# PHYSIOLOGICAL RESPONSES AND THEIR ASSOCIATIONS TO EXTERNAL LOAD IMMEDIATELY AND 12 HOURS AFTER OFFICIAL ICE HOCKEY MATCH IN FINNISH ELITE ICE HOCKEY PLAYERS

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**ABSTRACT**

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**Introduction.** Knowledge about physiological responses and sufficient recovery times after matches are needed in professional team sports to avoid illnesses, injuries and overtraining. In many team sports (e.g. soccer, rugby, handball and basketball), such knowledge has been attained, however, in ice hockey research in this area is lacking. Therefore, the aim of this study was to identify the physiological responses and their associations to external load in official Finnish elite league ice hockey match.

**Methods.** The study subjects were 38 Finnish elite league ice hockey players. The study was done in single group repeated measures design, where measurements were done before, during, immediately and 12 hours after the match. The pre- and post-match measurements included venous blood samples for leukocytes, lymphocytes and creatine kinase (CK), salivary samples for testosterone (T), cortisol (C), testosterone to cortisol -ratio (T/C), DHEA-S and IgA and the assessment of neuromuscular performance by measuring jump height, maximum power, flight times, take-off times and flight time to take-off time -ratios (FT/TT) from countermovement jump (CMJ). Also, match activity data, heart rates and session-RPE (sRPE) were recorded during the match.

**Results.** Maximum power, FT and FT/TT in CMJ increased at post 0h. At post 12h maximum power and FT returned to baseline, whereas FT/TT remained improved. CK increased at post 0h and further increased at post 12h. T and T/C decreased at post 0h with no change in C, whereas DHEA-S increased. No significant differences were found in T, C, T/C or DHEA-S between baseline and post 12h values. Circulating leukocytes increased and lymphocytes decreased at post 0h with no change in salivary IgA. At post 12h lymphocytes returned to baseline and leukocytes remained elevated. Greater skating intensity was associated with increased T and DHEA-S at post 12h. sRPE correlated with the amount of high-intensity skating, acute changes in T, DHEA-S and IgA and with changes in C and DHEA-S between post 0h and post 12h.

**Conclusions.** Ice hockey match does not seem to impair neuromuscular performance, but other markers indicate that the players are not fully recovered 12 hours after the match. External load is poorly associated with physiological responses to ice hockey match. Hence, more research is needed to better understand the individual responses to ice hockey matches.

Key words: ice hockey, match-load, internal load, external load, physiological responses

**ABBREVIATIONS**

| | |
|---|---|
| ATP | adenosine triphosphate |
| C | cortisol |
| CK | creatine kinase |
| CMJ | countermovement jump |
| DHEA | dehydroepiandrosterone |
| DHEA-S | dehydroepiandrosterone sulfate |
| FT/TT | flight time to take-off time -ratio |
| GPS | global positioning system |
| HR | heart rate |
| IgA | immunoglobulin A |
| LPS | local positioning system |
| NHL | national hockey league |
| NK | natural killer cell |
| PCr | phosphocreatine |
| RPE | rating of perceived exertion |
| SJ | squat jump |
| sRPE | session rating of perceived exertion |
| T | testosterone |
| T/C | testosterone to cortisol -ratio |
| URTI | upper respiratory tract infection |
| VAS | visual analogue scale |
| $VO_{2max}$ | maximal oxygen uptake |
| 1RM | one repetition maximum |

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# 1 INTRODUCTION

Professional team sport athletes are required to have a highly developed physical qualities to be able to play at the highest level of competitions (Burr et al. 2008; Vigh-Larsen et al. 2019). However, it has been well documented that physical qualities tend to decline in team sport athletes during in-seasons (Delisle-Houde et al. 2018; Laurent et al. 2014; Whitehead et al. 2019). This impaired physical performance during in-seasons is probably a result of imbalances between physical loading and sufficient recovery. If the state of insufficient recovery is continued for too long, the athletes may be in an increased risk for developing illnesses, injuries and overtraining syndrome. Therefore, to avoid these unwanted events, the coaching staff should be aware of the physical strain that is caused to the players by the training sessions and competitive matches. (Meeusen et al. 2013) This is especially important in professional team sports, as the players are exposed to busy schedules of matches and physical and tactical training and other psychological stressors (e.g. traveling, media, contract negotiations, sponsors) that impair recovery (Doeven et al. 2018; Quarrie et al. 2017). When the coaches and team support staff have the knowledge about the match-loads and needed recovery times after matches, they can more precisely plan productive training schedules and prepare the players for the upcoming matches (Morton 1997; Russell et al. 2016).

Even though the events during team sport matches are random and depend on many different factors, it is assumed that the intensity is nearly maximal and great physiological and psychological stress is imposed to the players. In fact, there are several studies that have examined the physiological changes and recovery times in different team ball games (e.g. soccer, rugby, handball and basketball). According to this data, match-play in fast-paced intermittent team ball games acutely decrease neuromuscular performance, cause muscle damage and alter endocrine and immune function and these changes take up to 24 to 72 hours to recover back to prematch levels. (Chatzinikolaou et al. 2014; Fatouros et al. 2010; Pliauga et al. 2015; Silva et al. 2013; Twist & Sykes 2011)

However, very limited data exist about acute match loads and recovery times in ice hockey. This can be considered as a big gap in the ice hockey literature, as ice hockey match-play differs from other team sports in its high-intensity profile, active playing time and unique characteristics of locomotion in skating (Lignell et al. 2018). Due to these factors it is most likely that ice hockey match-play also differs in its physiological responses and recovery profiles after matches, and so there is a high demand for research examining these questions. Therefore, this present study aims to answer what are the responses in neuromuscular performance, markers of muscle damage and in hormonal and immunological status immediately and 12 hours after official ice hockey match. This study also examines the associations of these physiological changes to the external loading during the match. The findings from this study may then be further used by ice hockey coaches and strength and conditioning professionals to better individualize training and recovery strategies for the players after matches.

# 2 PHYSIOLOGICAL DEMANDS OF ICE HOCKEY

## 2.1 Ice hockey as a sport

Ice hockey is a fast-paced team sport that involves several intervals of high-intensity intermittent skating, fast changes of direction, explosive accelerations and high-impact body contacts in combination with highly skilled technical tasks (Montgomery 1988; Cox et al. 1995; Lignell et al. 2018). Ice hockey match consists of three 20-minute periods of active playing time with 15-minute intermissions between periods. If the score is tied after 60 minutes of regulation time the match is continued to overtime and penalty-shot shootout until the winner is determined. (IIHF 2018.) At the elite level, the match consists of approximately 25-30 shifts of intense play with each bout typically lasting about 45 to 60 seconds and sometimes even up to 90 seconds. The playing bouts are separated by 2 to 5-minute recovery periods and the total playing time in a match for individual player is typically 15-25 minutes. (Montgomery 1988; Cox et al. 1995; Lignell et al. 2018)

In a study by Lignell et al. (2018) they measured high-intensity activities during match-play in top-class ice hockey players from the National Hockey League (NHL). They measured the total skating distance during the game and divided the distances to different categories using the following speed thresholds: very low skating (1-10.9 km/h), slow skating (11-13.9 km/h), moderate-speed skating (14-16.9 km/h), fast skating (17-20.9 km/h), very fast skating (21-24 km/h) and sprint skating (> 24 km/h). Further, low intensity skating was determined to be all the skating done in very low, slow and moderate-speed skating, whereas high intensity skating was categorized as all the skating done at fast, very fast and sprint skating velocities. They reported that the total average skating distance during the match was 4606 ± 219 m. Of the total amount of skating 31, 11 and 14% was covered in very low speed, low speed and moderate speed skating, respectively. The average amount of high-intensity skating during the match was 2042 ± 97 m of which 49, 27 and 24% was covered in fast, very fast and sprint skating, respectively. The total average number of high-intensity skating bouts during the match for individual player was 113 ± 7 which is approximately 7 (4-10) high-intensity skating bouts averaging 15 meters each minute. This data highlights the fast nature and highly

intermittent activity pattern of the top-class ice hockey game. This demands the players to have the ability to perform short, explosive, high-intensity bouts and to be able to recover sufficiently after each bout. (Lignell et al. 2018)

## 2.2 Energy metabolism during ice hockey match

Because of the fast and highly intermittent characteristics of ice hockey game the players are required to have well developed aerobic and anaerobic energy systems. The contribution of anaerobic energy production during one shift in ice hockey match is estimated to be approximately 69% and aerobic energy production 31% of the total energy produced during the shift (Seliger et al. 1972). Like in many other team sports, one shift in ice hockey game consists of repeated short maximal or near maximal working bouts followed by relatively short low-intensity recovery periods (Lignell et al. 2018). During the one maximal working bout lasting up to 6 seconds, most of the produced adenosine triphosphate (ATP) is resynthesized from phosphocreatine (PCr) and anaerobic glycolysis with very little emphasis on aerobic metabolism. This anaerobic energy production disturbs homeostasis in the working muscle cells as tissue stores of PCr, glycogen and oxygen decrease and metabolites such as lactate and inorganic phosphate starts to build up. During the recovery period, aerobic energy systems are used to restore these changes in the working muscles. If the recovery period is not long enough and the anaerobic energy stores are not sufficiently restored, the contribution of aerobic energy production during the next maximal effort is increased, thus compromising intensity of the performance (Figure 1.). (Glaister 2005)

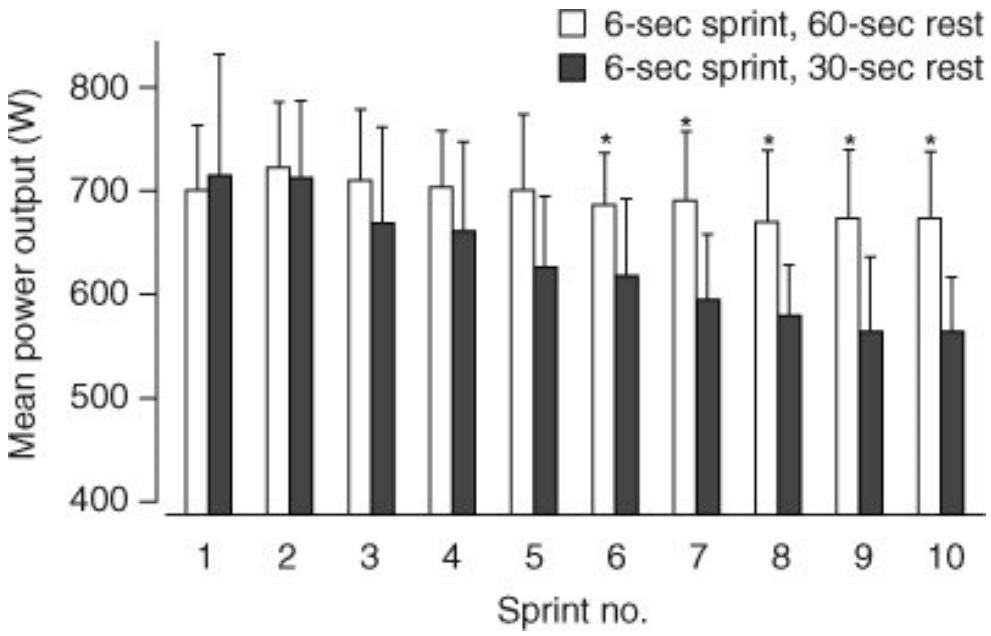



FIGURE 1. Mean power output during 10 x 6-second maximal treadmill sprints with 30- or 60-second recovery periods. (Glaister 2005)

Several methods have been used in attempt to accurately determine metabolic demands during ice hockey match. Commonly measured physiological parameters have been heart rate (HR) (Montgomery 1988; Spiering et al. 2003), blood lactate (Noonan 2010) and muscle glycogen stores (Green et al. 1978). The average heart rate during one shift is approximately 85% and the peak heart rate above 90% of maximal heart rate. Between the shifts heart rate then recovers to 55-70% of the maximal heart rate. (Montgomery 1988) Mean heart rate during the entire ice hockey match is about 68% of the maximal heart rate (Spiering et al. 2003). Measured blood lactate levels during ice hockey game range between 4.4 to 13.7 mmol/l with the mean value being 8.15 mmol/l (Noonan 2010). Muscle glycogen stores have been reported to diminish by 60% during ice hockey game (Green et al. 1978). These results highlight the intense and variable nature of ice hockey game. According to the blood lactate levels and decreased muscle glycogen stores it can be stated that anaerobic energy production is dominant during the shifts.

To evaluate the aerobic and anaerobic metabolic demands during the entire ice hockey match Stanula et al. (2014) measured the time spent at different intensity zones based on aerobic and

anaerobic heart rate thresholds obtained from incremental maximum oxygen uptake ($VO_{2max}$) test. The subjects were sixteen players from the Polish national junior team (under 20-years old) and the data was collected from 4 different games in 2 week's period at the World Championships. They determined individual intensity zones for each player according to the ventilatory threshold values measured in the $VO_{2max}$ test before the tournament. The intensity zones were following; low which was heart rates below aerobic threshold, moderate which was heart rates between aerobic and anaerobic thresholds and high which was heart rates above anaerobic threshold. What they found was that time spent at each intensity zone was different between forwards and defensemen. The percentage of total playing time at each intensity zone for forwards and defensemen were following low 58.75% vs. 44.29%, moderate 21.95% vs. 25.84% and high 19.30% vs. 29.87%, respectively. The data from this study indicates that approximately half of the total playing time during ice hockey game is spent below aerobic threshold and thus training of the aerobic energy systems should not be neglected. (Stanula et al. 2014) When looking at these results it should be noted that heart rates during the ice hockey match cannot be directly compared to heart rates measured during the incremental $VO_{2max}$ test, because other factors such as body temperature, static work of the upper body muscles and emotions during matches also affect heart rates besides oxygen cost of the match-play (Montgomery 1988).

### 2.3 Physiological characteristics of ice hockey players

Because of the metabolic demands of ice hockey, the players must have highly trained aerobic and anaerobic energy production systems. As the game of ice hockey has become faster and more aggressive over the years, the physical appearance and physiological fitness of the players has also changed. Now days the players are also required to have a large lean body mass and high levels of muscular strength. Thus, the players should have well-developed total body fitness level to be able to maintain sport-specific performance and to prevent injury and premature fatigue during matches. (Cox et al. 1995; Montgomery 2006)

### 2.3.1 Body composition of ice hockey players

The body composition of ice hockey players has changed markedly over the years. In a longitudinal comparison done in professional NHL ice hockey players it has been found that the players are currently 10 cm taller and 17 kg heavier than the players in the 1920s and 1930s. In 2003 the average height of the NHL players was 1.85 m and average body mass was 92 kg. The estimated body fat percentage of the players measured using skinfold method was 10.4%. Body fat percentage measures have not been done for the players in the 1920s and 1930s, but it is assumed that the greater body mass is explained by increases in muscle tissue since the players have also become stronger. (Montgomery 2006)

The body composition and anthropometrics also seems to differ among the level at which the players are playing. In Danish ice hockey players, it was found that there were differences between elite league and subelite league players in height (182.3 cm vs. 180.9 cm), body mass (85.7 kg vs. 80.8 kg) and muscle mass (41.9 kg vs. 38.8 kg). These differences might in part be explained by the fact that the subelite players were younger than the elite league players (19.4 years vs. 23.5 years). (Vigh-Larsen et al. 2019) However, if the Danish elite league players (Vigh-Larsen et al. 2019) are compared to the NHL players (Montgomery 2006), it can be seen that the NHL players who play at the highest level are taller and heavier than the Danish elite league players.

