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Repeated-Sprint Training in Hypoxia in Well-Trained Tennis Players

Master Thesis in Human Movement and Sport Sciences

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Abstract

Introduction: Repeated-sprint training in hypoxia (RSH) has been reported to improve intermittent high-power performance, in particular by enhancing fatigue resistance and thus parameters such as repeated-sprint ability (RSA). One of RSH's primary interest lies in the fact that adaptations are triggered with reduced hypoxic exposure (< 5 h) and with less than six to ten training sessions. Since tennis players have full in-season calendars with limited training time frames in-between tournaments, and given the explosive nature of the sport, it was hypothesized that RSH would be a suitable method to improve tennis-specific performance. **Methods**: Thirty well-trained tennis players (28.8 ± 5.9 years, 72.3 ± 9.9 kg, 179.1 ± 6.7 cm, FTF ranking: 1.54 ± 2.84, $\dot{V}O_{2max}$ 57.2 ± 6.2 ml/min/kg) were randomly distributed in three groups: two experimental groups who performed five sessions of four sets of five 8-s maximal sprints with ball strokes either in normoxia (RSN) or in normobaric hypoxia (RSH), at a simulated altitude of 3000 m, and a control group (CON). Subjects were tested before (PRE), after (POST1) and 21 days after (POST2) the intervention. Tests consisted in the Test to Exhaustion Specific to Tennis and a RSA test during which leg muscle and cerebral oxygenation were assessed with near-infrared spectroscopy. **Results**: From PRE to POST1 and to POST2, RSH improved the time to exhaustion by 18.2% and by 17.3% (p<0.001), respectively. They also increased by 24.4% (p=0.003) and by 19.8% (p=0.027) the time to the onset of blood lactate accumulation, while the two other groups did not change their performance significantly. RSH was also the only group to improve tennis performance at the last stage at POST1 (+32.8%, p=0.025). Regarding the RSA test total time, both groups ameliorated it, though RSH (-1.9%, p=0.009, at POST1; -2.2%, p=0.002, at POST2) to a greater extent than RSN (-1.2%, p=0.049 at POST1; -1.2%, p=0.041, at POST2) in comparison to PRE values. Only RSH improved the RSA test best sprint time (-2.33%, p=0.01 at POST1; -3.4%, p<0.001, at POST2). Leg muscle tissue saturation was lower at POST1 (-81.6 ± 7.5% vs -72.5

± 24.5%, p<0.001) only for the RSH group, but it was back to similar to PRE values at POST2, even though performance gains were maintained. Significant decrease in cerebral oxygenation were measured in all three groups at POST1 and POST2. **Conclusion**: Specific (i.e. with strokes) repeated-sprint training in hypoxia enhanced RSA and tennis-specific performance to a larger extent than similar training in normoxia and therefore appears valuable for well-trained tennis players.

Résumé

Introduction : Il a été reporté que la répétition de sprints en hypoxie (RSH) améliore les performances d'haute intensité, notamment en renforçant la résistance à la fatigue, et donc également certains paramètres liés à la capacité à reproduire des sprints (« repeated-sprint ability », RSA). Un des intérêts principaux de RSH réside dans le fait que des adaptations sont induites avec une exposition hypoxique limitée (< 5h), et avec moins de six à dix sessions d'entraînement. Étant donné que les joueurs de tennis ont durant la saison un calendrier chargé avec des créneaux d'entraînement limités dû aux nombreux tournois, et, étant donnée la nature explosive du sport, il a été hypothétisé que RSH saurait une méthode favorable pour améliorer la performance spécifique au tennis. **Méthodes** : Trente joueurs de tennis bien entraînés ((28.8 ± 5.9 ans, 72.3 ± 9.9 kg, 179.1 ± 6.7 cm, FTF ranking: 1.54 ± 2.84, $\dot{V}O_{2max}$ 57.2 ± 6.2 ml/min/kg) ont été répartis au hasard dans trois groupes : deux groupes expérimentaux qui ont effectué cinq sessions de quatre sets de cinq sprints de 8 s contenant des frappes de balles, soit en normoxie (RSN) soit en hypoxie normobare (RSH), a une altitude simulée de 3000 m, et un groupe contrôle (CON). Les sujets ont été testés avant (PRE), à la fin (POST1), et 21 jours après (POST2) l'intervention. Les tests étaient constitués du « Test to Exhaustion Specific to Tennis (TEST) » et d'un test RSA durant lequel l'oxygénation musculaire (quadriceps) et cérébrale étaient mesurées via une spectroscopie proche infrarouge. **Résultats** : Entre PRE et POST1 et POST 2 le groupe RSH a amélioré le temps jusqu'à l'épuisement de 18.2% et 17.3% (p<0.001), respectivement. Ils ont augmenté de 24.4% (p=0.003) et de 19.8% (p=0.027) le temps jusqu'au seuil d'accumulation du lactate sanguin. Il était également le seul groupe a amélioré sa performance technique lors du dernier palier du TEST lors de POST1 (+32.8%, p=0.025). Les deux autres groupes n'ont pas changé de manière significative les paramètres évoqués ci-dessus. Par rapport au temps total du test RSA, les deux groupes l'ont amélioré, bien que le groupe RSH (-1.9%, p=0.009, at POST1; -2.2%, p=0.002, at POST2) de manière plus

prononcée que le groupe RSN (-1.2%, p=0.049 at POST1; -1.2%, p=0.041, at POST2) en comparaison avec les valeurs PRE. Seul le groupe RSH a diminué le temps du meilleur sprint du test RSA (2.33%, p=0.01 at POST1; -3.4%, p<0.001, at POST2). La saturation tissulaire de la jambe était plus basse à POST1 (-81.6 ± 7.5 vs -72.5 ± 24.5, p<0.001) seulement pour le groupe RSH, mais était à nouveau à des valeurs proches de PRE à POST2, bien que les gains de performances étaient maintenus. Une diminution significative de l'oxygénation cérébrale a été mesurées dans les trois groupes. **Conclusion** : La répétition de sprint en hypoxie spécifique au tennis (i.e. avec frappes de balles) améliore la RSA et la performance tennistique dans une plus grande mesure qu'un entraînement similaire effectué en normoxie, et apparait donc comme relevant comme forme d'entraînement pour des joueurs de tennis bien entraînés.

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Abbreviation Index

Δ: Delta

ATP: Adenosine triphosphate

ADP: Adenosine diphosphate

BA: Ball accuracy [% of balls hit inside a defined zone]

BF: Ball frequency [in ball per min^{-1}]

BV: Ball velocity [in km/h^{-1}]

CON: Control

FT: Fast-twitch

FTF: French Tennis Federation

$F_IO_{2:}$ Fraction of inspired oxygen [in %]

HR: Hear rate [in beats per minute]

HR_{max}: maximum heart rate [in beats per min^{-1}]

HHb: Deoxy-hemoglobin [in µm]

[La]: blood lactate concentration [in mmol. L^{-1}]

NIRS: Near-infra-red spectroscopy

O_2Hb: Oxy-hemoglobin [in µm]

OBLA: Onset of blood lactate accumulation

RSA: Repeated-sprint ability

RSA_{best}: RSA test best sprint time [in s]

RSA_{TT}: RSA test cumulated sprint time [in s]

RSH: Repeated-sprint training in hypoxia

RSN: Repeated-sprint training in normoxia

RPE: Rate of perceived exertion

ROF: Rate of fatigue

S_{dec}: Percentage of sprint decrement [in %]

tHb: Total hemoglobin [in µm]

TL: Training load

TP: Tennis performance index [BA x BV, in a.u.]

TSI: Tissue saturation index [in %]

$\dot{V}CO_2$: Carbon dioxide production [in l/min^{-1}]

$\dot{V}CO_2/\dot{V}O_2$: Respiratory exchange ratio

$\dot{V}E$: Minute ventilation [in L/min^{-1}]

$\dot{V}O_2$: Oxygen uptake [in ml/min/kg]

$\dot{V}O_{2max}$: Maximal oxygen uptake [in ml/min/kg]

1 Introduction

1.1 Tennis performance and its characteristics

In high level tennis, performance and its limits are multifactorial. Indeed, different aspects such as the athletes' endurance and physiological capacities, their speed and agility, their technique, their tactics and psychological strength are intrinsically linked and have all an impact on the player's performance (Brechbuhl et al., 2018a; Kolman et al., 2018; Kovacs, 2006, 2007). Any lack of efficiency in one of these qualities would impact on one's ability to reach the highest level of tennis. However, defining which of these different parameters is the paramount component of performance is intricate for they are all inherent to a player's current level. This is primarily due to the fact that tennis is subject to unpredictability and a significant degree of variation. Unpredictability of rally length, match duration, position and type of shot played by the opponent and the resulting appropriate shot needed to be executed accordingly, overall strategy, weather and court surface (Girard and Millet, 2004; Kovacs, 2006). All these different parameters influence the physiological stress endured by the player.

Tennis has been previously defined as "*intermittent exercise, alternating short (4-10 seconds) bouts of high-intensity exercise and short (10-20 s) recovery bouts interrupted by several resting periods of longer duration (60-90 s)*" (Fernandez et al., 2006). However, these figures may largely vary from one point to the next. For instance, the average point duration on clay for male players at the international level is reported to be 7.5 ± 7.3 s (Mendez-Villanueva et al., 2010), and 7.2 ± 5.2 for women (Fernandez-Fernandez et al., 2008). The large standard deviations demonstrate the inter-point variability that shapes each game of tennis.

Likewise, the match duration is also contingent on variation, in addition to depending on whether it is a best out of three or out of five sets match. Although an average length of 1.5 h has been suggested (Bergeron et al., 1995; Kovacs, 2006), this number may loosely vary, and can reach greater lengths, such as the 2012 Australian Open men singles final (Djokovic vs Nadal), which saw the former triumph after 5 h and 53 min[1]; and can even reach extreme durations such as the 2010 Wimbledon men first round (Mahut vs Isner) which lasted a staggering record time of 11 h and 5 min[2].

Admittedly, these total durations comprise effective playing time (i.e., the time during which the ball is in play) but also end-changing time and recovery time in-between points, games and sets. This effective time has been previously measured and, depending on the sex and court surface, varies between 17.5 and 21.9% of the total time for players at the international level (Fernandez et al., 2006; Fernandez-Fernandez et al., 2008; Mendez-Villanueva et al., 2010; Morante and Brotherhood, 2006), whereas it is situated between 21 and 38.5% for regional level players (Morgans et al., 1987; Richers, 1995).

During this effective time, tennis players hit around 3 shots per rally, with ~80% of these shots hit within 2.5 m of their position in the middle of the baseline, ~10% within 2.5 and 4.5 m, and ~5% need a displacement greater than 4.5 m (Ferrauti et al., 2003). In Grand Slam tournaments (i.e., Australian Open, French Open, Wimbledon and US Open), between 1997 and 1999, rallies duration averaged 7.1 ± 2.0 s for women and 5.2 ± 1.8 s for men, which was statistically significant, revealing sex differences in the game (O' Donoghue and Ingram, 2001). Players run an average of 3 m per shot and between 8 and 15 m per rally, which result in a total of 1'300 to 3'600 m per hour of play (Deutsch et al., 1988; Fernandez-Fernandez et al., 2009), depending

[1] https://ausopen.com/history/memorable-moments/2012-great-tennis-match-all-time (visited on 19.02.19)

[2] http://www.wimbledon.com/en_GB/aboutwimbledon/history_2010s.html (visited on 19.02.19)

on the player's level and court surface (Girard and Millet, 2004; Murias et al., 2007; O' Donoghue and Ingram, 2001). They shift directions an average of four times in a given point (Deutsch et al., 1988). Most of these displacements and movements are executed in an explosive manner with maximal speed/power output intent, resulting in a total of between three hundred and five hundred explosives movements for each player in a three set match (Deutsch et al., 1988; Fernandez et al., 2006).

All these different figures tend to suggest that tennis is a high-intensity sport and would thus imply it requires energy supplies mainly dependent on anaerobic pathways. However, when analyzing players' heart rate (HR) during the game, it was found that players spent more than 85% of the time in low or moderate intensity (Gomes et al., 2016), with average HR ranging from 135 and 161 bpm depending on the level, sex and court surface, which corresponds to ~60-80% of HR_{max} (Christmass et al., 1998; Docherty, 1982; Elliott et al., 1985; Fernandez et al., 2006; Fernandez-Fernandez et al., 2007, 2008; Ferrauti et al., 1997, 2001a; Gomes et al., 2016; König et al., 2001; Morgans et al., 1987; Smekal et al., 2001).

Similarly, when contemplating oxygen uptake ($\dot{V}O_2$), few studies measured this parameter during an actual match, and therefore most scientific articles studied players in other forms, such as incremental tests. The few authors who did evaluate it in play-like conditions, demonstrated that during a game, players at the national and international level had an average $\dot{V}O_2$ between 23.1 ± 3 and 29.1 ± 5.6 ml/min/kg depending, once again, on sex, level and surface, which corresponds to ~ 46 to 59% of their maximal oxygen uptake ($\dot{V}O_{2max}$) (Fernandez et al., 2005b; Ferrauti et al., 1998; Seliger et al., 1973; Smekal et al., 2001). For regional level and young players, this value tends to be slightly higher, stretching from 30 ± 0 to 40,3 ± 5.7 ml/min/kg, depending on type of play (i.e., serve-volley player, baseline attacker, baseline defender), level, sex and court surface (Bernardi et al., 1998; Dansou et al., 2001; Girard and Millet, 2004).

Consequently, when scrutinizing the average HR and $\dot{V}O_2$, numbers tend to categorize tennis as an aerobic sport rather than anaerobic. In spite of the numerous high-intensity bouts, the sport has also a major and primary aerobic component. As a result, it could be logically inferred that tennis players necessitate high aerobic qualities, and have or need in consequence, very high maximal oxygen uptake ($\dot{V}O_{2max}$) values. Yet, although professional players playing at the international level are known to perform well in maximal oxygen uptake tests, with values ranging between 44 ml/kg/min and 69 ml/kg/min (Baiget et al., 2015; Banzer et al., 2009; Bergeron et al., 1991; Brechbuhl et al., 2017; Christmass et al., 1998; Copley, 1980; Docherty, 1982; Elliott et al., 1985; Fernandez et al., 2005a; Smekal et al., 2001), having higher values of $\dot{V}O_{2max}$ (<65 ml/kg/min) does not necessarily translate to a better tennis performance, and therefore training time might be better spent on other factors, then prioritizing the improvement of $\dot{V}O_{2max}$ (Kovacs, 2007).

Therefore, all in all, tennis players require a combination of speed, coordination, power, agility in addition to a medium-to-high anaerobic *and* aerobic fitness (Bergeron et al., 1991; Fernandez-Fernandez et al., 2009; Kovacs, 2007; Parsons and Jones, 1998; Ulbricht et al., 2016). Indeed, high-velocity/power-output moves such as the serve and powerful attacking strokes, as well as explosives changes of direction and quick accelerations necessitate a high anaerobic ATP production. However, the intensity being also majorly submaximal and in order to recover well between rallies, and sustain a significant power-output for a duration of several hours, a high level of endurance and thus aerobic fitness is also required (Fernandez-Fernandez et al., 2009; Kovacs, 2004, 2006, 2007).

Hence, both energy supply pathways are fully involved and vital, given that during a match, metabolic requirements will alternate between fast anaerobic pathways (i.e., 1) adenosine triphosphate (ATP) present within the muscle fibers, 2) phosphocreatine's direct adenosine diphosphate (ADP) phosphorylation into ATP, 3) glycolysis and glycogenolysis) to provide for

high-intensity bouts; and slow aerobic pathways (i.e., mitochondrial cellular respiration through the Krebs cycle) to cover the prolonged submaximal exercise and to replenish anaerobic energy sources (Elliott et al., 1985; Fernandez et al., 2006; Fernandez-Fernandez et al., 2009; König et al., 2001; Kovacs, 2004, 2006; Marieb and Hoehn, 2016; Smekal et al., 2001).

Likewise, muscle fiber composition also corroborates the duality of energy system pathways. It was assessed through immunohistochemistry that in professional tennis players, *vastus lateralis* muscle fibers composition was as follows: 62 ± 7% type I; 33 ± 4% type IIa and 5 ± 3% type IIx (Sanchis-Moysi et al., 2010). The level of type I fibers is relatively high, which suggests indeed an activity requiring an important aerobic component, yet not as high as typical long-distance endurance elite runners, who have been reported to have type I fiber percentage which can reach 74 to 85% (Simoneau and Bouchard, 1989; Trappe et al., 1995). On the other hand, with tennis players, the percentage of type IIa fibers is also relatively high, and may be compared to elite sprinters' percentage of type IIa fibers, which was confirmed to be 34% (Trappe et al., 2015). These fibers are paramount given their high force production and fatigue resistance (Stienen et al., 1996). This summarizes well the characteristics of tennis, sitting between aerobics needs, but with the main high-power outputs requiring an important quantity of fast fatigue-resistant fibers. Therefore, when prescribing training, both domains need to be stimulated and basic aerobic training should be superimposed with tennis-specific high-power anaerobic training.

1.1 Tennis-related fatigue

It was reported that in the 2012 Australian Open final opposing Novak Djokovic to Rafael Nadal during a 5 h 53 min match, both players ran more than 6.5 km, reached speeds superior to 20 km/h, and hit more than one thousand and one hundred groundstrokes at an average velocity of 97 km/h for Nadal, and 107 km/h for Djokovic (Reid and Duffield, 2014). Admittedly this is an extreme, but needless to say that due to its high-power output nature,

tennis activity has an important physiological impact on the body. The long match durations coupled with repetitive high-intensity bouts wears out the organism on different levels, which will in turn affect tennis performance.

Muscle fatigue has been described as an exercise-induced reduction in maximal voluntary muscle force, and/or inability to maintain a given force production (Gandevia, 2001). This may be caused peripherally at the muscle cell level, due to an excitation-contraction coupling alteration, and/or a disturbance in the surface membrane, and/or due to metabolic event (i.e., hydrogen (H^+) ions, inorganic phosphate, ADP, etc. accumulation, which disturb the Na^+/K^+ balance and Ca^{2+} cycling and thus actin-myosin interaction) (Fitts, 1994). Force production diminution may also be the result of central fatigue, generated by a decrease in the capacity of the nervous system to drive motoneurons efficiently. The phenomenon may be generated by a decline of motor unit firing rates at the muscle level; and/or by a reduction of cortical excitability at the cerebral cortex level; and/or by decline in supraspinal drive; and/or by an input alteration from muscle spindles, tendon organs and group III and IV muscle afferents innervating skeletal muscles at the spinal level (Gandevia, 2001).

On a tennis court this is meaningful, for any fatigue causing an alteration in the ability to contract muscle fibers or in their efficiency will translate into a limited power output, and thus a diminution of the playing efficiency and overall tennis performance (Girard and Millet, 2008). A 3-hour tennis protocol has been demonstrated to decrease maximal voluntary contraction (MVC) which was correlated with a decline in muscle activation, hence demonstrating that prolonged tennis activity alters voluntary force production due to a diminished neural input to the working muscles (Girard et al., 2011). Similarly, the same authors measured a reduction in leg stiffness, which was also correlated with a decrease in MVC after 3 h of tennis match play (Girard et al., 2006). The impaired leg muscle stiffness appears to be due to a repetition of the stretch-shortening cycle (Avela et al., 1999). This would in turn suggest that tennis activity

related fatigue might be partially caused by a decline in the muscle-tendon complex's capacity to generate force.

