Advancing hypoxic training in team sports: from intermittent hypoxic training to repeated sprint training in hypoxia

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ABSTRACT

Over the past two decades, intermittent hypoxic training (IHT), that is, a method where athletes live at or near sea level but train under hypoxic conditions, has gained unprecedented popularity. By adding the stress of hypoxia during 'aerobic' or 'anaerobic' interval training, it is believed that IHT would potentiate greater performance improvements compared to similar training at sea level. A thorough analysis of studies including IHT, however, leads to strikingly poor benefits for sea-level performance improvement, compared to the same training method performed in normoxia. Despite the positive molecular adaptations observed after various IHT modalities, the characteristics of optimal training stimulus in hypoxia are still unclear and their functional translation in terms of whole-body performance enhancement is minimal. To overcome some of the inherent limitations of IHT (lower training stimulus due to hypoxia), recent studies have successfully investigated a new training method based on the repetition of short (<30 s) 'all-out' sprints with incomplete recoveries in hypoxia, the so-called repeated sprint training in hypoxia (RSH). The aims of the present review are therefore threefold: first, to summarise the main mechanisms for interval training and repeated sprint training in normoxia. Second, to critically analyse the results of the studies involving high-intensity exercises performed in hypoxia for sea-level performance enhancement by differentiating IHT and RSH. Third, to discuss the potential mechanisms underpinning the effectiveness of those methods, and their inherent limitations, along with the new research avenues surrounding this topic.

INTRODUCTION

Prolonged altitude sojourns using the 'live high-train high' or the 'live high-train low' models[1 2] have been increasingly used in athletes involved in endurance and, more recently, in intermittent (eg, team and racket sports) disciplines in an attempt to gain a competitive edge.[2 3] However, the question as to how effectively prolonged altitude exposure can improve athletic performance and its underpinning physiological mechanisms and signalling pathways remains contentious.[4 5]

Over the past two decades, intermittent hypoxic training (IHT), that is, a method where athletes live at or near sea level but train under hypoxic conditions, has gained large popularity. Hence, IHT presents the advantages of minimal travel and relatively low expense and causes limited disruption to the athletes' normal training environment and lifestyle. Another advantage is that it also avoids the deleterious effect (decreased muscle excitability) of an extended stay in altitude.[6] By adding the stress of hypoxia during 'aerobic' or 'anaerobic' interval training (INT), it is believed that IHT would potentiate greater performance improvements compared to similar training at sea level. For long, erythrocytosis was believed to be the primary factor benefiting putative sea-level performance improvement after a sufficient (several weeks) hypoxic stimulus.[4 5 7] However, IHT viewed this from a new perspective with evidence that exercising even for a short period in hypoxia affects a large number of genes mediated by hypoxia-inducible factors (HIFs)[8] and the exercise performance with muscular adaptations arising (and not necessarily an improved oxygen carrying capacity).[9–12] Nevertheless, in other IHT studies, any potentiating effect of hypoxia in addition to training was ambiguous.[3 13–16] Although an improvement in anaerobic performance after IHT has been mentioned in four studies,[17–20] it is noteworthy that these studies were 'uncontrolled', and therefore the effects of training cannot be distinguished from those of hypoxia.[13] As such, it seems that after decades of research, "IHT does not increase exercise performance at sea level in endurance athletes any more than simply training at sea level."[21]

Until now, only scarce literature has assessed the potential benefits of altitude training in intermittent sports.[16 22 23] Therefore, the relevance of altitude training in team-sport athletes for improving players' specific fitness (repeated sprint ability (RSA)) has not been scientifically sounded as yet. Team-sport players (eg, football) perform a large number of high-intensity actions, including numerous sprints, often with incomplete recoveries, during the course of a game. As a consequence, developing their ability to repeatedly perform intense exercise bouts for sustained periods is important for crucial match actions.[24] For example, failure to recover after a sequence of intense actions may leave the team more vulnerable defensively by decreasing the chances to reach passes or increasing the time to take up a defensive position (tackles). Sport-specific training methods for team sports using the stress of hypoxia as a strong additional stimulus with specifically designed training models are arguably promising methods. For instance, repeated sprint training in hypoxia (RSH), defined as the repetition of several short (≤30 s) 'all-out' exercise bouts in hypoxia interspersed with incomplete recoveries (exercise-to-rest ratio <1:4), could be considered as such a sport-specific training strategy. Although RSH could be considered as a form of IHT, its efficacy is presumably based on