### 2.3.2 Aerobic endurance performance of ice hockey players

As stated previously, ice hockey players are required to have a high level of aerobic capacity to be able to compete at the elite level. This has been shown to be true as large differences have been observed between elite and subelite players in total distance covered during sport-specific on-ice Yo-Yo test, where players perform short skating bouts with increasing speed followed by 10 second resting period (Vigh-Larsen et al. 2019). The high level of aerobic capacity of competitive ice hockey players has also been shown in many other studies as the measured $VO_{2max}$ values of the players range between 55 to 60 ml/kg/min (Burr et al. 2008; Montgomery 2006; Peterson et al. 2015; Quinney et al. 2008; Vescovi et al. 2006). These

values are comparable to $VO_{2max}$ in soccer players despite the fact that in soccer the players cover 2 to 3 times greater distance and the total playing time is also 4 to 6 times longer during soccer matches (Tonnessen et al. 2013).

Even though the ice hockey game is highly intermittent and consists of short bursts of high intensity efforts, the aerobic capacity is needed for recovery between the shifts and thus effects performance. In fact, aerobic capacity has been associated with on-ice performance (Peterson et al. 2015), number of scoring changes (Green et al. 2006) and total distance covered during match-play (Lignell et al. 2018).

### 2.3.3 Strength and power performance of ice hockey players

As a fast-paced contact sport, ice hockey requires the players to have sufficient muscular strength and power in lower and upper body (Montgomery 1988). Lower body strength and power is needed for skating, accelerations, changes of direction and to maintain balance in body contacts. Lower body strength training will also help the players to have lower center of mass and thereby increasing the stability while skating. Upper body strength is related to on-ice performance by improving body checking, shooting and puck control. Total body strength and lean mass are needed in contact situations and to prevent injury. Sufficient muscle mass and strength gives protection in contacts with other players, sticks, pucks, boards and playing surfaces. Balanced muscular development is also important for injury prevention as imbalances in muscular strength between muscle groups may increase the risk of injury in joints and muscles. (Boland et al. 2019; Twist & Rhodes 1993)

Muscular strength is a physical quality that improves in professional ice hockey players as the players get older. This was found in a longitudinal study observing the physiological profiles of NHL players over the years. The upper body strength was measured as predicted one repetition maximum (1RM) in bench press and the data was collected between the years of 1993 and 2003. The mean 1RM in bench press for 17- to 19-years old professional ice hockey players was 106.95 ± 19.9 kg. From there the strength levels linearly increased for the older age groups and reached a peak in the 25- to 29-years old group when the mean bench press

1RM was 128.09 ± 19.7 kg. This increase of 21 kg in bench press 1RM was associated with the mean increase in body mass by 5 kg during that time period. (Montgomery 2006)

Upper body strength and power have been associated with puck speeds in slap shot and wrist shot in professional and semiprofessional ice hockey players. In a study by Bezak & Pridal (2017), they found that bench press 1RM and mean power produced with 40kg and 50kg in bench press correlated with puck velocities in slap shots and wrists shots. The main findings in their study was that mean bench press 1RM and average concentric power in bench press with 40kg and 50kg were 95.2 ± 13.8kg, 485.4 ± 50.7 W and 509.5 ± 63.3 W, respectively. The correlations were greater for power than for strength in both slap shot and wrist shots. These results show that sufficient upper body strength is needed in ice hockey, but more emphasis should be placed on upper body power production for improved shooting performance. (Bezak & Pridal 2017)

Also, lower body power seems to be a distinguishing factor between subelite and elite ice hockey players. It was reported by Vigh-Larsen et al. (2019), that elite players outperformed subelite players in countermovement jump (CMJ), agility -test and in sprinting performance. Also, top ranked elite team players performed better in CMJ and agility -test than elite players from bottom ranked teams. (Vigh-Larsen et al. 2019) The importance of lower body power output in professional ice hockey is further supported by the fact that players who perform better in standing long jump are more likely to be drafted at higher rounds at the NHL entry draft (Burr et al. 2008).

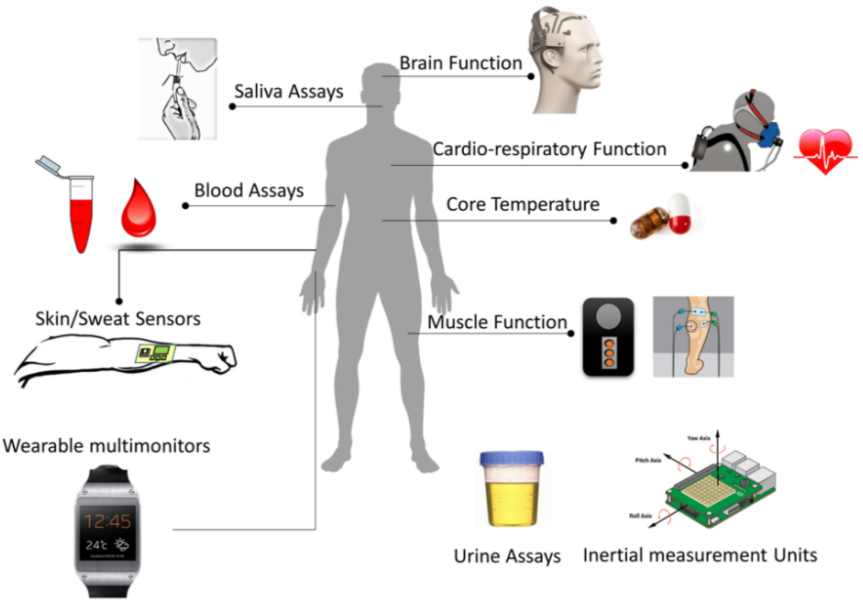
# 3 MEASURING MATCH-LOAD AND RECOVERY IN TEAM SPORTS

Professional team sport athletes undergo a busy schedule of physical and tactical training combined with several competitive matches during a week. For example, during the regular season Finnish elite league ice hockey players may have 2 to 3 matches each week (Liiga 2019). Although the events during matches are random and there is a lot of variance in the performance of the players, it can be assumed that the intensity during the matches is maximal and therefore cause the most strain on the players. Thus, it is crucial to achieve sufficient state of recovery after the matches before participating into physical training to prevent illness, injury or non-functional overreaching. (Bishop et al. 2008).

The evaluation of the state of recovery and athlete's preparedness for training can be achieved by monitoring training load. In addition, training load monitoring can also provide information about how athlete is adapting to the training program. Training load monitoring can be divided to measuring external and internal loads. External load is classified as the work done by the athlete and it is the foundation of many traditional training load monitoring systems. For example, external load can be the distance covered by the player during a match or the mean power output by cyclist during a race. External load is usually used to measure the capacity and capabilities of the athlete, whereas internal load is used to measure the physiological stress and adaptations imposed by the match or training session. (Halson 2014) When trying to understand the current state of athlete's recovery and the true training load, it is recommended to measure both external and internal loads. For example, if the same workout is done in recovered and fatigued state, the external loading might be exactly the same, but there might be great changes in the internal loading. (Bourdon et al. 2017)

Due to the recent advances in technology, training load monitoring has become a popular tool among sport scientists and coaches to guide their decision making in the pursuit of optimal performance. (Cardinale & Varley 2017) However, there can be some downsides in training load monitoring such as limited budget, time and knowledge to gather and analyze data by most practitioners and also the fact that there is no guarantee that the training load monitoring will lead to improved performance. (Burgess 2017; Halson 2014) Also, to date there is no

single variable, that would reflect the internal loading of the athlete and thus multiple different measurements are advised to be done of which some can be quite invasive and expensive (figure 2). This implementation of too many different devices and measurements can then interfere with the performance of the athlete and negate the purpose of the training load monitoring. Therefore, it is important to carefully decide which methods are being used to measure training load. (Cardinale & Varley 2017)




| Cardiorespiratory Parameters | Humoral Parameters | Neuromuscular and muscle metabolism Parameters |
|---|---|---|
| Heart Rate: **H,$$** | Venous Blood: **L,$$$** | Electromyography: **M,$$$** |
| Heart Rate Variability: **L,$$** | Capillary Blood: **M,$$** | Electroencephalography: **L,$$$** |
| Breathing related Parameters: **L,$$** | Sweat: **L,$$$** | Galvanic Skin Response: **M,$$** |
| Oxygen Uptake and derived parameters: **L,$$$** | Saliva: **M,$$$** | Near Infrared Spectroscopy (NIRS): **L,$$$** |
| | Urinary Markers: **M,$$$** | Brain NIRS: **L,$$$** |

**Practicality**: **H**igh, **M**edium, **L**ow; **Cost:** $,$$,$$$

FIGURE 2. Illustrating paragraph summarizing methods for measuring internal load. (Cardinale & Varley 2017)

## 3.1 Measuring external load

External load can be described as the work done by the athlete during training or competition that can be objectively measured. Depending on the sport and used technology external load measures can be duration, speed, power output, distance covered, accelerations, and sport-specific movements like tackles or shots performed. Valid and reliable external load monitoring is important for objectively determine the demands in training and competitions. (Cardinale & Varley 2017)

In team sports external loads are often measured with global positioning system (GPS) devices that measure covered distance, accelerations, decelerations, changes of direction and collisions (Black & Gabbett 2014; Burgess 2017; Cummins et al. 2013; Malone et al. 2017 T). However, the reliability of GPS decreases as the movement speed increases and thus fast movements like jumping, kicking, and tackling may not be detected with GPS. For that reason, now days almost every GPS device used in sports also contains a triaxial accelerometer to detect these fast movements (Cardinale & Varley 2017). Another problem with GPS is that it cannot measure movement inside buildings. That is why in sports played inside like in ice hockey, other methods like local positioning system (LPS) (Luteberget et al. 2018) and multiple-camera computerized tracking system (Lignell et al. 2018) have been used to measure external load.

## 3.2 Measuring internal load

Athletes internal loading to training or competition can be described as the summation of physiological and psychological responses to the stimulus. As different sports and different types of training can have very different physiological and psychological demands, it is recommended to use a holistic approach for measuring internal loading to exercise. The different physiological methods for measuring internal loading can be categorized into cardiorespiratory, humoral and neuromuscular parameters. (Cardinale & Varley 2017)

From the cardiorespiratory parameters heart rate is the most common method for measuring internal loading in athletes. Heart rate monitoring can estimate oxygen consumption during loading since heart rate and oxygen consumption have a linear relationship in steady-state conditions. (Halson 2014) Heart rate can also be used to classify the intensity of the effort when heart rate is presented as a percentage of maximum heart rate (Borresen & Lambert 2008). Other often used cardiorespiratory parameter is heart rate variability. Heart rate variability is however more useful for monitoring training status in prolonged period of time than acutely after exercise as it requires longitudinal monitoring on individual level. (Halson 2014)

Humoral parameters can yet be categorized into biochemical, hormonal and immunological responses to exercise. These parameters are mostly assessed from blood, saliva and urine samples. Probably the most used biochemical marker in sports research is creatine kinase (CK) which is associated to muscle damage. (Halson 2014) From hormonal responses to exercise it has been recommended to use testosterone to cortisol -ratio to measure internal loading as this ratio decreases in relation to intensity and duration of the exercise (Heidari et al. 2019; Meeusen et al. 2013). Measured immunological parameters in relation to loading in team sports have been leucocytes, lymphocytes, immunoglobulin A (IgA) and inflammation markers C-reactive protein and interleukin-6 (Orysiak et al. 2017; Thorpe et al. 2017). It has been stated that none of these humoral markers should be used alone to determine acute loading but rather in an integrated fashion to get a comprehensive overall picture about the physiological reactions to different types of stimuli (Cardinale & Varley 2017).

Vertical jumps are popular method for assessing recovery of neuromuscular function after competitions in team ball games. Different types of vertical jumps can be used to measure lower body strength and power production, but CMJ is the most widely used variation in team sports. Depending on the method used to measure CMJ, many different parameters can be used to estimate neuromuscular function. These parameters can be jump height, peak power, mean power, mean force, flight time, contraction time or flight time to contraction time -ratio. Besides vertical jumps, neuromuscular function has also been assessed in team ball games by using other tests like sprint times, maximal voluntary contraction and line drill tests. (Doeven et al. 2018)

A very cost-efficient and easy to implement method for assessing internal load is the use of rating of perceived exertion (RPE) method developed by Borg. This method was further developed into session-RPE (sRPE) by Foster et al. (2001). Session-RPE takes into account the intensity and duration of the training session. When using sRPE, the athletes are first asked to rank the intensity of the training session on the scale from 0 to 10, where 0 equals rest and 10 means maximal effort. This number is then multiplied by the length of the training session in minutes to get a number describing the training load of the session. This method has been proven to be valid and reliable when measuring training loads in different sports and physical activities in men and women at different ages and levels of expertise. It has even been proposed that it could be used as a "stand alone" method for measuring training load. However, if possible, it should be recommended to use it in combination with some other physiological parameter, such as heart rate. (Haddad et al. 2017)

# 4 PHYSIOLOGICAL RESPONSES TO MATCH-PLAY IN TEAM SPORTS

There is very little research about the acute responses to ice hockey match-play in elite league players. According to the available literature it seems that ice hockey match-play may cause acute decreases in neuromuscular performance as the average speed of high intensity skating is lower in the third period compared to first and second periods (Lignell et al. 2018). No direct research on neuromuscular performance acutely after official match-play has been done in ice hockey players. The data about humoral parameters indicates that responses in muscle damage, immune function and inflammation are not as severe in ice hockey as they are in soccer (Lignell et al. 2018; Mohr et al. 2016). This might be explained by shorter duration of ice hockey game and lesser impact during locomotion in skating than in running (Lignell et al. 2018).

Even though the internal loading during ice hockey match might not be as severe as in other team ball games, it seems that the overall recovery during ice hockey season is not sufficient. In male collegiate ice hockey players, it was found that neuromuscular performance measured as a jump height in CMJ and squat jump (SJ) decreased and perceived fatigue increased significantly during the 18 week long season (Whitehead et al 2019). Declined physical performance and lean body mass during ice hockey in-season have also been observed in two other studies (Delisle-Houde et al. 2018; Laurent et al. 2014). As the knowledge about acute loading and post-match recovery from ice hockey match-play is spare, the research about acute physiological responses to exercise in general and to match-play in other intermittent team ball games are reviewed next in more detail.

## 4.1 Neuromuscular performance

Prolonged and/or intense exercise typically cause muscular fatigue, which can be manifested as temporary declines in force, velocity and power produced by the working muscles. The magnitude of impairment in neuromuscular performance depends on several factors such as duration and intensity of the exercise as well as on the individual's age, physical fitness, nutritional status and fiber type composition of the muscles involved. All of these factors can

also affect what is the underlying cause of declined performance and more often there are several different physiological mechanisms that work synergistically at different sites of the neuromuscular system that cause the impaired performance. These alterations leading to declined muscular performance can happen in the muscle itself (peripheral fatigue) or in the nervous system controlling the muscle (central fatigue). Eight potential sites in voluntary force production process have been identified in which fatigue might occur (figure 3). These sites are 1) excitatory input to higher motor centers, 2) excitatory drive to lower motor neurons, 3) motor neuron excitability, 4) neuromuscular transmission, 5) sarcolemma excitability, 6) excitation-contraction coupling, 7) contractile mechanisms and 8) metabolic energy supply and metabolite accumulation. However, in highly trained and motivated athletic population, impaired sarcolemma excitability, declined metabolic supply and increased metabolite accumulation are the most likely causes of impaired neuromuscular performance. (Farrell et al. 2012, 171-172) Since measuring the underlying mechanism behind muscular fatigue is often impractical in field settings when match responses are being studied, simple tests that measure neuromuscular function is rather preferred.