It has also been established that 3h of tennis match play induced an increase in muscle soreness as well as a decrease in maximal voluntary force (MVF) (Lees, 2003).

These different types of fatigue measured physically, occur on the court and affect key aspects of the game. On top of decreasing ball velocity, the diminished power output also results in slower movements, and therefore inadequate positional play and mistimed or mis-hit shots, which highly contribute to a reduction in shot accuracy (Lees, 2003). Indeed, 2 h of demanding tennis activity result in increased errors in first serves and ground strokes, as well as a hitting accuracy reduction in ground strokes up to 81% (Davey et al., 2002, 2003; Vergauwen et al., 1998). Similarly, it has been suggested that the decrement in running speed caused by fatigue leads to inaccurate stroke preparation, and a decrease in stroke speed (Ferrauti et al., 2001b). Furthermore, muscular fatigue has also been known to increase reaction time, which is paramount in tennis (Forestier and Nougier, 1998).

Hence, prolonged tennis activity induces fatigue, which manifests by having an incidence on key components of tennis performance such as force/power production, speed, accuracy, displacements, and reaction time. Therefore, in order to maximize tennis performance, the improvement of fatigue resistance, as well as the delay of its onset ought to be a key objective of tennis training (Kovacs, 2007).

1.2 Tennis training and improving fatigue resistance

Thus, given all the previous figures, the nature of tennis and the required energy pathways, training and practice should be in accordance with match play performances. Consequently, in addition to on-court technical training, an effective physical preparation is also essential. Its aims firstly ought to develop extensive endurance to cover the aerobic dimension and increase

recovery capacities in-between intense bouts of activity; and secondly, a comprehensive training session should also enhance a player's ability to perform anaerobic repeated dynamic movements and should contain rapid displacements, acceleration, deceleration, agility drills and explosive jumps, which are omnipresent in the game (Kovacs, 2007). As Kovacs (2006) further explains: "*it is important to train tennis players in the specific movement patterns that are encountered during match play. If specificity principles are used to design training programmes, it would also seem sensible to train tennis athletes using sprint activities that are no longer than the furthest distance that the athlete would run, per shot, during a point.* *A programme consisting of stop-start sprints of no more than 20 meters would be appropriate* [my underlining]". Indeed, since most displacements in tennis are executed with maximal speed intent, incorporating sprints in regular training appears appropriate. Similarly, Fernandez-Fernandez et al. (2010) also suggest that in order to trigger adaptations to the cardio respiratory system a high percentage of a player's training load should incorporate "*repetitive displacements (with stroke) of high intensity (90-95% HR_{max})*".

Improvement of the repeated-sprint ability (RSA) through repeated-sprint work is characterized by 10-20 short bouts (<10 s) of maximal sprints, interspersed with brief periods of insufficient recovery (<60 s), for a total work:rest ratio between 1:4 and 1:6 (Glaister, 2005; Spencer et al., 2005). Since tennis matchplay work:rest ratio varies between 1:3 and 1:5, such a training ratio seems in accordance with tennis specificity (Fernandez et al., 2006; Kovacs, 2007). Additionally, it has been demonstrated that repeated-sprint training has a positive effect on RSA, and its time-efficiency ratio makes it a particularly interesting and valid method of training for tennis activity (Fernandez-Fernandez et al., 2012).

Furthermore, this holds even greater meaning knowing that an elite tennis player's calendar can be incredibly busy and full with several tournaments per months, in addition to containing a certain amount of uncertainty due to ranking cut-offs in tournament entry, and/or depending on

the level reached—or not—in said tournament (Reid and Schneiker, 2008). Therefore, the number of training practices in-between tournaments on top of being hard to plan ahead, can be quite limited. It has been suggested that a cycle of three weeks interspersed with two weeks of recovery plus physical and technical preparation would be best to prepare athlete physically and mentally (Fernandez-Fernandez et al., 2009). As most players rightfully favor technical and tactical trainings over physical fitness development during the high season, it implies that strategies to augment the physical trainings benefits/time invested ratio is of the essence (Fernandez-Fernandez et al., 2015).

Hence, the period available ought to be optimized in order to maximize gains and performance, and consequently, a traditional and classical periodization might not always fit in a narrow two-week window between two tournaments. Two weeks of repeated-sprint training, however, have been suggested to be sufficient to increase $\dot{V}O_2$ kinetics, ameliorate fractional muscle O_2 extraction and improve high-intensity exercise tolerance (Bailey et al., 2009). As the objective is to maximize gains over a relatively short period in order to spend more time on technical aspects, performing such a repeated-sprint training in hypoxia could add additional effects by enhancing the insult to the body and increase the physiological adaptations and thus inherent performance.

1.3 Hypoxia, definition and generalities

Hypoxia is defined as a discrepancy between the oxygen required by the body tissues and the insufficient oxygen provided (Schumacker and Cain, 1987).

1.3.1 Terrestrial hypoxia

Under normal conditions at sea level (normobaric normoxia, NN), the barometric pressure (Pb) is 760 mmHg, and the air composition is 20.946% O_2, 0.03% CO_2, 78.084% N_2 0.934 Ar, in addition to others noble gazes, which are negligible due to microscopic volumes (i.e., less than

30 parts per million) (Brimblecombe, 1996). Contrary to popular beliefs, when climbing a mountain, the air composition does not change, the quantity of oxygen is exactly the same; it is however, the Pb that decreases. According to Dalton's law of partial pressures, in a mixture of non-reacting gazes, the total pressure exerted is equal to the sum of the partial pressures of the individual gazes (Dalton, 1802). In accordance to that law, the partial pressure of oxygen present in the air (PO_2) may be calculated with the following equation: $\boldsymbol{PO_2 = F_iO_2 \times Pb}$, where F_iO_2 is the fraction of inspired oxygen present in the air. Thus, at sea level, the $\boldsymbol{PO_2 = 20.946\% \times 760\ mmHg = 159\ mmHg.}$ Consequently, when the Pb drops with altitude, the F_iO_2 remains the same, but the PO_2 drops proportionally, which affects the quantity of oxygen delivered to the cells. Indeed, between the entry of the respiratory track and the mitochondria where the oxygen is delivered, a cascade occurs in order to decline the oxygen tension to facilitate the exchange. This oxygen cascade decreasing PO_2 is resumed in Figure 1.

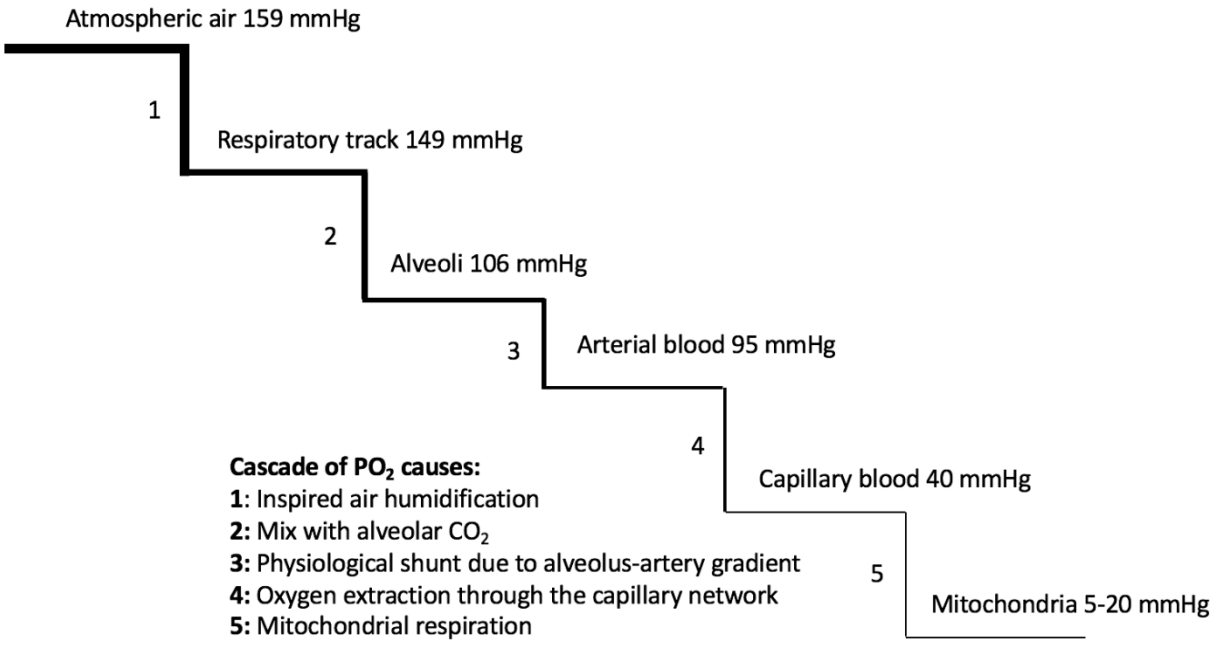

Figure 1. Oxygen cascade decrease in PO_2 from atmospheric air to the mitochondria, in normobaric normoxia, and its causes. Adapted from (Millet and Schmitt, 2011).

Figure 1 demonstrates what happens under ordinary circumstances at sea level, in NN. However, at 3000 m for instance, where the Pb is 526 mmHg, the initial PO_2 initiating the cascade would only be 110 mmHg. The higher the altitude, the lower the Pb and PO_2, which will affect the oxygen cascade and thus lower the oxygen delivered to the cells downstream. This is where the lack of oxygen caused be altitude takes place (see Figure 2).

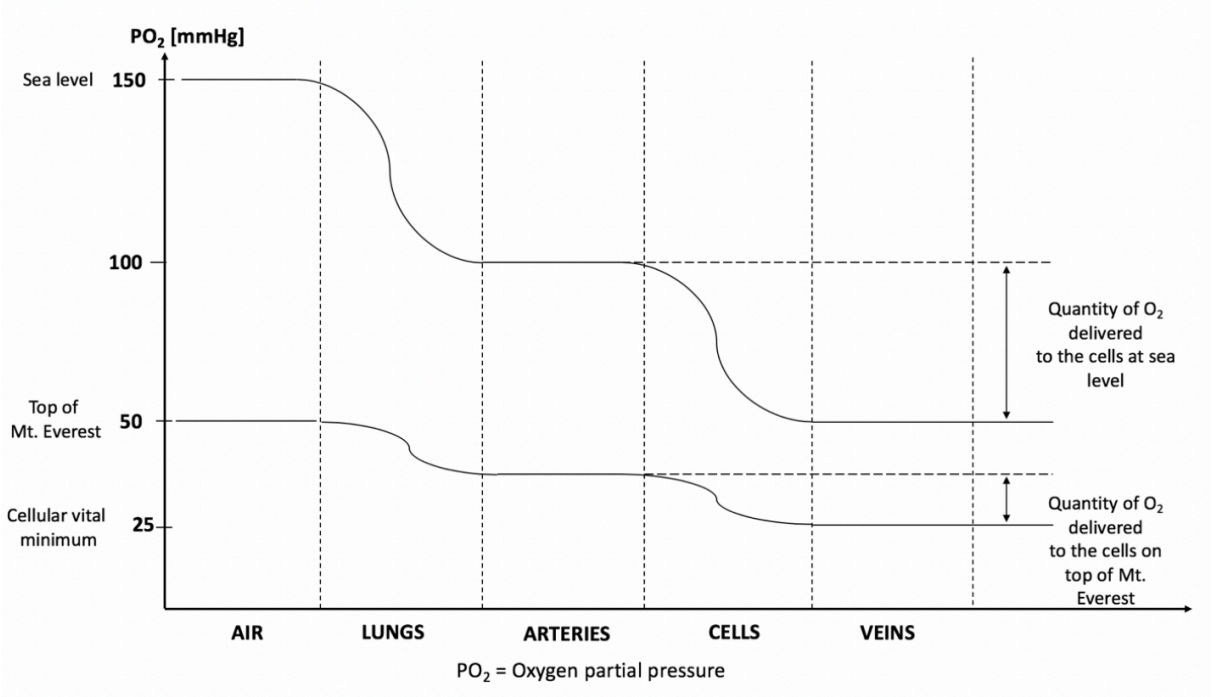

Figure 2. Effect of hypoxia on the decrease in PO_2 and its effect on the oxygen cascade. Adapted from (Millet and Schmitt, 2011).

An environment is considered hypoxic as soon as the PO_2 attains less than 150 mmHg, with an associated Pb of 716 mmHg, which corresponds to an altitude of 500 m. This is defined as hypobaric hypoxia (HH).

1.3.2 Artificial hypoxia

Hypoxia may also be artificially induced through different methods. As training in natural hypoxia entails practical difficulties such as the obligation to relocate to an altitude training facility, which depending on the region may be practically difficult and/or costly, other means

have been developed to achieve similar conditions. The following equation has been thus developed following the Equivalent Air Altitude model: $\boldsymbol{PO_2 = F_iO_2 \times (Pb - 47\ mmHg)}$, where 47 mmHg is the partial pressure of H_2O particles present in the alveoli at a corporal temperature of 37 °C (Conkin and Wessel, 2008). Since the PO_2 is dependent of Pb and F_iO_2, if decreasing Pb is not possible, then the F_iO_2 has to diminish in order to lower the PO_2. Hence, it is possible to replicate hypoxia by mechanically diminishing the fraction of oxygen present in the air, which is designated as normobaric hypoxia (NH). This may be achieved by either oxygen filtration, or by N_2 dilution. Both cases result in a diminished PO_2, although the Pb stays the same. These systems can be applied to rooms, chambers or even masks (i.e., with an Altitrainer®, for instance) which allows an athlete to train/and or sleep in hypoxia whilst actually still physically being at sea level.

1.4 Training in hypoxia

Training in hypoxia was first more deeply looked into and developed in preparation for the 1968 Mexico Olympic Games. Since then, the interest in its benefits has largely risen to become a necessity in order to be successful in certain activities, such as endurance sports. The purpose being that extensive exposure (i.e., three to four weeks) to systemic hypoxia will increase erythropoiesis, which will in turn augment the red cells volume and thus also hemoglobin ([Hb]) mass. This enhanced [Hb] mass will then increase O_2 carrying capacity and improve performance at sea level (Chapman et al., 1998; Stray-Gundersen et al., 1995).

The very original method consisted of living and training in altitude (i.e., Live High-Train High, LHTH), but its major downside is that the lack of oxygen forces the diminution of training intensities: at 2500 m, interval-training is 12-15% slower when compared to sea-level, and maximal aerobic power is decreased by ~1% every 100 m gained in altitude after 1500 m (Buskirk et al., 1967). Therefore, new methods have been defined for limiting these drawbacks,

such as Live High - Train Low (LHTL), which enables an athlete to train at low altitude and maintain high intensities, while living in altitude and still benefit from hypoxic adaptations (Levine and Stray-Gundersen, 1997; Stray-Gundersen et al., 2001). Similarly, for the past fifty years, a multitude of methods have blossomed, varying the components and their inherent possible adaptations, to become a whole panorama (resumed in Figure 3) of means to train in hypoxia.

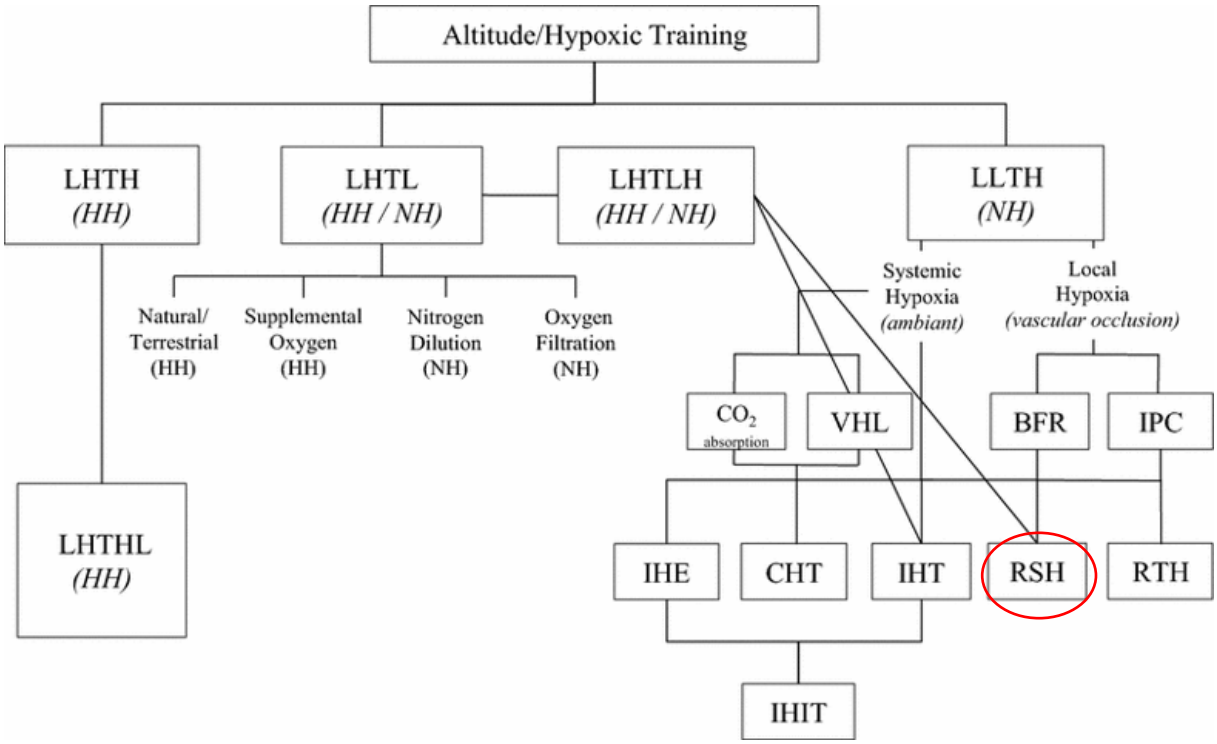

Figure 3. Updated panorama of the different hypoxic/altitude training methods used for a range of athletes. Adapted from Girard et al. (2017). BFR blood flow restriction, CHT continuous hypoxic training, CO_2 absorption rebreathing with a mask, HH hypobaric hypoxia, IHE intermittent hypoxic exposure, IHIT IHE during interval-training, IHT interval hypoxic training, IPC ischemic pre-conditioning, LHTH live high-train high, LHTL live high-train low, LLTH live low-train high, LHTHL live high-train high and low, LHTLH live high-train low and high, NH normobaric hypoxia, RSH repeated-sprint training in hypoxia, RTH resistance training in hypoxia, VHL voluntary hypoventilation at low lung volume.

1.5 Repeated-sprint Training in Hypoxia

Although tennis players may also benefit from other forms of hypoxic training, one may argue that repeated-sprint training in hypoxia would be relevant for developing anaerobic power specific to tennis. It consists in repetitions of short (<30 s) all-out sprints with incomplete recoveries performed in hypoxia (Faiss et al., 2013b). RSH's key advantage is its relatively low cost and its capacity to be implemented virtually anywhere, therefore minimizing the practical drawbacks of other forms of altitude trainings as well as shaping well into athletes' habits and training regiments. In addition, this training protocol is comparably short time-wise. This method has been demonstrated to increase performance and in particular high-power performance and its decrease to a lesser extent when repeated, as compared to the identical training performed in normoxia. Such results have been measured in different sports. Indeed, after a RSH protocol on rugby players, Galvin et al. (2013) measured a 33% increase in the Yo-Yo Intermittent Recovery test (YYIR) for the RSH group, while the group who performed the repeated-sprint training in normoxia (RSN) only increased by 14%.

