]

Characteristics of elite team-sport athletes

Elite TS athletes must adapt to varied physical demands during the course of a match. Typically, athletes compete on average at 70% of their maximal aerobic O_2 uptake (VO_{2max}),[27] and cover an average distance of ~9–13 km/match.[7 23 24 27] This suggests that, in addition to the intermittent high-intensity nature of TS, high aerobic power is important for success. In fact, while elite TS athletes do not exhibit the specific physical/physiological capacities of elite endurance and sprint athletes, they do possess high levels of 'aerobic' and 'anaerobic' potentials.[14 17]

EXERCISING AND COMPETING AT ALTITUDE

Paradoxically, although TS athletes regularly compete at altitude, our knowledge of the effects of hypoxaemia on performance is almost exclusively based on endurance athletes performing endurance exercise.[28–33] Understanding what physical components and how activity profiles are affected by altitude in TS athletes specifically is critical, and this quest is supported, for example, by the FIFA.[34] To remain specific to TS, we report the studies that have used TS athletes exclusively as participants.

Match activity profiles at altitude

Owing to the large distance covered in games and the numerous accelerations to be performed, competing at altitude is likely to exacerbate fatigue in TS athletes and influence the outcome of matches. In fact, field data demonstrate large inequalities in the probability to win for a sea-level team playing against a home team at moderate/high altitude.[15]

The first study, to the best of our knowledge, to report altitude-induced performance decrement analysed match data from the official game statistics of the 2010 FIFA World Cup held in South Africa, where 64 matches were played by 105 teams at sea level, 660, 1200–1400 and 1401–1753 m.[35] The results show a 3.1% lower total distance covered by teams during matches played at 1200–1400 and 1401–1753 m, compared with those played at sea level. Interestingly, however, the players' top-running speed was not different across altitudes, suggesting that at least single high-intensity work capacity is well maintained during matches. More recent data collected in preparation for the 2011 FIFA U20 World Cup corroborate these findings, reporting that the total running distance and LVR were reduced by 9% and 8%, respectively, during matches played 4 days after arrival in Denver, Colorado (1600 m), compared with sea-level matches.[22] Interestingly, this study also indicates a 15% reduction in HiVR at altitude. Furthermore, a rolling 5 min sample period was used to identify the peak 5 min period of the match and the 5 min period occurring immediately after the peak 5 min period, which gives an indication of transiently lower output, possibly fatigue, during matches. With this technique, there was a 21% reduction in peak HiVR as well as HiVR in the subsequent 5 min stanza (−55%) at 1600 m than at sea level. While the frequency of accelerations performed during matches was not clearly affected at this low altitude, the acceleration frequency in the 5 min subsequent to the peak 5 min period dropped considerably (−51%) at 1600 m, suggesting transient neuromuscular fatigue.[22] Other data consolidate the finding that altitude hinders TS athletes' running performance during matches; the recent International Study on Soccer at Altitude 3600 m (ISA3600), in this supplement,[36] also reports compelling negative effects of high altitude on soccer players, independent of whether they are high-altitude or sea-level natives.

It is also worth considering that findings from controlled lab studies do not translate directly to match performance of players at altitude. TS are complex and chaotic, whereas laboratory studies are well controlled, with known work and rest durations. Participants in those studies can plan their physical output with some certainty. TS athletes, however, may preserve their capacity to perform the hardest actions, such as CAC, in matches by modulating the amount of low-intensity activity undertaken, especially in the face of environmental challenges like altitude.[36] Therefore, laboratory studies cannot accurately predict the changes in match activity that actually occur in TS situations. Keeping this in mind, it is therefore critical when considering the effects of, for example, altitude on match running that the running profile of players is measured *during* competition. Sporting regulatory bodies should allow these data to be collected in all matches to assist in our understanding.