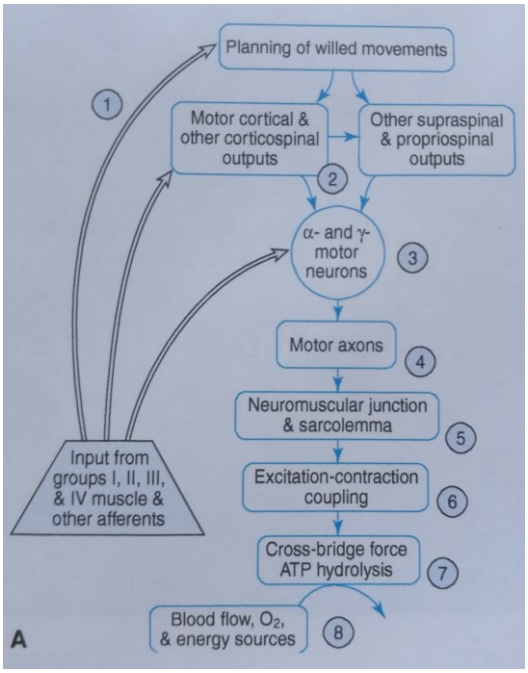



FIGURE 3. Potential sites in neuromuscular system to cause muscular fatigue. (Farrell et al. 2012, 172)

Most often used method for assessing neuromuscular function after match-play is jump height in CMJ where declines in jump height indicates impaired neuromuscular performance. When jump height has been measured after matches in team ball games (e.g. soccer, rugby, basketball) it has been found that jump height is reduced immediately after the match and remains below baseline levels for approximately 48 hours. Other often used method for measuring neuromuscular performance have been sprint times. Usual sprint distances have been 5, 10, 20, 30 and 40 meters. More variation has been observed in sprint time recovery after matches as the recovery periods range from 24 to 96 hours. (Doeven et al. 2018)

It seems that the type of sport does have an impact on the acute neuromuscular responses and the time course of recovery of the performance. The time course of recovery in CMJ height after basketball match have been reported to exceed 48 hours that is typically seen in other team ball games (Chatzinikolaou et al. 2015; Pliauga et al. 2015). This might be explained by the high number of jumps performed during basketball matches. In a similar fashion sprint times take longer to recover back to baseline after soccer matches than after basketball and handball matches. For soccer sprint times take more than 72 hours to recover to baseline (Fatouros et al. 2010; Ispirlidis et al. 2008) as for basketball (Pliauga et al. 2015) and handball (Chatzinikolaou et al. 2014) the recovery times are between 48 to 72 hours. This is probably explained by the longer duration and fewer number of substitutes in soccer.

Physical fitness also seems to be one factor that determine the magnitude of neuromuscular fatigue after matches in team sports. Johnston et al. (2015) found that in 16-years old junior rugby players neuromuscular fatigue measured by reductions in peak power in CMJ was greater in the players who had performed worse in Yo-Yo intermittent recovery test level 1. The reduction in neuromuscular performance was lesser in the players with higher fitness level despite the fact that they covered more distance and performed at higher intensities than the players with lower fitness level. (Johnston et al. 2015)

Measuring height in CMJ to determine neuromuscular function has had some critique as it may not be sensitive enough to detect changes in training load (Thorpe et al. 2017). Flight time to take-off time -ratio (FT/TT) has been shown to be more sensitive variable of recovery

when measuring CMJ. The true recovery status of neuromuscular function may not be detected if only height is measured from CMJ as it has been found that reductions in FT/TT takes longer to return to baseline than jump height. (Rowell et al. 2017) Therefore, it can be recommended to include FT/TT as one variable when measuring acute changes and recovery of neuromuscular function.

## 4.2 Muscle damage after exercise

Creatine kinase is widely used biochemical indirect marker to evaluate muscle damage in sport science research (Heidari et al. 2019). Creatine kinase is enzyme that is located inside muscle cells and it is used to produce ATP. When muscle cell is damaged, CK is leaked out of the muscle into blood stream and thus increased concentrations of CK in blood can reflect muscle damage caused by exercise. Resting levels of serum CK levels are usually higher in athletes than in sedentary population and this is explained by the greater lean muscle mass and regular exercise in athletes (Brancaccio et al. 2008). Median value for CK concentration for professional athletes in resting conditions have been reported to be 284 U/L (Mahmutyazicioglu et al. 2018) and recommended reference interval for athletes is suggested to be from 82 to 1083 U/L in men and 47 to 513 U/L in women. These values are dramatically higher than the upper limits reported for inactive population (171 U/L for males and 145 U/L for females). (Mougios 2007)

In a systematic review by Doeven et al. (2018), it was found that CK concentrations in blood starts to increase immediately after matches in different team ball games. The CK concentration continues to elevate for several hours and peak values are reached usually at 24 hours post-match. Large variance between the highest CK concentrations was observed as the reported peak values ranged from 100 U/L to 1411 U/L. After the peak value is reached, CK concentration then starts to gradually decrease and finally returns back to baseline levels between 42 to 120 hours after the match. These large variations in CK kinematics in blood after matches is probably explained by differences in physical demands between different team ball games as well as in the physical characteristics of the players. (Doeven et al. 2018)

In professional Australian football players, it was found that increased CK concentrations were associated with the number of high force impacts, accelerations, decelerations and sprinting distance during official match (Gastin et al. 2019). This can be explained by the large eccentric strain on the body during high speed running as eccentric loading is suggested to be the major cause of muscle damage during exercise (Barid et al. 2012; Howatson & Milak 2009). In high force eccentric loading as in running, sarcomeres inside working muscle cells might be overstretched and damaged from the loading. This can lead to Z-disc fragmentations and changes in muscle cell membrane permeability which in turn leads to CK leakage into blood. (Brancaccio et al. 2008) High impact collisions such as tackles are associated with increases in CK levels as they might cause damages and bruises in the area of the contact (Gastin et al. 2019).

Like ice hockey, Australian football and rugby can also be described as fast-paced intermittent contact sports. This would suggest that muscle damage would also be similar in these sports after match-play. This might not be the case as measured CK levels in professional ice hockey players after match-play are markedly lower than in Australian football and rugby players (338 ± 45 U/L in ice hockey vs. 691 ± 345 U/L in Australian football and 951 ± 392 U/L in rugby). (Gastin et al. 2019; Lignell et al. 2018; McLellan et al. 2011) Main reason for this is probably shorter duration of game-time and less eccentric loading during skating compared to running.

### 4.3 Hormonal responses to exercise

The endocrine system is an important modulator in athletic performance, fuel metabolism and skeletal muscle remodeling. Hormones are secreted to circulation from endocrine organs and they act by binding to specific receptors on the target cell's surface, cytoplasm, nucleus or mitochondrion, where after they stimulate or suppress intracellular signaling. During exercise hormones regulate mobilization of energy stores to metabolic fuels and also increase the fuel uptake and utilization by working muscles. After exercise hormones are then used to regulate the restoring of those used energy stores as well as the repairmen and remodeling of the skeletal muscles and other tissues involved in the exercise. These hormonal responses to

exercise then enhance performance and the body's ability to better endure the mechanical loading. (Farrell et al. 2012, 467)

### 4.3.1 Testosterone and exercise

Testosterone is often measured when hormonal responses to exercise are investigated. Testosterone is an anabolic steroid hormone and it is part of the androgen family. Secretion of testosterone is regulated by the hypothalamic-pituitary-gonadal axis and it is synthesized and secreted in the testes from Leydig cells in males and in females from the ovaries. Small amount of testosterone is also produced in the adrenal glands. Testosterone promotes anabolic status in muscle tissue by increasing muscle hypertrophy via upregulation of muscle protein synthesis and downregulation of muscle protein breakdown. Testosterone also exerts it's anabolic effects by increasing secretion of growth hormone. In circulation, testosterone is mostly bound to sex hormone-binding globulin and albumin (95-98%) and the remaining amount (2-5%) is unbound free testosterone. The unbound free testosterone is biologically available for target tissues and it is responsible for the anabolic effects of testosterone. (Papacosta & Nassis 2011)

Traditionally it has been thought that testosterone levels increase acutely after heavy resistance training (Kraemer & Ratamess 2005), whereas decreases are observed after endurance training (Urhausen et al. 1995). However, the results from resent meta-analysis indicates that the type of exercise is not as important for the acute testosterone response after exercise, but rather the intensity of the exercise. Intensity threshold of 60% of $VO_{2max}$ or 60% of 1RM was found to be needed to observe acute increases in testosterone concentrations. (D'Andrea et al. 2020) Quite a lot of research exists about acute responses in testosterone concentrations after matches in team sports, but there does not seem to be a consistent pattern in the responses. Some studies have reported increases (Gravina et al. 2011; McLellan et al. 2010), some decreases (Cormack et al. 2008; Cuniffe et al. 2010; Romagnoli et al. 2016; West et al. 2014) and some have reported no changes (Silva et al. 2013; Chatzinikolaou et al. 2014, Ispirlidis et al. 2008; Kreamer et al. 2008).

### 4.3.2 Cortisol and exercise

Cortisol is also a steroid hormone and it is a member of glucocorticoid family. It is secreted from adrenal cortex and it is regulated by hypothalamic-pituitary-adrenal axis. Cortisol secretion is increased under stressing conditions such as physical exercise. Chronically elevated cortisol levels are associated with depression, anxiety and physical training. Cortisol is characterized as a catabolic hormone as it decreases muscle protein synthesis, upregulates muscle protein breakdown and inhibits immune function and inflammatory processes. (Papacosta & Nassis 2011) By upregulating muscle protein breakdown, cortisol increases the amount of circulating amino acids which are then used in gluconeogenesis to produce glucose. This usually happens during increased energy expenditure. (Farrell et al. 2014, 484-485)

Exercise intensity and duration are associated with increased cortisol secretion. Intensity threshold of 60% of $VO_{2max}$ has been reported for exercise induced increases in cortisol secretion and beyond this threshold large elevations in cortisol concentration can be observed. (Hill et al. 2008; Maresh et al. 2006) As in other exercise, cortisol levels increase acutely after matches in intermittent team sports. For example, peak cortisol concentrations have been measured immediately after soccer, handball and rugby matches (Chatzinikolaou et al. 2014; Cuniffe et al. 2010; Elloumi et al. 2003; Ispirldis et al. 2008; McLellan et al. 2010; McLellan et al. 2011; Romagnoli et al. 2016). There is quite a lot of variation in the recovery times for cortisol after matches, as cortisol levels have been reported to decrease back to baseline values within 14 to 72 hours (Chatzinikolaou et al. 2014; Cuniffe et al. 2010; McLellan et al. 2011; McLellan et al. 2010; Ispirldis et al. 2008; Silva et al. 2013, West et al. 2014). Longer time periods (over 48 hours) seems to be needed for cortisol levels to return to baseline after rugby and soccer matches (Silva et al. 2013, McLellan et al. 2011; McLellan et al. 2010West et al. 2014).

### 4.3.3 Testosterone to cortisol -ratio

As testosterone represents anabolic and cortisol catabolic state of the body, it has been proposed to use testosterone/cortisol (T/C) -ratio as a measure of fatigue caused by training. Aldercreutz et al. (1986) proposed that over 30% decreases in T/C -ratio could be used as a diagnostic tool for detecting overtraining in athletes. Later this idea has been disputed as decreases in this ratio are not always accompanied by impaired performance. T/C -ratio can rather be used as an indicator of physical loading acutely after exercise. (Meeusen et al. 2013; Urhausen et al. 1995) Responses in T/C -ratio has been measured after rugby matches in few occasions. In these studies, it was found that T/C -ratio decreased significantly after match and recovered back to baseline values within 4 to 36 hours (Cunniffe et al. 2010; Elloumi et al. 2003; McLellan et al. 2010; West et al. 2014). In soccer recovery times seem to be longer for T/C -ratio as Silva et al. (2013) reported that 72 hours were needed for T/C to return back to baseline values. Even though there are some conflicting results about testosterone responses in team ball games, T/C -ratio might be one useful method for evaluating physical loading after matches, but conclusions should probably not be made solely based on this marker.

### 4.3.4 Dehydroepiandrosterone and dehydroepiandrosterone sulfate

Dehydroepiandrosterone (DHEA) is another androgenic steroid hormone besides testosterone, that has had some attention in exercise science literature. DHEA is produced in the adrenal gland and two different forms of it are found in circulation. The other is unconjugated form and the other is conjugated as its sulphate ester, dehydroepiandrosterone sulfate (DHEA-S). Much more DHEA-S can be found in circulation in comparison to DHEA, and it is the most abundant steroid hormone in circulation. In circulation, DHEA is mostly bind to albumin, with minimal binding to sex hormone binding globulin, whereas DHEA-S can only bind to albumin. DHEA-S can be considered as precursor for DHEA and DHEA further acts as a precursor for other stronger sex hormones. Many DHEA-metabolizing enzymes are located in peripheral tissues, where they can transform DHEA to testosterone, dihydrotestosterone and estrogens. (Corrigan 2002)

Not much research exists about acute DHEA-S responses to exercise, but according to the available literature DHEA-S seems to increase acutely after exercise bout. Tremblay et al. (2005) found that DHEA-S increased in a dose-response manner after steady-state exercise done at 55% of $VO_{2max}$, when the exercise duration was extended. Also, higher intensity endurance events have been found to increase DHEA-S, as increased concentrations have been found after marathon (Bonen & Keizer 1987), triathlon (Malarkey et al. 1993) and swimming (Velardo et al. 1991). Very high-intensity anaerobic exercise also seems to elevate DHEA-S concentrations, since Enea et al. (2009) found that 30-second maximal Wingate -test increased DHEA-S in untrained young women and also in trained women who used oral contraception. Studies examining DHEA-S responses after matches in team ball games are spare, but in one study done with international level female soccer players found, that during 3-day tournament at which total of six matches were played, DHEA-S was significantly elevated from baseline at the third day of the tournament (Aizawa et al. 2006).

### 4.4 Immune function after exercise

The function of the immune system is to protect body against infections. Physical strain from exercise can mediate changes in immune function mostly through nervous and endocrine systems. Heavy exercise causes so called “open window” of depressed immune function which can last several hours after the exercise. During this immunodepression, circulating numbers of several immune variables are decreased and this can lead to an invasion of microbial agents such as viruses and eventually cause infection and illness. If recovery time between training sessions is not long enough to ensure the recovery of the immune system, then the degree of immunodepression after the second training bout is even greater. (Peake et al. 2017)

The immune system can roughly be divided into innate and acquired immune defense. Innate defense can be described as the first line of immune defense and it comprises of physical and chemical barriers (e.g. skin and mucosal membrane) and phagocytes (e.g. neutrophils, monocytes, etc.). The function of the innate immune defense can be described as rapid and predictable. The acquired immune defense, on the other hand, is very specialized but its

functions are slower than innate immune systems. The acquired immune defense consists of lymphocytes which can be classified into T and B lymphocytes and natural killer cells. Even though immune system is divided into innate and acquired defenses, they work very much in integrated fashion to fight against pathogens. (Walsh 2018)

Single exercise session causes large increases in circulating white blood cells (leukocytes) and the levels are increased also during the recovery time. This increase in circulating leukocytes is called leukocytosis and it is in part mediated by the duration and intensity of the exercise. Also, high body temperature (environment, protective gear, etc.) might increase this exercise induced leukocytosis. The increase can be observed in almost all leukocyte subpopulations. However, during the recovery period there is an opposite reaction in circulating neutrophils and lymphocytes as the number of neutrophils continue to increase whereas lymphocytes start to decrease below baseline levels. The neutrophil levels after exercise are comparable to the levels during bacterial infections but they usually return to baseline within 24 hours. If the intensity and duration of exercise is particularly high, the decrease in lymphocytes might begin already during the exercise session. The lymphocyte levels are restored back to baseline levels quite quickly, usually within 4-6 hours after exercise. (Gleeson 2007; Rowbottom & Green 2000) These acute immune function responses to exercise are presented in figure 4.