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different mechanisms than on the existing IHT methods (discussed below), therefore justifying the addition of the RSH modality in the altitude training nomenclature.[25]

The aims of the present review are threefold: first, to summarise the main mechanisms for INT and repeated sprint training in normoxia (RSN). Second, to critically analyse the results of the studies involving high-intensity exercises performed in hypoxia for sea-level performance enhancement by differentiating IHT and RSH. Third, to discuss the potential mechanisms underpinning the effectiveness of those methods, and their inherent limitations, along with new research avenues surrounding this topic.

A computer-based literature search was conducted in April 2013 using the PubMed electronic database using combinations of specific keywords: 'altitude', 'hypoxic', 'training', 'intermittent hypoxia', 'repeated sprints', 'interval training', 'exercise' and 'performance'. Recently, an international consensus group of the IOC[26] underlined the further need "to study the effects of training in hypoxia and live high-train low modalities on performance at sea level, low and moderate altitude using a placebo-controlled double-blind design." Well aware of the methodological issues (ie, importance of ruling out placebo effects[21 27]) pertaining to the conclusion of some altitude training studies, the present review is limited to studies including a control (CON) group in their experimental design, allowing the effects of training and hypoxic stimulus to be clearly differentiated.

INT VERSUS RSN

The efficiency of INT[28 29] has been investigated extensively. It can broadly be subdivided into (1) short or long aerobic[28] versus anaerobic[29] INT and (2) short or long intervals versus sprint intervals.[30] INT consists of 'repeated short-to-long bouts of rather high-intensity exercise interspersed with recovery periods'.[28] While any INT session will naturally challenge the metabolic and neuromuscular systems, it is beyond the scope of this review to detail all the stressed factors.[24 30–32] However, we support the recent statement that "the cardiorespiratory (ie, VO_2) data, but also cardiovascular work, stored energy and cardiac autonomic stress responses are the primary variables of INT", whereas "anaerobic glycolytic energy contribution and neuromuscular load/musculoskeletal strain are secondary."[30] Indeed, the expected benefits of INT are primarily to maximise VO_{2max} and therefore cardiac output and the arterial-mixed venous oxygen difference[32] as well as the VO_2 kinetics,[31] which are important determinants of endurance performance. Overall, INT performed at intensities[33] and exercise-to-rest ratios[34] that elicit maximal volume and pressure overloads on the myocardium and VO_2 responses near maximal oxygen uptake (VO_{2max}) are quite likely optimal in terms of cardiac output, blood flow, shear stress, recruitment and increased oxidative capacity of fast twitch (FT) fibres. This imposes the need to maintain the longest time >90% VO_{2max}.[35]

During repeated sprints in normoxia, the factors responsible for the performance decrements (eg, decline in sprint speed/power across repetitions) include limitations to energy supply (eg, phosphocreatine (PCr) resynthesis and aerobic and anaerobic glycolysis), metabolite accumulation (eg, inorganic phosphate, Pi; hydrogen ion, H^+) and neural factors (eg, neural drive and muscle activation).[36 37] Among these factors, the ability to resynthesise PCr is probably the central determinant of RSA. Hence, the oxidative pathway is essential for the PCr resynthesis rate,[38] and the decrease in PCr concomitant to the rise in Pi and AMP stimulates the anaerobic glycolytic contribution at the start of a sprint. If the increase in H^+ accumulation is also known to impair RSA, recent findings[39] suggest that this fitness component is determined to a larger extent by the muscle energy supply (eg, short-term (<1 min) PCr resynthesis rate) than by the H^+ removal.

In team sports, the clinical relevance of improving RSA is debated,[40] but it is a common belief that such adaptations would be beneficial for improved match-related physical performance. For instance, the mean time recorded during an RSA test predicts the distance of high-intensity running and the total sprint distance during a professional football match.[41] Furthermore, football players experience temporary fatigue during a game (eg, lower amount of sprinting, high-intensity running and distance covered after a sequence of repeated and intense actions), which may determine the outcome of crucial situations (eg, decreased technical and tactical behaviour and wrong cognitive choices).[42 43] This suggests that improving RSA would maximise team-sport physical performance and that it is important to better understand training strategies that can enhance this fitness component.

