

Repeated sprint training in hypoxia – an innovative method

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Repeated Sprint Training in Hypoxia – An Innovative Method

Wiederholtes Sprinttraining bei Hypoxie – Eine innovative Methode

Summary

- ▶ **The year 2018** marked the 50th anniversary of the Mexico Olympic games, which represents the starting point of scientific research on hypoxic training. Since the original “Live High – Train High”, many altitude/hypoxic training methods have been developed. The aim of the present review is to present the most recent method called “Repeated Sprint training in Hypoxia” (RSH). RSH is of unprecedented interest in the altitude training area with 25 studies published in the 5-year period following the pioneer article in 2013, and with only two studies that did not report any beneficial effects.
- ▶ **Potential mechanisms** include transcriptional factors involved in oxygen-signaling and oxygen-carrying capacity and mitochondrial metabolism enzymes, improved behavior of fast-twitch fibers notably via compensatory vasodilatation, improved vascular relaxation and greater microvascular oxygen delivery as well as faster rate of phosphocreatine resynthesis.
- ▶ **In general**, RSH leads to superior repeated-sprint ability (i.e., faster mean sprint times or higher power outputs associated with a better resistance to fatigue during a repeated-sprint test) in normoxic conditions. RSH where hypoxia is induced by voluntary hypoventilation at low lung volume (named VHL) may also improve repeated-sprint performance more than in normoxia.
- ▶ **Practically**, RSH benefits have been demonstrated for a large range of team- (rugby, football, LaCrosse, Australian Football, field hockey), endurance (cycling, track and field, cross-country ski), racket (tennis) or combat (Jiu-Jitsu) sports.

KEY WORDS:

Altitude, High-Intensity, Team-Sports, Performance, Hypoxia

Zusammenfassung

- ▶ **Das Jahr 2018** markierte den 50. Jahrestag der Olympischen Spiele in Mexico City, dem Beginn wissenschaftlicher Untersuchungen von Hypoxie-Trainingsmaßnahmen. Seit dem ursprünglichen Konzept des „Live High – Train High“ ist eine Vielzahl von Höhen- und hypoxischen Trainingsformen entwickelt worden. Das Ziel dieses Reviews ist es, die neueste Methode, d. h. „Wiederholtes Sprinttraining in Hypoxie“ (Repeated Sprinttraining in Hypoxia, RSH) zu präsentieren. RSH trifft aktuell auf ein äußerst großes Interesse im Bereich des Höhentrainings, was anhand von 25 internationalen Studien in den vergangenen fünf Jahren deutlich wird, von denen nur zwei keine Leistungssteigernden Effekte fanden.
- ▶ **Mögliche Mechanismen** könnten veränderte Transkriptionsfaktoren sein, die in den Sauerstoff-Signalweg, die Sauerstoff-Transportkapazität, enzymatische Aktivität des mitochondrialen Stoffwechsels, eine stärkere kompensatorische Dilatation insbesondere in den fast-twitch Fasern, eine verbesserte vaskuläre Relaxation, eine größere microvaskuläre Sauerstoffabgabe und eine schnellere Kreatinphosphat Re-Synthese involviert sind.
- ▶ **Generell führt RSH** zu einer verbesserten Leistung bei Sprintwiederholungen (d.h., schnellere Sprintzeiten oder geringere Leistungsminderung bei wiederholten Sprints) unter normoxischen Bedingungen. Desgleichen wird, wenn die Hypoxie durch gewollte Hypoventilation bei geringem Lungenvolumen (VHL) erzeugt wird, die wiederholte Leistungsfähigkeit stärker verbessert als bei Training in Normoxie.
- ▶ **In der Praxis** profitieren unterschiedlichste Athleten von RSH, d. h. Teamsportler (Rugby, Fußball, LaCrosse, Australian Football, Feldhockey) Ausdauersportler (Rennradfahrer, Leichtathleten, Skilangläufer) Tennisspieler und Kampfsportler.

SCHLÜSSELWÖRTER:

Höhe, hohe Intensität, Team-Sport, Leistung, Hypoxie

Introduction

The year 2018 was the 50th anniversary of the Mexico Olympic games (2340 m, barometric pressure 580 mmHg) that was a pivotal point in sport history. Following the dominance of altitude acclimatized athletes during this event, altitude/hypoxic training has become increasingly popular among individual endurance athletes.