A group of trained amateur cyclists gained a 38% augmentation in number of sprints attained before exhaustion in a RSA test as well as significantly greater successive changes in total hemoglobin concentration's amplitude during sprints following eight sessions of RSH over four weeks (Faiss et al., 2013b).

Also on trained amateur cyclists who performed a 6-week sprint training protocol either in NH or NN, Puype et al. (2013) measured a ~6% increase in mean power during a 10-min time trial as well as an 8% increase in $\dot{V}O_{2max}$ for both groups. However, the power associated with a blood lactate value of 4 mmol. L^{-1}, which the authors considered to be the anaerobic threshold, was inflated by 7% only in the RSH group.

A group of young elite soccer players who underwent a 5-week shuttle-run sprint training in hypoxia improved by -38% the fatigue slope of an RSA test, whilst for the group who did the same protocol in normoxia it was increased by +9% (Gatterer et al., 2014).

On a similar population, another study quantified a diminution of ~4% in both 1st sprint and cumulated sprints time in a repeated-agility test (6x 20 m, 30 s rest), whereas the group who executed the same training in normoxia only gained 2%, following a 5-week protocol (Brocherie et al., 2015a).

Following six weeks of repeated double-poling sprint training in either hypoxia or normoxia, two groups of highly trained cross-country skiers improved by 25% and 21% the maximal power output of sprints during a RSA test, respectively (statistically non-significant), however, whilst the RSN group maintained the same amount of sprints performed before exhaustion, the RSH group increased it by 57% (Faiss et al., 2015).

A research group measured a 5% gain in peak and mean power output for the RSH group, whereas only a 1.5% increase was noted for the RSN group for collegiate level female lacrosse players, subsequent to a 8-session repeated cycling-sprint protocol spread over four weeks (Kasai et al., 2015). In another protocol studying collegiate track and field sprinters, the same research group also reported a significant 2.5% increase in the mean power output in 10-s maximal sprint test as well as a ~3% increase in maximal pedaling frequency for the group who trained in hypoxia, while no significant changes were observed in the normoxia-trained group. In addition, enhanced values of muscle energy substances were evaluated, namely muscle glycogen and PCr, and they were significantly increased in both hypoxic and normoxic conditions, though not significant when compared between groups (Kasai et al., 2017).

Ensuing six sessions of RSH, a group of well-trained rugbymen benefited from a lesser degree of fatigue (i.e., minus ~2%) in an RSA test when compared to the group who trained in normoxia (Hamlin et al., 2017).

Following a 14-day LHTL protocol, two groups underwent an additional repeated-sprint training of six sessions either in NH or in NN. Although positive results have been measured in both groups, the group who cumulated the two hypoxic trainings decreased cumulated sprint time to a bigger extent, and only this group's benefits remained significant at Post-2 (Brocherie et al., 2015b).

In a recent study analyzing world-level rugby union players, it was interestingly noticed that as little as four RSH session were sufficient to improve maximal and mean power significantly during an RSA cycling test, while the RSN's values remained statistically unchanged (Beard et al., 2019a).

Regarding negative results, although no studies have measured a negative outcome following RSH, some studies have found no additional effect of hypoxia on an RSN training (Goods et al., 2015; Montero and Lundby, 2016). Therefore, given the previous results, implementing RSH in a training plan for a sport that necessitate repetitions of high-power movement appears to be well indicated. The benefits/time-involved ratio makes it particularly interesting for athletes who have unpredictable and full-time schedules such as tennis players.

1.6 Physiological benefits of RSH

Unlike longer-term forms of hypoxic training which aim at triggering an enhanced erythropoietic response and thus improve oxygen (O_2) convection capacity, RSH on the other hand uses a too short hypoxic exposure to improve red cells mass and thus O_2 convection, which necessitate several hundred hours of exposure (Levine and Stray-Gundersen, 2006). However, RSH ameliorates the diffusion of O_2 between the muscle cells and the bloodstream. Indeed, RSH's foremost physiological adaptations lies in an increased of blood perfusion level of fast-twitch (FT) muscle fibers, which enhances their O_2 extraction and utilization, and thus alters their behavior, making them more fatigue-resistant (Faiss et al., 2013). The underlying

mechanisms have been suggested to be due to an improved vasodilatation induced by the hypoxia itself (Calbet and Lundby, 2009; Casey and Joyner, 2012); and to a higher O_2 extraction capacity from FT fibers at a lower arterial O_2 pressure (McDonough et al., 2005). Additionally, it has been revealed that RSH also impacts the molecular level. Indeed, after eight training sessions of RSH in cycling, the expression of HIF-1α was upregulated (Faiss et al., 2013). HIF-1α is one of the mRNAs responsible in the oxygen-sensing pathway, notably in the mediation of changes in genes expression that regulates physiological mechanisms such as erythropoiesis and angiogenesis following acute and chronic exposure to hypoxia (Semenza, 2007; Semenza et al., 2006). The same authors also measured changes in other mRNAs expressions which they suggest result in a better pH regulation capacity, as well as in a shift from aerobic to glycolytic activity in the muscle, which partially explains the modified behavior of the FT muscle fibers and their enhanced resistance to fatigue (Faiss et al., 2013).

Galvin et al. (2013) have also demonstrated that RSH provokes an ameliorated cerebral deoxygenation during sprints, though central drive was maintained, which the authors suggest results in an improved work capacity in NN following RSH.

1.7 Benefits of RSH for tennis players

RSH in well-trained tennis players has been performed and measured in the past, and its results were significantly positive. Indeed, following a five-session RSH training protocol over twelve days, the RSH group improved by nearly 15% the time to exhaustion in the so-called Test to Exhaustion Specific to Tennis (TEST); increased by 40% the time to the OBLA; increased by 23.6% the time to the second ventilatory threshold (VT2); and the ball accuracy (BA) improved by 13.8% during the TEST (Brechbuhl, et al., 2018). The authors suggest that though not measured, improvements in BA may be due to enhanced cerebral oxygenation. These important physiological and technical improvements are meaningful and beneficial to players, as they

translate to a delayed fatigue which, in addition to a better BA, results in an enhanced performance on the court (Baiget et al., 2017; Brechbuhl et al., 2017).

Moreover, following an in-season RSH microcycle, a rookie professional tennis player improved in different aspects, such as a -4.5% decrease in single sprint time, a decrease of -3.1% in RSA total time, and -16.7% in sprint decrement, a YYIR total distance covered gain of 21.4%, in addition to an improvement from 4 to 12 ATP points from pre to post RSH intervention (Brechbuhl et al., 2018c).

Yet, even though the results presented by Brechbuhl et al. (2018) are greatly significant and promising, the question remains whether such results could be potentiated even further. Indeed, their RSH protocol consisted of shuttle-run sprints, without ball stroking. However, one of RSH's study that recorded the most significant improvements in the delay of fatigue during maximal power movements (i.e., + 57% from PRE to POST tests) concerned cross-country skiing double-poling sprint, and thus upper body movements (Faiss et al., 2015). Therefore, it could be argued that as tennis players also majorly use upper-body movements, an RSH protocol that would include ball hits and thus upper-body movements as well, could expand the extent of the benefits. The specificity of such a protocol itself would induce possible extra gains than only shuttle-sprints, as on-court training protocol has been evidenced to induce better technical improvements such as higher groundstroke accuracy and longer time to fatigue in comparison to an off-court training with similar load (Srihirun et al., 2014).

Moreover, as explained earlier, one of RSH's main adaptation is improving fatigue resistance of FT fibers (Faiss et al., 2013b), however, in tennis players' lower limbs muscles (i.e., *vastus lateralis*) 'only' 1/3 of the fiber-type distribution consists of type 2 fibers, the rest being type 1, whereas in upper-body muscles (i.e., *triceps brachii*), 2/3 of the fibers are type 2 and thus FT fibers (Sanchis-Moysi et al., 2010). Therefore, a protocol that would require upper-body

movements in addition to shuttle-run sprints could theoretically be beneficial from RSH to a greater extent than mere lower-body sprints.

1.8 Purpose & Hypotheses

To the best of the authors' knowledge, no previous study has measured the effect of RSH with ball strokes on well-trained tennis players. The purpose of the present study was thus to analyze the impact of an RSH protocol containing actual forehand and backhand strokes on tennis-specific performance and RSA. Moreover, the secondary aim was to measure variations in leg muscles and cerebral blood oxygenation to see whether they correlate with possible technical and/or physiological benefits.

Given the actual state of the literature regarding the topic, the present work tested the following hypotheses:

a) Repeated-sprint training improves RSA-related parameters and high-intensity exercise fatigue resistance.
b) The extent of the improvements induced by the repeated-sprint training in hypoxia is greater than similar training in normoxia.
c) RSH containing ball strokes improves technical aspects (i.e., ball velocity and ball accuracy) measured in a test to exhaustion specific to tennis.
d) Physiological and technical gains in the RSA test and in the test to exhaustion specific to tennis are correlated to enhanced changes in muscle and cerebral oxygenation.

2 Methods

2.1 Subjects

Thirty healthy, well-trained regional level tennis players volunteered to take part in this study (thirty men and six women; 28.8 ± 5.9 years old, 72.3 ± 9.9 kg, 179.1 ± 6.7 cm, FTF ranking: 1.54 ± 2.84, $\dot{V}O_{2max}$ 57.2 ± 6.2 ml/min/kg). Subjects were recruited through ads (Appendix 1) sent to French Tennis Federation (FTF) licensed tennis clubs in the Île-de-France region. Inclusion requirements for participants were as follow:

i. Be affiliated with the French Social Security System;
ii. Have their age comprised between 18 and 40;
iii. Be licensed at the FTF;
iv. Have a ranking bellow 15 according to the FTF ranking system;
v. Be well trained. Were considered well-trained, athletes who trained at least three times per week, excluding matches and tournament, for a period of at least six months prior to the start of the study;
vi. No hypobaric nor normobaric hypoxic exposure prior to the start of the study.
vii. Be declared apt to perform hypoxic training following an examination by the FTF medical staff. The medical examination consisted in anthropometrics measures, palpations, an auscultation, a medical questionnaire, a resting electrocardiogram associated with heart beat frequency and arterial pressure, and finally a cardiac echography. If two cardiovascular risks or more were discovered for a potential subject, an exercise stress test would be conducted before considering them for participation.

Exclusion criterion included:

viii. Vulnerable individuals as defined by articles L. 1121-5 à L.1121-8 et L.1122-1-2 of the French Public Health Code (i.e., minors, expecting women, individuals without their discernment, etc.);

ix. Medical contraindication to physical activity;

x. Presence of any diagnosed disease;

xi. Ongoing medical treatment that could potentially alter the protocol effect and its analysis;

xii. Taking part in another scientific research study;

xiii. Lack of consent.

Participants were all healthy and gave written consent (Appendix 2) after having been exhaustively informed about the whole experimental protocol as well as its inherent possible risks, during an information session wherein they could also have their potential questions answered. The research protocol was approved by the local ethic committee ("Comité de Protection des Personnes", ANSM authorization N°: 2017-A02865-48) on January 31st, 2018. After meeting the requirements and successfully passing the recruitment and the medical examination, subjects were randomly assigned to three groups: two experimental groups, and one control group (CON). Both experimental groups underwent an identical repeated-sprint training with ball strokes (further detailed below), one of which was performed in normobaric hypoxia (RSH), at a simulated altitude of 3000 m, whilst the other was in normoxia (RSN). In order to prevent any placebo/nocebo effect, participants were not aware which group they belonged to. The CON group did not take part in the training protocol, they only completed the testing visits.

Both RSH (N=11) and RSN (N=11) groups were composed of nine men and two women, while the CON group (N=8) was composed of six men and two women. The three groups had similar characteristics, which are resumed in Table 1.

Table 1. Groups characteristics.

Group	Age [years]	Height [cm]	Weight [kg]	FTF Ranking	$\dot{V}O_2$ max [ml/kg/min]
RSH (N=11)	25.7 ± 6.8	179.3 ± 4.9	72.6 ± 8.9	1.91 ± 2.55	57.1 ± 7.9
RSN (N=11)	31.4 ± 4.5	174.9 ± 6.7	69.5 ± 8.7	1.55 ± 3.25	57.7 ± 5.6
CON (N=8)	29.6 ± 5.1	184.8 ± 5.2	76.0 ± 12.6	1.0 ± 2.93	56.7 ± 4.7

Aside of the experimental protocol, all three groups maintained their usual respective tennis training for the duration of the study. This training load (TL) was collected in order to detect any significant load differences which could alter the experimental protocol effect. The TL was calculated by multiplying each subject's training sessions duration in minutes by the rate of perceived exertion (1-10) for each training session (Foster, 1998).

2.2 Experimental design

The study in its entirety (i.e., the recruitment, the tests and the training protocol) took place at the French Tennis Federation National Training Center (4 Place de la Porte Molitor, 75016 Paris, France), at a temperature of ~21°C, and a relative humidity of ~55%. Prior to the training protocol, the three groups performed pre-tests (PRE). Afterwards, the RSH and RSN groups underwent a repeated-sprint training which consisted of five 1 h-sessions spread over two weeks. Another two set of tests were executed after the training protocol, the first at twenty days after the pre-tests (POST1) (i.e., right after the protocol), and the second at forty-one days after the pre-tests (POST2) (twenty-one days after the intervention). Owing to the time the hypoxic room necessitates to reach the simulated altitude of 3000 m (nearly 2 h), both groups trained on separate days: the RSH group trained days 1-3-5-9-11 of the protocol, while the RSN group trained days 2-4-8-10-12 (see Figure 4).

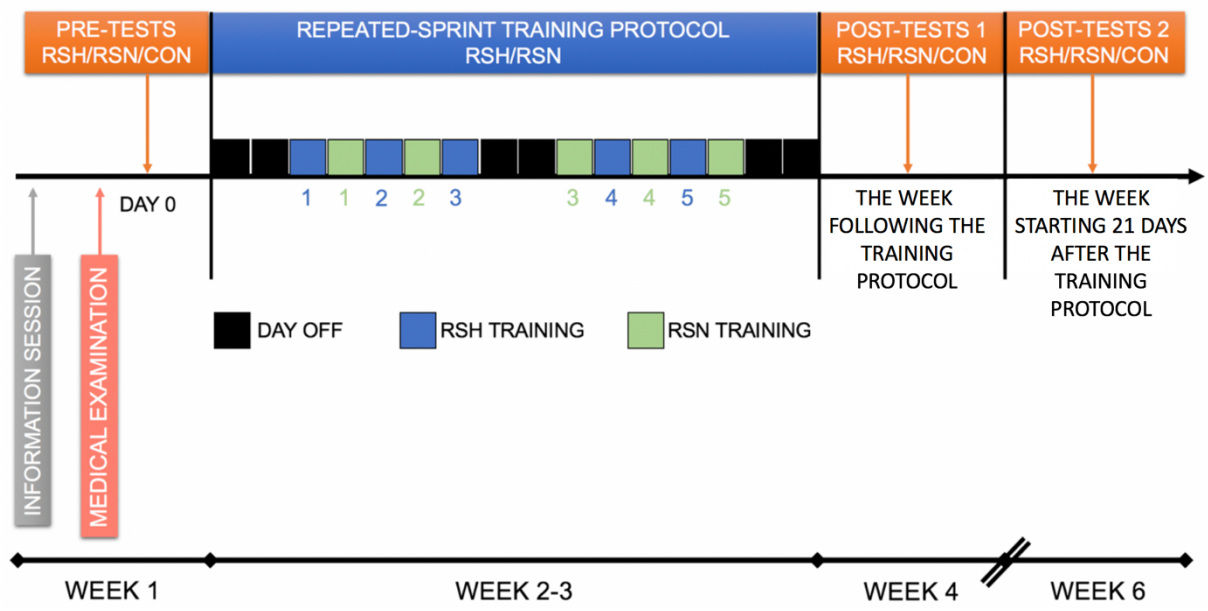

Figure 4. Study design.

2.2.1 Training protocol

The training protocol was performed in a normobaric hypoxic room (size 15.04 m×8.54 m; b-Cat®, Tiel, Netherlands). Altitude was simulated through O_2 extraction by means of a filter and a compressor system. The hypoxic device either simulated an altitude of 3000 m above sea level, with a F_iO_2 of 14.5% for the RSH group; or simulated an altitude of 200 m, with a F_iO_2 of 20.9% for the RSN group. Regardless of the altitude simulated, in both conditions the device produced the same sound to blind the participants.

Trainings lasted ~50 min. After a standardized 15 min warm-up, which consisted of 10 min on an ergocycle, followed by a 5 min sprint-specific warm-up (i.e., butt kicks, high knees, increasing accelerations and lower limbs stretches), subjects performed four sets of five repetitions of ~8 s sprints interspersed with 22 s of passive recovery. A 5 min passive recovery separated each set (see Figure 5).

The sprints were constituted of two back and forth sprints along an imprinted line the exact length of the baseline of a singles tennis court, hence 8.23 m, with ball hitting on each end of

the line. Subjects hit two forehands on the right side and two backhands on the left side (for right-handed players, the opposite for the left-handed). The balls to be hit were distributed manually by a professional tennis coach, who was the same for all participants. A given ball was thrown as soon as the previous shot had been hit, or as soon as the subject started running from the left side of the line for the first one. In order to maximize training effect, participants were asked to sprint and hit the tennis balls as fast and as hard as they could. They were also strongly verbally encouraged by the ball distributor. They completed the training with a 10 min passive recovery (see Figure 5).

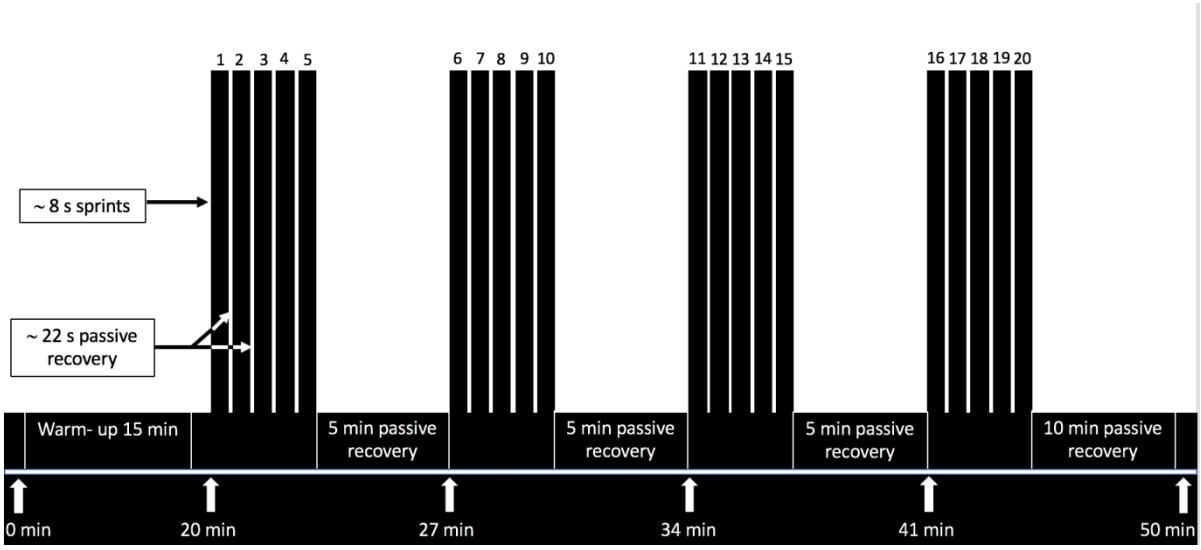

Figure 5. One training session protocol.