Although the brief description above of the main determinants of INT versus RSN highlights that those training methods aim at developing predominantly the aerobic pathway and RSA, respectively, the practical question of their optimal combination in team sports is widely debated[37 44] with two diverging approaches[45]; that is, an integrative 'mixed' method mainly based on IHT/RSN[37] contrasting with an 'isolated' method based on the parallel development of maximal aerobic speed and maximal sprinting speed.[44] The same debate was translated within the area of the optimal use of hypoxic training in team sports[3 15] and needs to better describe the main adaptive mechanisms of IHT and RSH. This is the objective of the next sections.

CURRENT TRENDS: IS IT TIME TO MOVE BEYOND IHT?

Performance improvement with IHT

In table 1, we report 23 controlled studies (ie, 20 IHT and 3 RSH) including training protocols performed in hypoxia versus normoxia. Interestingly, an additional benefit on performance-related variables of IHT compared with the same training performed in normoxia is present in only four of those studies. First, Martino *et al*[48] reported a faster 100 m swim time and larger improvement in peak power output during an arm Wingate test after 21 days of training including swim sprints at an altitude of 2800 m, compared to sea level. Since a detailed description of the training sessions is not available, the mechanisms inducing additional hypoxia-related benefits cannot be ascertained. Second, Hendriksen and Meeuwsen[54] highlighted a 5% increase in peak power output during a Wingate cycling test after 10 days of aerobic training in hypobaric hypoxia, while performance in the normoxic training group did not change. Third, Dufour *et al*[59] reported an improved endurance performance capacity in competitive distance runners after 6 weeks of high-intensity aerobic training at 3000 m (ie, 5% increase in their VO_{2max} and 35% longer time to exhaustion running at a speed associated with VO_{2max}), but not performance change in the group training in normoxia. Finally, Manimmanakorn *et al*[23] reported in one of the few studies conducted with team-sport athletes that a knee extension/flexion IHT performed over a 5-week period provided an additional benefit for improving maximum voluntary contraction torque during prolonged leg extensions. A remarkable observation across the above-listed studies is that the additional benefits of IHT seem to be partly related to an upregulation of the glycolytic potential and to an

Table 1 Summary of current research findings relative to the use of intermittent hypoxic training (IHT) or repeated sprint training in hypoxia (RSH)