Collectively, these first data collected on TS athletes competing at altitude are in good agreement with the scarce evidence available on *simulated* TS athlete performances at natural altitude. For example, compared with sea-level activity, the performance of rugby athletes during a 20 m shuttle run (−4%) and a rugby-specific repetitive power test (−16%) is decreased at 1550[37] and 1700 m, respectively.[38]

Laboratory studies

Owing to its high-energy demand, the skeletal muscle is sensitive to changes in partial pressure of O_2. Laboratory-based research indicates that a reduction in systemic O_2 delivery contributes to curtailment of CAC and repeated-sprint ability through varied metabolic and neuromuscular mechanisms. For

Billaut F, *et al*. *Br J Sports Med* 2013;**47**:i22–i25. doi:10.1136/bjsports-2013-092834

Downloaded from bjsm.bmj.com on November 28, 2013 - Published by group.bmj.com

example, total mechanical work during ten 10 s cycle sprints interspersed with 30 s of rest was reduced (−8%) in hypoxic (FiO_2 0.13, ~3600 m) versus normoxic conditions.[13] In hypoxia, a ~9-fold to 10-fold increase in deoxygenation of active muscles (assessed via near-infrared spectroscopy) occurs rapidly during the first repetition of a series of sprints.[13 39] However, as this deoxygenation quickly plateaus, and is relatively similar in normoxia and hypoxia, the results have been interpreted to mean that the level of muscle deoxygenation per se, and presumably muscle O_2 extraction, is unlikely to limit CAC.[13 39] On the other hand, the capacity of the muscle to get reoxygenated during recovery periods between efforts has been incriminated in the performance decline in hypoxia.[40] For instance, impaired muscle O_2 supply has been associated with hindered PCr resynthesis.[41] Interventions that reduce (eg, hypoxia) or prevent (eg, blood flow occlusion) O_2 supply to muscles reduce and prevent, respectively, PCr resynthesis.[42 43] Furthermore, the reoxygenation rate of the skeletal muscle has similar recovery kinetics to PCr after exercise.[44 45] Therefore, since PCr resynthesis is one of the main factors in performance during repeated efforts,[46 47] the slower repletion of this critical substrate is a key mechanism by which hypoxia impairs TS athletes' running performance at altitude.

Other studies have demonstrated greater anaerobic energy release and metabolic perturbations after single-sprint[11] and repeated-sprint[48 49] exercise in hypoxia. This suggests that sprinting in hypoxia leads to greater and/or earlier metabolic fatigue (and presumably muscle fatigue) than in normoxia. Another factor that could contribute to exacerbated fatigue and lower intermittent HiVR at altitude is the downregulation of the Na^+, K^+–ATPase in hypoxia.[50] For instance, when Na^+, K^+–ATPase are inhibited, the Na^+/K^+ leak-to-pump ratio is increased, muscle tetanic force is depressed and muscle fatigue is accelerated.[51] Furthermore, the influence of the build-up of energy metabolites such as inorganic phosphate and hydrogen ions on force development and calcium sensitivity, which is accelerated in hypoxia, has been well established during exercise.[51] Although no data are available, particularly on sprint exercise, it is quite likely that these mechanisms are at play during TS matches at altitude and contribute to hinder HiVR.

The effects of hypoxia on the nervous system function have been well described,[28] but once again, data collected from sprint exercise are very limited. A greater attenuation of the surface electromyographic activity (serving as a surrogate for the central motor drive to skeletal muscles) has been reported during repeated sprints in hypoxia.[13] Motor neuron activity can be affected via two mechanisms in hypoxia. For example, an indirect inhibitory effect of the sensory afferent feedback (groups III/IV muscle afferents) originating from the fatiguing locomotor muscles occurs during endurance exercise.[29 52] As mentioned above, the accelerated development of peripheral limb fatigue during sprints performed in hypoxia versus normoxia may strongly influence the regulation of central nervous system response and contribute to lower muscle recruitment.[13] There is little doubt that these peripheral influences are increasingly important at altitude and would exert a detrimental effect on LVR and HiVR over an extended period of time, such as during TS matches. However, the consequences of arterial hypoxaemia on the complex relationship between peripheral perturbations, muscle fatigue development, neural adjustments and performance have yet to be examined during repeated sprints in TS athletes in an intervention trial. There is also the possibility of a direct effect of brain hypoxia on muscle recruitment. For instance, motor neuron activity is dramatically influenced by O_2 availability,[53–55] and insufficient oxygenation of the cerebral cortex can impair neurotransmitter turnover and depress neuronal electrical activity,[56] which has been incriminated in the occurrence of central fatigue.[56 57] Of direct interest to TS athletes is that tests of repeated-sprint ability (10×10 s sprints with 30 s rest) in hypoxic conditions (FiO_2 0.13) result in larger cerebral deoxygenation compared to normoxia,[13 39] which leads to a larger decline in muscle recruitment in hypoxia.[13] Changes in cerebral deoxyhaemoglobin concentration explained 83% of the variance in electromyographic activity in hypoxia.[13] Therefore, it is possible that TS athletes playing at altitude exhibit such brain deoxygenation during high-intensity periods of a match that may contribute to curtailing their CAC.[22]