2.3 Testing

2.3.1 Test to Exhaustion Specific to Tennis

In order to evaluate the technical and physiological responses to the effect of the training protocol, the 'Test to Exhaustion Specific to Tennis' (TEST) (Brechbuhl et al., 2016) was adopted. The interest of using TEST was twofold. Firstly, unlike other field tests, TEST is tennis-specific and hence contains actual ball strokes, play-like lateral displacements, and

intermittent exercise, which can all be observed in a real tennis game. Secondly, as it was employed with a portable gas exchange analyzer, it allowed to determine the cardio-respiratory responses to the incremental exercice. Finally, it also measured the ball accuracy (BA) and the ball velocity (BV) of every ball stroke during the test. Thus, the TEST enabled the assessment of any modification in aerobic capacities, in addition to any technical alteration (Brechbuhl et al., 2017).

The TEST consisted in a mechanical ball machine (Hightof®, Echouboulains, France) throwing balls (Roland Garros®, Babolat, Lyon, France) alternating the right and left corner, at a constant BV of 86 km/h^{-1}, and at a constant ball frequency (BF) during a given stage but increasing in BF after each stage. The stages lasted 1 min and were interspersed with 30 s passive recoveries. The balls thrown were programmed to land on the court 3 m in front of the baseline, and 2 m sideways of the lateral lines. Players were obligated to hit shots in a designated pattern: alternating forehand and backhand strokes (i.e., forehand strokes on the right side, backhand strokes on the left side for right-handed players, the opposite for the left-handed players), and play cross-court topspin shots, aiming to land the ball in a targeted 3.115 m x 4.55 m rectangle 1 m away from the service line and 1 m away from the line separating the court lengthways, delimited on the exterior by the baseline and the singles sideline. The TEST design and settings are displayed in Figure 6. Sliced hits were proscribed due to their influence on the ball speed and landing position.

The TEST commenced with a 2 min-familiarization/warm-up stage wherein balls where distributed in the central area of the court to limit lateral displacements and fatigue, at a BF of 16 balls/min^{-1}. A 30 s standing passive recovery followed, which was repeatedly recurrent in-between every stage.

The main TEST protocol began with the first stage at a BF of 10 balls/min^{-1} and then incremented by 2 balls/min^{-1} every stage until stage seven, equivalent to 22 balls/min^{-1}, and thereafter increased only by 1 ball/min^{-1} between stages, until exhaustion (see Figure 6).

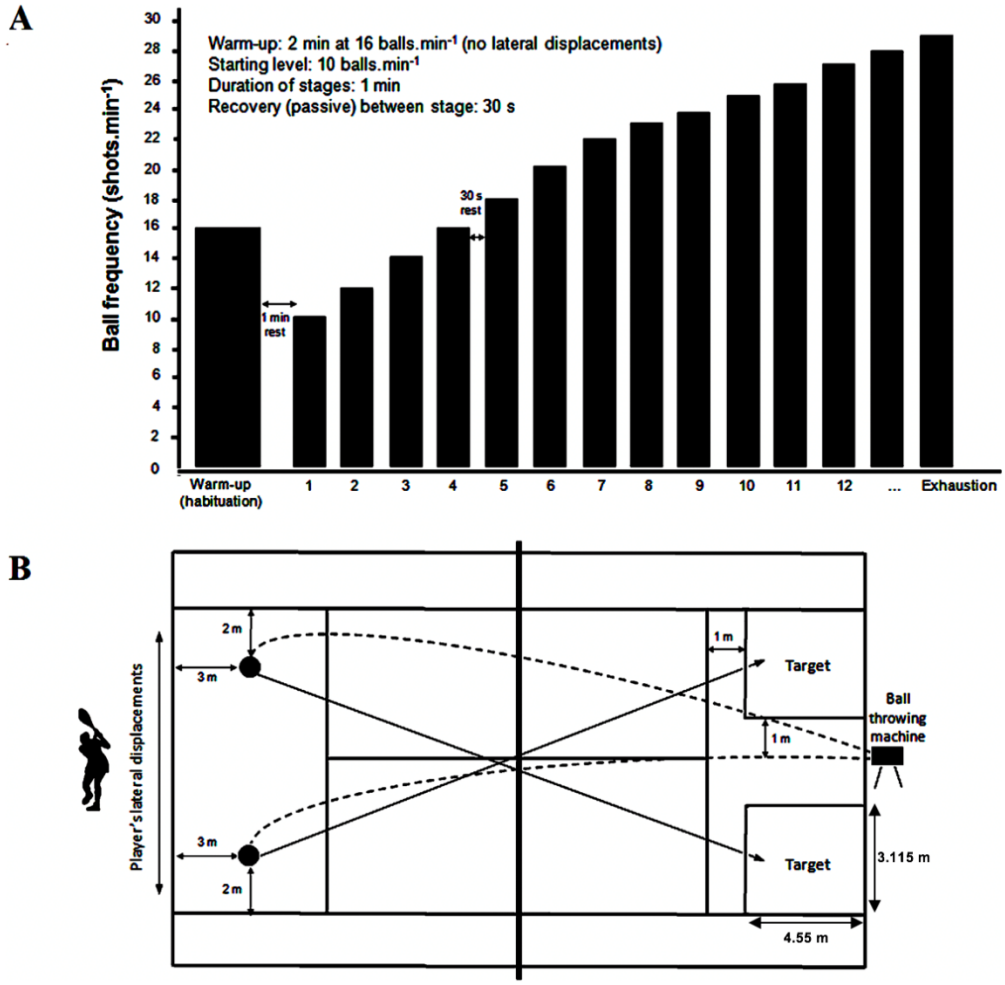

Figure 6. TEST design (A), and the schematic outline of its setting (B), adapted from (Brechbuhl et al., 2016).

The TEST took place on a technological court (PlaySight® system (PlaySight Interactive, Ltd., Kokhav Ya'ir, Israel), which measured BV and BA (i.e., landing positions). From this TEST, a tennis performance index (TP) was assessed, which was the product of BA and BV (Brechbuhl et al., 2016). Subjects were thus instructed to hit each ball with the best velocity-accuracy ratio, implying a full commitment on every stroke. Players were verbally encouraged during the TEST and also received an oral feedback by the investigators after each stage, informing them

of inadequate ball velocities (BV lower than 80 km/h^{-1}) and/or inadequate precision (30% or more balls landing outside the targeted area).

The TEST was terminated either by the subject's exhaustion and voluntarily cessation, or by the investigators if said subject failed to hit the ball appropriately twice in a row; or if they received two warnings regarding BV and BA insufficiency (Brechbuhl et al., 2016). To guarantee an optimal and standardized ball rebound, balls were changed every six tests.

During the TEST, in order to evaluate cardio-respiratory responses, subjects wore (see Figure 7) an automated portable gas analyzer (Metamax II CPX system, Cortex® , Leipzig, Germany) and HR monitor (Suunto Ambit2® , Vantaa, Finland) measuring breath-by-breath ventilation, and thus also assessing oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory exchange ratio ($\dot{V}CO_2/\dot{V}O_2^{-1}$), and minute ventilation ($\dot{V}E$). The gas and volume calibration of the device were conducted before each test by a trained specialist, following the manufacturer's instructions. Capillary blood lactate concentrations ([La]) measurements were taken before the start of the TEST, after every second stage, and directly after exhaustion using a Lactate Pro device (LT-1730; Arkray®, Kyoto, Japan).

Figure 7. Portable gas analyzer equipment.

2.3.2 Repeated-sprint Ability

Repeated-sprint ability was assessed with an RSA test, during which changes in peripheral and cerebral tissue oxygenation were measured with two Near Infra-Red Spectroscopy (NIRS) devices (see details below).

Figure 8. Setting of the RSA test.

Sprints were timed with two photoelectric cells connected to an electronic timer (Witty, Microgate, Bolzano, Italy) and were rounded to the nearest hundredth of a second.

Before the RSA test started, subjects performed a 15 min standardized warm-up which was exactly the same as the warm-up performed before the training protocol. Following the warm-up, the NIRS devices and a HR monitor (Suunto Ambit2® , Vantaa, Finland) were positioned onto the subjects (see below for details), and resting capillary blood [La] were measured using a Lactate Pro device (LT-1730; Arkray®, Kyoto, Japan).

Prior to the beginning of the RSA test, subjects completed three maximal reference sprints interspersed with 2 min of passive recovery. These reference sprints served as benchmark to detect any form of pacing strategy during the RSA test, which could distort the results. The timing of the first sprint of the RSA test had to attain at least 95% of the best timing of the three reference sprints. If that criterion was not met, subjects were asked to sit down for a duration of 2 min before they could attempt a second try. This only happened to one subject, albeit who

managed to reach the 95% on his second try. A 3-min passive seated recovery followed the reference sprints as to not generate any fatigue before the actual test.

The RSA test consisted of eight back and forth sprints the length of an imprinted baseline of a singles tennis court (8.23 m), resulting in a total distance of 16.46 m per sprint, interspersed with 20 s of passive standing recovery. In order to validate a sprint, subjects had to place at least one foot behind the opposite line (see Figure 8). They were given a warning if they failed to do so. They could use neither the walls nor their hands on the ground as assistance when they rotated 180°. They were strongly verbally encouraged by the investigators.

Regarding starting position, subjects were instructed to stand with their left leg behind a white tape located 20 cm behind the starting line to make sure that the photoelectric cells were not triggered by a parasitic movement before the beginning of the actual sprints. The start of the first sprint was at subjects' will, but the remaining seven were given by an investigator who gave the 10 s mark, and then counted down out loud the last 5 s of the recovery. HR was measured before the test (HR pre-RSA) and straight after the end of the eighth sprint (HR post-RSA). A 3-min seated passive recovery followed the test, after which capillary blood [La] and HR (HR post-3 min RSA) were assessed again, as well as the rate of perceived exertion (RPE). Three scores were determined from the RSA test: *I)* best sprint time (RSA_{BEST}), *II)* cumulated sprints time (RSA_{TT}) and *III)* percentage of sprint decrement (S_{dec}) which was calculated with the following equation:

$$\mathbf{S_{dec}\ (\%) = [(RSA_{TT} / (RSA_{best} \times number\ of\ sprints)) - 1] \times 100}$$ as developed by Glaister et al. (2008).

2.3.3 Near Infra-Red Spectroscopy

During the RSA test, changes in tissue oxygenation were evaluated via NIRS. NIRS technique is founded on the capacity of near-infrared (NIR) light (i.e., between 700 and 1000 nm) to penetrate biological tissues including skin, bone and muscle. As Boushel and Piantadosi (2000)

futher explain: "*The amount of light recovered after illuminating the tissue depends on the degree of scattering in the tissue and the amount of absorption by the chromophores in the tissue. Only three molecules are known to affect NIR light absorption during changes in tissue oxygen tension, haemoglobin, myoglobin and cytochrome c oxidase. Differences in the oxygen-dependent absorption spectra of the iron and/or copper centres of these molecules make it possible to measure changes in the relative amounts of oxidized copper and oxygenated haeme species present in muscle [. . .] In most cases, however, NIRS does not allow for precise quantitative measurement of concentration differences but instead provides trends in the responses of the oxylabile chromophores to changes in oxygen availability*". However, it is possible to estimate the approximate tissue concentrations of oxy- and deoxy-hemoglobin by measuring the optical pathlength of NIR photons passing through the different tissues (Boushel and Piantadosi, 2000; van der Zee et al., 1992; Wyatt et al., 1990).

For the present study two NIRS devices were used: the PortaMon and PortaLite (Artinis, Zetten, The Netherlands). Both devices were first tightly wrapped in a transparent plastic in order to prevent humidity from impregnating the apparatus and affecting the signal quality. Secondly, double-sided tape was applied in order to obtain adherence between the devices and the subject's skin (see Figure 9).

The PortaMon, which measured muscle oxygenation, was placed on the lower third of the *m. vastus lateralis,* which is vascularized by the lateral circumflex femoral artery. The area had been previously prepared, shaven and disinfected. Elastic tape was employed to secure the device to the subject's leg. A thick brown bandage was then firmly affixed around to cover the receiver from the daylight as well as to ensure the equipment would not budge during the sprints (see Figure 9). For inter-test reproducibility purposes, the exact position of the probe was marked down with a permanent black pen and subjects were instructed to maintain that mark

in-between tests; however, most subjects failed to do so, which is a possible limit to the measurements.

After cleaning and disinfecting the subject's forehead in order to remove any moisture, the PortaLite was positioned on the left side of the forehead, 3 cm from the medial line, 2 cm above the supraorbital margin, recording the oxygenation activity of the left prefrontal cortex, which is vascularized by the anterior cerebral artery. Similar to the PortaMon process, the PortaLite was then taped to the subject's forehead to diminish any displacement, and a black headband, firmly attached, completed the setup to provide immovability and opacity. The wire passed behind the subject's hear, was taped to their t-shirt, and the device's housing was put in their pocket, which was taped shut to prevent the device from dropping during the sprints (see Figure 9).

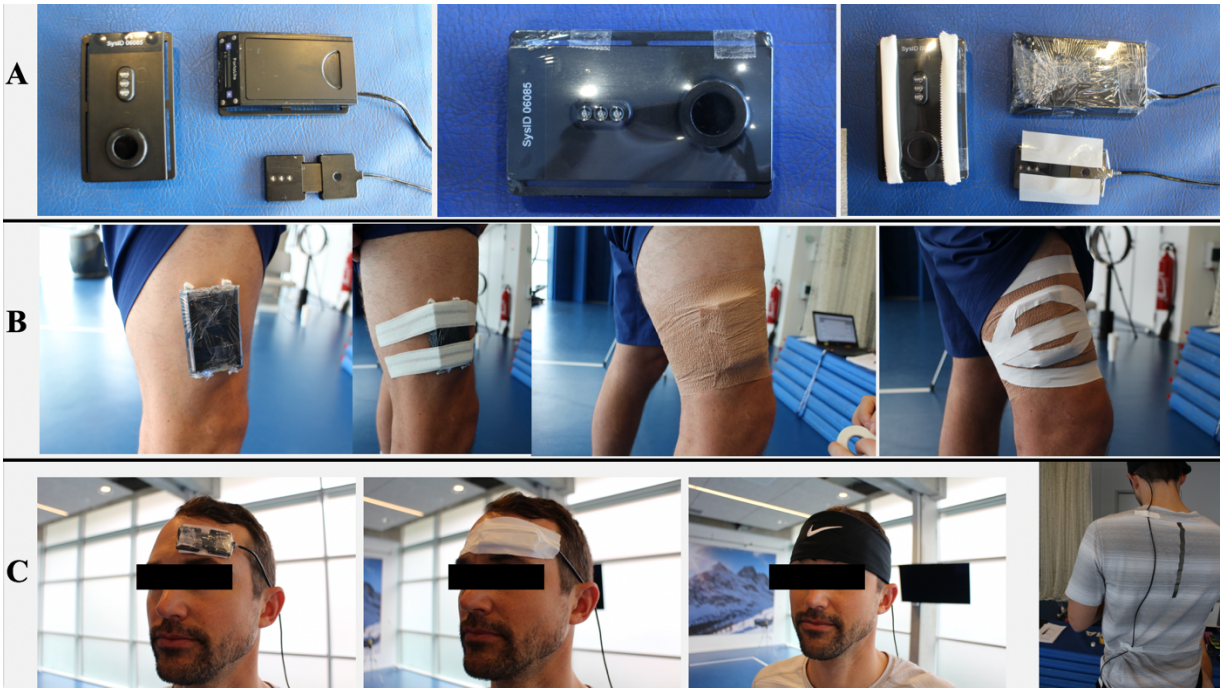

Figure 9. NIRS set up. Both devices wrapped in plastic, then with double sided tape (A); PortaMon for muscle oxygenation analysis placed on the vastus lateralis (B); and the PortaLite for oxygenation, placed near the prefrontal cortex (C).

Both devices comprised three light source transmitters, which used two wavelengths of 760 and 850 nm, positioned at 30, 35 and 40 mm from the receiver respectively.

The differential pathlength factor was standardly set to 4.0 for the PortaMon, and 6.0 for the PortaLite, as per similar other research (Duncan et al., 1995; Faiss et al., 2013b; van der Zee et al., 1992; Willis et al., 2017).

The two apparatus recorded signals at their maximum frequency, hence 10 and 50 Hz for the PortaMon and PortaLite, respectively, but were down-sampled to 10 Hz for further analysis (Oxysoft 3.0.53, Artinis, The Netherlands). The data was then exported to Microsoft Excel (Microsoft, Redmond, WA, United States) for analysis. In order to smoothen the signal and minimize noise artifacts, a 4^{th}-order low-pass zero phase Butterworth filter with a 0.2 Hz cutoff frequency was applied (Yu et al., 1999). A Microsoft Excel macro was applied to automatically detect the maximum and the minimum for each sprint. The starting point was manually entered, using the filtered deoxy-hemoglobin curve as visual reference (See Figure 10). This analysis allowed to identify the different phases, i.e., the sprints and recoveries of the RSA test. The difference, or delta, of concentrations between the minimum and the maximum constituted the main data that were analyzed in this study. Five oxygenation parameters were gathered from equivalent analysis, namely the delta (Δ) concentrations of oxy-hemoglobin (Δ[O_2Hb]), deoxy-hemoglobin (Δ[HHb]), total hemoglobin (Δ[tHb]), tissue saturation index (ΔTSI, %) and absolute TSI maximum (abs TSI_{max}).

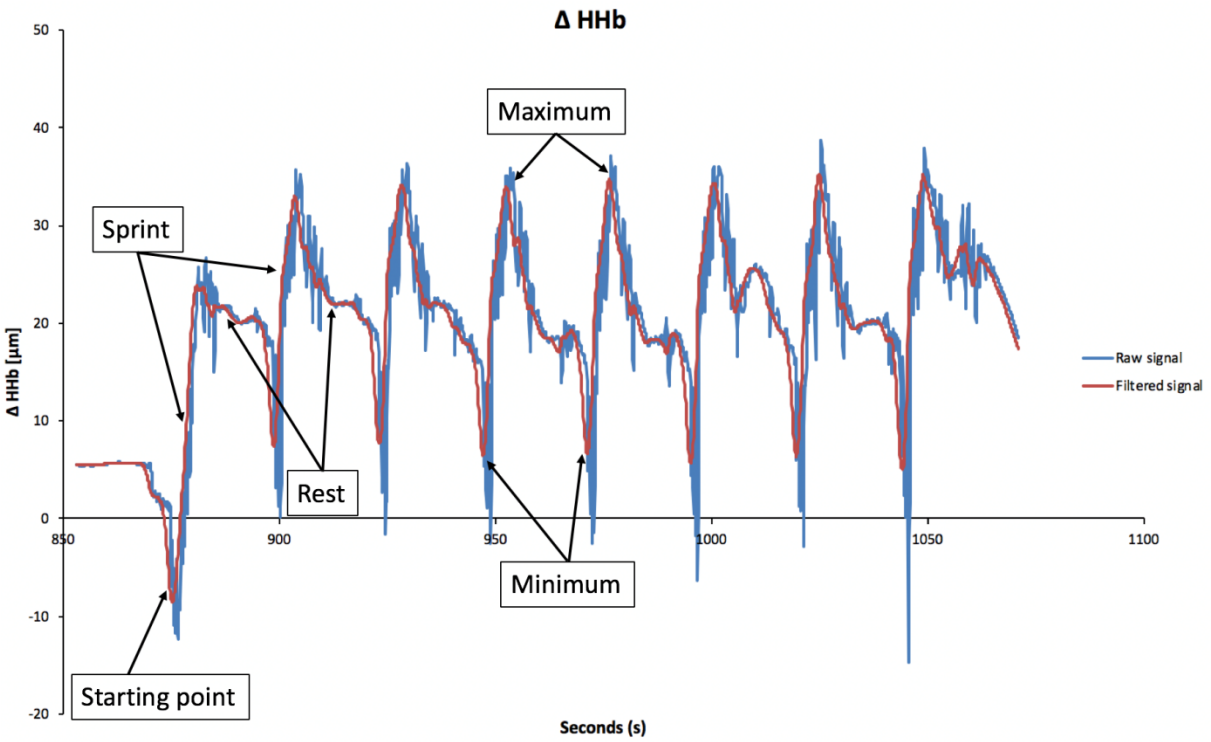

Figure 10. Visual representation of a typical leg deoxy-hemoglobin curve during the RSA test. In blue the raw signal measured by the PortaMon, in red the filtered signal used for analysis.