Author (year)	Participants	Design (number of training sessions, type, altitude and training content)	Groups	Statistically significant results ($p<0.05$)
Roskamm *et al* (1969)[46]	Untrained	24 over 4 weeks, cycling, 2250 m (N=6) or 3450 m (N=6; HH). 30 min aerobic training	IHT, N=12 INT, N=6	10–17% VO_{2max} 6% VO_{2max}
Terrados *et al* (1988)[47]	Elite cyclists	12–20 over 3–4 weeks, cycling, 2300 m (HH). Aerobic training and some intervals (15 s at 130% of aerobic peak power output)	IHT, N=4 INT, N=4	33% PPO 22% PPO
Martino *et al* (1995)[48]	Elite swimmers	Swim sprints at 2800 m (HH) during 21 days at altitude. No details available	IHT, N=20 INT, N=13	*–6% 100 m swim time, 34% PPO arm Wingate test* NS changes
Emonson *et al* (1997)[49]	Untrained	15 over 5 weeks, cycling, 2500 m (HH). 45 min at 70% of VO_{2max}	IHT, N=9 INT, N=9	12% VO_{2max} 12% VO_{2max}
Katayama *et al* (1998)[50]	Untrained	10 over 2 weeks, cycling, 4500 m (HH). 30 min at 70% of normoxic VO_{2max} level	IHT, N=7 INT, N=7	7% VO_{2max} 5% VO_{2max}
Bailey *et al* (2000)[51]	Runners	4 weeks at ~2000 m (NH). Aerobic training, no details	IHT, N=18 INT, N=14	15% VO_{2max} 5% VO_{2max}
Geiser *et al* (2001)[52]	Untrained	30 over 6 weeks, cycling, 3850 m (NH). 30 min at 77–85% of maximum heart rate	IHT, N=18 INT, H=15	11% VO_{2max}, 17% 30 min TT mean PO 9% VO_{2max}, 19% 30 min TT mean PO
Karlsen *et al* (2002)[53]	Cyclists	9 over 3 weeks, cycling, 3000 m (NH). 120 min aerobic training	IHT, N=8 INT, N=8	NS changes in VO_{2max} or 30 min TT NS changes in VO_{2max} or 30 min TT
Hendriksen and Meeuwsen (2003)[54]	Triathletes	10 over 10 days, cycling, 2500 m (HH). 105 min aerobic training	IHT, N=8 INT, N=8	*5% PPO cycling Wingate test* NS increase
Truijens *et al* (2003)[55]	Swimmers	15 over 5 weeks, swimming, 2500 m (NH). 12.5 min >100% VO_{2max} (30 s or 60 s bouts)	IHT, N=8 INT, N=8	NS changes *6% VO_{2max}*
Ventura *et al* (2003)[56]	Cyclists	18 over 6 weeks, cycling, 3200 m (NH). 30 min aerobic training	IHT, N=7 INT, N=5	NS changes in VO_{2max} or 10 min TT NS changes in VO_{2max} or 10 min TT
Morton and Cable (2005)[16]	Team-sport players	12 over 4 weeks, cycling, 2750 m (NH). 10×1 min at 80% of 2 min PPO	IHT, N=8 INT, N=8	8% cycling Wingate test PPO, 7% VO_{2max} 6.5% cycling Wingate test PPO, 8% VO_{2max}
Roels *et al* (2005)[57]	Cyclists and triathletes	14 over 7weeks, cycling, 3000 m (NH). 6–8×2–3 min at 100% of aerobic PPO	IHT, N=11 IHIT, N=11 INT, N=11	4% 10 min TT mean PO *9% VO_{2max}*, 5% 10 min TT mean PO 5% 10 min TT mean PO
Roels *et al* (2007)[58]	Cyclists and triathletes	15 over 3 weeks, cycling, 3000 m (NH). 9×60 min at 60% VO_{2max} and 36 min with intervals of 2 min at 100% aerobic PPO (2 min bouts)	IHT, N=10 INT, N=9	7% aerobic PPO 7% aerobic PPO, 8% 10 min TT mean PO
Dufour *et al* (2006)[59]	Runners	12 over 6 weeks, running, 3000 m (NH). 24–40 min <VO_{2max}	IHT, N=9 INT, N=9	*5% VO_{2max}, 35% T_{lim} at vVO_{2max}* NS changes
Hamlin *et al* (2010)[22]	Cyclists and triathletes	10 over 10 days, cycling, 3200–4400 m (NH). 90 min aerobic training followed by two 30 s Wingate tests	IHT, N=9 INT, N=7	3% PO cycling Wingate test NS changes
Lecoultre *et al* (2010)[60]	Cyclists	12 over 4 weeks, cycling, 3000 m (NH). 4×12–18 min at 100–120% of aerobic PPO, 4×30–48 min <VO_{2max} and 4×100 min aerobic training	IHT, N=7 INT, N=7	7% 40 km TT mean PO 6% 40 km TT mean PO
Mao *et al* (2011)[61]	Active males	25 over 5 weeks, cycling, 2750 m (NH). 30 min aerobic training	IHT, N=12 INT, N=12	16% VO_{2max} 10% VO_{2max}
Manimmanakorn *et al* (2013)[23]	Female team-sport players	15 over 5 weeks, knee flexion and extension, ~4500 m (NH). 6 sets of low resistance knee extensions and flexions to failure with 30 s between sets	IHT, N=10 INT, N=10	*15% MVC3, 17% MVC30, 129% REPS201RM* 86% REP201RM
Holliss *et al* (2013)[62]	Active males	15 over 3 weeks, leg extension, 3000 m (NH). 10×60–70 s intense exercise with 20–30 s passive recovery. One leg IHT, the other leg INT	IHT, N=9 INT, N=9	25% leg extension, incremental T_{lim} 27% leg extension, incremental T_{lim}
Puype *et al* (2013)[63]	Moderately trained cyclists	18 over 6 weeks, cycling, 3000 m (NH). 4–9 sprints of 30 s interspersed with 4.5 min recovery at 50 W	RSH, N=10 RSN, N=10 CON, N=10	6% sprint PO, 6% VO_{2max}, 6% 10 min PO, 7% LT4 5% sprint PO, 6% VO_{2max}, 6% 10 min PO, NS NS changes
Galvin *et al* (2013)[64]	Rugby players	12 over 4 weeks, treadmill running, 3500 m (NH). 10 sprints of 6 s interspersed with 30 s recovery	RSH, N=15 RSN, N=15	*33% Yo-Yo Intermittent Recovery 1 test performance* 14% Yo-Yo Intermittent Recovery 1 test performance