PREPARING TO FACE HYPOXIC CHALLENGES

The information gained by quantifying and explaining performance decrements at altitude should provide coaches and sport scientists with knowledge to better prepare TS athletes to compete at altitude. However, until now, scarce literature has discussed the potential benefits of altitude training in TS,[14 15] and caution should be exerted regarding the relevance of such regimens for improving players' specific fitness (ie, CAC). Below, we provide some information that can be used to optimise preparation of TS athletes facing hypoxic stress during matches. We focus specifically on CAC because it is a key fitness characteristic associated with scoring in matches[9] and because it is greatly affected (~50% decrement) by altitude.[22]

Maintaining CA capacity

Adaptations potentially favourable to high-intensity exercises and CAC may come from intermittent hypoxic training (IHT) in which athletes exercise in hypoxia.[14] The resynthesis of PCr—known as a key metabolic pathway for CAC[46 47] may be positively impacted by adding a hypoxic stimulus during training. In fact, IHT involving a single leg extension results in faster PCr resynthesis after high-intensity exercise than similar training in normoxia.[58] The authors hypothesised that the faster PCr resynthesis observed after IHT was probably due to an enhanced muscle O2 delivery. Since hypoxia impairs the muscle reoxygenation capacity, which is directly linked to PCr resynthesis,[44 45] it is possible that an IHT block that can accelerate the repletion of this substrate would benefit the recovery of power output and, therefore, CAC at altitude.

Compared with sea-level training, IHT induces additional metabolic stress[59] and provoke specific molecular adaptations.[60 61] The lower O_2 partial pressure in muscle tissue during IHT may induce a larger upregulation of the hypoxia-inducible factor 1á,[58 60 61] which has in turn been shown to increase the fibre cross-sectional area, oxidative enzyme activity, mitochondrial density and capillary/fibre ratio.[61–64] Recently, the idea was put forward that IHT involving sprint exercise would increase blood perfusion, thereby enhancing O_2 utilisation and improving the behaviour of fast-twitch fibres.[65] Molecular adaptations in muscle tissue together with higher blood perfusion (presumably via nitric oxide-mediated vasodilatation mechanisms)[66 67] after IHT could also positively influence sprint endurance in hypoxic environments and increase the chances of success for an 'away' team residing at sea level.

Downloaded from bjsm.bmj.com on November 28, 2013 - Published by group.bmj.com

Reducing the general impact of altitude

Teams aiming at reducing the general impact of hypoxia when travelling at altitude for a match should seek acclimatisation to the altitude of the match.[14 15] In this situation, the most effective strategy is likely to stage several days to weeks at the altitude of the match, particularly if travel across multiple time zones is also required.[15 37] For example, it was recently reported that the negative impact of an altitude of 1600 m on total distance covered during a match (−9.1%) and HiVR (−15.2%) in youth TS athletes was progressively reversed after only 6 days of residence at this low altitude (total distance: 6.2%, HiVR: 18.3%).[22] For moderate altitude (2000–3000 m), 1–2 weeks may be suitable to maximise physiological acclimatisation (eg, O_2 transport and acid–base balance),[68] although such data are not available in a population of TS athletes. Competition at high altitude (>3000 m) poses other challenges. However, there is evidence of incomplete acclimatisation in TS athletes after more than 2 weeks at altitude.[69 70]

CONCLUSIONS

There are numerous hypoxia-induced, negative physiological perturbations that suggest that TS athlete activity will be reduced at altitude. These perturbations are central (ie, within the central nervous system) and peripheral (ie, within the skeletal locomotor muscles) in nature, and both reduce CAC as well as exacerbate fatigue in repeated-sprint tasks at altitude. However, these studies do not have strong ecological validity for the reality of the chaotic nature of TS, where athletes may engage in strategies to protect the capacity to perform hard tasks. A combination of field and laboratory studies is therefore required to provide a clearer answer to the effects of altitude on field-based TS athletes.

What is known on this subject

- Acute exposure to altitude impairs aerobic capacity and endurance performance.
- Team-sport athletes compete in tournaments held at altitudes sufficient to impair physical performances.