2.4 Statistical analysis

Unless specified otherwise, all data are presented as mean ± standard deviation (SD). Changes in tissues oxygenation during the RSA test were evaluated with a linear mixed model three-way repeated-measures analysis of variance (ANOVA) (time x group x sprint number). Fixed effects were: time (PRE, POST1, POST2), group (CON, RSN, RSH) and the sprint number (1, 2, 3, 4, 5, 6, 7, 8), whereas subjects were considered as the random effect. In case of significant interactions, Tukey's *post-hoc* multiple comparisons of means method was applied to assess group differences. The confidence level was set at 95%. Sprints times, TEST parameters, HR and blood [La] concentrations were analyzed with a linear mixed model two-way repeated measures ANOVA (time x group), with all pairwise comparison (Tukey's *post hoc* multiple comparisons of means method). The Shapiro-Wilk test was applied to examine normality.

Sphericity was verified with Mauchly's Test of Sphericity. When significance was found, if the Greenhouse-Geisser epsilon (ϵ) estimate correction was equal or below 0.75, it was used, if it was higher, Huynh-Feldt ϵ estimate correction was employed. No deviation from homoscedasticity was present after inspection using Levene's Test. The statistics for the changes in blood oxygenation were performed using the software R (R Core team 2017, Foundation for Statistical Computing, Vienna, Austria), whereas the TEST results and remaining RSA parameters' statistical significance were evaluated with the software Jamovi (the Jamovi project, Version 1.0, 2019). The effect size was measured with eta squared (η^2), <0.01 was considered as small, 0.06 was considered medium, and >0.14 was considered as a large effect (Cohen, 2013). The null hypothesis was rejected at $p<0.05$.

3 Results

The total TL aside of the training protocol was 10270 ± 6473 for the RSH group, 8366 ± 5447 for the RSN group and 11678 ± 7445 for the CON group, which did not significantly differ from one another (p>0.05).

3.1 Performance

3.1.1 Test to Exhaustion Specific to Tennis

The physiological and technical responses to the TEST are summarized in Table 2.

Table 2. Physiological and technical results from the Test to Exhaustion Specific to Tennis (TEST).

Group	Time	$\dot{V}O_{2max}$ [ml/min^{-1}/kg^{-1}]	TIME TO OBLA [s]	BV 100% [km/h]	BA 100% [%]	TP 100% [a.u.]
RSN	PRE	57.8 ± 5.7	410 ± 87.1	91.1 ± 7.55	41.5 ± 9.91	37.9 ± 10.3
	POST1	57.3 ± 5.9	425 ± 112	92.9 ± 8.75	39.5 ± 15.2	37.3 ± 16.0
	POST2	58.8 ± 6.5	452 ± 78.3	99.1 ± 8.33*	41.9 ± 8.52	41.8 ± 10.3
RSH	PRE	57.2 ± 7.8	373 ± 98.5	99.6 ± 10.7	37.1 ± 13.0	37.2 ± 14.8
	POST1	58.7 ± 7.0	464 ± 118**	101 ± 8.75	49.1 ± 12.4*	49.4 ± 13.1*
	POST2	58.5 ± 7.2	447 ± 103*	99.8 ± 7.53	48.1 ± 9.86	47.9 ± 9.29
CON	PRE	56.2 ± 5.6	405 ± 76.9	95.6 ± 8.85	34.5 ± 9.98	32.9 ± 9.41
	POST1	56.3 ± 6.1	435 ± 76.9	96.0 ± 7.83	39.4 ± 8.67	38.0 ± 8.93
	POST2	57.0 ± 6.8	420 ± 45.4	102 ± 7.89	34.1 ± 12.7	34.9 ± 14.4

Values for maximal oxygen uptake ($\dot{V}O_{2max}$) [ml/min/kg], time to the onset of blood lactate accumulation (OBLA) [s], ball velocity during the last stage (BV 100%) [km/h], ball accuracy during the last stage (BA 100%) [%] and

*tennis performance index during the last stage (TP) [%]. Mean ± SD. ***(p<0.001), **(p<0.01), *(p<0.05) significant main effect in time different from PRE.*

One of the main adaptations induced by the training protocol was the time to exhaustion (shown in Figure 11), which had a significant effect of time (p=0.008, η^2=0.03), and a significant interaction of time x group (p=0.002, η^2=0.056). The RSN group had a slight non-significant improvement of 1.2% (p>1.00, NS) from PRE to POST1, and 5.8% (p=0.77, NS) between PRE and POST2. The RSH showed the most substantial time gain, which was increased by 18.2% between PRE and POST1 (p<0.001) and remained significantly elevated by 17.3% at POST2 (p<0.001).

Although there was no significant change in maximal lactate for any of the groups (p=0.494, non-significant (NS)), there was however, a significant effect of time to attain the onset of blood lactate accumulation (OBLA) (p<0.001, η^2=0.051). The RSH group increased it by 24.4% from PRE to POST1 (p=0.003), and by 19.8% from PRE to POST2 (p=0.027), whereas the other two groups did not significantly change their time to OBLA.

The RSN group was the only one to improve BV at the last stage (BV 100%) between PRE and POST2 by 8.8% (p=0.048), though not between PRE and POST1.

Regarding TP at the last stage (TP 100%), there was a significant effect of time (p=0.015, η^2=0.042). Only the RSH group improved it by 32.8% (p=0.025) from PRE to POST1, and by 28.8% from PRE to POST2, though not statistically significant (p=0.078, NS). Since TP is the product of BV and BA, this improvement originates from an increase in BA during the last stage (BA 100%) by 32.3% from PRE to POST1 (p<0.025), and by 29.6% from PRE to POST2 (p=0.055, NS).

The CON group did not demonstrate any significant change in any of the previously mentioned parameters throughout the tests.

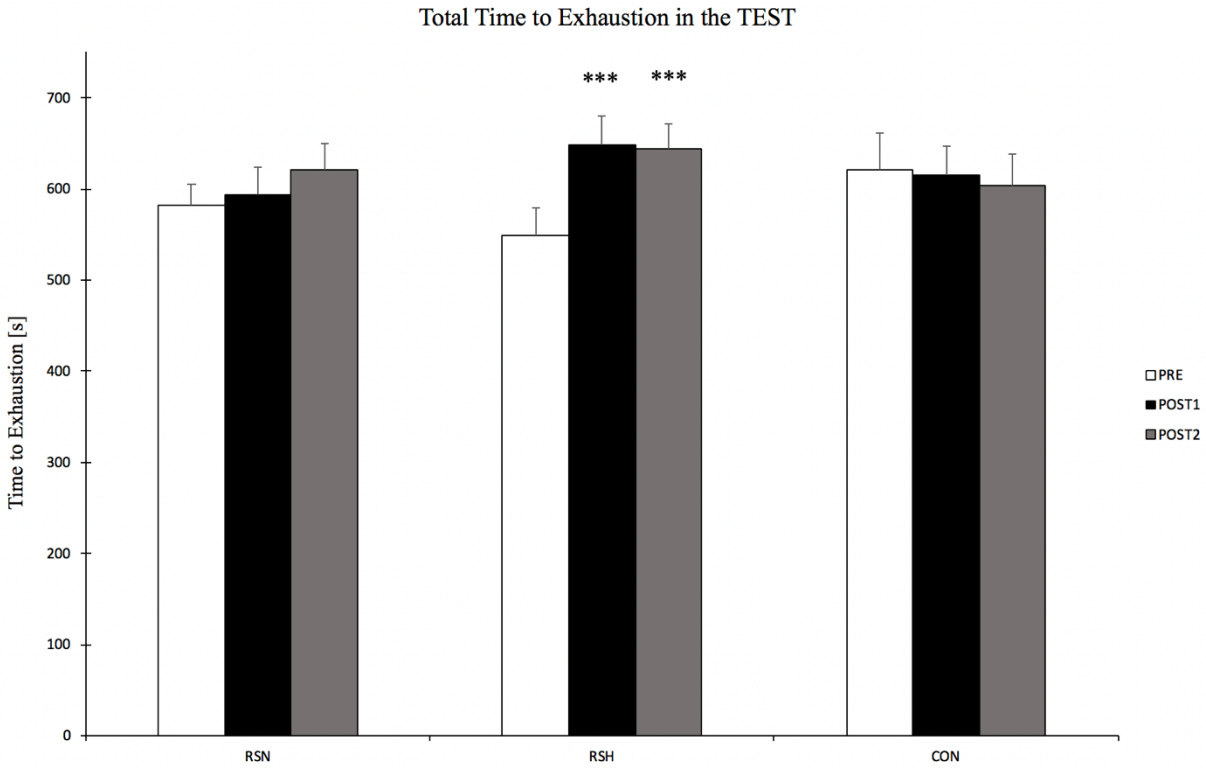

*Figure 11. Boxplot representing the total time to exhaustion (TTE) during the test to exhaustion specific to tennis (TEST). Mean ± SD. ***($p<0.001$) main effect in time different from PRE.*

None of the groups significantly modified their HR_{max}, their maximal [La], their $\dot{V}O_{2max}$, or any other ventilation parameters following the experimental protocol.

3.1.2 Repeated-sprint Ability test

The results from the RSA test are resumed in Table 3. The results suggest a positive effect of the sprinting protocol, as both training groups improved some of their RSA parameters.

For the RSH group, RSA_{BEST} decreased from PRE to POST1 by 2.33% (p=0.01), and from PRE to POST2 by 3.4% (p<0.001). The RSN group decreased it by 0.8% both from PRE to POST1 (p=0.76, NS), and from PRE to POST2 (p=0.62, NS). The CON group did not change its performance (p>0.05, NS).

Table 3. Results of the repeated-sprint ability (RSA) test.

Group	Time	RSA_{BEST} [s]	RSA_{TT} [s]	S_{dec} [%]
	PRE	3.96 ± 0.26	32.5 ± 1.96	2.77 ± 1.11
RSN	**POST1**	3.93 ± 0.26	32.1 ± 1.95*	2.41 ± 1.23
	POST2	3.93 ± 0.25	32.1 ± 1.97*	2.41 ± 1.16
	PRE	3.86 ± 0.12	31.6 ± 1.15	2.48 ± 1.19
RSH	**POST1**	3.77 ± 0.17**	31 ± 1.2**	2.95 ± 0.84
	POST2	3.73 ± 0.14***	30.9 ± 1.12**	3.35 ± 1.77
	PRE	3.92 ± 0.29	32.3 ± 2.15	2.75 ± 0.94
CON	**POST1**	3.91 ± 0.2	32.2 ± 1.90	2.95 ± 1.38
	POST2	3.92 ± 0.26	32.2 ± 1.98	2.9 ± 1.08

*Best sprint time (RSA_{BEST}); cumulated time of all sprints (RSA_{TT}) and percentage of sprint decrement (Sdec)]. Mean ± SD. ***(p<0.001), ** (p<0.01), *(p<0.05 significant main effect in time different from PRE.*

Regarding RSA_{TT}, the RSN group gained a small decrease of 1.2% from PRE to POST1 (p=0.049), which was maintained at POST2 (p=0.041) (see Figure 12). The RSH group demonstrates a similar pattern, though to a greater extent, as they improved the total time by 1.9% from PRE to POST1 (p=0.009), and by 2.2% from PRE to POST2 (p=0.002). The CON group remained stabled throughout the three testing periods. There were some changes in S_{dec} for the for the RSN and RSH groups, although due to quite important standard deviations, none of them were statistically significant in time (p=0.78, NS).

There was no significant change in HR pre-RSA, HR post-RSA, HR post-3 min RSA, RPE, [La] pre-RSA or [La] post-3 min RSA.

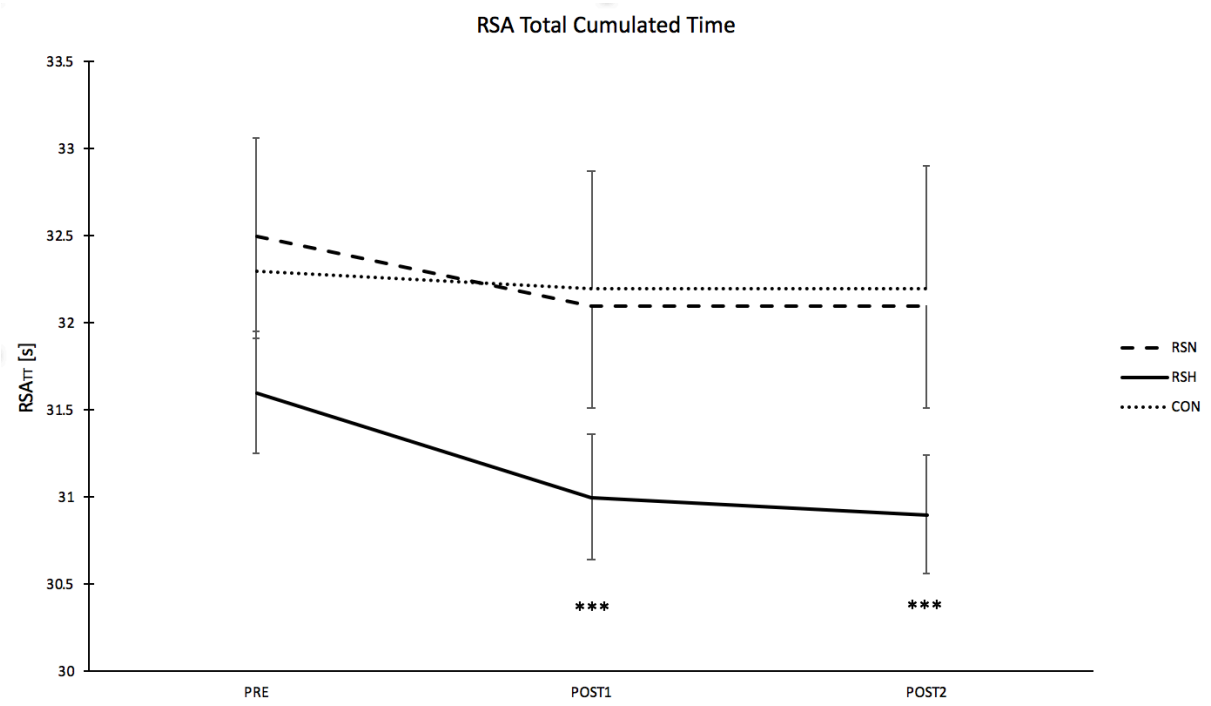

*Figure 12. Representation of the total cumulated time of the repeated-sprint ability test (RSA). Mean ± standard error (SE). ***(p>0.001) significant main effect in time different from PRE.*

3.2 Tissue oxygenation variations

3.2.1 Muscle oxygenation variations

There was no statistically significant interaction of time x sprint number (N°), group x sprint N°, or time x group x sprint N° (p>0.05, NS). There were several interactions of time x group, in leg oxygenation variations, which are shown in Table 4. Since there were not any interaction with the sprint N°, the data is presented as the average of all eight sprints.

Table 4. Near Infra-Red Spectroscopy measurements of m. Vastus Lateralis (Leg) oxygenation variations.

Group	Time	Δ[O_2Hb] [µm]	Δ[HHb] [µm]	Δ[tHb] [µm]	ΔTSI [%]	abs TSI_{max} [%]
	PRE	-9.2 ± 9.8	9.6 ± 6.0	-4.6 ± 11.0	-82.6 ± 6.1	59.8 ± 8.4
RSN	**POST1**	-13.6 ± 7.7***	11.5 ± 7.4	-11.9 ± 6.7***	-81.9 ± 9.9	56.2 ± 9.6
	POST2	-14.1 ± 8.1***	9.2 ± 8.0$^{\#}$	-10.1 ± 5.0***	-80.3 ± 9.9	58.9 ± 6.6
	PRE	-9.4 ± 11.6	9.3 ± 7.2	-4.6 ± 10.2	-81.6 ± 7.5	57.0 ± 10.6
RSH	**POST1**	-11.5 ± 7.4*	7.5 ± 9.6	-8.4 ± 6.9***	-72.5 ± 24.5***	53.4 ± 9.5
	POST2	-12.8 ± 6.7***	8.5 ± 7.2	-10.9 ± 5.9$^{***,\#}$	-78.7 ± 8.6$^{\#\#\#}$	56.6 ± 6.9
	PRE	-8.4 ± 7.4	10.6 ± 8.0	-4.5 ± 7.0	-80.4 ± 6.9	57.1 ± 14.7
CON	**POST1**	-14.4 ± 7.1***	10.5 ± 8.1	-11.0 ± 5.3***	-80.8 ± 7.1	54.7 ± 9.2
	POST2	-12.53 ± 6.6***	9.7 ± 8.7	-10.2 ± 6.3***	-79.9 ± 8.6	56.1 ± 11.4

*Delta (Δ) concentrations of oxy-hemoglobin (Δ[O2Hb]), deoxy-hemoglobin (Δ[HHb]), total hemoglobin (Δ[tHb]), Δ tissue saturation index (ΔTSI) and absolute TSI maximum (abs TSI_{max}). Mean ± SD. ***(p<0.001), **(p<0.01, *(p<0.05) significant main effect in time different from PRE; ###(p<0.001), ##(p<0.01), #(p<0.05) significant main effect in time different from POST1.*

The training protocol had an effect on muscle tissue oxygenation. There was a significant interaction of time x group for ΔTSI (p<0.001), reported in Figure 13. Indeed, following the training in hypoxia, the RSH group significantly desaturated more at POST1 in comparison to the PRE tests. The ΔTSI dropped by 11.2% (p<0.001) at POST1, and interestingly it increased back up (+8.6% (p<0.001) at POST2). It is interesting to note that the SD for the RSH group at POST1 more than tripled in comparison to the PRE test, and it re-stabilized to similar values to

PRE in the POST2 test. Both the RSN and the CON groups demonstrated no significant change in that parameter over the three tests.