Continued

Table 1 Continued

Author (year)	Participants	Design (number of training sessions, type, altitude and training content)	Groups	Statistically significant results (p<0.05)
Faiss *et al* (2013)[65]	Moderately trained cyclists	8 over 4 weeks, cycling, 3000 m (NH). 3×5 all-out 10 s sprints interspersed with 20 s recovery at 120 W	RSH, N=20	6% sprint PO, *38% completed sprints in RSA test*
			RSH, N=20	7% sprint PO, no change in completed sprints
			CON, N=10	NS changes

This table is limited to investigations with a group training in hypoxia (IHT, IHIT or RSH) and a group training in normoxia (INT or RSN). CON group without training present in two studies. Altitude described as either HH or NH. A significant difference between groups is shown in italics (p<0.05).
CON, control group; HH, hypobaric hypoxia; IHT, intermittent hypoxic training; IHIT, intermittent hypoxia interval training; INT, intermittent training in normoxia; LT4, power output corresponding to 4 mmol blood lactate; MVC3, peak maximum voluntary contraction in 3 s; MVC30, area under the peak 30 s maximal voluntary contraction curve; NH, normobaric hypoxia; NS, non-significant; PO, power output; PPO, peak power output; REPS201RM, repetitions at 20% of 1 repetition maximal load; RSA, repeated sprint ability test to exhaustion; RSH, repeated sprint training in hypoxia; RSN, repeated sprint training in normoxia; TT, time trial; T_{lim}, time to exhaustion; VO_{2max}, maximal oxygen uptake; vVO_{2max}, velocity associated with VO_{2max}.

increased anaerobic capacity (eg, larger increase in Wingate performance). These adaptations might help athletes engaged in intermittent sports to improve their match-related performance.

Besides, in another study conducted with team-sport athletes, similar improvements in aerobic and anaerobic power outputs were observed when training was performed in hypoxia and normoxia.[16] Other well-designed controlled studies highlighted the benefits of IHT on aerobic performance but failed to demonstrate an additional benefit of conducting the training in a hypoxic environment.[46 55 57 58 66] With the many different training strategies and methods available, the possibility that IHT might "enhance endurance performance when subsequent exercise is conducted in hypoxia" in football players as stated in a recent comprehensive review[3] was therefore questioned by our team.[15]

Physiological mechanisms and limitations of IHT

IHT is quite likely to have a minimal effect on erythropoiesis since a large 'hypoxic dose' is required for significantly "stimulating the erythropoietic pathway to the point that it enhances post-altitude sea-level endurance performance."[4 5] In support of this assumption, previous IHT studies failed to observe any significant change in the total haemoglobin mass, red cell volume or any other red cell indices compared with a CON group[62 67] (see ref. [2] for further discussions).

Compared with sea-level training, IHT has the potential to induce a further physiological strain[68] and specific molecular adaptations,[11 12 69] though not necessarily associated with improved exercise capacity. The rationale of using IHT relies on the hypothesis that these muscle adaptations surpass those triggered by normoxic exercise. In particular, the lower partial pressure of oxygen (PO_2) in muscle tissue during IHT when compared with INT would lead to a larger upregulation of HIF-1α.[11 12 62] In untrained or moderately-trained participants, muscular adaptations occurring in response to IHT include—but may not be limited to—an increased citrate synthase activity, mitochondrial density, capillary-to-fibre ratio and fibre cross-sectional area as well as upregulation of factors of mitochondrial biogenesis or enzymes implicated in carbohydrate and mitochondrial metabolism, oxidative stress defence and pH regulation.[10 11 47 52 59 70 71] However, as stated recently,[9] one may question the functional significance of these physiological adaptations (eg, larger increase in citrate synthase activity in IHT than in INT) since the effects of IHT on endurance performance measured in normoxia are 'minimal and inconclusive in trained athletes'.[21]