Therefore, the early 1970s was the starting point of the scientific investigation on the effectiveness of altitude training. At this time, several altitude training centres (e.g., Font-Romeu in France, Saint-Moritz in Switzerland, Colorado Spring in USA, Kunming in China) were developed for the exclusive purpose of “Live High – Train High” ▶

REVIEW

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(LHTH) and its associated hematological benefits in endurance aerobic sports – i.e., an increase in hemoglobin mass (Hb_{mass}) – which most often results in an improvement in maximal oxygen consumption ($\dot{V}O_{2\text{max}}$) (44). It has been speculated recently that the effect of altitude/hypoxic training on Hb_{mass} would depend on baseline level; i.e., athletes with already high initial values would not increase their Hb_{mass} markedly following the intervention; conversely, athletes with a low initial Hb_{mass} value would demonstrate greater Hb_{mass} gains (41). However, this analysis is based on inaccurate data (19, 48) and this theory has therefore been refuted (35). Hence, 3–4% Hb_{mass} gains have been observed in highly-trained athletes with already high initial Hb_{mass} values ($>14\text{g}\cdot\text{kg}^{-1}$) before the intervention (27, 44).

Since early 1970s' and the popularity of LHTH, many altitude/training methods have been developed such as "Live High-Train Low" (LHTL) or more recently "Live Low-Train High" (LLTH) (Table 1) (23, 38). The 50th anniversary of Mexico Olympic Games was the opportunity to scrutinize the major evolution of altitude training methods during these last 50 years. For this purpose, an international symposium was organized in Font-Romeu on 7–8th July 2018, where Ben Levine stated that "three inputs have been paramount over this period: 1. The introduction of Living High – Training Low (LHTL) in the 1990s; 2. The development of different normobaric hypoxic (NH) facilities that make easier for athletes to use hypoxic training everywhere; and 3. The development of RSH in 2010s".

The aim of the present review is therefore to present updated information on the most recent hypoxic method, the so-called "Repeated Sprint training in Hypoxia" (RSH).

RSH – Definition

The RSH paradigm requires the completion of maximal, short duration (typically $\leq 30\text{s}$) efforts interspersed with incomplete recovery periods ($\leq 60\text{s}$) in hypoxic environment (6, 23). A fundamental difference with "Intermittent Hypoxic Training" (IHT; i.e., interval-training performed in hypoxia) is the "all-out" effort required by RSH, which demands a very high recruitment of fast-twitch fibers. Single sprint performance is well preserved up to altitudes even higher than 3500 m (23) and for maximal efforts up to 60 s. However, repeating sprints in hypoxia results in performance decrement (2) with its magnitude being dependent on both the exercise duration and the exercise:recovery ratio, which in turn determines the oxidative and glycolytic contribution (13, 37).

Since 2007, it has been demonstrated that voluntary hypoventilation at low lung volume (VHL) could induce severe arterial oxygen desaturation (51, 52) leading to muscle (52) or cerebral (50) deoxygenation. In these conditions, the hypoxic state has been shown to be similar to what is observed at an altitude of about 2400m (49). The VHL approach is now recognized as one altitude training method potentially useful for a wide range of sporting activities (38). Noteworthy, two recent repeated-sprint studies have reported that training under hypoxic stress that is created through VHL produces larger improvement in repeated sprint ability (RSA) than training near sea level with an unrestricted breathing pattern (16, 45).