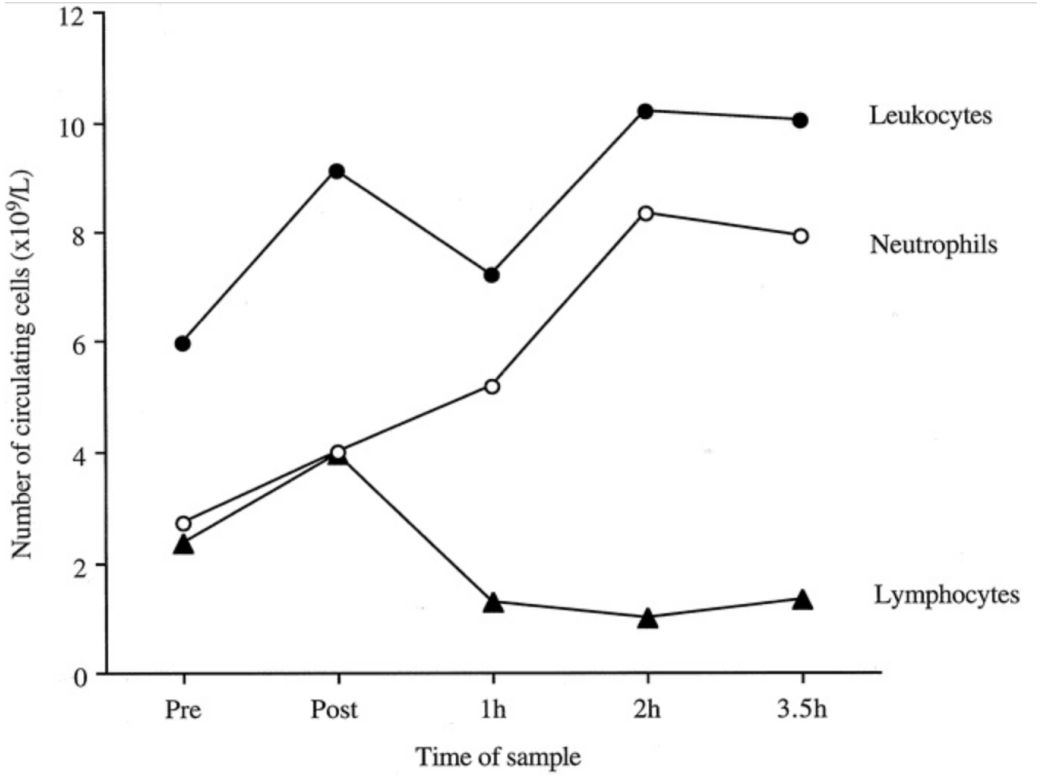



FIGURE 4. Acute immune function responses to exercise. (Rowbottom & Green 2000)

The immune responses to exercise are in part modulated by hormonal changes during exercise. There are two major neuroendocrine pathways, hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system, which are activated during exercise and lead to alterations in circulating immune cells. (Webster & Glaser 2008) In response to exercise, HPA axis is responsible for the release of glucocorticoids (cortisol) and the sympathetic nervous system is responsible for the release of catecholamines (mainly epinephrine and norepinephrine). Both cortisol and catecholamines then affect the number, function and activity of circulating immune cells. (Brenner et al. 1998) Typically, catecholamines are released early at the beginning of the exercise, whereas increases in cortisol typically happen after the exercise session. This early release of catecholamines results in an increase in circulating neutrophils and natural killer (NK) cells (Pyne 1994), whereas cortisol is responsible for suppression of NK- and T-cell function and decreased lymphocyte count which usually happens after the exercise (Cupps & Fauci 1982; Nieman 1994; Nieman 1997). Besides these neuroendocrine pathways, there are few other reasons that could be responsible

for changes in functions and numbers of circulating immune cells. For example, increased production of reactive oxygen species, exposure to airborne pathogens due to increased ventilation and increase in gut bacterial toxins in circulation could also lead to immunodepression after exercise. (Gleeson 2007)

Exercise of high intensity or long duration have also been found to decrease mucosal immunoglobulin A (IgA) secretion. IgA is secreted from cells in the mucosal lymphoid tissue and it is characterized as an important part of the first line of defense against pathogens. (Rowbottom & Green 2000) IgA is a glue-like substance that has an antibody activity against viruses, bacteria and common allergens. Early research has demonstrated that salivary IgA is decreased by over 50% after intensive and long duration cross country skiing (Tomasi et al. 1982) and bicycling (Mackinnon et al. 1987). Also, high intensity interval training has been shown to decrease salivary IgA in relation to the intensity of the exercise (Mackinnin et al. 1993). However, it seems that relatively long durations and high intensities are needed to cause decreases in salivary IgA as exercise done at 50-80% of $VO_{2max}$ for 15 to 90 minutes does not seem to cause alterations in salivary IgA (Mackinnon & Hooper 1994; McDowell et al. 1991).

When acute responses in immune function has been studied after match-play in team ball games, the responses seem to follow a quite predictable pattern. Immediately after soccer match, leukocytes increase significantly and return to baseline usually within 24 hours (Andersson et al. 2010; Fatouros et al. 2010; Gravina et al. 2011; Ispirlidis et al. 2008; Romagnoli et al. 2016). Also, two studies have found significant decreases in lymphocyte counts immediately after soccer matches (Cunniffe et al. 2010; Gravina et al. 2011). Acute responses in salivary IgA concentrations have been measured after Australian rules football. Coad et al. (2015) measured salivary IgA after three different pre-season matches and they found, that when the overall external load remained constant during the first two matches, no changes in salivary IgA were found. However, after the third match the external load was significantly greater than at the previous matches and this increase in external load was accompanied with significant decreases in salivary IgA. After the third match, it took 36 hours for the salivary IgA to return to baseline values. The authors concluded that the observed decrease in mucosal immune function is probably a result of the combination of

three consecutive matches and the acute increase in external match load in the last match. (Coad et al. 2015)

To summarize these results, it seems that match-play in high intensity intermittent team ball games causes acute immunodepression which may last up to 36 hours. This decreased immune function seems to be dependent on the intensity and duration of the match-load. During this time of impaired immune function after matches, the players may be more vulnerable to upper respiratory tract infections (URTI). In fact, it has been found, that during 24-week long ice hockey season, the players had more symptoms of URTI when their blood leukocyte count was higher and blood lymphocyte count and salivary IgA concentrations were lower. (Orysiak et al. 2016)

# 5 PURPOSE OF THE STUDY, RESEARCH QUESTIONS AND HYPOTHESES

The purpose of the study was to evaluate the acute neuromuscular and physiological responses to official ice hockey match-play and follow the recovery for 12 hours in Finnish elite league ice hockey players. The study concentrated on changes in neuromuscular performance, hormonal and immunological status and biochemical markers of muscle damage and their association with external loading during the match. To authors knowledge this is the first study to measure acute changes in these parameters after official ice hockey match-play.

The research questions and hypotheses are following:

1. Does official ice hockey match-play impair neuromuscular performance immediately and 12 hours after the match and is the impairment associated with greater external load?

   H1: Neuromuscular performance is impaired acutely and remains below baseline values 12 hours after the match. In other intermittent team ball games such as football, handball and rugby, neuromuscular performance is usually impaired for approximately 48 hours after match-play (Chatzinikolaou et al. 2014; Fatouros et al. 2010; Silva et al. 2013; Twist & Sykes 2011). Greater external loading during the match is associated with declined neuromuscular performance, as largest decreases in neuromuscular performance are typically observed after soccer matches where external loads are greater than in other team sports (Doeven et al. 2018).

2. Does official ice hockey match-play cause muscle damage, measured as serum CK, immediately and 12 hours after the match and are the increases in serum CK associated with external load?

   H2: Ice hockey match-play causes muscle damage as serum CK is increased immediately after match and is further increased at 12 hours post-match. When CK

concentrations in blood have been measured in different intermittent team ball games, it has been found that the CK concentrations starts to increase immediately after match-play and continues to elevate for several hours and peak values are usually reached at 24 hours post-match. (Chatzinikolaou et al. 2014; Djaoui et al. 2016; McLellan et al. 2010; Nedelec et al. 2014; Romagnoli et al. 2016; Russell at al. 2016; Silva et al. 2013; Twist & Sykes 2011) Increases in CK concentration are associated with external load during the match as has been observed in Australian football players after match-play (Gastin et al. 2019).

3. Does official ice hockey match-play effect anabolic and catabolic hormonal status measured as T, C, DHEA-S and T/C -ratio immediately and 12 hours after the match and are these changes associated with external load?

   H3: Catabolic hormonal activity is increased after the match as T:C ratio decreases immediately after the match and remains below baseline values at 12 hours post-match. In soccer and rugby, T:C ratio is decreased after match-play and returns to baseline values within 4 to 72 hours (Cunniffe et al. 2010; Elloumi et al. 2003; McLellan et al. 2010; Silva et al. 2013; West et al. 2014). C is increased immediately after the match and remains elevated after 12 hours and increases are associated with external load (Chatzinikolaou et al. 2014; Cuniffe et al. 2010; Elloumi et al. 2003; Ispirldis et al. 2008; McLellan et al. 2010; McLellan et al. 2011; Romagnoli et al. 2016) T and DHEA-S are elevated immediately after the match in relation to external load (Aizawa et al. 2006, D'andrea et al. 2020; Tremblay et al. 2005).

4. Does official ice hockey match-play effect immune function, especially circulating leukocyte and lymphocyte counts and salivary IgA, immediately and 12 hours after the match and are these changes associated with external load?

   H4: Blood leukocytes are increased immediately after the match and remain elevated after 12 hours (Andersson et al. 2010; Fatouros et al. 2010; Gravina et al. 2011; Ispirlidis et al. 2008; Romagnoli et al. 2016). Blood lymphocytes are elevated

immediately after the match and returned to baseline by 12 hours post-match (Gleeson 2007; Rowbottom & Green 2000). Salivary IgA is decreased after the match in relation to external load (Mackinnon et al. 1987; Mackinnon et al. 1993; Tomasi et al. 1982).

# 6 METHODS

## 6.1 Subjects

The study subjects were 38 Finnish elite league ice hockey players from two different teams. The subjects represented all outfield positions and goaltenders were not included. The characteristics of the subjects are represented in table 1. Before volunteering to the study, the players were informed about the purpose, benefits and procedures associated in the study. Written informed consent was obtained from all the players before they participated in the measurements. The study was approved by the Ethical Committee of The Central Finland Health Care District.

TABLE 1. Descriptive information of the subjects.

| Age (years) | Height (cm) | Body mass (kg) | Body fat (%) | Fat free mass (kg) | Experience in elite league (years) |
|---|---|---|---|---|---|
| 26.3 ± 4.8 | 182.1 ± 6.3 | 85.9 ± 6.9 | 14.5 ± 2.9 | 73.3 ± 5.5 | 4.7 ± 5.1 |

## 6.2 Study design

The study was done in single group repeated measures design, where measurements were done before, during, immediately and 12 hours after official Finnish elite league ice hockey match. Premeasurements were done at the morning of the game day at 9:00 a.m. (post-9h). At the premeasurements blood samples for leukocytes, lymphocytes and CK and saliva samples for T, C, DHEA-S and IgA were collected. After the sample collection, the players did 20-minute controlled warmup before performing CMJ with maximal effort. Saliva samples for T, C and DHEA-S were also collected at 4:00 p.m. (post-2.5h) before the warmup for the official match because of the circadian rhythm of the hormones. The game started at 6:30 p.m. and during the match, physical activity data was collected with local positioning system (LPS) and the players' heart rates were monitored. Immediately after the match at 9:00 p.m. the players

performed CMJ and blood and saliva samples were collected (post-0h). At the morning of the following day at 9:00 a.m. the same measurements were repeated as at the morning of the match day (post-12h).

## 6.3 Measurements

### 6.3.1 Anthropometry

The subjects were measured for height, body mass and bodyfat percentage (BF%) before the start of the official season. The players paid a visit to the laboratory between 3 to 7 weeks before the match measurements took place. During the measurements, players were wearing underwear and they were advised to empty their bladder and not to eat a meal 4 hours prior to the measurements. Height was measured using manual tape and body mass, fat free mass and BF% estimation was measured using multiple-frequency bioelectrical impedance device (Tanita MC-780 MA, Seoul, South Korea). The impedance values were used in equation provided by the manufacturer to estimate body composition.

### 6.3.2 Countermovement jump

A force plate (ForcePlatform FP8, HUR, Finland) was used to measure flight times, take-off times, takeoff velocities, jump heights and maximum power in countermovement jumps. CMJ heights were calculated using takeoff velocities. Flight times and take-off times were used for flight time/take-off time -ratio (FT/TT) for more sensitive estimation of neuromuscular function. The performance of the CMJ started from upright position, from which the subjects did fast countermovement by flexing hip, knee and ankle joints to preferred depth to reach maximal jump height. Immediately after CMJ the subjects forcefully extended the hip, knee and ankle joints to jump vertically off the ground. During the flight the legs had to remain straight under the hips and bending of the knees was not allowed during landing. Hands had to remain on the hips during the entire movement. The jumps were supervised by experienced testing personnel and the jumps that did not meet the instructions were excluded. Three attempts were allowed for each subject and the best result was included in the analysis.

### 6.3.3 Blood samples

Blood samples were collected in seated position from the antecubital vein with venipuncture. Blood was collected into two tubes from which other was containing ethylenediaminetetraacetic acid (EDTA) for measuring leukocytes and lymphocytes from whole blood samples and other was used for measuring CK from serum samples. To separate serum, the blood was allowed to clot for 30 minutes after which it was centrifuged for 15 minutes with 3600 rpm. Whole blood samples were analyzed within 24 hours using automated Sysmex XP 300 analyzer (Sysmex, Kobe, Japan). Serum samples were stored at -80 °C until measured. Serum CK concentration was assessed using colorimetric analysis with Konelab XTi20 device (Thermo, Vantaa, Finland).

### 6.3.4 Saliva samples

Saliva samples were collected via cotton swabs (Salivette®, Sarstedt, Nümbrecht, Germany). The subjects were advised not to eat food or drink fluids other than water, wash their teeth or use tobacco products during one hour before sample collection. During sample collection subjects were instructed to pour the swabs from collecting tubes into their mouths without touching the swabs with their hands. The subjects were instructed to chew the swab for at least 1 minute to stimulate salvation. Skin contact was avoided when the swab was removed back to the collecting tubes. The samples were then centrifuged for 3 minutes at 1000 x g and stored at -80 °C until analyzed. Saliva testosterone and DHEA-S concentrations were analyzed with enzyme-linked immunosorbent assays (Testosterone Saliva ELISA and DHEA-S Saliva ELISA, IBL, Hamburg, Germany). Saliva cortisol was assessed via ECLIA (Immulite 2000, Siemens, Llanberis, UK) and IgA via spectrofotometric method (Konelab XTi20, Thermo, Vantaa, Finland).

### 6.3.5 Heart rate monitoring

Heart rate of the players was measured using heart rate belt. Both teams used their own heart rate monitoring systems which were Firstbeat Sports (Firstbeat Technologies Oy, Jyväskylä, Finland) and Polar Team Pro (Polar, Kempele, Finland). Raw data from Polar Team Pro monitoring system was exported to Firstbeat Sports software which was used to analyze data from both teams. Heart rate was measured during the whole match and values were reported as beats per minute and also as a percentage with respect to maximal heart rate. The used maximal heart rates of the players were the highest heart rates that were obtained during $VO_{2max}$ test or during training sessions.