Several authors have reported additional adaptations potentially favourable to high-intensity exercises. These include improvements in muscle O_2 homeostasis and tissue perfusion induced by improved mitochondrial efficiency, control of mitochondrial respiration,[71 72] angiogenesis[73] and muscle buffering capacity.[74] However, the translation into enhanced performance is not always observed and when it does occur, it may be irrelevant for team sports. Hence, non-specific IHT protocols or inappropriate performance tests—that is, evaluating endurance capacity (with VO_{2max} tests or time trials) but neglecting indices of match-related performance such as RSA—have been mainly conducted so far.

With the exception of studies performed at an intensity corresponding to the second ventilatory threshold,[12 59 72] where the increased expression of factors involved in glucose uptake, oxidative stress defence and pH regulation was associated with an increased endurance performance capacity, most of the IHT studies (including those with some muscle adaptations) did not report any additional performance benefit of IHT over INT. In untrained participants, the effect of training seems to predominate, overwhelming any additional effect of hypoxia.[75] Furthermore, Levine[75] convincingly argued that, compared to similar training in normoxia, IHT quite likely induces a lower stimulus for the active musculature since the lowered power output[76] and the reduced oxygen flux resulting from hypoxia would be associated with a downregulation of muscle structure and function.

Performance improvement with RSH

Some of the methodological limitations related to IHT have been overcome in recent studies investigating a new hypoxic training strategy named RSH.[63–65] RSH is based on the repetition of 'all-out' efforts of short (≤30 s) duration interspersed with short incomplete recoveries. This model differs from IHT since the intensity of the training stimulus is maximal and therefore allows one to maintain high FT recruitment so that positive results can be expected when adding hypoxia to training. RSH is particularly interesting since, under hypoxic conditions (<3800 m), a single sprint performance of short duration (<10 s) is generally preserved, whereas fatigue resistance during RSA tests is reduced with earlier and larger decrements in mechanical work.[77–79]

Recently, we[65] showed that RSH delays fatigue during a repeated sprint test to exhaustion. In that study, 50 trained athletes were randomly dispatched in three different intervention groups (RSH: 3000 m, FiO_2 14.5%; RSN: 485 m, FiO_2 20.9% and CON: no specific sprint training) and tested twice (before and after a 4-week training protocol including two repeated sprint training sessions per week) for the determination of

endurance performance, anaerobic capacity and RSA. If endurance performance (during a 3 min 'all-out' time trial) was not increased, RSN and RSH improved the average power output during 10 s sprints (by 6–7%) and a 30 s Wingate test (by 3–5%), although a major additional benefit of RSH compared with RSN was found. The number of sprints completed during an RSA test to exhaustion was improved by 40% only after RSH: an average of 9 sprints was performed before training in both groups but 13 after RSH and still 9 after RSN. The relevance of the observed improvement in RSA in team-sport athletes is unanswered yet since the direct translation of RSA to the team game result is questionable.[40]

Puype *et al*[63] then showed that RSH improved by 7% the power output, corresponding to 4 mmol blood lactate during a maximal incremental test, while it did not change after RSN. However, in that study, the gains in power output during a 10 min time trial (6–7%) or VO_{2max} during an incremental test (6%) were similar after RSH and RSN. Interestingly, the phosphofructokinase activity was markedly increased (59%) only after RSH, quite likely reflecting an upregulation of muscle glycolytic capacity. Since the performance tests were limited to longer aerobic efforts, they cannot be linked directly to physical performance improvement.

Furthermore, Galvin *et al*[64] recently showed in rugby players a 19% additional benefit of RSH compared with RSN in high-intensity intermittent running performance (Yo-Yo IR1 test[80]). This substantially higher performance improvement has important practical implications since the Yo-Yo test correlates very well with physical performance and the amount of high-intensity running in several team sports such as soccer, basketball, rugby and handball.[64]

Thus, RSH was shown to be as efficient as RSN in improving power output on a single sprint (5–7%) when including 10 s sprints interspersed with 20 s recoveries[65] or 30 s sprints with 270 s recoveries[63] (table 1). Additionally, but only after RSH, cycling power output corresponding to 4 mmol of lactate during an incremental test[63] and high-intensity intermittent running performance were significantly improved[64] only after RSH while fatigue development was delayed during a repeated cycling sprint test performed until exhaustion.[65]