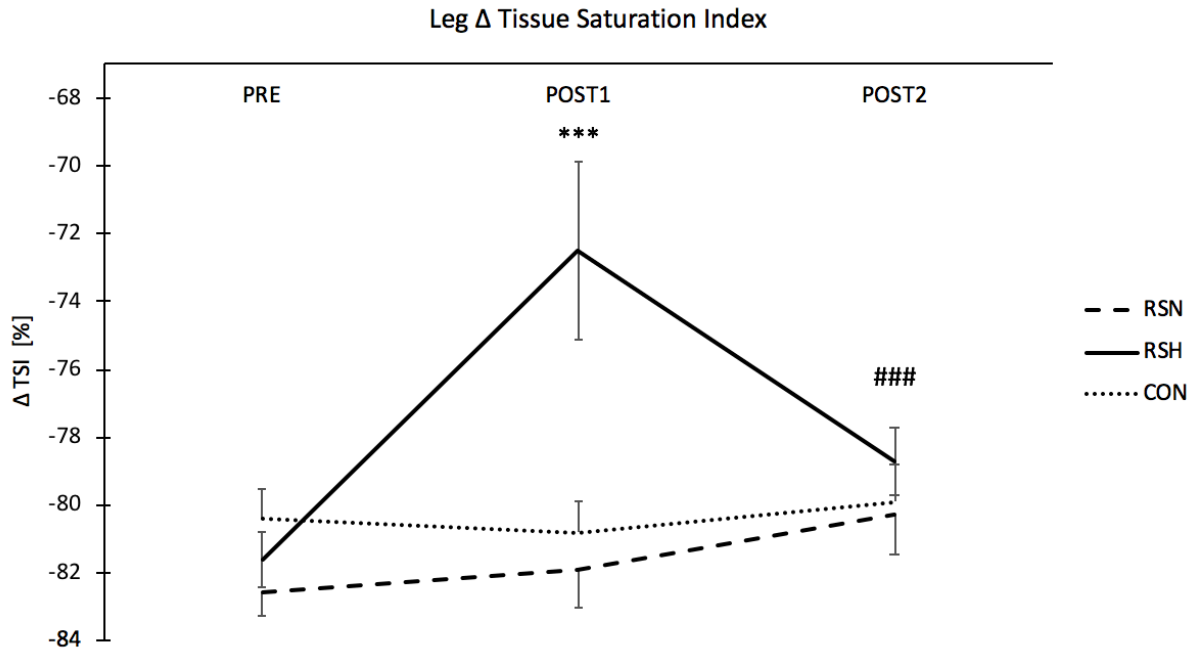

*Figure 13. Representation of the delta tissue saturation index (ΔTSI) in vastus lateralis (leg) during repeated-sprint exercise. Mean ± SE. ***($p<0.001$) significant main effect in time different from PRE, ###($p<0.001$) significant main effect in time different from POST1.*

Although the average saturation diminished for the RSH group, the abs TSI_{max} did not significantly change for any of the groups. The Δ[HHb] concentrations increased by 19.8% (p=0.063, NS) between PRE and POST1 for the RSN group but decreased by 20% (p=0.025) from POST1 to POST2 for the RSN group. In an opposite fashion, the RSH group decreased it by 19.4% (p=0.064, NS) between PRE and POST1, but it rose again by 13.3% (p=0.92, NS) though both variations were statistically non-significant.

Given the large variability and thus possibly unreliability of both $\Delta[O_2Hb]$ and Δ[tHb] concentrations measurements throughout the three tests and the three groups, these two parameters were not used for the analysis.

3.2.2 Cerebral oxygenation variations

Similar to the muscle oxygenation parameters, for the cerebral oxygenation, there was no significant interaction of time x sprint number (N°), group x sprint N°, or time x group x sprint N° (p>0.05, NS). There were several interactions of time x group in cerebral oxygenation variations, which are reported in Table 5. Since there were not any interaction with the N° of sprint, the data is also presented as the average of all eight sprints.

Table 5. Near Infra-Red Spectroscopy measurements of the prefrontal cortex (cerebral) oxygenation variations.

Group	Time	Δ[O_2Hb] [µm]	Δ[HHb] [µm]	Δ[tHb] [µm]	ΔTSI [%]	abs TSI_{max} [%]
	PRE	-6.9 ± 5.2^{+}	3.6 ± 1.9	-5.8 ± 6.5	-88.4 ± 10.1	85.3 ± 11.0
RSN	**POST1**	-11.0 ± 5.8***	3.6 ± 1.8+	-12.2 ± 7.0***	-95.9 ± 8.4***	91.8 ± 8.1***
	POST2	-13.1 ± 6.7***	4.0 ± 2.6	-15.1 ± 9.4***	-95.8 ± 7.2$^{***, +}$	91.9 ± 5.0***
	PRE	-8.5 ± 6.8	3.5 ± 2.0	-8.6 ± 7.7	-86.1 ± 7.8	82.0 ± 10.0
RSH	**POST1**	-14.2 ± 6.4***	4.0 ± 1.8^{+}	-14.7 ± 7.8***	-91.3 ± 8.2***	86.1 ± 8.6***
	POST2	-14.4 ± 5.7***	4.0 ± 1.2	-13.4 ± 6.4***	-89.5 ± 5.1**	83.9 ± 5.1***
	PRE	-12.1 ± 7.8	4.7 ± 2.5	-11.1 ± 9.8	-86.1 ± 10.9	80.8 ± 9.5
CON	**POST1**	-17.2 ± 7.6***	6.6 ± 4.8***	-17.4 ± 10.7***	-89.5 ± 7.6*	84.3 ± 7.0***
	POST2	-15.3 ± 7.9$^{**, \#}$	5.6 ± 5.3$^{**, \#}$	-16.2 ± 10.1***	-91.2 ± 8.5$^{***, \#\#\#}$	86.5 ±6.9$^{***, \#\#\#}$

*Delta (Δ) concentrations of oxy-hemoglobin (Δ[O2Hb]), deoxy-hemoglobin (Δ[HHb]), total hemoglobin (Δ[tHb]), tissue saturation index (TSI), and absolute TSI maximum (abs TSI_{max}). Mean ± SD. ***(p<0.001), **(p<0.01, *(p<0.05) significant main effect in time different from PRE; ###(p<0.001), ##(p<0.01), #(p<0.05) significant main effect in time different from POST1. +(p<0.05) significant effect of group different from CON.*

There was a significant effect of time x group for cerebral ΔTSI (p<0.001), graphed in Figure 14. The RSN group decreased it by 8.5% (p<0.001) between PRE and POST1, and it remained lower at POST2 (p<0.001) in comparison to PRE. For the RSH group, the parameter decreased by 6% (p<0.001) from PRE to POST1 but went back up to being 3.9% (p<0.001) in POST2 in comparison to PRE. The CON group followed a similar pattern, with a decrease of 3.9 (p=0.04) from PRE to POST1, and an even more marked decrease of 5.9% from PRE to POST1 (p<0.001).

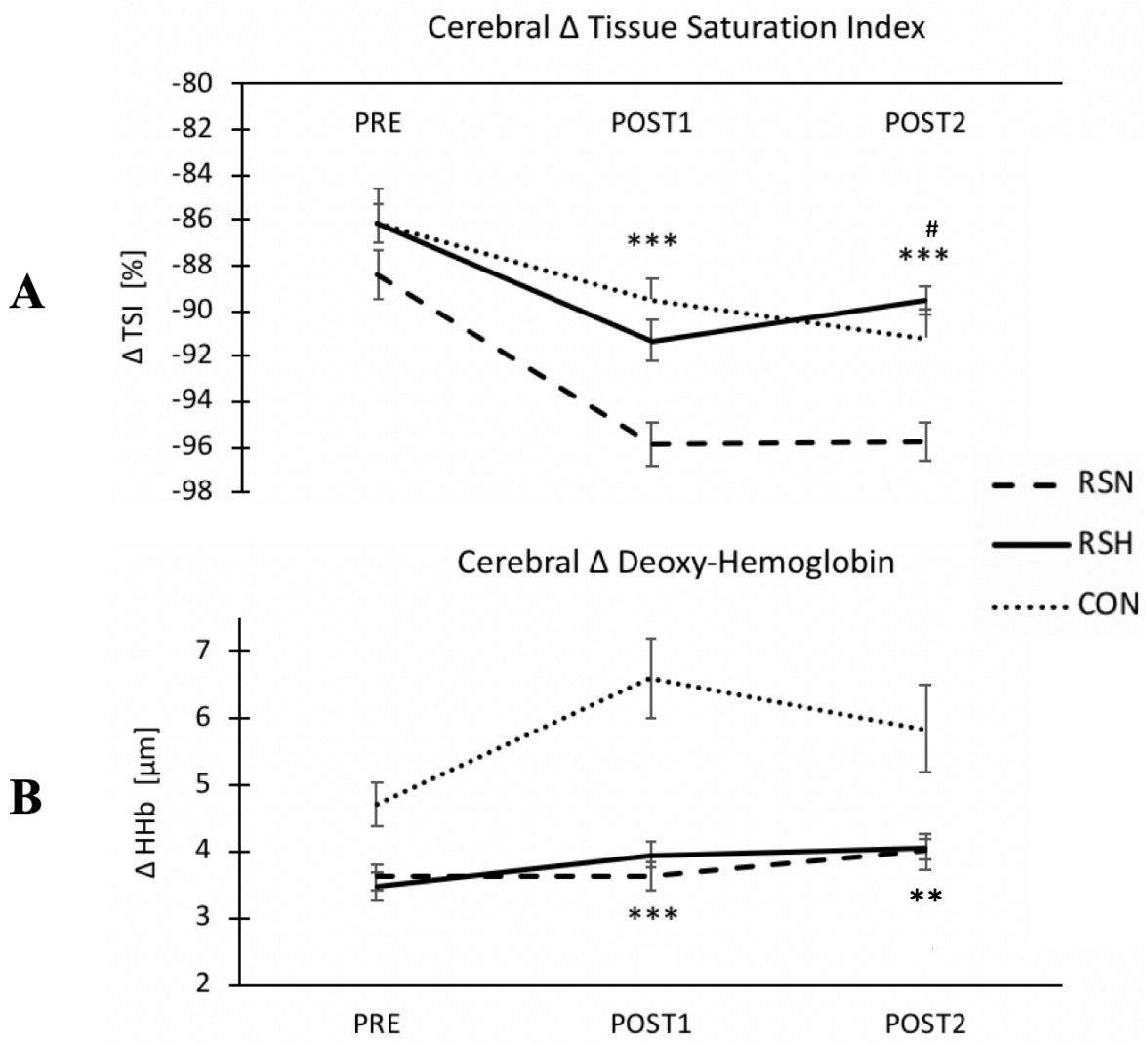

*Figure 14. Representation of oxygenation parameters in prefrontal cortex (cerebral) during repeated-sprint exercise. Delta tissue saturation index (ΔTSI) (A); and delta deoxy-hemoglobin (Δ[HHb]) (B). Mean ± SE. *** p <0.001, ** p <0.01 significant main effect in time, different from PRE, # p < 0.05 significant main effect in time, different from POST1.*

Unlike the muscle abs TSI_{max}, which was showed no significant changes, the variation in cerebral abs TSI_{max} evolved, with a significant effect of time (p<0.001), with all three groups having higher values at POST1 and POST2 in comparison to PRE. Regarding Δ[HHb], both the RSN and RSH group remained at similar values throughout the tests. The CON group however, increased that parameter by 40% (p<0.001) from PRE to POST1, and by 19.1% (p=0.004) between PRE and POST2 (see Figure 14).

4 Discussion

The purpose of this study was to assess the effects of five sessions of repeated-sprint training in hypoxia on well-trained tennis players. It generated larger improvements in tennis-specific performance, technical and physiological parameters as well as in sprinting performance than the same training performed in normoxia.

4.1 Effect of repeated-sprint training in hypoxia on tennis-specific performance

4.1.1 Physiological parameters

One of the principal findings of the present study was the increased time to exhaustion in the TEST by almost 18.2% for the RSH group, whereas it remained statistically unchanged for the RSN and CON group. This result is aligned with that of previous studies, who also measured an increased time to exhaustion following RSH (Brechbuhl et al., 2018a; Faiss et al., 2013b, 2015; Galvin et al., 2013). This improvement cannot be explained by a better absolute aerobic power since there was no change in $\dot{V}O_{2max}$ or other ventilation parameters. Interestingly, although it may at first appear as an enhanced fatigue resistance when the effort is near-maximal, the augmented time to OBLA suggests that towards the end, the subjects from the RSH group spend a similar amount of time in a physical severe state, it is however, the onset of that state that is right-shifted. This is in accordance to the similar research conducted by Brechbuhl et al., (2018a), who also found an increased time to exhaustion in the TEST, as well as a delayed OBLA. Therefore, the RSH group increased their submaximal work load intensity for a same blood [La]. Puype et al., (2013) had found same results with cyclists, who enhanced the power associated with OBLA by 7%. This improvement of the time to attain OBLA subsequent to RSH could come from certain mRNAs genes expression up-regulation. Indeed, following eight cycling sprint sessions in hypoxia, Faiss et al., (2013b) measured an expression increase of 35% in carbonic anhydrase III (CA3), a 12% increase in lactate dehydrogenase

(LDH) and a 20% augmentation in monocarboxylate transporter 4 (MCT-4) in the RSH group. LDH is important for energy metabolism and CA3 has a key function in pH regulation (Gore et al., 2007; Messonnier et al., 2007). Furthermore, in addition to stressing the importance of pH regulation and lactate removal in one's ability to perform supramaximal exercise, Messonnier et al. (2007) suggest that these parameters not only depend on lactate accumulation and H^+ production, but also on muscle intracellular buffering mechanisms. An improved hypoxia-induced muscle buffer capacity has been linked to the following twofold mechanisms: the hypoxic ventilatory response (i.e., hyperventilation to increase alveolar PO_2) causes a hypocapnia which results in a decrease in H^+, which ultimately increases the acid-base balance (Gore et al., 2007). In turn, an augmentation of renal bicarbonate excretion occurs, which is known to be a lactic acid buffer (Beaver et al., 1986). The same authors suggest that this could partially explain why hypoxic training has been shown to increase muscle buffering (Clark et al., 2004; Mizuno et al., 1990), and diminish the extent of reduction in the pH (Gore et al., 2007), and hence moving the lactate curve to the right.

The possible enhancement of lactate clearance may also be improved by RSH through the increase in MCT-4. It is known to play a central role in glucose uptake, lactate transport and lactic acid efflux (Wilson et al., 1998). It has been reported that it is involved in intracellular lactate shuttle (Brooks, 1998), and therefore its increase could improve cytosolic lactate transport to the same cell mitochondria for oxidization (Dubouchaud et al., 2000). Moreover, as LDH facilitates lactate oxidation in the mitochondria (Dubouchaud et al., 2000), its expression up-regulation following RSH measured by Faiss et al. (2013b) also supports this lactate transport facilitation, and thus also possibly its clearance.

Similarly, the increase in expression in CA3 following RSH could also be accounted for the delayed OBLA. CA helps catalyze the chemical conversion from CO_2 and H_2O to HCO_3^- and

H^+, and additionally, it has been suggested that extracellular CA contributes to the pH regulation by aiding lactate and H^+ transport through the sarcolemma (Geers and Gros, 2000).

In addition, although blood [La] max and [La] post-3 min following the RSA test did not vary after the intervention, the RSA_{TT} was improved for the RSH group, hence it could be logically argued that the subjects developed more power for the same blood [La], hence supporting either a better lactate clearance, or an improved muscle tolerance to the same concentration.

On another note, the up-regulation of some of these genes such as MCT-4 is of significance. Indeed, not only it plays a key role in lactic acid efflux, but, although present in all muscle cells, it has a predominant presence in non-oxidative muscles (Juel and Halestrap, 1999). Therefore, the increase in its expression suggests an increase in glycolytic activity, in particular in predominantly non-oxidative muscle fibers. This shift might be explained by the fact that at sea-level conditions, the rate of glycolysis upon the start of exercise is faster in comparison to oxidative phosphorylation, and the short nature of repeated-sprint training does not offer sufficient time for the oxidative pathway to fully contribute to the production of ATP needed. It has also been indicated that the amount of ATP that is provided by either of the energy pathways is much dependant on fibre-type; even though one is never completely dissociated from the other, FT fibres rely more on glycolysis, while red muscles primarily depend on oxidative phosphorylation (Juel and Halestrap, 1999). It has also been reported that the hypoxia itself increases mitochondrial oxidative stress and therefore diminishes its efficiency in the oxidative phosphorylation process (Magalhães et al., 2005). Therefore, if during the repeated-sprints in hypoxia, the minor ATP portion that in normoxic conditions would normally be produced by aerobic pathways is impaired, the glycolytic system has to compensate in order to meet the ATP requirement. This falls in line with the research from Puype et al., (2013), who also measured an increase of phosphofructokinase (PFK) activity by 59% in the group who performed sprint training in hypoxia. PFK expression has been demonstrated to be stimulated

in succession to high-intensity physical activity requiring important anaerobic ATP production through glycolysis, especially if performed in hypoxia (Jacobs et al., 1987; Puype et al., 2013; Vogt et al., 2001). Puype et al. (2013) explain this increased fraction of anaerobic ATP production to offset the diminution of oxidative energy turnover caused by the hypoxia. They also suggest that this extra stress on glycolytic flux may provoke an up-regulation of the anaerobic pathway. Faiss et al. (2013b) drew the same conclusion from their mRNA analysis, theorizing a shift from the aerobic to anaerobic pathway.

Consequently, a shift in energy pathways coupled with an enhanced buffer capacity and lactate clearance may explain the time lag in the OBLA from PRE to POST tests.

4.1.2 Technical parameters

TP during the final stage of the TEST was improved in the RSH group. This increase was only the result of a better BA, since BV did not significantly change. These results are in line with those of Brechbuhl et al. (2018b), who found similar changes. Interestingly though, part of the rationale driving the present study, was that RSH has been previously reported to increase fatigue resistance, particularly in FT (Faiss et al., 2013a, 2015; Galvin et al., 2013; Hamlin et al., 2017); and since muscle groups present in the upper-body are predominantly composed of FT fibers, a RSH involving actual tennis strokes was hypothesised to trigger adaptations that would induce a power increase and thus possibly significantly better BV. Surprisingly, the present results do not support this hypothesis for BV.

Even less expected, the RSN group improved BV by 8.8% from PRE to POST2. However, one could speculate that given the similitude of values between PRE and POST1, the gain in BV arises from a learning effect of the test. The *p*-value statistically confirming the variation being barely significant (p=0.048), this result ought to be interpreted with caution. Moreover, the CON group, though clearly not significant, also shows a small tendency in improvement in BV

over the course of the three tests, which also supports the hypothesis of a possible learning effect of the test.

One may speculate that the RSH-induced stimulus on the upper-body might have not been severe enough to improve fatigue resistance in arm muscles. Indeed, this study design contained four tennis strokes per sprint; that is twenty strokes per set, which results in eighty ball hits per training session. In comparison, the protocol conducted by Faiss et al. (2015), which demonstrated potent results in terms of upper-body fatigue resistance (i.e., improvement of 57% in number of sprints achieved before exhaustion), consisted in one hundred and twenty sprints (six sessions of four sets of five 10-s sprints. Cross country skiers are known to have maximal poling frequencies up to 1.5 Hz (Stöggl et al., 2007), which means that during one training session, Faiss et al. (2015)'s subjects performed around three hundred maximal pulls. That is 375% more upper-body movements per training session than the ones executed in the present study. Similarly, Beard et al. (2019b) measured improvements in peak power following only four sessions of upper-body double poling sprints in hypoxia. However, their subjects completed four sessions of three sets of eight 10-s sprints, which also results in over three hundred maximal pulls per session, which despite the number of sessions being one less, is still threefold greater than the number of upper-body movements in the present protocol. Therefore, it could be debated that although the present experimental protocol contained specific tennis strokes, the number of hits might have not been sufficient to trigger the targeted adaptations. Further research investigating the effects of a greater upper-limbs total work in hypoxia on BV would be interesting.