What this study adds

- Altitude-induced arterial hypoxaemia hinders the capacity to perform consecutive sprints.
- Match running performance is lower at altitudes as low as 1200 m.
- Team-sport athletes competing at altitude should prepare for the challenges of hypoxaemia to increase their chances of success.

Contributors FB and RJA have both contributed to the writing and the editing of the manuscript.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

Open Access This is an Open Access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 3.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/3.0/

REFERENCES

1 Aughey RJ. Increased high-intensity activity in elite Australian football finals matches. *Int J Sports Physiol Perform* 2011;6:367–79.
2 Bradley PS, Sheldon W, Wooster B, *et al*. High-intensity running in English FA Premier League soccer matches. *J Sports Sci* 2009;27:159–68.
3 Buchheit M. Repeated-sprint performance in team sport players: associations with measures of aerobic fitness, metabolic control and locomotor function. *Int J Sports Med* 2011;32:1–10.
4 Aughey RJ. Applications of GPS technologies to field sports. *Int J Sports Physiol Perform* 2011;6:295–310.
5 Osgnach C, Poser S, Bernardini R, *et al*. Energy cost and metabolic power in elite soccer: a new match analysis approach. *Med Sci Sports Exerc* 2010;42:170–8.
6 Aughey RJ, Falloon C. Real-time versus post-game GPS data in team sports. *J Sci Med Sport* 2010;13:348–9.
7 Mohr M, Krustrup P, Bangsbö J. Match performance of high-standard soccer players with special reference to development of fatigue. *J Sports Sci* 2003;21:519–28.
8 Varley MC, Aughey RJ. Acceleration profiles in elite Australian soccer. *Int J Sports Med* 2013;34:34–9.
9 Faude O, Koch T, Meyer T. Straight sprinting is the most frequent action in goal situations in professional football. *J Sports Sci* 2012;30:625–31.
10 Dempsey JA, Wagner PD. Exercise-induced arterial hypoxemia. *J Appl Physiol* 1999;87:1997–2006.
11 Balsom PD, Gaitanos GC, Ekblom B, *et al*. Reduced oxygen availability during high intensity intermittent exercise impairs performance. *Acta Physiol Scand* 1994;152:279–85.
12 Billaut F, Smith KJ. Prolonged repeated-sprint ability is related to arterial O_2 desaturation in man. *Int J Sports Physiol Perform* 2010;5:197–209.
13 Smith KJ, Billaut F. Influence of cerebral and muscle oxygenation on repeated-sprint ability. *Eur J Appl Physiol* 2010;109:989–99.
14 Billaut F, Gore CJ, Aughey RJ. Enhancing team-sport athlete performance: is altitude training relevant? *Sports Med* 2012;42:751–67.
15 Gore CJ, McSharry PE, Hewitt AJ, *et al*. Preparation for football competition at moderate to high altitude. *Scand J Med Sci Sports* 2008;18(Suppl 1):85–95.
16 Bishop DJ, Girard O. Determinants of team-sport performance: implications for altitude training by team-sport athletes. *Br J Sports Med* 2013;47:i17–23.
17 Girard O, Mendez-Villanueva A, Bishop D. Repeated-sprint ability—part I: factors contributing to fatigue. *Sports Med* 2011;41:673–94.
18 Di Salvo V, Gregson W, Atkinson G, *et al*. Analysis of high intensity activity in Premier League soccer. *Int J Sports Med* 2009;30:205–12.
19 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.
20 Marcora SM. Perception of effort during exercise is independent of afferent feedback from skeletal muscles, heart and lungs. *J Appl Physiol* 2009;106:2060–2.
21 Noakes TD, St Clair Gibson A, Lambert EV. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans. *Br J Sports Med* 2004;38:511–14.
22 Garvican LA, Hammond K, Varley MC, *et al*. Lower running performance and exacerbated fatigue in soccer played at 1600 m. *Int J Sports Physiol Perform* 2013;114:1362.
23 Bangsbö J, Mohr M, Krustrup P. Physical and metabolic demands of training and match-play in the elite football player. *J Sports Sci* 2006;24:665–74.
24 Rampinini E, Coutts AJ, Castagna C, *et al*. Variation in top level soccer match performance. *Int J Sports Med* 2007;28:1018–24.
25 Deutsch MU, Kearney GA, Rehrer NJ. Time—motion analysis of professional rugby union players during match-play. *J Sports Sci* 2007;25:461–72.
26 Deutsch MU, Maw GJ, Jenkins D, *et al*. Heart rate, blood lactate and kinematic data of elite colts (under-19) rugby union players during competition. *J Sports Sci* 1998;16:561–70.
27 Bangsbö J. The physiology of soccer—with special reference to intense intermittent exercise. *Acta Physiol Scand Suppl* 1994;619:1–155.
28 Amann M, Kayser B. Nervous system function during exercise in hypoxia. *High Alt Med Biol* 2009;10:149–64.
29 Amann M, Eldridge MW, Lovering AT, *et al*. Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *J Physiol* 2006;575:937–52.
30 Hahn AG, Gore CJ. The effect of altitude on cycling performance: a challenge to traditional concepts. *Sports Med* 2001;31:533–57.
31 Kayser B, Narici M, Binzoni T, *et al*. Fatigue and exhaustion in chronic hypobaric hypoxia: influence of exercising muscle mass. *J Appl Physiol* 1994;76:634–40.
32 Noakes TD. Factors 'limiting' exercise performance in hypoxia. *Med Sci Sports Exerc* 2006;38:191–2.
33 Wehrlin JP, Hallen J. Linear decrease in VO_{2max} and performance with increasing altitude in endurance athletes. *Eur J Appl Physiol* 2006;96:404–12.
34 Bartsch P, Saltin B, Dvorak J. Consensus statement on playing football at different altitude. *Scand J Med Sci Sports* 2008;18(Suppl 1):96–9.