### 6.3.6 Match analysis

The external loading and the performance of the players during the match was measured using real-time local positioning system that is based on Angle-of-Arrival signal processing method (collection frequency of 25 Hz and latency 100 ms, Quuppa Intelligent Locating System$^{TM}$). The system uses 16 antennas that are fixed on the roof of the ice hall and the antennas capture the radio signal transmited by the tags (figure 5) installed in the players jerseys. Analysis of the location is based on the angle of the radio signal (figure 6). The radio signal is sent from the tags to the antennas by using Bluetooth Low Energy -technology (BLE, Bluetooth 4.0 or Bluetooth Smart). The Quuppa antennas then send the raw data to a server that uses software program by Bitwise to calculate the tag position. The algorithms by Bitwise was then used to calculate total skating distance, average skating speed, maximal skating speed and the time spent, and distance covered at different intensity zones during the match. This method has been proven to be accurate and reliable for measuring players movements in team ball games played indoors (Figueira et al. 2018).


FIGURE 5. Quuppa Intelligent Locating System™ tags that were installed in the player's jerseys.

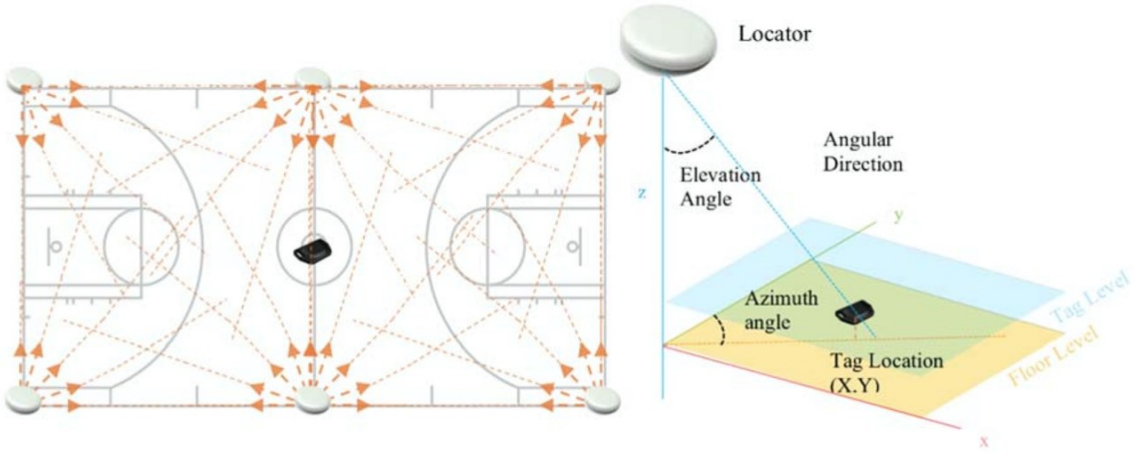



FIGURE 6. Antennas measure the tag location based on the Angle-of-Arrival signal processing method (Figueira et al. 2018).

The intensity zones were chosen according to the data collected by the software program provider (Bitwise). The velocity thresholds for the chosen intensity zones were based on the data collected by Bitwise from multiple official Finnish elite league ice hockey matches and

to the recommendation by Sweeting et al. (2017) and Malone et al. (2017). According to the recommendations by Sweeting et al. (2017), evenly wide velocity cluster were chosen to determine the intensity zones. Total of six skating intensity zones, each 5 km/h wide, were used. By the same way as in the study by Lignell et al. (2018), the time and distance covered at zones 1-3 were combined to represent low intensity skating, whereas zones 4-6 were combined to represent high intensity skating. The categorization of the intensity zones is illustrated in table 2.

TABLE 2. Categories for different intensity zones.

| **Zone** | **Descriptor** | | **Threshold** | |
|---|---|---|---|---|
| | | | **(km/h)** | **(m/s)** |
| **1** | Very low-speed skating | **Low intensity skating** | 0 - < 5 | 0 - < 1.39 |
| **2** | Slow-speed skating | | ≥ 5 - < 10 | ≥ 1.39 – < 2.78 |
| **3** | Moderate-speed skating | | ≥ 10 - < 15 | ≥ 2.78 - < 4.17 |
| **4** | High-speed skating | **High intensity skating** | ≥ 15 - < 20 | ≥ 4.17 - < 5.56 |
| **5** | Very high-speed skating | | ≥ 20 - < 25 | ≥ 5.56 - < 6.94 |
| **6** | Sprint skating | | ≥ 25 | ≥ 6.94 |

### 6.3.7 Questionnaires

The players were asked to subjectively rate their perceived exertion, recovery and muscle soreness at different timepoints during the study. The perceived recovery was asked on the morning of the matchday and on the morning after the match. The perceived recovery was rated using CR-10 RPE scale where higher values indicated better recovery. At the same timepoints the players were also asked to rate their perceived muscle soreness in the lower limbs by using visual analog scale (VAS). The players were asked to mark a spot on a 100mm line where the extremes on left side indicated “no muscle soreness at all” and right side indicated “the worst imaginable muscle soreness due to physical exercise”. Immediately after the match players were asked to rate their perceived exertion using CR-10 RPE scale, where

higher values indicated greater strain during the match. The perceived exertion was further used to calculate sRPE according to recommendations by Foster et al. (2001). Total active playing time where intermissions, breaks and recovery time between the shifts were excluded, was used as the exercise duration in sRPE calculation.

## 6.4 Statistical analyzes

The sample distribution was assessed using Shapiro-Wilk test. For the parametric variables the comparisons between the means at different time points were done using repeated measures ANOVA. For non-parametric variables related-samples Freidman's two-way analysis of variance and Wilcoxon signed rank test were used. The correlations between changes in physiological variables and external loading during the match were analyzed using Pearson correlation coefficient. The results are presented in the text and tables as mean ± SD. The criterion level for statistical significance was set at $p \leq 0.05$. Statistical significance is illustrated in the tables and figures by using star symbols (*** = $p<0.001$, ** = $p<0.01$, * = $p<0.05$). All statistical analyses were done using Statistical Package for the Social Sciences (SPSS for macOS, version 26, IBM, Chicago, IL, USA).

# 7 RESULTS

## 7.1 Match characteristics

Total playing time during the match was 15.5 ± 2.7 min (9.4-21.8 min) and the total skating distance was 3650 ± 107 m (2088-5022 m). The mean average and maximal skating speeds during the whole match were 14.1 ± 0.2 km/h (12.1-16.8 km/h) and 31.8 ± 0.3 km/h (28.4-35.7 km/h), respectively. The mean average amount of low and high intensity skating during the match were 1062 ± 256 m and 2202 ± 506 m, respectively. The measured external loads for each period and for the whole match are presented in table 3.

Skating distance decreased by 8% in the third period compared to the first period. Also, the average skating speed in the third period was 6% slower than in the first period and 7% slower than in the second period. At the same time, the time spent at low intensity skating during the third period was 9% longer than in the first and second periods. Also, the covered distance at low intensity skating was 6% greater in the third period than in the second period.

TABLE 3. Mean ± SD external loads for the first, second and third periods and for the whole match. a=p<0.05 difference between 1st and 3rd periods, b=p<0.05 difference between 2nd and 3rd periods.

| | 1st period | 2nd period | 3rd period | Whole match |
|---|---|---|---|---|
| **Skating distance (m)** | 1251 ± 266 | 1264 ± 241 | 1154 ± 280**[a] | 3650 ± 657 |
| **Playing time (min)** | 5.2 ± 1.0 | 5.2 ± 1.1 | 5.1 ± 1.2 | 15.5 ± 2.7 |
| **Skating distance (m/min)** | 240 ± 17 | 241 ± 24 | 227 ± 26**[ab] | 235 ± 19 |
| **Average speed (km/h)** | 14.4 ± 1.0 | 14.5 ± 1.5 | 13.5 ± 1.6***[ab] | 14.1 ± 1.1 |
| **Maximal speed (km/h)** | 30.3 ± 2.0 | 30.5 ± 1.9 | 30.4 ± 2.3 | 31.8 ± 1.6 |
| **Time at low-intensity skating (min)** | 2.3 ± 0.6 | 2.3 ± 0.8 | 2.5 ± 0.8**[ab] | 7.1 ± 1.7 |
| **Time at high-intensity skating (min)** | 2.3 ± 0.6 | 2.2 ± 0.6 | 2.0 ± 0.7 | 6.5 ± 1.4 |
| **Distance at low-intensity skating (m)** | 354 ± 112 | 344 ± 111 | 364 ± 124***[b] | 1062 ± 256 |
| **Distance at high-intensity skating (m)** | 761 ± 200 | 755 ± 208 | 685 ± 219 | 2202 ± 506 |

The average RPE after the match was 6 ± 2 and sRPE was 91 ± 28. There were weak correlations between sRPE and total time spent at high intensity skating (figure 6), total distance covered at high intensity skating (figure 7) and with total skating distance during the match (figure 8).

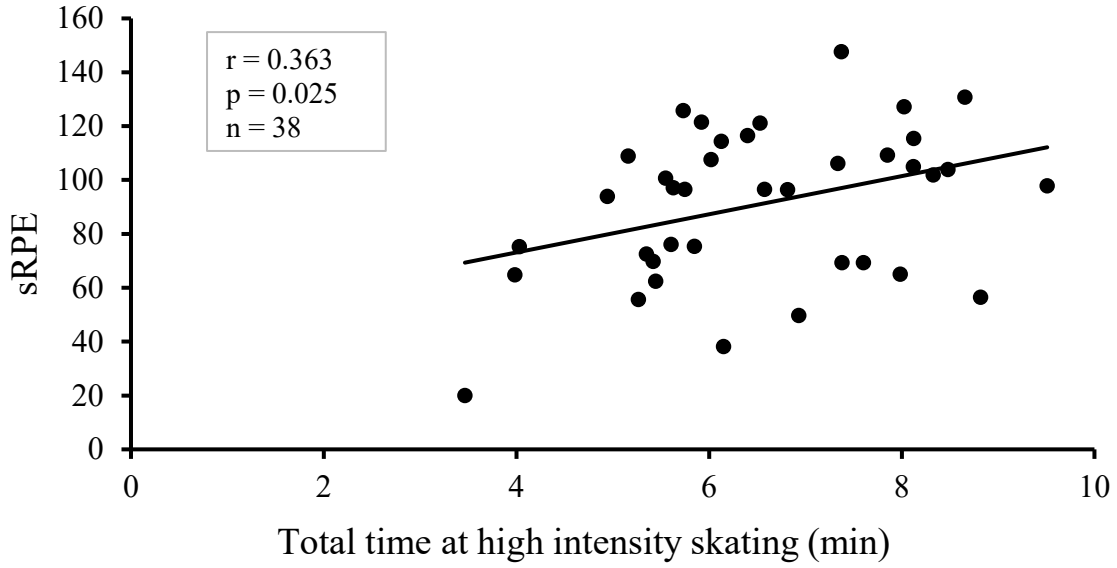



FIGURE 7. Correlations between sRPE and total time at high intensity skating during the match.

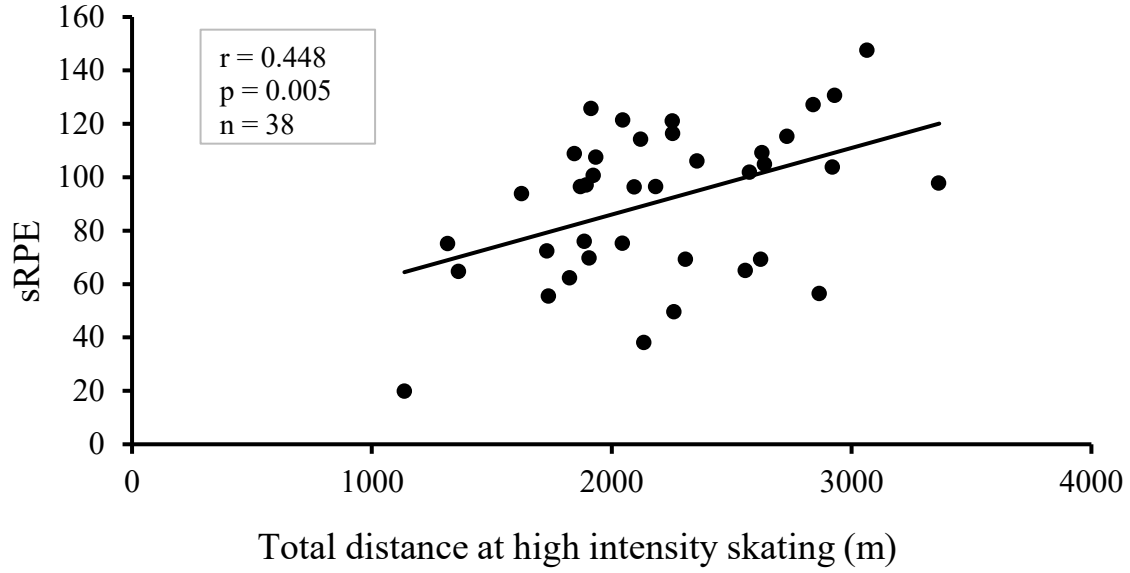



FIGURE 8. Correlations between sRPE and total distance at high intensity skating during the match.

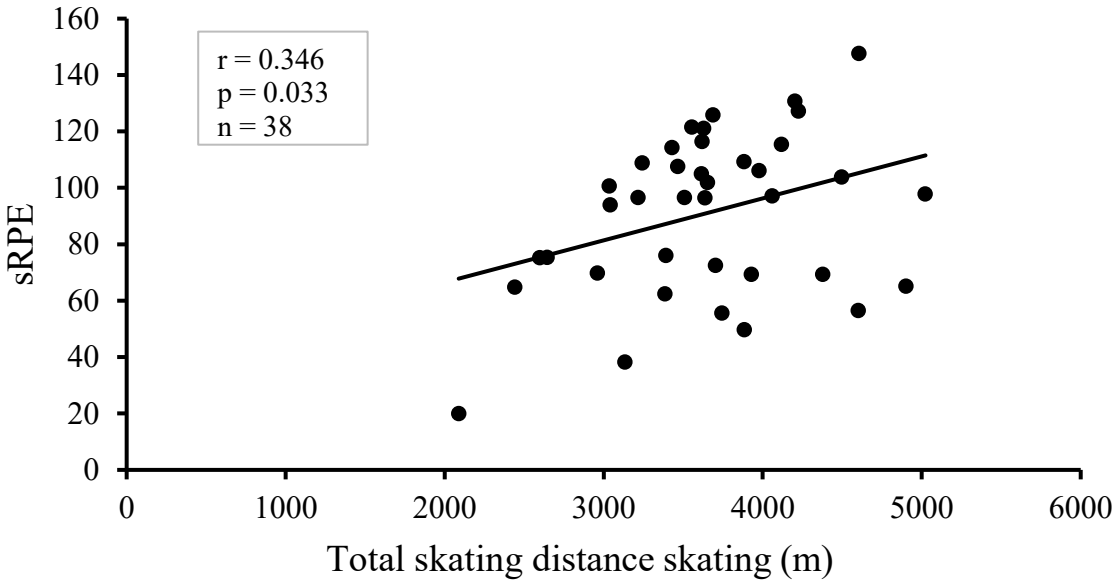



FIGURE 9. Correlations between sRPE and total skating distance during the match.