However, tennis players do not solely rely on their upper-body to be performant, and though upper-body movements might have not been the most fatigued body parts, lower limbs on the contrary were solicited to a great extent, and the RSA parameters improvements seem to attest adaptations in that regard.

Concerning BA, it was hypothesized that improvements would arise partially due to an increase in cerebral blood oxygenation variations (Brechbuhl et al., 2018b). However, based on the cerebral NIRS present measurements, changes in BA cannot be attributed or correlated to this parameter. Indeed, due to the presence of significant inter- and intra-group changes for all parameters and all three groups, these variations cannot be imputed to the experimental protocol. The only parameters which seems to manifest a certain tendency which differs from the CON group for both training groups is Δ [HHb]. Yet, since the curve of this parameter is similar between RSH and RSN groups but only the RSH group ameliorated their BA, the accuracy benefit may not be attributed to cerebral Δ [HHb].

These unexpected cerebral oxygenation results might have been caused by several factors, the first being the exercise effect of the test itself. Indeed, when assessed during incremental exercise, prefrontal cerebral oxygenation has been demonstrated to peak between moderate and hard intensity, but surprisingly, to drop at very hard intensity (Rooks et al., 2010). Accordingly, the intensity of the RSA test might have been too substantial to measure peak cerebral oxygenation. Though practically complicated, it would have been propitious to measure cerebral oxygenation variations during the TEST in order to evaluate it throughout the incremental intensity and correlate them with changes in BA.

Another factor that could have affected the present cerebral results is the signal measurement itself. It has been shown that cerebral oxygenation measurements are affected by subjects' age and furthermore, the inter-measure sensor location variation could induce up to 9% measurement differences (Kishi et al., 2003; Mancini et al., 1994). For obvious reasons, PRE test probe location could not be marked on subjects' faces, and there were most likely some positioning variances between tests, which may have been a limit to this measurement.

Likewise, although the impact on signal strength of skin enveloping soft tissues has been reported to be less than 5% (Hampson and Piantadosi, 1988), the greater presence of soft tissue

in addition to the cranial bone might increase the absorption of the NIR light and impair its capacity to penetrate cerebral tissue (Boushel and Piantadosi, 2000). The same authors also clarify that NIRS only measures cerebral oxygenation changes of the tissue directly located underneath the optodes. This is particularly meaningful, as it has been reported that in hypoxia, cerebral blood flow is heterogeneously distributed throughout brain regions. Indeed, not only have brain region-dependant differences in blood flow been measured, but more importantly, the increase in blood flow during hypoxia can vary up to 20% depending on the brain structure (Binks et al., 2008). According to these authors' results, the frontal lobe, which was evaluated in the present study, is one of the parts of the brain where the smallest augmentation (i.e., 11.3%) in blood flow occurs (in opposition to the 28.9% increase determined in the right nucleus accumbens for instance). Therefore, since it is established that: *I*) brain tissue O_2 level is correlated to cerebral blood flow (Jaeger et al., 2005), and *II*) cerebral blood flow increases in hypoxia (Binks et al., 2008; Curtelin et al., 2018), if certain areas of the brain gain in blood flow more than others in hypoxic conditions, they could also most likely benefit from increased oxygenation levels in comparison to those lesser irrigated parts; and similarly, if significant blood flow differences between brain structures subsist, then possible region discrepancies in blood oxygenation levels may also exist. Therefore, changes in oxygenation measured in the frontal lobe might not represent that of others brain regions or overall cerebral oxygenation levels, which could have had an impact on BA. Given these findings, further cerebral oximetry assessment from multiple brains areas during both RSH protocol and the TEST could adduce insight in the cerebral oxygenation kinetics during high intensity exercise in hypoxia and its possible role in accuracy benefits.

Further, it has been demonstrated that during both sprinting and incremental exercise in hypoxia, more attenuated changes in oxygenation occurred at 3800 m when compared to 400 m, which Willis et al. (2017) suggest to be a possible cerebral autoregulation of increased

perfusion in order to compensate for the diminished arterial O_2 content (Subudhi et al., 2007). This adaptation could have played a role in improving cognitive functions involved in tennis accuracy.

Finally, aside of cerebral oxygenation parameters, BA could have been improved due to fastest sprinting times measured in the RSA test. Indeed, the 2.33% improvement in RSA_{best} equals to a speed increase from 4.26 m/s^{-1} to 4.41 m/s^{-1}. This is of great significance, as a decrease in running speed has been reported to result in inaccurate stroke preparation as well as changes in stroke intention (which the authors define as avoiding error vs hitting winners) (Ferrauti et al., 2001b). Correspondingly, the opposite is most likely true, as in this 15 cm per second gains could induce better stroke preparation, which in turn could make the difference between a clean (i.e., at the centre of the racquet strings) and a poor impact, which greatly affects stroke quality and accuracy.

4.2 Effect of repeated-sprint training in hypoxia on repeated-sprint ability

The repeated-sprint training had a positive effect on RSA parameters for both training groups with a 1.2% and 1.9% improvement in RSA_{TT} for the RSN and RSH group, respectively. Yet, only the RSH group improved RSA_{best}. These findings confirm the hypothesis that repeated-sprint training ameliorates RSA parameters, and the extent of this benefit is enhanced when executed in hypoxia. It also reinforces previous similar results (Beard et al., 2019a; Brocherie et al., 2017; Kasai et al., 2017, 2019). The present results however, differ from those of Brechbuhl et al. (2018b), who did not report any putative effect of hypoxia on RSA parameters. However, although their training protocol was similar (though without the ball hits), their RSA test consisted of 20-m shuttle sprint without any direction change. Given that their protocol entailed 16.46 m (i.e., two lengths of a tennis court baseline) sprints with a 180° rotation in the middle, it could be argued that subjects developed more the deceleration-reacceleration part of

the sprint than maximal sprinting speed *per se*, as 8.23 m is most likely not enough to attain maximal sprinting speed, since it has been reported in young soccer players that during a 40 m sprint, maximal speed was attained between 30 and 40 m (Buchheit et al., 2012). Yet, the RSA test from Brechbuhl et al. (2018b) did not assess direction change, whereas the present study did. The fact that the present RSA test was more tennis specific could explain the discrepancy in RSA results despite the strictly similar training protocol and same population.

If the subjects did indeed develop deceleration and reacceleration parameters regardless of maximal sprinting speed, it would be still be particularly relevant for tennis-specific transferability. As in match play elite players run an average of 3 m per shot, and change four times directions per point (Deutsch et al., 1988; Fernandez-Fernandez et al., 2009), their maximal sprinting speed will in all likelihood hardly ever be reached, but on the other hand, they will need to constantly accelerate, decelerate and reaccelerate for every given shot. Moreover, as explained earlier, a better stroke preparation and stability at the moment of the hit may come from a decreased time for the same displacement. Therefore, improving these abilities is undoubtedly paramount for tennis performance.

One of the assumptions behind this study was the noteworthy role of the variation in muscle oxygenation in RSA parameters improvements. The 11.2% drop in ΔTSI at POST1 for the RSH group suggests a significant effect of oxygenation change explaining RSA benefits. These perfusion amplitude variations following RSH concur with previous similar studies (Faiss et al., 2013b, 2015; Galvin et al., 2013; Willis et al., 2017). Interestingly though, ΔTSI was lower at POST1, but at POST2, it was back up to values similar to PRE even though RSA performance remained improved. Likewise, Δ [HHb] increased for the RSN group at POST1, but was back to baseline numbers at POST2, but their RSA_{TT} was still ameliorated to same degree as in POST1. Therefore, the present data indicate that variations in oxygenation parameters

unquestionably have an influence on sprinting performance, but they are not the sole factor involved.

Other adaptations, though not measured in the present study, have been proposed to explain possible gains triggered by RSH. De Smet et al. (2016) measured a decrease in type IIx fibers in *m. vastus lateralis* and an increase in type IIa (which was greater when combined to oral nitrate intake) following five weeks of sprint interval training in hypoxia. Given the important force production and fatigue resistance of type IIa fibers (Stienen et al., 1996), a shift in fiber composition may explain higher explosive performance.

During exercise, as workload increases and O_2 tension diminishes, there is a proportional compensatory vasodilatation and increase in mean arterial pressure to meet skeletal muscles O_2 demand (Andersen and Saltin, 1985). What is more, mere acute hypoxic exposure also triggers a vasodilatation which raises blood flow to compensate for the drop in arterial O_2 saturation, but it has been demonstrated that in the course of exercise in hypoxia, the resulting compensatory vasodilatation is greater than the sum of both exercise and hypoxia individual responses (Casey and Joyner, 2012; Lundby et al., 2009). Since sprinting activity is purely anaerobic, slow-twitch muscle fibers' participation in such explosive movements is ostensibly negligible; hence, it is consequently FT fibers that benefit from the increased blood flow and inherent enhanced blood perfusion. Additionally, in normoxic conditions, an important functional reserve in muscle tissue O_2 diffusing capacity exists which is primarily utilized near exhaustion, and it has been indicated that this functional reserve was recruited during exercise in hypoxia (Calbet et al., 2015). The same study stipulates that in incremental exercise to exhaustion in hypoxia, muscle O_2 uptake is foremost limited by convectional rather than diffusive mechanisms, and ergo the kinetics of O_2 diffusion from hemoglobin to the cells is not the limiting factor to $\dot{V}O_2$ peak in hypoxia. This diminution in O_2 convection during repeated-sprints to exhaustion in hypoxia has also been reported (Willis et al., 2017). Therefrom the

authors conclude that in sprinting activity in hypoxia, limitation to O_2 uptake lies in mitochondrial respiration regulation (Calbet et al., 2015). This aerobic limitation within the cells could contribute to the shift to glycolytic pathways described earlier, which could be responsible for performance gains. This also support the previous hypothesis that in RSH, one of the main adaptations is the increased O_2 extraction ability, which most likely compensates for the limitation of mitochondrial respiration and partially results from increased blood flow (Faiss et al., 2013a, 2015).

4.3 Practical application

In conclusion, given the threefold increase in the standard deviation of ΔTSI at POST1 for the RSH group, the present data are also aligned with previous research on hypoxic training describing important inter-subject variability (Chapman et al., 1998; Levine, 2002; Levine and Stray-Gundersen, 1997), and this should be taken into consideration before prescribing hypoxic training.

Nonetheless, the purpose of this study was to assess the effects of RSH on well-trained tennis players and the present results suggest that as little as five sessions of RSH spread over two weeks is enough to trigger adaptations beneficial for both sprinting and tennis-specific performance. Although repeated-sprint practice also improves certain aspects, namely RSA, the putative effect of hypoxia undoubtedly potentiates the enhancement to a greater extent and provokes further adaptations. Of particular interest is the duration of the benefits which are still maintained twenty-one days after the intervention. Since elite tennis players are known to have limited in-season timeframes for training due to a full calendar (Reid and Schneiker, 2008), a two-week RSH protocol appears suitable to improve performance and maintain it for at least two dozen days. Moreover, it does not conflict with players regular aerobic and technical training. It has been suggested that implementing such a protocol two or three times per season

to initiate and maintain amelioration would be relevant for tennis players (Brechbuhl et al., 2018b).

4.4 Limits and perspectives

As with every scientific research using high technology devices and equipment for measurements, there is always a certain margin of error in reliability and reproducibility. Even though NIRS evaluation has been assessed to be reliable and reproducible (Boushel and Piantadosi, 2000; Jones et al., 2016; Mancini et al., 1994), it still has its flaws and limitations including, but not limited to, the repercussion of adipose tissue thickness; the unknown contribution of myoglobin in the overall signal; the effect of skin tissue perfusion and differences in skin pigmentation (Jones et al., 2016).

Also, cerebral oxygenation variations were evaluated during the RSA test for convenience, for subjects already wore the portable gas exchange measurer during the TEST, but the authors remain cognizant that conceivably, given the difference in energy pathways involved for both tests, a variance in cerebral oxygenation could be possible, hence fluctuations quantified in the RSA test might have been otherwise in the TEST. Therefore, further evaluation is needed on the impact of cerebral oxygenation amplitude and blood flow changes and their effect on accuracy following RSH.

Another possible limit to the present results is the timing of the protocol and the tests. Indeed, for practical reasons (i.e., mainly due to the time required for the hypoxic chamber to attain the targeted altitude, and to avoid mixing hypoxic and normoxic environments), RSN and RSH groups trained on alternative days. Therefore, the RSH group ended their training protocol one day before the RSN group finished theirs. Similarly, it necessitated a whole week to test every subject, which means that depending on the day of the week they were tested, the time between the end of the intervention and POST1 and POST2 was not equal for every participant.

Although subjects were randomly distributed over the testing week for POST1 and the order was kept the same for POST2 to minimize this impact, it still remains a possible bias to the present results.

In spite of the already promising results, taking into account the well-established more severe physiological response of HH in comparison to NH (Bourdillon et al., 2017; Degache et al., 2012; Hauser et al., 2017; Millet et al., 2012; Saugy et al., 2014), it could be sensibly argued that adaptations and thus performance benefits, could be potentiated if RSH were to be executed in genuine altitude. Prospective research in this regard would be favorable for the field of hypoxic training.

4.5 Conclusion

The aim of this study was to measure the effect of a two-week mesocycle of RSH on well-trained tennis players. It was hypothesized that such a protocol would induce significant ameliorations and adaptations which would transfer and benefit players in both tennis-specific and sprinting performance, but the extent of those would be greater in hypoxia when compared to the exact same training in normoxia. It was also assumed that these said adaptations would mainly be the result of improved muscle and cerebral tissue oxygenation. Although the results compellingly support and confirm the first two hypotheses, tissue oxygenation variations measurements effectuated in the present study demonstrate that it definitely plays a role in the adaptations, but it is however, not the sole mechanism involved in the intricate response and physiological alteration to high intensity hypoxia training. Nevertheless, despite underpinning physiological adaptations being still somewhat unclear, the actual performance gains quantified concur with the growingly important body of literature advocating the benefits of RSH on high intensity performance and the delay of its fatigue (Millet et al., 2019).

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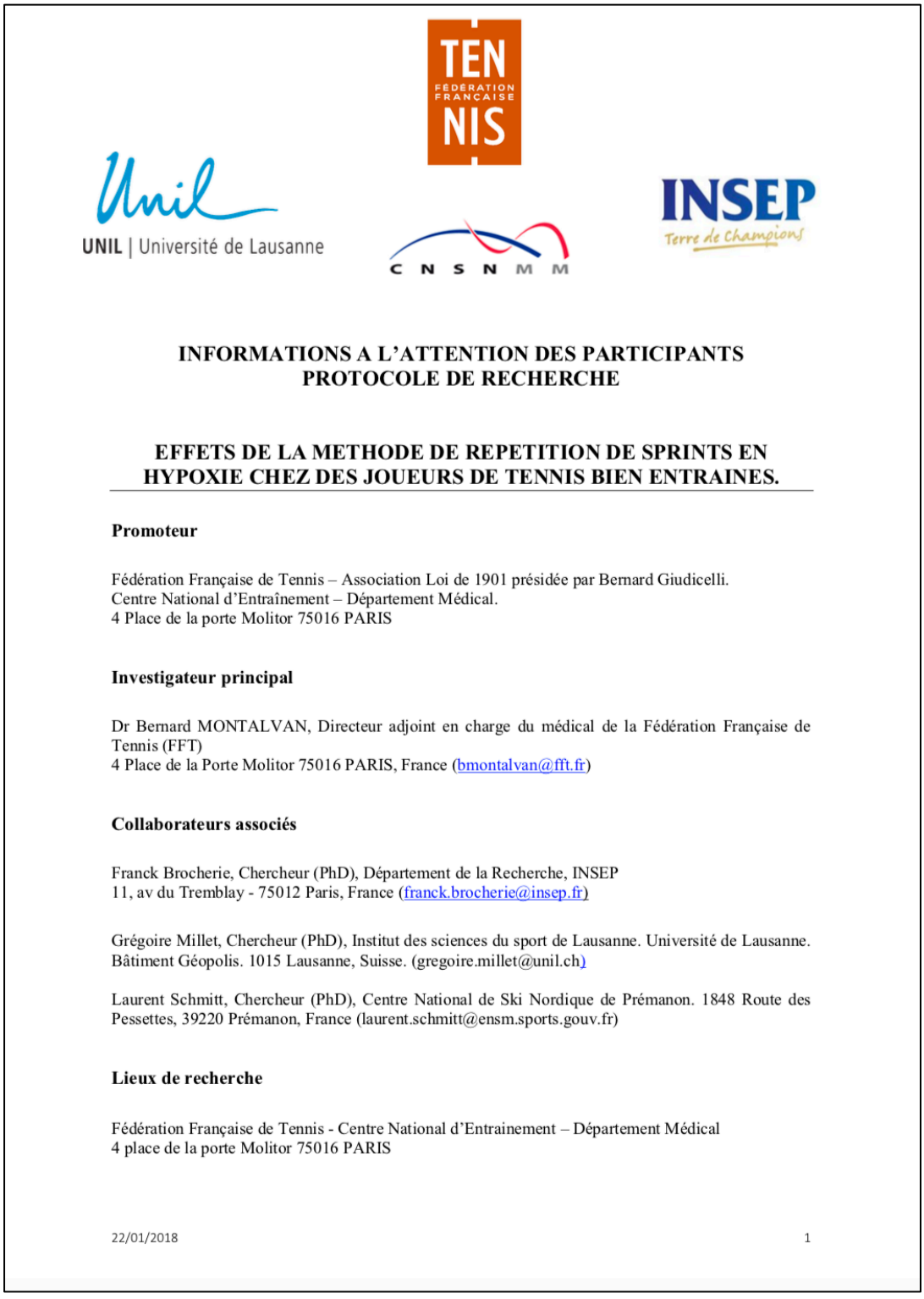
6 Appendix

6.1 Appendix 1. Information for participants



INFORMATIONS A L'ATTENTION DES PARTICIPANTS
PROTOCOLE DE RECHERCHE

EFFETS DE LA METHODE DE REPETITION DE SPRINTS EN HYPOXIE CHEZ DES JOUEURS DE TENNIS BIEN ENTRAINES.

Promoteur

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22/01/2018 1

Cher Monsieur,

Nous vous proposons de participer volontairement à une recherche biomédicale dont l'objectif est d'analyser les **effets de la méthode de répétition de sprints en hypoxie chez des joueurs de tennis bien entraînés**

1. OBJECTIF PRINCIPAL DU PROTOCOLE DE RECHERCHE

Bien que les compétences techniques et tactiques spécifiques au sport soient des facteurs prédominants, le tennis se caractérise par d'intenses sollicitations physiques qui nécessitent une condition physique bien équilibré (mélange de vitesse, d'agilité, de force et de puissance combinée à des capacités anaérobies et aérobies modérées à élevées) afin de maintenir une qualité de frappe alors que la fatigue tend à se développer au cours d'un match. Tandis que les matchs de tennis en général indiquent généralement une intensité d'exercice modérée [c.-à-d. 60-70% de la consommation maximale d'oxygène ($\dot{V}O_{2max}$), 70-80% de la fréquence cardiaque maximale], la reproduction des conditions de jeu réelles a rapporté des valeurs de lactatémie atteignant 8,6 mmol.L-1 chez les joueurs de tennis professionnels. Cela suggère une participation importante des processus glycolytiques pour répondre à l'approvisionnement en énergie pendant une partie de tennis. Différentes études ont exploré les effets de la répétition de sprints en hypoxie (RSH) sur la performance sportive en sport collectif. En revanche personne n'a spécifiquement étudié les performances liées au tennis. RSH apparaît comme une stratégie d'entraînement prometteuse dans les sports de raquette pour éventuellement améliorer la performance liée aux matchs. Avec RSH, des niveaux accrus de perfusion sanguine dus à la vasodilatation compensatoire en réponse à la diminution de la teneur en oxygène et à un comportement amélioré des fibres à contraction rapide sont attendus par rapport à la répétition de sprints en normoxie (RSN). Une étude a notamment montré une augmentation très significative du flux sanguin musculaire suite à un entraînement sous forme RSH en mobilisant le haut du corps. Par ailleurs, il a été montré que le flux sanguin cérébral s'adaptait à l'exercice en hypoxie.
Les réponses de l'entraînement en altitude sont complexes parce que le stress hypoxique et de l'entraînement sont combinés. L'hypoxie représente un facteur de stress supplémentaire avec la réduction de la PIO_2 (pression partielle de l'oxygène inspiré) et une diminution transitoire des activités parasympathiques et nerveuses sympathiques. A ce jour aucune publication ne concerne l'effet de RSH sur l'activité neurovégétative.
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Actuellement, l'élite des joueurs français utilisent de plus en plus cette nouvelle technique d'entraînement. Cependant, cette pratique mérite de plus amples éclairages sur les bénéfices déjà ressentis et observés.