RSH – Mechanisms

Acute hypoxia induces an increased skeletal muscle sympathetic discharge leading to an enhanced vasoconstrictor activity during exercise. Despite this mechanism, contracting

skeletal muscle blood flow and coronary sinus blood flow increase, contributing to the maintenance of cardiac and peripheral O_2 delivery (26). This increase in blood flow is the consequence of a compensatory vasodilatation that aims at maintaining constant the total O_2 delivery to the muscle (10). At submaximal exercise intensity in hypoxia, this "compensatory" vasodilatation is well-established (10) and aims to ensure an augmented blood flow and maintenance (or limit the alteration) of oxygen delivery to the active muscles. Nitric oxide (NO) produced by endothelium seems the primary vasodilatory candidate since significant blunting of the augmented vasodilatation was reported with nitric oxide synthase (NOS) inhibition during hypoxic exercise (11). However, the source of NO contributing to compensatory dilatation seems less dependent on β -adrenergic mechanisms as exercise intensity increases. There are other candidates for stimulating NO release during higher intensity hypoxic exercise, such as ATP released from erythrocytes and/or endothelial derived prostaglandins. Overall, this enhanced hypoxic exercise hyperemia is proportional to the hypoxia-induced drop in arterial O_2 content and therefore its magnitude is larger at high than at low exercise intensity.

RSH is a recent training method mainly based on the above-described mechanisms and differs from interval-training in hypoxia performed at or near maximal aerobic power (13). Indeed, RSH performed at maximal intensity likely leads to a greater muscle perfusion and oxygenation (6) and specific muscle transcriptional responses (7, 14). Several mechanisms have been proposed to explain the effectiveness of RSH: during sprints in hypoxia, the compensatory vasodilatation and associated higher blood flow would benefit more to the fast-twitch fibers than the slow-twitch fibers (13). Consequently, RSH efficiency is likely to be fiber-type selective and intensity dependent; i.e. requiring maximal intensity and short exercise bouts (13, 37). With repeated maximal-intensity hypoxic efforts, specific skeletal muscle tissue adaptations may arise through the oxygen-sensing pathway (i.e., capillary-to-fiber ratio, fiber cross-section area, myoglobin content and oxidative enzyme activity such as citrate synthase) that do not occur in normoxia or to a lesser degree if they do occur (28, 53). Potential mechanisms involve transcriptional factors involved in oxygen-signaling and oxygen-carrying capacity and mitochondrial metabolism enzymes, improved behavior of fast-twitch fibers (34), notably via compensatory vasodilatation and faster rate of phosphocreatine resynthesis (7, 14, 28, 53). Based on preliminary results on mice performing supramaximal exercise in hypoxia, we speculate that RSH likely also improves responsiveness of the vascular bed (32).

RSH – Effects on Performance

To date, the increasing popularity of the RSH method is shown by the publication of 26 papers on this topic in the last five years and its effective use by many professional sports (e.g., Welsh rugby, Roland Garros tennis training centre, Aspire football Academy, Australian football squads, cross-country sprint skiers). Table 2 presents the updated review of these 25 studies + 1 meta-analysis on RSH (up to 1st January 2019) in a chronological order.

Firstly, despite its novelty, RSH is of high interest in applied exercise physiology: with 25 experimental studies published in the 5-years period following the pioneer RSH article in 2013 (14). This represents more studies than for LHTL (18 studies between 1997 (33) and 2002).

Table 1

Historical summary of altitude/hypoxic training methods (adapted from (23)).

| METHODS | DATE | SPORTS | PUBLICATIONS |
|---|-------|-------------|--|
| Live High – Train High (LHTH) | 1960s | Endurance | (Dill and Adams 1971) (12) |
| Intermittent Hypoxic Training (IHT) | 1960s | Endurance | (Roskamm et al. 1969) (43) |
| Live High – Train Low (LHTL) | 1997 | Endurance | (Levine and Stray-Gundersen 1997) (33) |
| Resistance Training in Hypoxia (RTH) | 2000s | Power | (Friedmann et al. 2003) (17) |
| Repeated-Sprint in Hypoxia (RSH) | 2013 | Team/racket | (Faiss et al. 2013) (14) |
| Live High – Train Low and High (LHTLH) | 2015 | Team/racket | (Brocherie et al. 2015) (9) |
| Live High – Train High and Low (LHTHL) | 2015 | Endurance | (Rodriguez et al. 2015) (42) |
| RSH with Voluntary Hypoventilation at Low lung volume (RSH-VHL) | 2017 | Team/racket | (Trincat et al. 2017) (45) |

Secondly, this method is of interest in a wide range of team- (rugby league, rugby union, football, LaCrosse, Australian Football, field hockey), endurance (cycling, track and field, cross-country ski), racket (tennis) or combat (Jiu-Jitsu) sports.