Downloaded from bjsm.bmj.com on November 28, 2013 - Published by group.bmj.com

35 Nassis GP. Effect of altitude on football performance: analysis of the 2010 FIFA World Cup Data. *J Strength Cond Res* 2013;27:703–7.
36 Aughey RJ, Hammond K, Varley MC, *et al*. Soccer activity profile of altitude versus sea-level natives during acclimatisation to 3600 m (ISA3600). *Br J Sports Med* 2013;47:i107–113.
37 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;11:593–9.
38 Weston AR, Mackenzie G, Tufts MA, *et al*. Optimal time of arrival for performance at moderate altitude (1700 m). *Med Sci Sports Exerc* 2001;33:298–302.
39 Smith KJ, Billaut F. Tissue oxygenation in men and women during repeated-sprint exercise. *Int J Sports Physiol Perform* 2012;7:59–67.
40 Billaut F, Buchheit M. Repeated-sprint performance and vastus lateralis oxygenation: effect of limited O_2 availability. *Scand J Med Sci Sports* 2013;23:e185–93.
41 McMahon S, Jenkins D. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. *Sports Med* 2002;32:761–84.
42 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;86:1367–73.
43 Kime R, Hamaoka T, Sako T, *et al*. Delayed reoxygenation after maximal isometric handgrip exercise in high oxidative capacity muscle. *Eur J Appl Physiol* 2003;89:34–41.
44 Chance B, Dait MT, Zhang C, *et al*. Recovery from exercise-induced desaturation in the quadriceps muscles of elite competitive rowers. *Am J Physiol* 1992;262: C766–75.
45 McCully KK, Iotti S, Kendrick K, *et al*. Simultaneous in vivo measurements of HbO_2 saturation and PCr kinetics after exercise in normal humans. *J Appl Physiol* 1994;77:5–10.
46 Gaitanos GC, Williams C, Boobis LH, *et al*. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol* 1993;75:712–19.
47 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:e51977.
48 Calbet JA, De Paz JA, Garatachea N, *et al*. Anaerobic energy provision does not limit Wingate exercise performance in endurance-trained cyclists. *J Appl Physiol* 2003;94:268–76.
49 Morales-Alamo D, Ponce-González JG, Guadalupe-Grau A, *et al*. Increased oxidative stress and anaerobic energy release, but blunted Thr172-AMPKá phosphorylation, in response to sprint exercise in severe acute hypoxia in humans. *J Appl Physiol* 2012;113:917–28.
50 Aughey RJ, Gore CJ, Hahn AG, *et al*. Chronic intermittent hypoxia and incremental cycling exercise independently depress muscle in vitro maximal Na^+-K^+-ATPase activity in well-trained athletes. *J Appl Physiol* 2005;98:186–92.
51 Perrey S, Rupp T. Altitude-induced changes in muscle contractile properties. *High Alt Med Biol* 2009;10:175–82.
52 Amann M, Proctor LT, Sebranek JJ, *et al*. Somatosensory feedback from the limbs exerts inhibitory influences on central neural drive during whole body endurance exercise. *J Appl Physiol* 2008;105:1714–24.
53 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* 2007;581:389–403.
54 Dillon GH, Waldrop TG. In vitro responses of caudal hypothalamic neurons to hypoxia and hypercapnia. *Neuroscience* 1992;51:941–50.
55 Szubski C, Burtscher M, Loscher WN. The effects of short-term hypoxia on motor cortex excitability and neuromuscular activation. *J Appl Physiol* 2006;101:1673–7.
56 Chaudhuri A, Behan PO. Fatigue and basal ganglia. *J Neurol Sci* 2000;179:34–42.
57 Nybo L, Rasmussen P. Inadequate cerebral oxygen delivery and central fatigue during strenuous exercise. *Exerc Sport Sci Rev* 2007;35:110–18.