Le but de cette étude va être d'évaluer l'impact et l'efficacité de la méthode RSH avec frappes de balles intégrées sur le flux sanguin et ses potentielles corrélations avec une meilleure capacité de performance dans l'activité spécifique. La mesure de cette capacité à mieux faire face à la difficulté de l'effort sera évaluée à travers le Test d'Effort Spécifique au Tennis. Le flux sanguin sera mesuré lors du test d'habileté à répéter des sprints, ainsi que la variabilité cardiaque le matin des journées de pré et post-tests.

2. MODALITES D'INCLUSION AU PROTOCOLE

Pour participer à cette étude, vous devez avoir entre 18 et 40 ans et être affilié au régime de la Sécurité Sociale ou bénéficiaire d'un tel régime. Votre inclusion au projet de recherche se fera après une prise de connaissance rigoureuse du projet. Vous devrez également avoir été déclaré indemne de toute pathologie cardio-vasculaire ou musculo-squelettique lors de la visite médicale de pré-inclusion, et de ce fait ne présenter aucune contre-indication aux techniques mobilisées pour l'étude. Cette visite comportera un examen clinique (palpation, auscultation, mesures anthropométriques) et une exploration fonctionnelle comprenant :

- Un questionnaire médical
- Un électrocardiogramme (ECG) de repos, associé à une mesure de la fréquence cardiaque et de la tension artérielle.
- Une échographie cardiaque.
- Si au moins deux facteurs de risques cardiovasculaires sont présents, un test d'effort sera nécessaire pour envisager la validation d'une participation à l'étude

3. METHODOLOGIE ET DEROULEMENT DE L'ETUDE

Le but de cette étude va être d'évaluer l'impact et l'efficacité de la méthode dite de « répétition de sprints en hypoxie » (RSH) comparée à la répétition de sprints en normoxie (RSN) et à un groupe contrôle via différentes mesures physiologiques et de performance. Les participants volontaires seront répartis par tirage au sort dans un des trois groupes (RSN, RSH, Contrôle), le jour de la réunion d'information.

Ces mesures seront :

- ***Tests de performance*** en effectuant des tests neuromusculaires sans charge additionnelle sur 3 mouvements standards (Contre Mouvement Jump (CMJ), Squat- Jump (SJ), Saut réactivité). A la suite, on mesure l'habileté à répéter des sprints sur une course type aller-retour sur la largueur d'un terrain de simple. Après 1h de repos pourra débuter le test d'effort spécifique au tennis qui dure 15 à 25 minutes.

- ***Test de variabilité cardiaque (HRV)*** le matin des journées de test.

- ***Perceptives et psychologiques*** : via vos réponses à des questionnaires ce qui nous permettra d'analyser et d'évaluer votre perception de l'effort physique.

- ***Objectives***

  En mesurant de manière indirecte, via un ***prélèvement sanguin par le bout du doigt*** (≈ 25µL), la concentration de lactate dans le sang.

  En mesurant la **fréquence cardiaque**, nous permettant alors d'observer l'intensité de l'effort fourni.

  En mesurant les **quantités de gaz inspirés et expirés** afin de déterminer la mise en jeu métabolique pendant l'effort fourni.

  En mesurant la **quantité de flux sanguin cérébral et musculaire** afin d'estimer une capacité accrue de resynthèse des substrats énergétiques ainsi qu'un meilleur traitement du lactate pendant l'effort par exemple.

De plus, vous serez amené à effectuer la technique d'entraînement suivante :

Répétition de sprint en hypoxie : Il s'agit de réaliser 4 séries de 5 répétitions d'un effort à intensité maximale qui consiste à couvrir 3 fois la largeur d'un terrain de simple. Chaque course est ponctuée par une frappe de balle. Un départ est donné toutes les 30 secondes pour un effort d'une durée de 7 secondes environ. 5 minutes de récupération passive sont observées entre chaque série.

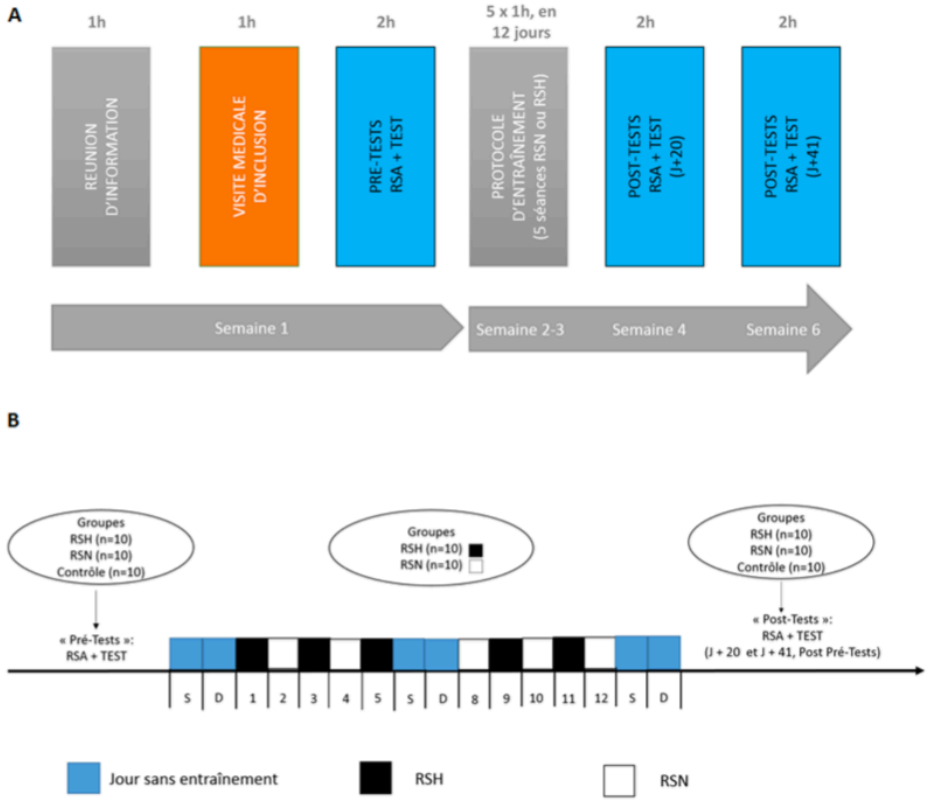

Figure 2 : déroulement du protocole expérimental (A). Protocole d'entraînement de 12 jours (B).

Tout le protocole, décrit sur la figure 2 ci-dessus, se déroulera au Centre National d'Entraînement. Vous y effectuerez chacune des séances suivantes :

- Une réunion d'information générale et un **apprentissage du protocole du test HRV**
- Une **première journée avec** votre visite médicale d'inclusion suivie des pré-tests
- **La période d'entraînement** avec 5 séances sur rdv (temps sur place : 1 heure)
- **Deux journées de post tests** *(1 à J + 20, l'autre à J + 41)* afin de mesurer les effets de l'entraînement et leur maintien dans le temps.

4. EXPOSE DES BENEFICES

L'étude ne présente aucun bénéfice pour les participants

5. EXPOSE DES EFFETS INDESIRABLES

Vous pourrez ressentir des courbatures lors des jours suivants les séances RSH.
Effet indésirable peu probable : vomissement occasionné par une forte lactatémie.
Douleur à l'endroit de la piqûre pour la mesure de la lactatémie (très rare).

6. INDEMNISATION

Une **indemnité compensatoire de 200€** sera versée à chaque participant volontaire ayant réalisé l'intégralité des séances et des tests prévus dans le protocole. Nous prendrons également en charge les frais à hauteur de 0,56 euros/km pour les résidents en banlieue parisienne (les 200 premiers kilomètres, puis 0,10 euros/km), et nous rembourserons l'équivalent du prix d'un ticket de métro pour les résidents parisiens.
L'indemnité sera complète (200€ + frais de déplacement) pour les sujets sortis de l'expérimentation pour des effets indésirables.
L'indemnisation de la participation pour les sujets quittant l'étude à leur demande sera calculée au prorata de la participation (X présences / 3 pour le groupe « contrôle » + frais de déplacement, ou X présences / 8 pour les groupes RSH ou RSN + frais de déplacement).

7. ASPECTS LEGAUX

Si vous souhaitez participer, il vous est demandé de signer librement un formulaire de consentement. Vous êtes invité à prendre le temps de réflexion nécessaire et à en discuter, si vous le souhaitez, avec le médecin de votre choix et/ou avec vos proches. Vous êtes bien entendu libre de refuser de participer à cette étude sans avoir à vous justifier. Même en ayant donné votre consentement, vous resterez totalement libre de vous retirer de l'étude à tout moment sans avoir à vous justifier. Par ailleurs, votre signature du consentement ne décharge d'aucune façon les investigateurs et le promoteur de leurs responsabilités.

Vous avez la possibilité de poser des questions au Dr Bernard Montalvan, à tout moment avant et en cours d'étude. Les motifs d'un éventuel arrêt ou sortie d'étude à l'initiative du promoteur ou des investigateurs vous seront transmis lors de votre visite de pré-inclusion. Toute information nouvelle survenant pendant votre participation et pouvant éventuellement modifier votre décision de participation à cette recherche vous sera immédiatement donnée. Les informations concernant votre santé peuvent vous être communiquées au cours ou à l'issue de la recherche directement ou indirectement par l'intermédiaire d'un médecin que vous aurez préalablement désigné.

Si vous acceptez de participer à cette étude vous n'aurez pas le droit de participer simultanément à une autre recherche. De ce fait, vous intégrerez le fichier national automatisé des "volontaires qui se prêtent à une recherche" (article L 1121-16 du Code de la Santé Publique) où figureront : les trois premières lettres de votre nom patronymique, les deux premières lettres de votre prénom, votre date de naissance, les dates de début et de fin de participation à l'étude. Les résultats globaux de l'étude vous seront communiqués dans un délai de 6 mois après la fin de la période expérimentale.
La Fédération française de Tennis a souscrit un contrat de Responsabilité Promoteur de recherche impliquant la personne humaine chez HDI GLOBAL SE sous le numéro 0100534514058170103.

8. CONFIDENTIALITE DES DONNEES

Le traitement informatisé des données nominatives est conforme aux dispositions de la loi n°2004-801 du 6 août 2004 relative à la protection des personnes physiques à l'égard des traitements de données à caractère personnel et modifiant la loi n°78-17 du 6 janvier 1978 relative à l'informatique, aux fichiers et aux libertés. Vous pourrez exercer votre droit d'accès et de rectification garanti par les articles 39 et 40 de cette dite loi en vous adressant aux investigateurs de l'étude. Vous pouvez accéder au texte de cette loi sur le site internet de la Commission Nationale de l'Informatique et des Libertés (CNIL) : www.cnil.fr.

Les photographies et les films réalisés au cours de l'étude seront uniquement cadrés sur le bras. Par conséquent, l'anonymat des participants sera préservé.

Les données recueillies pour cette recherche seront confidentielles. Elles ne pourront être consultables que par les investigateurs, leurs collaborateurs et leur équipe médicale, les personnes mandatées par le promoteur et soumises au secret professionnel et les personnes mandatées par les autorités sanitaires et judiciaires. S'ils le souhaitent les participants pourront demander la transmission des résultats globaux de l'étude.

Cette recherche est soumise aux dispositions qui figurent dans le Code de la Santé public, titre II du livre premier relatif aux recherches biomédicales. Vous pouvez avoir accès à ce code sur le site internet de Légifrance : www.legifrance.gouv.fr.

Le CPP IDF 6 Groupe hospitalier Pitié-Salpétrière a donné un avis favorable à la mise en œuvre de cette recherche le ……/……/ 2018.

L'Agence Nationale de Sécurité du Médicament (ANSM) a autorisé la mise en œuvre de cette recherche le 11/10/2017.

Le promoteur a contracté un contrat d'assurance spécifique (n° de contrat : 0100534514058 170103) pour cette recherche auprès de HDI GLOBAL SE France.

6.2 Appendix 2. Consent form for participants

DECLARATION DE CONSENTEMENT

N° d'autorisation délivré par l'ANSM : 2017-A02865-48

EFFETS DE LA METHODE DE REPETITION DE SPRINTS EN HYPOXIE CHEZ DES JOUEURS DE TENNIS BIEN ENTRAINES.

Promoteur

Fédération Française de Tennis – Association Loi 1901 présidée par Bernard Giudicelli.
Centre National d'Entrainement – Département Médical
4 Place de la porte Molitor 75016 PARIS

Investigateur principal

Dr Bernard MONTALVAN, Directeur adjoint en charge du médical de la Fédération Française de Tennis (FFT)
4 place de la Porte Molitor 75016 PARIS, France (bmontalvan@fft.fr)

Collaborateurs associés

Franck Brocherie, Chercheur (PhD), Département de la Recherche, INSEP
11, av du Tremblay - 75012 Paris, France (franck.brocherie@insep.fr)

Grégoire Millet, Chercheur (PhD), Institut des sciences du sport de Lausanne. Université de Lausanne. Bâtiment Géopolis. 1015 Lausanne, Suisse. (gregoire.millet@unil.ch)

Laurent Schmitt, Chercheur (PhD), Centre National de Ski Nordique de Prémanon. 1848 Route des Pessettes, 39220 Prémanon, France (laurent.schmitt@ensm.sports.gouv.fr)

Lieux de recherche

Fédération Française de Tennis - Centre National d'Entrainement – Département Médical
4 place de la porte Molitor 75016 PARIS.

Je soussigné M. *(prénom, NOM)* ……,accepte librement et volontairement de participer à la recherche intitulée « Effets de la méthode d'entraînement de répétition de sprints en hypoxie chez des joueurs de tennis élite », dont le promoteur est la Fédération Française de Tennis (Centre National d'Entraînement), 4 place de la Porte Molitor, 75016 Paris, et l'investigateur principal est Bernard Montalvan.
Je certifie que :

1. INFORMATIONS

- j'ai été informé de l'intérêt et du déroulement de l'étude par oral et par écrit.
- j'ai pu poser toutes les questions utiles à ma bonne compréhension des informations reçues et j'ai obtenu des réponses claires et précises.
- j'ai bénéficié d'un temps de réflexion suffisant pour prendre ma décision, entre les informations et le consentement.

2. INTERRUPTION DE LA PARTICIPATION

- je suis libre de refuser de participer à cette étude.
- j'ai parfaitement conscience que je peux interrompre à tout moment ma participation à cette recherche quelles que soient mes raisons et sans endosser aucune responsabilité. Cependant dans le cas échéant, je m'engage à en informer l'investigateur principal.
- je pourrais être exclu de l'étude si je ne respecte pas les termes du protocole ou si un médecin le juge nécessaire.

3. OBLIGATIONS

- je suis affilié ou bénéficie d'un régime de sécurité sociale.
- je ne suis pas privé de liberté par une décision judiciaire ou administrative et je ne fais pas l'objet d'une tutelle.

4. EXAMEN MEDICAL PREALABLE

- je bénéficierai d'un examen médical préalable à l'étude. Les résultats éventuels me seront communiqués directement ou par l'intermédiaire d'un médecin de mon choix.

Docteur :……Adresse :……

5. LEGISLATION

- Je conserve un exemplaire de la note d'information et du présent formulaire de consentement qui sera conservé 15 ans par le promoteur.
- mon consentement ne décharge pas les organisateurs de la recherche de leurs responsabilités à mon égard et je conserve tous mes droits garantis par la loi.
- j'ai compris que les données recueillies ayant trait à mon état de santé, à mes habitudes de vie, à ma situation administrative sont strictement confidentielles et ne peuvent être consultées que par le promoteur, le médecin qui me suit, l'investigateur principal ainsi que les personnes qui collaborent à la recherche, ces personnes étant soumises au secret professionnel.
- selon les dispositions de la loi du 4 mars 2002, je serais informé des résultats globaux de l'étude par l'investigateur.
- en conformité avec la loi n° 2004-806 du 9 août 2004 relative à la politique de santé publique, le CPP Sud Méditerranée IV a émis un avis favorable quant à la réalisation de l'étude en date du ……..
- le promoteur de l'étude a souscrit un contrat d'assurance (n° de contrat : 0100534514058 170103) garantissant sa responsabilité pour cette recherche auprès de : HDI Gerling France.

6. INFORMATISATION DES DONNEES

- j'accepte le traitement informatisé de mes données en conformité avec la loi « Informatique et Liberté » du 6 janvier 1978, modifiée par la loi n°94-548 du 1er juillet 1994 et 2002-303 du 4 mars 2002.
- les données enregistrées lors de cette étude sont confidentielles, leur consultation est exclusivement destinée aux personnes qui collaborent à la recherche désignées par l'investigateur principal.
- je peux exercer, à tout moment, un droit d'accès et de rectification de mes données personnelles
- je suis informé que mes données personnelles (les trois premières lettres de mon nom patronymique, les deux premières lettres de mon prénom, ma date de naissance, les dates de début et de fin de participation à l'étude) seront inscrites au fichier national des « volontaires qui se prêtent à une recherche » en vertu de l'article L 1121-19 du Code de la Santé Publique.
- j'ai été informé que mes données personnelles seront rendues anonymes, avant d'être intégrées dans un rapport ou une publication scientifique.

7. PRISE DE PHOTOS OU DE FILMS

- je donne mon accord pour que des photos ou des films soient pris et utilisés pour les besoins de l'investigateur.

8. INTERDICTION DE PARTICIPER A UNE AUTRE ETUDE

- j'ai été informé que je ne pourrai pas participer à une autre étude pendant ma participation à cette étude

9. SIGNATURE

Je conserve un exemplaire de la note d'information et du présent formulaire de consentement.

Fait à…….

Date :……	Signature :

Signature de l'investigateur qui certifie avoir expliqué le but, les modalités ainsi que les risques potentiels de la recherche.

Date :……....	Signature :

6.3 Appendix 3. Rate of Perceived Exertion form for participants

How difficult it felt

2) What is your overall perceived exertion?

3) How difficult does it feel to breathe?

4) How much limb discomfort do you have?

0	Nothing at all
0.5	Very, very slight (almost none)
1	Very slight
2	Slight
3	Moderate
4	Somewhat severe
5	Severe
6	
7	Very severe
8	
9	Very, very severe
10	Maximal