Physiological mechanisms and promises of RSH

We hypothesised that RSH would induce beneficial adaptations mainly due to the improved blood perfusion level inducing an enhanced O_2 utilisation and an improved behaviour of FT fibres. With maximal effort intensities, specific skeletal muscle tissue adaptations (molecular level) may arise through the oxygen-sensing pathway (ie, capillary-to-fibre ratio, fibre cross-sectional area, myoglobin content and oxidative enzyme activity such as citrate synthase) that either do not occur in normoxic conditions or, if they do, they do so to a lesser degree.[10–12] Additionally, exercising in hypoxia is known to trigger a compensatory vasodilation to match an increased oxygen demand at the muscular level.[81]

Increasing evidence indicates that neuromuscular (muscle contractility and/or activation), biomechanical (running economy) and metabolic (muscle and/or cerebral deoxygenation/reoxygenation kinetics) factors may also play key roles in the hypoxia-induced mechanisms in response to maximal-intensity intermittent exercises. For instance, it is generally accepted that neuromuscular transmission and action potential propagation along with muscle fibres (sarcolemma excitability) remain unchanged with acute hypoxia in relaxed muscles or during brief contractions.[82] Indirect evidence rather suggests that the increased rate of fatigue seen at altitude may be the result of a more rapid accumulation of Pi during each sprint and a reduced rate of its removal during recovery.[38 83] Repeated sprints result in large changes in PCr and H^+ concentrations. However, the restoration of power output during repeated sprints seems to be influenced more by the muscle energy supply (eg, PCr resynthesis) than by the recovery of muscle pH.[39] However, enhanced buffer capacity or upregulation of genes involved in pH control has also been reported after RSH[63 65]

Moreover, performance decrements are also likely to be explained by a reduced neural drive to the active musculature, (estimated by surface electromyography) arising secondary to a stronger reflex inhibition due to brain hypoxia[84] or a hypoxia-induced increased level of intramuscular metabolites known to stimulate group III–IV muscle afferents.[83] Furthermore, larger cerebral deoxygenation levels[77] and slower reoxygenation rates during recoveries[85]—which strongly correlate with the exacerbated reduction in mechanical work in hypoxia during an RSA test—have also been observed with acute altitude exposure. As exercise intensity increases, glycolytic FT muscle fibres are preferentially recruited,[86] while at lower intensity (eg, <VT2) oxidative slow twitch (ST) and FT muscle fibres are solicited.

During sprints in hypoxia, the compensatory vasodilation (with an increase in blood flow) that aims at maintaining constantly the total O_2 delivery to the muscle is quite likely maximal since exercise intensity is essential in the amplitude of this compensatory mechanism.[81] FT fibres are quite likely to benefit more than ST fibres from the higher blood perfusion. Hence, owing to their greater fractional O_2 extraction if highly perfused,[87] the enhanced microvascular O_2 delivery to FT would 'make FT to behave more like their oxidatively efficient ST counterparts'.[88] So, RSH efficiency is likely to be fibre-type selective and intensity dependent and therefore based on mechanisms presumably different from those associated with IHT. We speculate here that the improved responsiveness of the vascular bed and the improved blood perfusion through nitric oxide (NO)-mediated vasodilation mechanisms[81] could be paramount in RSH. Further investigations into the NO pathway (neuronal NO synthase (nNOS) and endothelial NO synthase (eNOS)) are required in RSH to determine whether mechanisms other than NO-mediated vasodilation are also involved. Moreover, fibre-type selective peripheral vascular effects of nNOS-derived NO have been reported during high-speed treadmill running, whereas these effects were not seen at slower speeds.[89] It is, however, striking to note in two recent studies[89 90] a similar fibre-type mechanism on dietary nitrate (NO_3^-) supplementation that enhances blood flow. With NO_3^- supplementation, blood flow and vascular control were indeed augmented mostly in FT,[90] partly due to the lower microvascular PO_2 in contracting FT.[87] Interestingly, an elevated microvascular PO_2 is known to reduce PCr breakdown[38] and speed PCr recovery kinetics.