Thirdly, in general, superior performance outcomes, in particular for RSA, in normoxic conditions have been associated with RSH vs. RSN (repeated sprint training in normoxia) studies. For instance, RSH produces faster mean sprint times and/or smaller speed decrements compared to RSN (See Table 2 and (6)), likely resulting from an improved fatigue resistance. Following 6-8 RSH sessions, the number of sprints completed before task failure increased in well-trained cyclists (14), cross-country skiers (15) or rugby players (16) during an “open-loop” protocol. Improvement in RSA has also been reported in “close-loop” protocol (1, 16, 21, 30, 31, 40, 47) meaning an increased velocity or power output during a repeated-sprint test. Recently, larger improvement in time to exhaustion during a tennis-specific incremental test was also reported in tennis players following RSH vs RSN (16). Negligible enhancement in glycolytic capacity is generally reported with RSH. However, RSH-VHL might be more effective for improving glycolysis due the hypercapnic effect of the apnea phase (45). Similarly, $\dot{V}O_{2\max}$ improvement caused by RSH are also likely minimal (6, 14).

Fourthly, only two studies (24, 39) of 25 did not report any additional beneficial effects of RSH: the completion of 12–15 cycling RSH sessions over 4-5 weeks did not lead to further improvement in RSA, compared to similar normoxic training (24, 39). Arguably, the use of non-specific cycling RSH training in team- or racket- sport athletes performing predominantly run-based activities (24) and methodological shortcomings (i.e., absence of protective pacing measures leading to submaximal intensity, especially over long repeated-sprints sets) may potentially explain the absence of additional effect of RSH vs. RSN.

Conclusions and Perspectives

More than 50 years after the first scientific publications on altitude/hypoxic training and the launch of prestigious altitude training centers (e.g., Font-Romeu in France; Saint Moritz in Switzerland) for the preparation of the Mexico 1968 Olympic Games, major progress has been made in improving athletic performance by hypoxic training measures and understanding its underlying mechanisms. RSH is undoubtedly a promising and well-tolerated training model (6, 8) although its underlying mechanisms remain partly hypothetical at this stage. Further researches are required in order to confirm if the effectiveness of RSH comes from an improved muscle blood perfusion, which in turn would benefit from optimized oxygen extraction by fast-twitch fibers.

Recently, positive adaptive mechanisms on endothelial function leading to larger vascular relaxation have been described in mice performing supramaximal exercise in hypoxia, when compared to similar exercise in normoxia (32), paving the way for further investigation on the therapeutic use of high-intensity hypoxic exercise in patients (36). ■

Conflict of Interest

The authors have no conflict of interest.

Table 2 – Part 1

Published studies on RSH (n=26). M=Male, F=Female. The significantly (P<0.05) larger benefits of RSH are presented in bold. NH=Normobaric Hypoxia; Con=Control group without RS training; RSA=Repeated Sprints Ability; RSE=Repeated Sprint Exercise; RSH=Repeated-Sprint Training in Hypoxia; RSN=Repeated-Sprint Training in Normoxia; SIH=Sprint-Interval Training in hypoxia; PO=Power Output; P_{max}=maximal PO; P_{mean}=Mean PO; LT4=Lactic Threshold, i.e. lactate concentration of 4 mmol. L-1, Yo-Yo IR, Yo-Yo IR test; Pcr=Phosphocreatine; (tHb)=total hemoglobin/myoglobin; VHL=voluntary hypoventilation; OBLA=onset of blood lactate accumulation; La=Blood lactate concentration.