58 Holliss BA, Fulford J, Vanhatalo A, *et al*. Influence of intermittent hypoxic training on muscle energetics and exercise tolerance. *J Appl Physiol* 2013;114:611–19.
59 Buchheit M, Kuitunen S, Voss SC, *et al*. Physiological strain associated with high-intensity hypoxic intervals in highly trained young runners. *J Strength Cond Res* 2012;26:94–105.
60 Zoll J, Ponsot E, Dufour S, *et al*. Exercise training in normobaric hypoxia in endurance runners. III. Muscular adjustments of selected gene transcripts. J Appl Physiol 2006;100:1258–66.
61 Vogt M, Puntschart A, Geiser J, *et al*. Molecular adaptations in human skeletal muscle to endurance training under simulated hypoxic conditions. *J Appl Physiol* 2001;91:173–82.
62 Hoppeler H, Klossner S, Vogt M. Training in hypoxia and its effects on skeletal muscle tissue. *Scand J Med Sci Sports* 2008;18(Suppl 1):38–49.
63 Dufour SP, Ponsot E, Zoll J, *et al*. Exercise training in normobaric hypoxia in endurance runners. I. Improvement in aerobic performance capacity. *J Appl Physiol* 2006;100:1238–48.
64 Roels B, Thomas C, Bentley DJ, *et al*. Effects of intermittent hypoxic training on amino and fatty acid oxidative combustion in human permeabilized muscle fibers. *J Appl Physiol* 2007;102:79–86.
65 Faiss R, Léger B, Vesin J-M, *et al*. Significant molecular and systemic adaptations after repeated sprint training in hypoxia. *PLoS ONE* 2013;8:e56522.
66 Casey DP, Joyner MJ. Compensatory vasodilatation during hypoxic exercise: mechanisms responsible for matching oxygen supply to demand. *J Physiol* 2012;590:6321–6.
67 Casey DP, Joyner MJ. Local control of skeletal muscle blood flow during exercise: influence of available oxygen. *J Appl Physiol* 2011;111:1527–38.
68 Wachsmuth NB, Völzke C, Prommer N, *et al*. The effects of classic altitude training on hemoglobin mass in swimmers. *Eur J Appl Physiol* 2013;113:1199–211.
69 Sargent C, Schmidt WF, Aughey RJ, *et al*. The impact of altitude on the sleep of young elite soccer players (ISA3600). *Br J Sports Med*; 2013;47:i86–92.
70 Wachsmuth N, Kley M, Spielvogel H, *et al*. Changes in blood gas transport of altitude native soccer players near sea-level and sea-level native soccer players at altitude (ISA3600). *Br J Sports Med*; 2013;47:i93–9.

Downloaded from bjsm.bmj.com on November 28, 2013 - Published by group.bmj.com

Update in the understanding of altitude-induced limitations to performance in team-sport athletes

François Billaut and Robert J Aughey

Br J Sports Med 2013 47: i22-i25
doi: 10.1136/bjsports-2013-092834

Updated information and services can be found at:
http://bjsm.bmj.com/content/47/Suppl_1/i22.full.html

These include:

References This article cites 66 articles, 20 of which can be accessed free at:
http://bjsm.bmj.com/content/47/Suppl_1/i22.full.html#ref-list-1

Article cited in:
http://bjsm.bmj.com/content/47/Suppl_1/i22.full.html#related-urls

Open Access This is an Open Access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 3.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/3.0/

Email alerting service Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic Collections Articles on similar topics can be found in the following collections

Open access (89 articles)

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/