### 7.2 Heart rate responses

The average heart rate during the whole match was 137 ± 9 bpm, which represented 71 ± 3% of the players maximum heart rate. Peak heart rates were reached during both the first and

second periods. Minimum heart rates during the second period were 7% higher than in the first period and 4% higher than in the third period. The average heart rate was 4% lower during the third the third period in comparison to the second period. Also, maximum heart rates were 2% lower during the third period compared to the first and second periods. (Table 4)

TABLE 4. Mean ± SD heart rate responses to first, second and third periods and average heart rates during the whole match. a=p<0.05 difference between 1st and 3rd periods, b=p<0.05 difference between 2nd and 3rd periods, c=p<0.05 difference between 1st and 2nd periods.

| | **1st period** | **2nd period** | **3rd period** | **Whole match** |
|---|---|---|---|---|
| **HR min (bpm)** | 102 ± 12 | 109 ± 11***c | 105 ± 9*b | |
| **HR min %** | 53 ± 5 | 56 ± 5***c | 55 ± 4*b | |
| **HR average (bpm)** | 137 ± 11 | 140 ± 11 | 135 ± 9***b | 137 ± 9 |
| **HR average %** | 71 ± 5 | 73 ± 4 | 70 ± 4**a | 71 ± 3 |
| **HR max (bpm)** | 187 ± 8 | 187 ± 8 | 184 ± 7***ab | |
| **HR max %** | 97 ± 2 | 97 ± 2 | 96 ± 2***ab | |

### 7.3 Responses in neuromuscular performance

There was no significant difference in the CMJ height immediately after the match when compared to pre-match values. Also, there was no significant difference in the CMJ height between pre 9h and post 12h measurements. However, the CMJ height was 2% lower 12h post-match compared to post 0h measurements. CMJ heights are illustrated in figure 9. Maximum power in CMJ was 4% greater immediately after the match when compared to the baseline values. Maximum powers in CMJ are presented in figure 10.

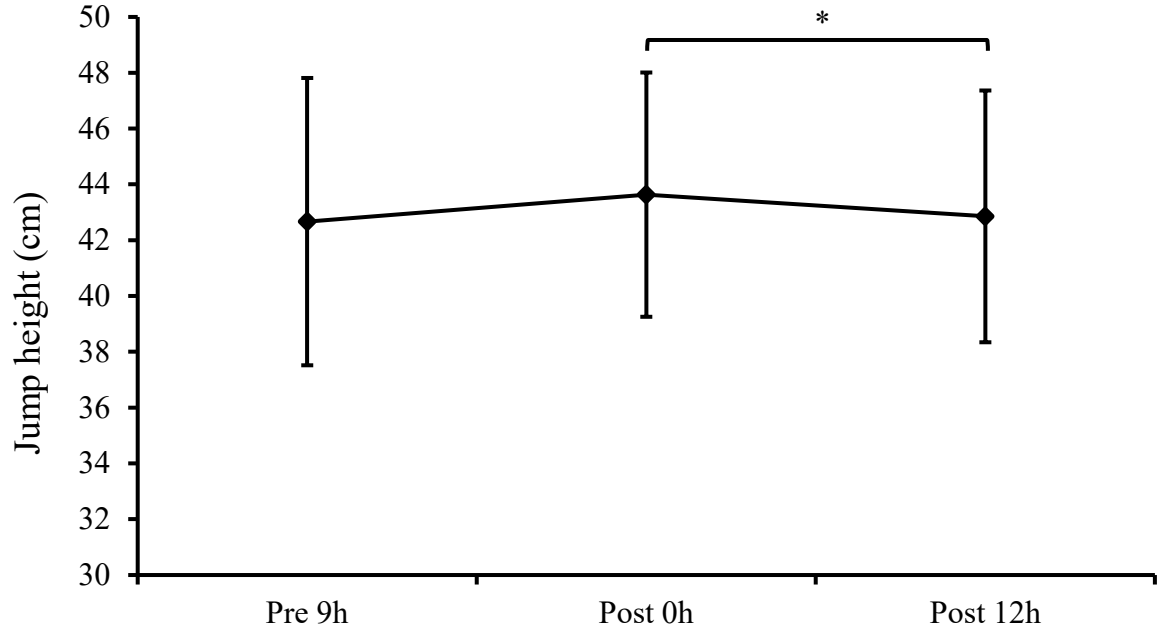



FIGURE 10. Mean ± SD CMJ height at pre 9h, post 0h and post 12h.

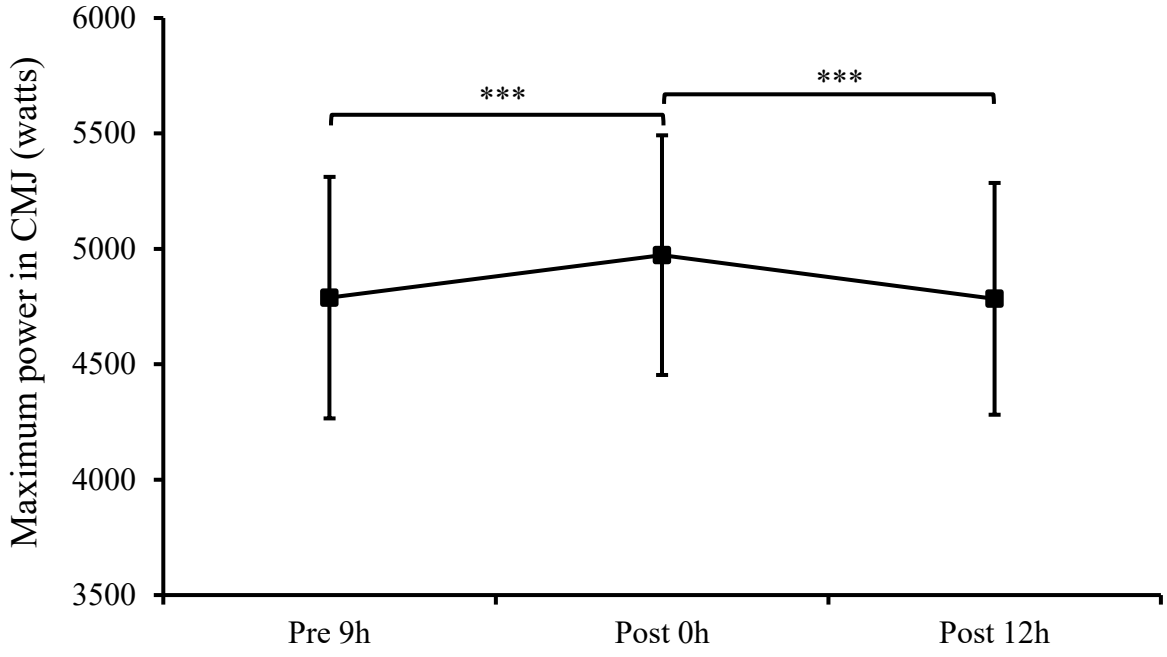



FIGURE 11. Mean ± SD maximum power in CMJ in watts at pre 9h, post 0h and post 12h.

Flight times, take-off times and flight time/take-off time-ratios in CMJ are presented in table 5. Flight times increased by 2% immediately after the match and by post 12h the flight times

had returned back to baseline levels. Flight time to take-off time ratio increased by 3% at post 0h and remained 3% above baseline values at post 12h.

TABLE 5. Mean ± SD flight times, take-off times and flight time/take-off time ratios in CMJ at pre 9h, post 0h and post 12h measurements. a=p<0.05 difference between pre 9h and post 0h, b=p<0.05 difference between pre 9h and post 12h.

| | **Pre 9h** | **Post 0h** | **Post 12h** |
|---|---|---|---|
| **Flight time (ms)** | 594 ± 33 | 607 ± 28**[a] | 598 ± 30 |
| **Take-off time (ms)** | 799 ± 71 | 795 ± 80 | 781 ± 66 |
| **Flight time/take-off time ratio** | 0.75 ± 0.08 | 0.77 ± 0.09**[a] | 0.77 ± 0.08*[b] |

There was a weak, but statistically significant correlation (r = 0.338, p = 0.047) between total playing time during the match and change in the CMJ height from baseline to post 0h. No other correlations were found between external loading during the match and changes in neuromuscular performance after the match.

### 7.4 Responses in markers of muscle damage

Serum CK concentrations increased by 22% immediately and by 39% 12 hours after match compared to pre-match values. Serum CK concentrations at different timepoints are presented in figure 11.

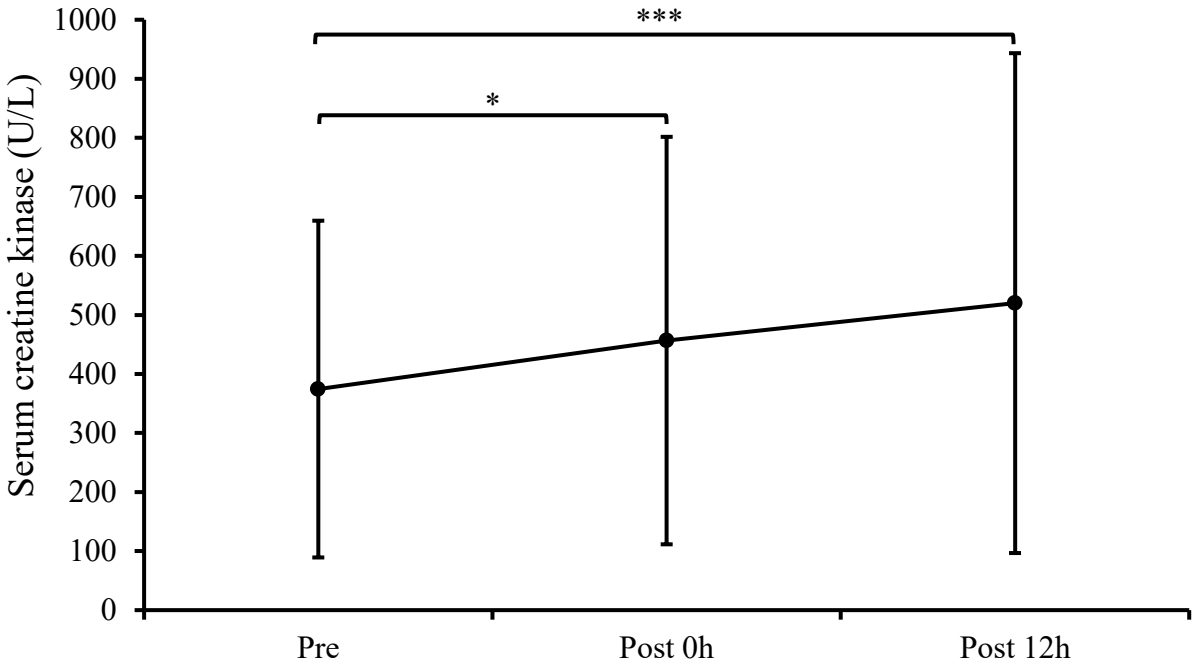


FIGURE 12. Mean ± SD serum creatine kinase concentrations at pre 9h, post 0h and post 12h.

Subjective muscle soreness measured by VAS was significantly ($p<0.001$) greater at the morning after the match compared to the morning of the matchday. At the morning of the matchday VAS was 22.5 ± 15.6 mm and at the following morning VAS was 42.0 ± 21.2 mm.

No significant correlations were found between changes in markers of muscle damage after the match and external loads during the match or with sRPE. Also, there was no statistically significant correlation between changes in CK and changes in VAS after the match.

### 7.5 Hormonal responses

Due to the circadian rhythm of the measured hormones, comparisons were done only between pre 9h and post 12h measurements and pre 2.5h and post 0h measurements. There was a 22% reduction in salivary testosterone immediately after the match when compared to pre 2.5h values. There was also a trend for decreased salivary testosterone at post 12h in comparison to the pre 9h measurements, but the difference was not statistically significant ($p = 0.06$). Salivary testosterone concentrations are presented in figure 12.

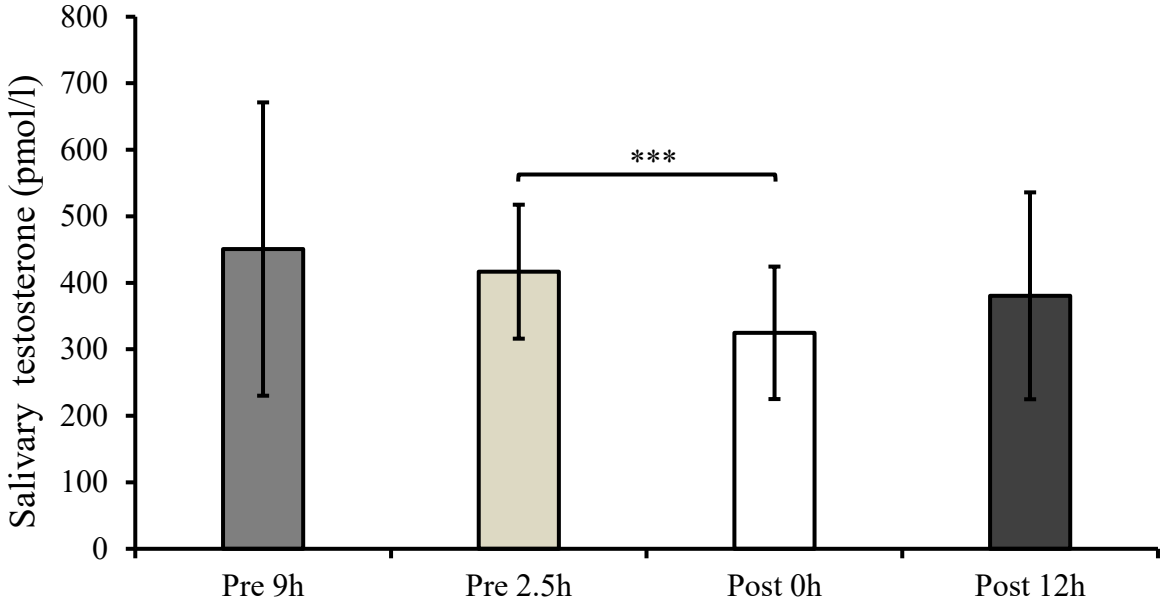



FIGURE 13. Mean ± SD salivary testosterone concentrations at pre 9h, pre 2.5h, post 0h and post 12h measurements.

In salivary cortisol there was no significant differences between pre 9h and post 12h measures or between pre 2.5h and post 0h measures. The salivary cortisol concentrations are presented in figure 13.

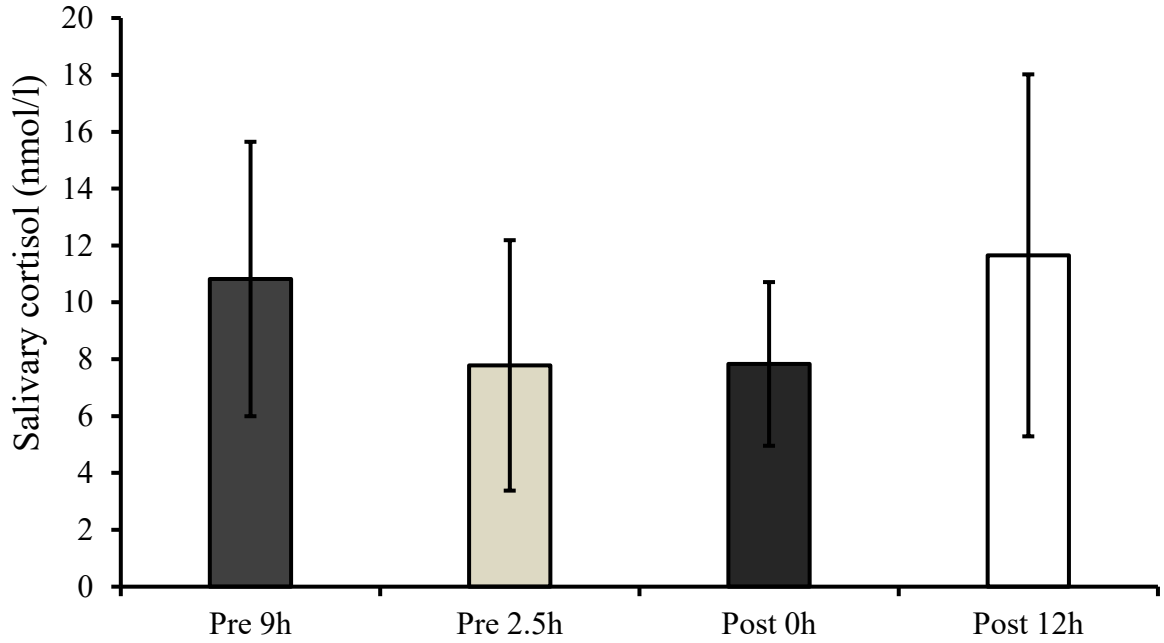



FIGURE 14. Mean ± SD salivary cortisol concentrations at pre 9h, pre 2.5h, post 0h and post 12h measurements.