| AUTHORS (YEAR) | SUBJECTS | TRAINING PROTOCOL (SESSIONS, TYPE, ALTITUDE, CONTENT) | GROUPS | MEANING DIFFERENCES |
|------------------------------|--|---|-------------------------------------|--|
| Faiss et al. (2013) (14) | Moderately trained cyclists M | 8 sessions, 4 wk., cycling, 3000m (NH). 3x5x10s–20s passive recovery. | RSH, N=20 RSN, N=20 CON, N=10 | +6% P _{mean} sprints +38% more sprints during RSE. +7% P _{mean} sprints, no more sprints during RSE. NS changes. No RSE improvement. |
| Galvin et al. (2013) (18) | Rugby players M | 12 sessions in 4 wk., treadmill, 3500m (NH). 10x6s–30s passive recovery. | RSH, N= 15 RSN, N=15 | +33% Yo-Yo +14% Yo-Yo |
| Gatterer et al. (2014) (22) | Football players M | 7-8 sessions in 5 wk., running, 3000m (NH). 3x10sx4.5 m round-trip shuttle - 20s passive recovery. | RSH, N=5 RSN,=8 | +20% Yo-Yo IR, -38% slope fatigue curve during RSA. +21% Yo-Yo IR, +9% slope fatigue curve during RSE. |
| Gatterer et al. (2015) (20) | Football players M | 8 sessions in 12 days, shuttle-run, 3300m, (NH). RSE=6x40-m (6x20-m back & forth, 20s passive recovery. | RSH, N=7 RSN, N=7 | Improvement RSE 0.91% mean time (s) Improvement RSE 0.39% mean time (s) |
| Brocherie et al. (2015) (5) | Well trained youth football players M | 10 sessions in 5 wk., racing, 2900m (NH). 5x4x5s–45s passive recovery. | RSH, N=8 RSN, N=8 | RSH vs. RSN; -4% Time of 1 st sprint, -4% time of the cumulative sprint, -2% Time of 1 st sprint, -2% Cumulative sprint time. |
| Faiss et al. (2015) (15) | Highly-trained XC-skiers (11=M, 6=F) | 6 sessions in 2 wk., double-pole ergometer cross-country skiing, 3000m (NH). 4x5x10s–20s passive recovery. | RSH, N=9 RSN, N=8 | +25% P _{max} Sprints +57% more sprints during RSE. +21% P _{max} sprints, no more sprints during RSE. |
| Kasai et al. (2015) (30) | Lacrosse players F | 8 sessions in 4 wk., ergo-cycle, 3000m, (NH). 2x10x7s–30s passive recovery. | RSH, N=16 RSN, N=16 | +5% P _{max} sprints on ergometer after RSH vs. 1.5% in RSN. +9.7% P _{mean} Sprints vs. 6%=VO _{2max} in both groups. |
| Goods et al. (2015) (24) | Australian football players M «semi-elite» | 15 sessions in 5 wk., ergo-cycle, (3000m, (NH). 3x9x5s–active recovery (self-selected pace). | RSH, N=9 RSN, N=10 | +4.7% P _{max} and +10.3% P _{mean} +8.6% P _{max} and +13.6% P _{mean} sprints -1.1% P _{max} and 1.4% P _{mean} , for the controlled group. +2.3% (RSH), +1.8% (RSN), +1.1% (controlled) on the average time of sprints (running) between before and after Training. |
| Montero & Lundby (2017) (39) | Moderately trained endurance athletes M | 12 sessions in 4 wk., ergo-cycle, 3000m (NH). 4x5x10s–20s active recovery. | Crossover RSN – RSH, N=15 | RSH Post 1 (3-5 days); +6.7% PPO, +14% MPPPO, +12.1% MPO. Post 2 (12 days); +6.8% PPO, +18.7% MPPPO, +18.1% MPO. RSN Post 1 (3-5 days); +7.2% PPO, +16.3% MPPPO, +9.9% MPO. Post 2 (12 days); +6.9% PPO, +16.3% MPPPO, +14.2% MPO. No differences between RSH & RSN RSH-induced increase in Δ(tHb) during RS in hypoxia compared with normoxia. |
| Hamlin et al. (2017) (25) | Well-trained rugby players M | 6 sessions in 3 wk., ergo-cycle, 3000m (NH). Followed by 3 post-tests in 2 wk., then 2 sessions in 1 wk. Followed by 2 Post-tests in 2 weeks. 4x5x5s–25s active recovery. | RSH, N=8 RSN, N=10 | – 2.0%, – 2.2%, – 1.6% RSE post 3 wk., post 4 wk., post 5 wk., respectively, in favor or RSH No difference between the groups on the performance of the YIR1. |
| Brocherie et al. (2017) (8) | Elite field hockey players | LHTL, 6 sessions in 2 wk., running, 3000m, (NH). 4x5x5s–25s passive recovery. | RSH, N=11 RSN, N=12 | RSH vs. RSN; -3.6% difference in RPE (average), -7.8% difference in overall peripheral discomfort and -23.2% difference in lower-limb discomfort |