Adding a hypoxic stimulus to training can modulate the PCr resynthesis during exercise. In support of this suggestion, Holliss *et al*[62] reported that single leg-extension IHT results in a faster PCr recovery from high-intensity exercise in hypoxia (with only a tendency observed in normoxia). However, exercise tolerance during an incremental test to the limit of exhaustion either in normoxia or hypoxia was not different between IHT and INT. The authors speculated that the faster PCr resynthesis observed after IHT was probably not due to an enhanced mitochondrial biogenesis but most likely due to a greater enhancement of muscle O_2 delivery. Overall, a faster PCr resynthesis

resulting from RSH would manifest because of better maintenance of power production (better recovery between efforts) during intermittent, high-intensity exercises.

The latter could arguably contribute to the increased RSA performance observed in normoxia after RSH.[65] By challenging the functional reserve in the muscle oxygen diffusing capacity most likely utilised in hypoxia,[91] repeated maximal efforts in hypoxia have the potential to stimulate beneficial adaptations in terms of PCr resynthesis and oxygen utilisation mediated by HIFs at the muscular level. By extension, the positive impact of RSH on glycolytic performance and skeletal muscle adaptations may lead to putative strong benefits for team sports like football, rugby union or Australian football, where the ability to repeat high-speed runs during an entire game is essential for overall performance.[92] At this stage, however, specific mechanisms that may enhance performance with RSH are still to be determined with further studies.

CONCLUSION

A thorough analysis of studies that have used IHT leads to strikingly poor benefits for sea-level performance improvement, compared to the same training protocol performed in normoxia.

Despite the positive molecular adaptations observed after various IHT modalities, the characteristics of optimal training stimulus in hypoxia are still unclear and their functional translation in terms of whole-body performance enhancement is minimal.

To overcome some of the inherent limitations of IHT (lower training stimulus due to hypoxia), recent studies have investigated a new training method based on the repetition of 'all-out' sprints in hypoxia, the so-called RSH. The succession of maximal efforts under hypoxic conditions was shown to be beneficial for maximal performance improvement and especially to delay fatigue when sprints with incomplete recoveries were repeated until exhaustion.

RSH is therefore proposed as a promising training strategy in intermittent sports to eventually improve match-related performance. Since team sports are characterised by intense exercise bouts repeated throughout a game, delaying fatigue and improving the ability to repeat sprints are crucial for the improved physical involvement of players.

Until now, there is scant evidence of the additional benefits of high-intensity training performed in hypoxia compared to the same training in normoxia on RSA. Until new evidence is provided, it is felt that compared to IHT, RSH is based on different fundamental mechanisms that are likely to be fibre-type selective, while the positive adaptations are probably dependent on the compensatory vasodilatory effects on the behaviour of FT fibres.

Yet, further studies with large sample sizes and double-blinded designed protocols are needed to endorse the efficacy of RSH. Then, in order to robustly assess the true benefits of RSH versus traditional IHT, both training strategies must be directly compared in the same experimental test setting. Judging the impact of RSH on athletic performance in various team sports could be best improved by testing, for example, specific work-to-rest ratios or the efficacy of different 'hypoxic doses'. Finally, if the efficacy of RSH is confirmed in more ecological situations (including overground sprints in hypoxic marquees rather than cycling an ergometer), it could then be readily implemented in the yearly periodisation of intermittent disciplines.

What are the new findings

- This review critically analyses the results of the studies involving high-intensity exercises performed in hypoxia for sea-level performance enhancements by differentiating intermittent hypoxic training (IHT) and repeated sprint training in hypoxia (RSH).
- IHT leads to strikingly poor benefits for sea-level performance improvement, compared to the same training protocol performed in normoxia.
- RSH is a promising training strategy that has been shown to delay fatigue when sprints with incomplete recoveries are repeated until exhaustion.

How might it impact on clinical practice in the near future

- This review will help athletes and teams in intermittent sports by providing an overview of the current scientific knowledge about intermittent hypoxic training and repeated sprint training in hypoxia (RSH).
- New studies are proposed to judge the efficacy of RSH in team sports and to determine the specific mechanisms that may enhance the team game results with RSH.

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Advancing hypoxic training in team sports: from intermittent hypoxic training to repeated sprint training in hypoxia

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