There was a 42% reduction in T/C-ratio immediately after the match compared to pre 2.5h values. T/C-ratios at different timepoints are presented in figure 14.

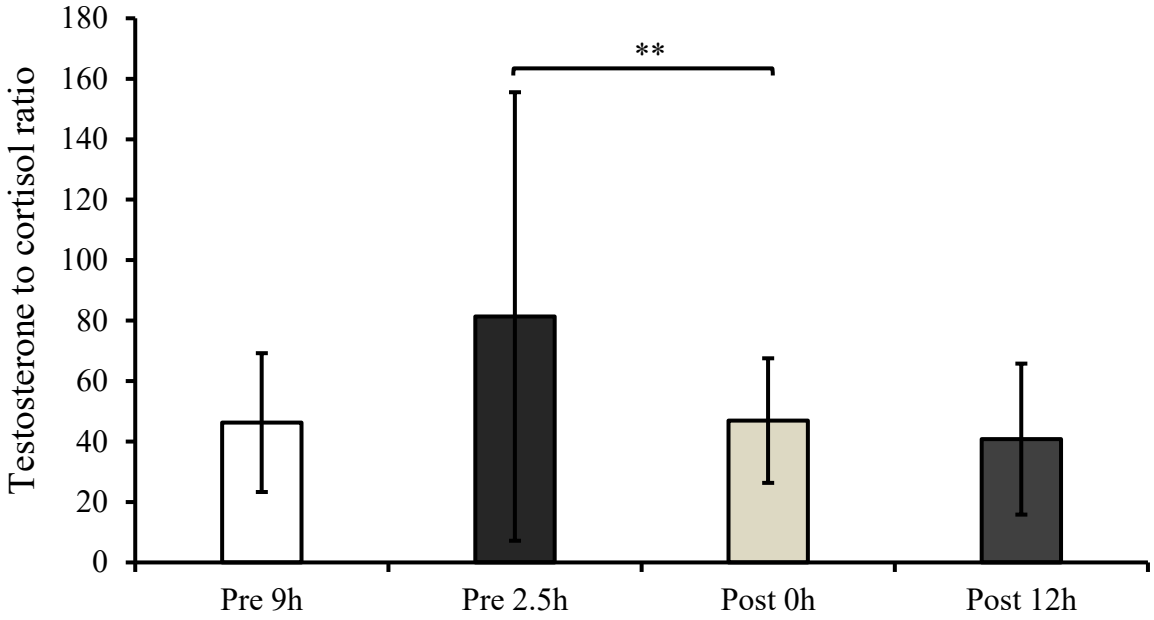



FIGURE 15. Mean ± SD testosterone to cortisol ratios at pre 9h, pre 2.5h, post 0h and post 12h measurements.

Salivary DHEA-S increased by 28% immediately after the match compared to pre 2.5h values. There was a trend for decreased salivary DHEA-S concentration in the morning samples, but the difference was not statistically significant ($p = 0.051$). Salivary DHEA-S concentrations are presented in figure 15.

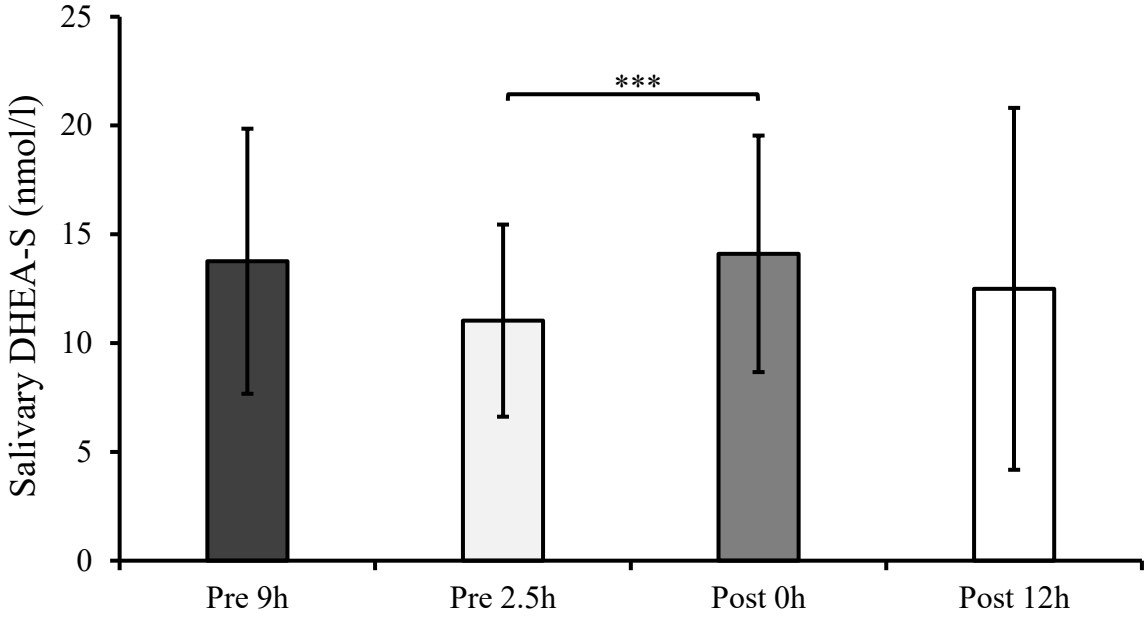


FIGURE 16. Mean ± SD salivary DHEA-S concentrations at pre 9h, pre 2.5h, post 0h and post 12h measurements.

There was a moderate correlation between match sRPE and acute change in salivary testosterone (figure 16), and weak correlations between sRPE and acute change in DHEA-S (figure 17), change in cortisol between morning samples (figure 18) and change in DHEA-S between morning samples ($r = 0.359$, $p = 0.029$). Total skating distance during the match had a weak correlation with change in DHEA-S between morning samples ($r = 0.363$, $p = 0.027$). Skating distance/playing time -ratio correlated moderately with the change in salivary testosterone between morning samples (figure 19) and weakly with the change in DHEA-S between morning samples (figure 20). Also, average skating speed during the match correlated moderately with changes in salivary testosterone between morning samples (figure 21) and weakly with the change in DHEA-S between morning samples (figure 22). Total playing time during the match did not correlate statistically significantly with any changes in the measured hormones.

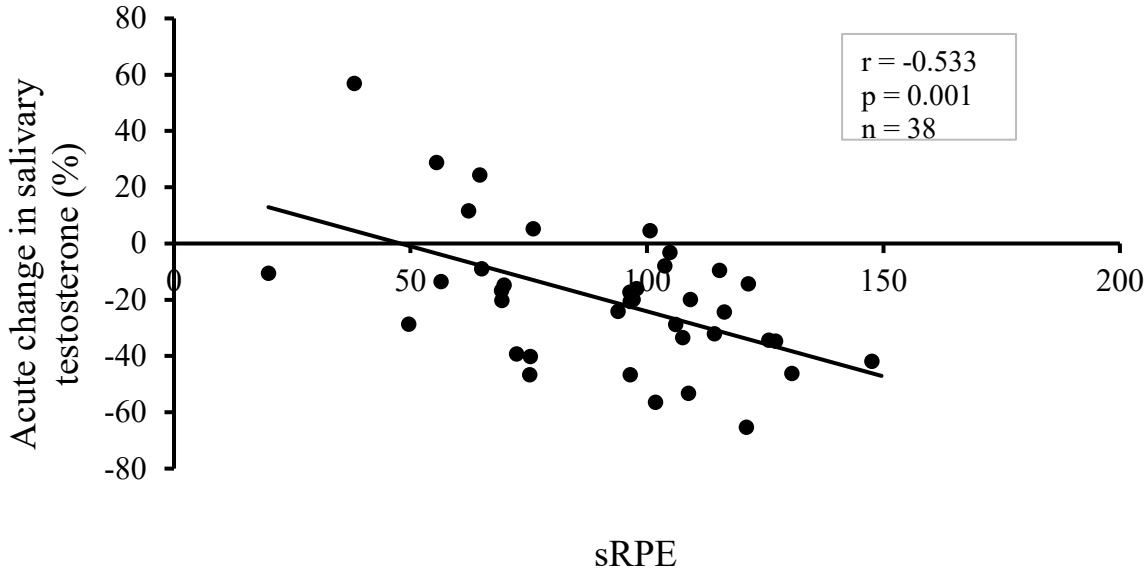



FIGURE 17. Correlation between acute change in salivary testosterone after the match and sRPE.

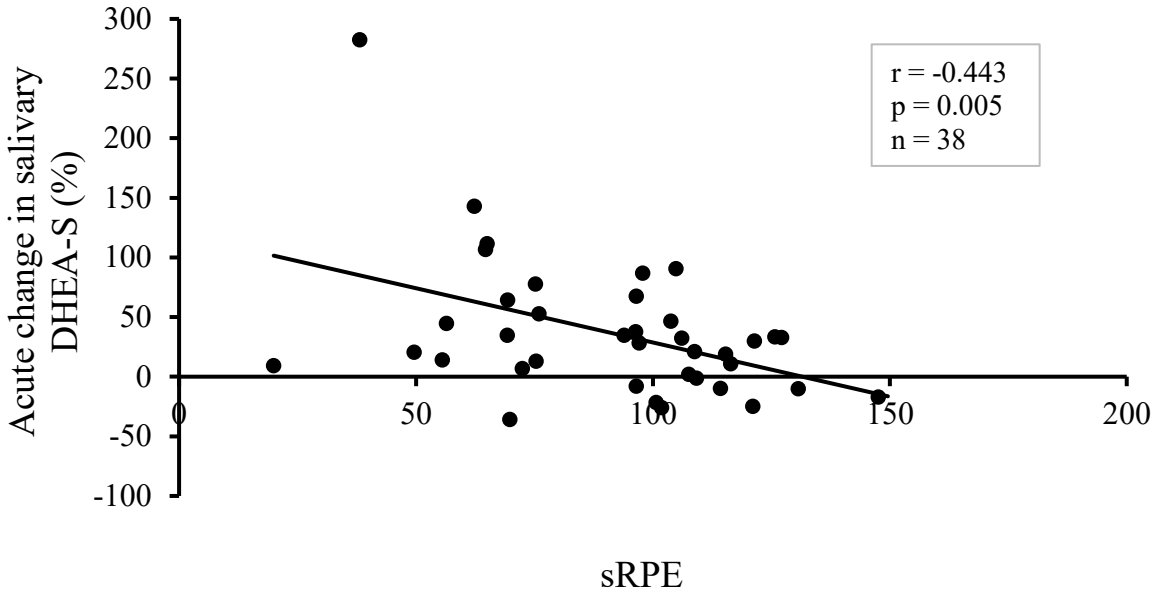



FIGURE 18. Correlation between acute change in salivary DHEA-S after the match and sRPE.

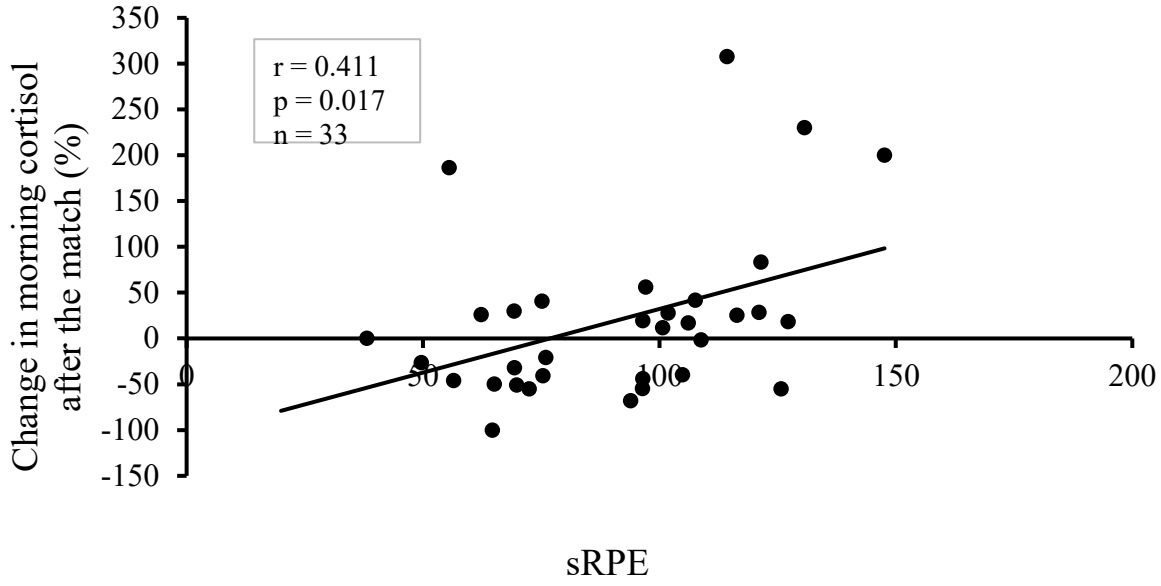



FIGURE 19. Correlations between change in cortisol between morning samples and sRPE.

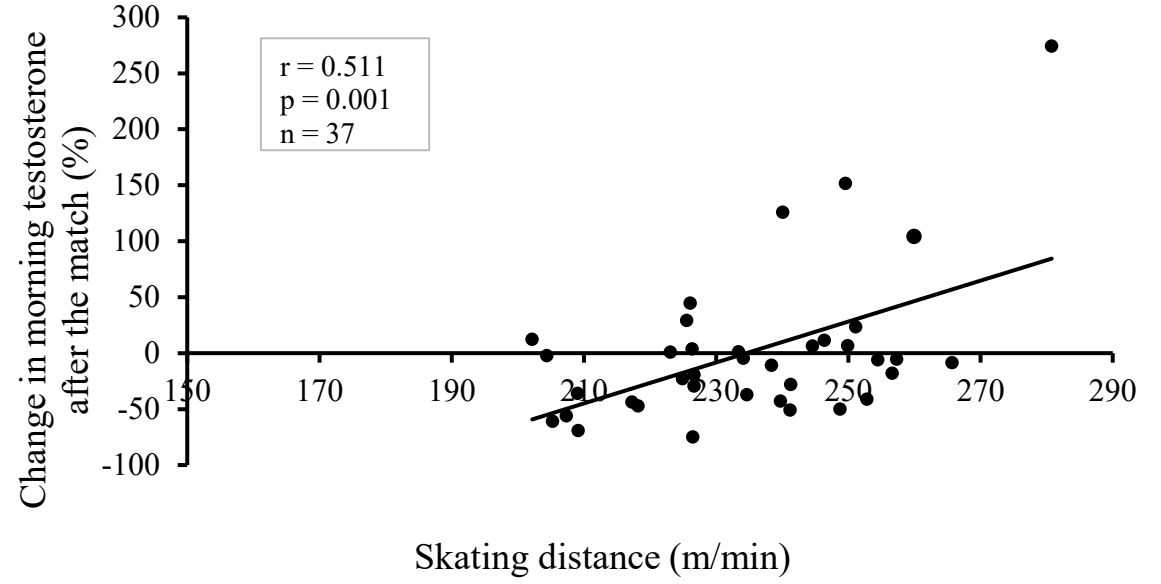



FIGURE 20. Correlation between change in morning salivary testosterone values and skating distance/playing time -ratio during the match.