Table 2 – Part 2

| AUTHORS (YEAR) | SUBJECTS | TRAINING PROTOCOL (SESSIONS, TYPE, ALTITUDE, CONTENT) | GROUPS | MEANING DIFFERENCES |
|--------------------------------------|---|---|--|---|
| Trincat et al. (2017) (45) | Competitive swimmers (9=M, 7=F) | 6 sessions in 2 wk that included 2x16x15m with 30s send off. RSH-VHL low level active recovery. | RSH-VHL,=8 RSN,=8 | RSH-VHL +35% number of sprints increased and (La) max compared to RSN |
| Woorons et al. (2017) (52) | Well trained subjects (8=M, 1=F) | RSH-VHL cycling. 2x8s–24s passive recovery. | RSH-VHL,=5 RSN,=4 | PO similar. Higher muscle reoxygenation during recovery in 2nd half of RSE. |
| Kasai et al. (2017) (29) | University sprinters M | 6 consecutive days, 3000m (NH) vs normoxia, 2 sessions/ day Morning: RS (5x6s – 24s recovery) 3x + (4x20s 5–15 min) Afternoon: 3x5x6s–36s recovery + 4x20s–40s passive recovery | RSH, N=10 RSN, N=9 | + 3% max pedaling frequency and Pmax. Increases in muscle glycogen (+79.9%) and Pcr (+3.9%). |
| Brocherie et al. (2017) (6) | Meta-Analysis | | N=202 | Pmean during RSE further enhanced (P=0.05) with RSH vs RSN. Best performance improved to same extent between RSH and RSN. |
| Zwaard et al. (2018) (46) | Elite field hockey players M | LHTL, 6 sessions in 2 wk. 3000m, (NH). 4x5x5s–25s passive recovery | LHTLH=6 LHTL=6 LLTL=6 | LHTLH +35% Succinate dehydrogenase, LHTLH and LHTL improved combination of fiber size and oxidative capacity. |
| Fornasier-Santos et al., (2018) (16) | Highly trained rugby union players M | RSH-VHL 8 sessions in 4 wk., running 2-3x8x40m on 30s- Semi-active recovery (walking) | RSH-VHL,=11 RSN,=10 | Number of sprints increased +64% |
| Oriishi et al. (2018) (40) | College 400-800m runners F | 4 sessions in 6 days., running, RSH=LH (200m), TH (3000m) RSN=LLTL | RSH=7 RSN=8 | P _{max} in the MART +2.5% Lactate Concentration decreased (p<0.05) at submax velocities. |
| Brechbuhl et al. (2018) (3) | Well trained tennis players (16=M, 4=F) | 5 sessions in 12 days, 3000m, (NH). 4x5 "sport specific" sprints – 50s passive recovery | RSH, N=9 RSN, N=9 | Time to exhaustion and OBLA improved. +13.8% in ball accuracy testing. |
| Brechbuhl et al. (2018) (4) | Rookie tennis player M | 6 sessions in 14 days, shuttle-run, 3000m (NH). 4x5x6s–24s passive recovery | RSH,=1 (Case-study) | No changes at +3 days post-RSH Improved physical fitness (single sprint time (–4.5%), RSA total time (–3.1%) and sprint decrement (–16.7%), YYIR2 total distance covered (+21.4%)) at +21 days |
| Wang et al. (2018) (47) | Recreational-active M | 8 sessions in 4 wk., ergo-cycle, 3000m, (NH). β-Alanine supplementation (NB) or normoxia placebo (NP) in both RSH or RSN groups. 3x5x10s–20s active recovery. | RSH-NB=10 RSH-NP=9 RSN-NB=11 RSN-NP=8 | +4% PO in RSE for RSH vs RSN groups. |
| Gatterer et al. (2019) (21) | Amateur team sports players M | 9 sessions, x3 wk., ergo-cycle, 2200m, (NH). RSH: 3x5x10s–20s 5-min recovery SIH: 4x30s–5-min recovery | RSH, N=6 SIH, N=5 | RSE running time improved by -0.14 s and -0.11 s after RSH and SIH (p=0.012). RSH improved reoxygenation during RSE |
| Brocherie et al. (2018) (7) | Elite field hockey players M | 6 sessions, running, 3000m (NH). 4x5x5s–25s passive recovery. | LHTLH,=8 LHTL,=11 LLTL,=9 | Larger LHTLH-induced adaptations in molecular responses in O2 signaling (HIF-1α) and transport (VEGF, Mb) and mitochondrial biogenesis and metabolism (PGC-1α, TFAM, CS) compared with LHTL and LLTL. |
| Brechbuhl et al. (2018) (4) | Rookie tennis player M | 6 sessions in 14 days, shuttle-run, 3000m (NH). 4x5x6s–24s passive recovery | RSH,=1 (Case-study) | No changes at +3 days post-RSH Improved physical fitness (single sprint time (–4.5%), RSA total time (–3.1%) and sprint decrement (–16.7%), YYIR2 total distance covered (+21.4%)) at +21 days |
| Wang et al. (2018) (47) | Recreational-active M | 8 sessions in 4 wk., ergo-cycle, 3000m, (NH). β-Alanine supplementation (NB) or normoxia placebo (NP) in both RSH or RSN groups. 3x5x10s–20s active recovery. | RSH-NB=10 RSH-NP=9 RSN-NB=11 RSN-NP=8 | +4% PO in RSE for RSH vs RSN groups. |

Table 2 – Part 3

| AUTHORS (YEAR) | SUBJECTS | TRAINING PROTOCOL (SESSIONS, TYPE, ALTITUDE, CONTENT) | GROUPS | MEANING DIFFERENCES |
|-----------------------------|--|---|---------------------------------|---|
| Gatterer et al. (2019) (21) | Amateur team sports players M | 9 sessions, x3 wk., ergo-cycle, 2200m, (NH). RSH: 3x5x10s–20s 5-min recovery SIH: 4x30s–5-min recovery | RSH, N=6 SIH, N=5 | RSE running time improved by -0.14 s and -0.11 s after RSH and SIH (p=0.012). RSH improved reoxygenation during RSE |
| Brocherie et al. (2018) (7) | Elite field hockey players M | 6 sessions, running, 3000m (NH). 4x5x5s–25s passive recovery. | LHTLH,=8 LHTL,=11 LLTL,=9 | Larger LHTLH-induced adaptations in molecular responses in O ₂ signaling (HIF-1 α) and transport (VEGF, Mb) and mitochondrial biogenesis and metabolism (PGC-1 α , TFAM, CS) compared with LHTL and LLTL. |
| Kasai et al. (2019) (31) | University sprinters M | 5 consecutive days, 3000m (NH) vs normoxia, 2 sessions/ day Morning: RS (5x6s–24s recovery) 3x + (4x20s 5-15 min) Afternoon: 3x5x6s–36s recovery + 4x20s–40s passive recovery | RSH, N=9 RSN, N=9 | Running time 0-10m improved (before, 1.39 \pm 0.01s; after, 1.34 \pm 0.02s, P<0.05) Increase in PCr content (31.5 \pm 1.3 to 38.2 \pm 2.8 mM, P<0.05). |
| Beard et al. (2019) (1) | Elite Rugby union players M | 4 sessions, cycling, 3000m (NH). 3x8x10s–20s passive recovery | RSH, N=10 RSN, N=9 | RSA P _{max} +6.3% P _{mean} + 6.5% improved |
| Woorons et al. (2019) (50) | Highly-trained Jiu-Jitsu fighters (7=M, 3=F) | RSH-VHL shuttle sprint running. 2x6s–15s passive recovery. | RSH-RSN, N=10 | SPO ₂ lower (89.8 vs 97.7 %; p < 0.01) and cerebral oxygenation (-6.1 \pm 5.4 vs -1.5 \pm 6.6 μ a) were lower than in RSN. higher during recovery periods. RSE performance not impaired with RSH-VHL. |

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