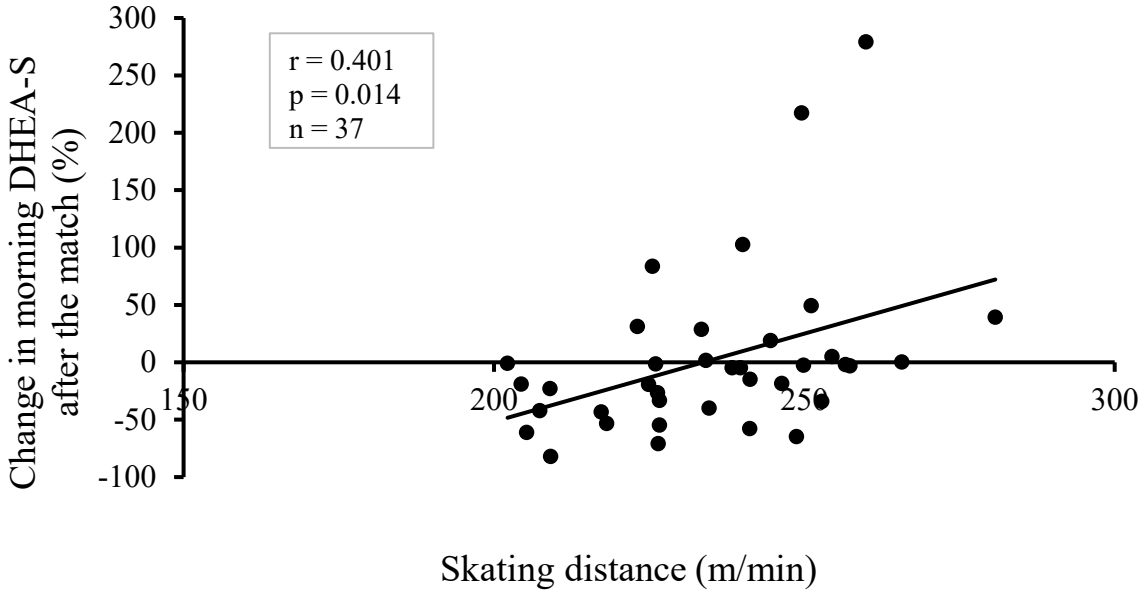



FIGURE 21. Correlation between changes in morning salivary DHEA-S and skating distance/playing time -ratio during the match.

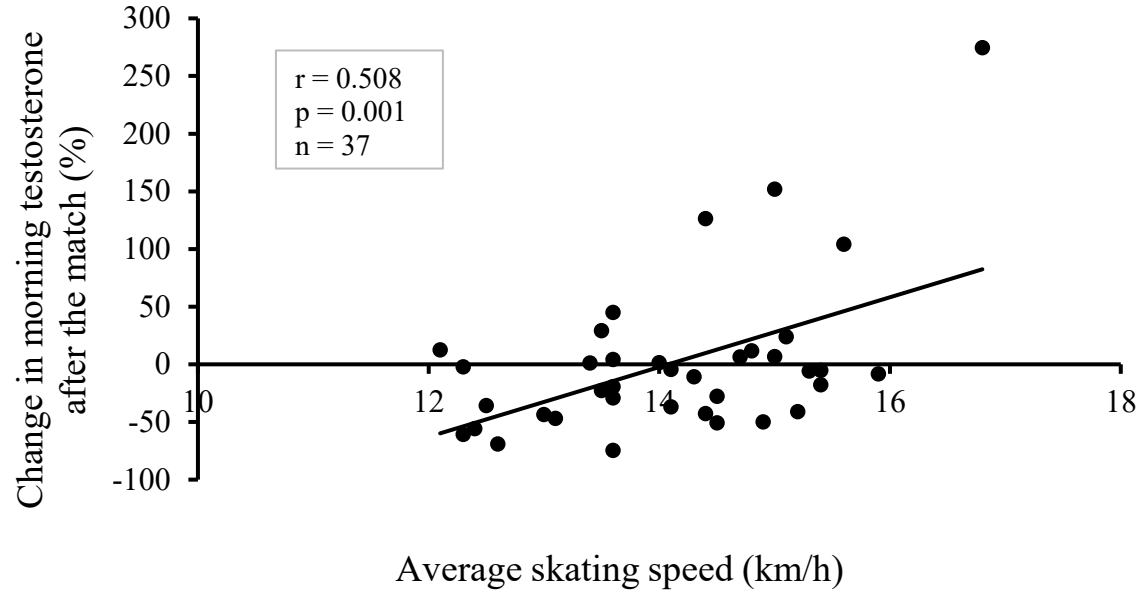



FIGURE 22. Correlation between changes in morning salivary testosterone and average skating speed during the match.

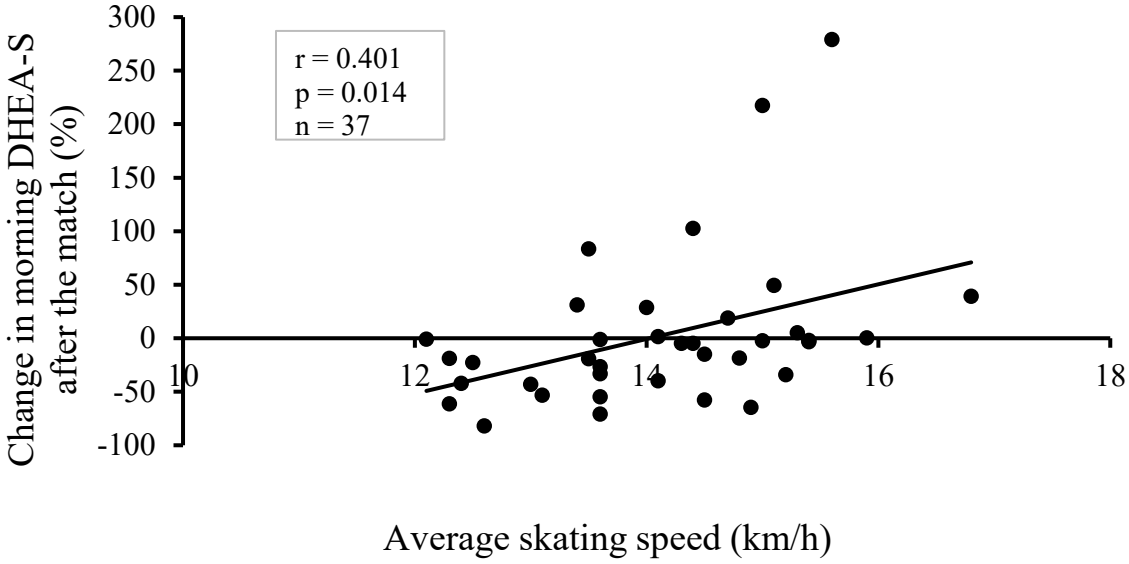



FIGURE 23. Correlation between changes in morning salivary DHEA-S and average skating speed during the match.

### 7.6 Immune responses

Blood leukocyte count increased by 84% immediately after the match and remained 10% above the baseline values at 12 hours post-match (figure 23). There was also a 24% decrease in blood lymphocyte count immediately after the match, but the difference was no longer evident 12 hours after the match (figure 24). In salivary IgA there was no statistically significant differences between timepoints (figure 25).

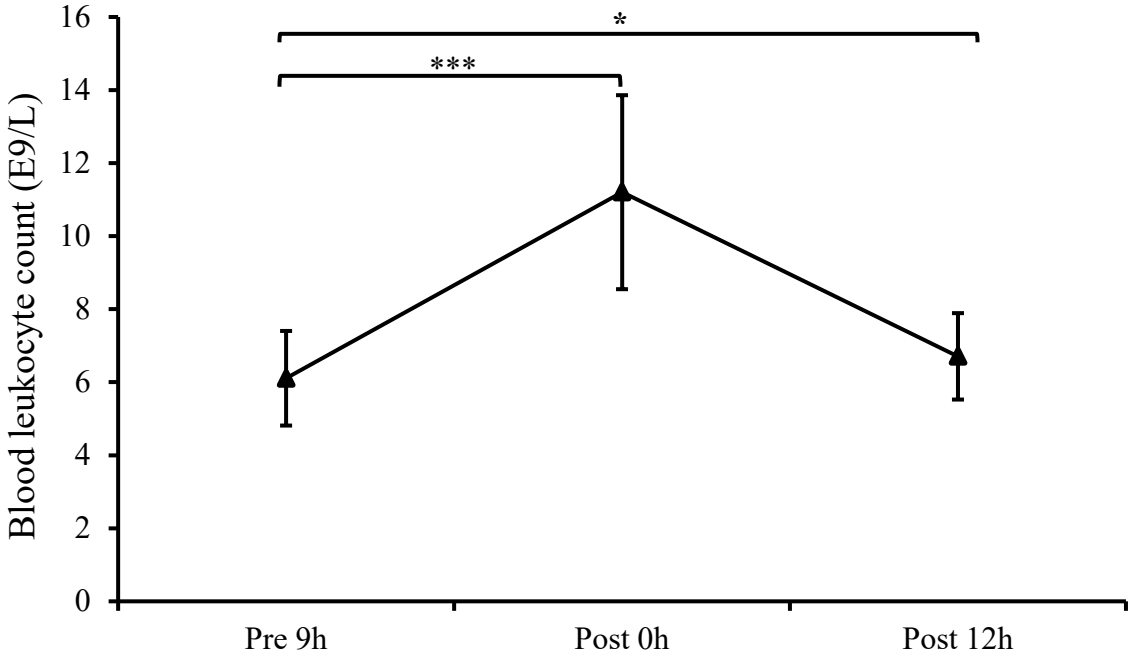



FIGURE 24. Mean ± SD blood leukocyte count at pre 9h, post 0h and post 12h.

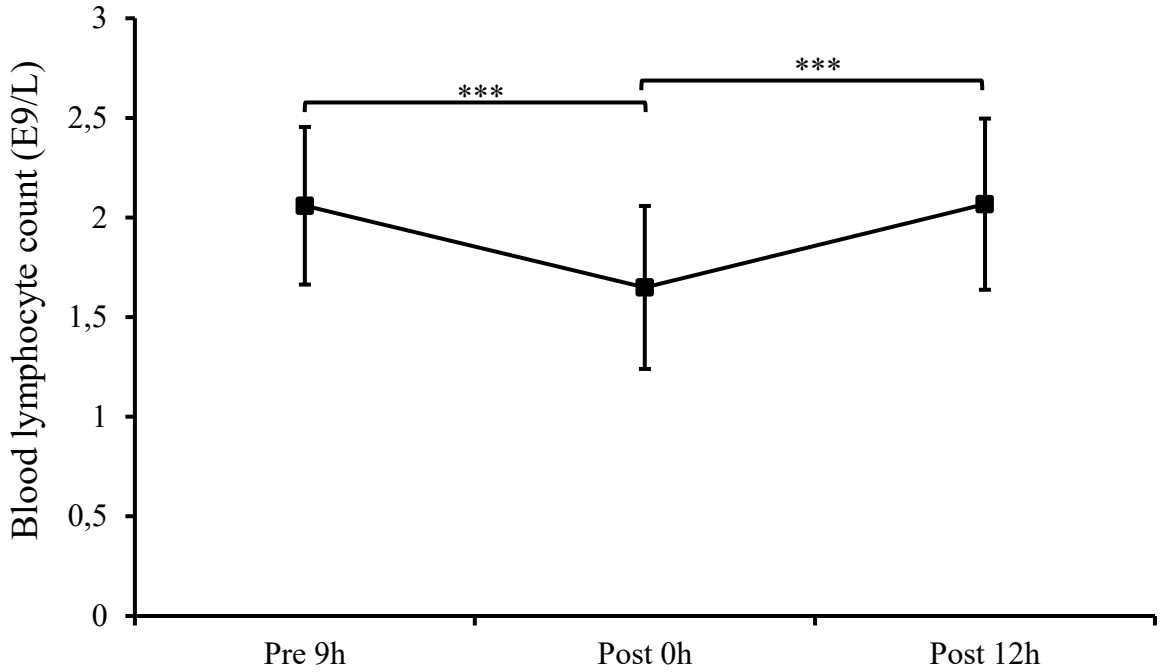



FIGURE 25. Mean ± SD blood lymphocyte count at pre 9h, post 0h and post 12h.

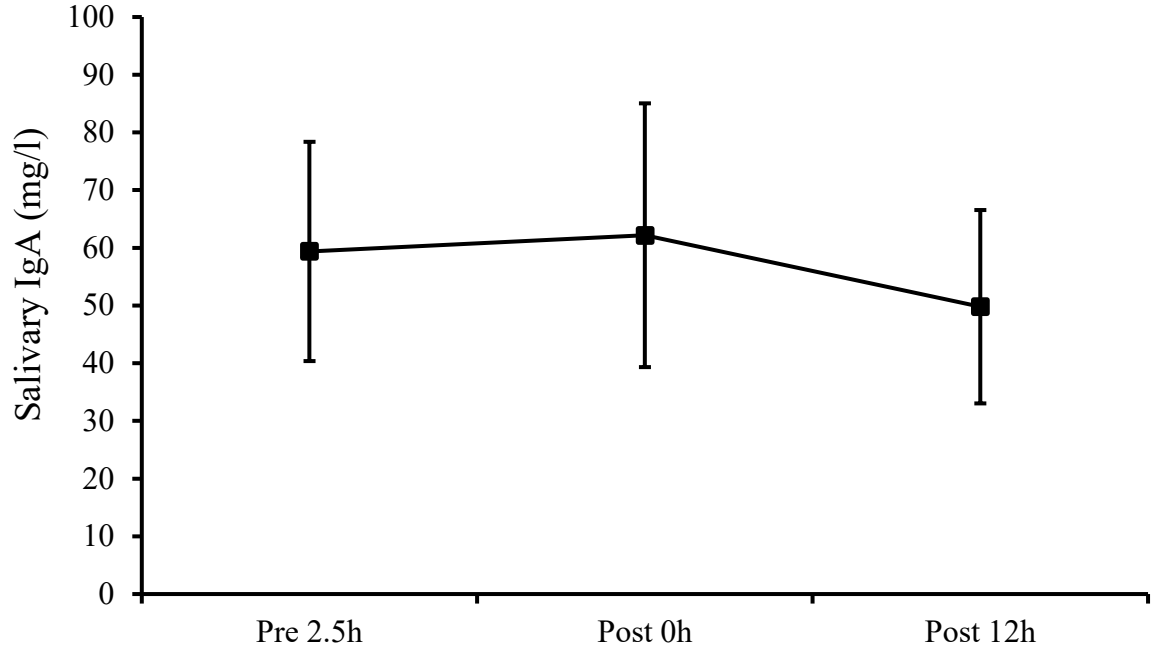



FIGURE 26. Mean ± SD salivary IgA at pre 9h, post 0h and post 12h.

There was a weak negative correlation (figure 26) between match sRPE and the change in salivary IgA from pre 2.5h to post 0h. No other correlations were found between measured immune responses and external loading during the match.

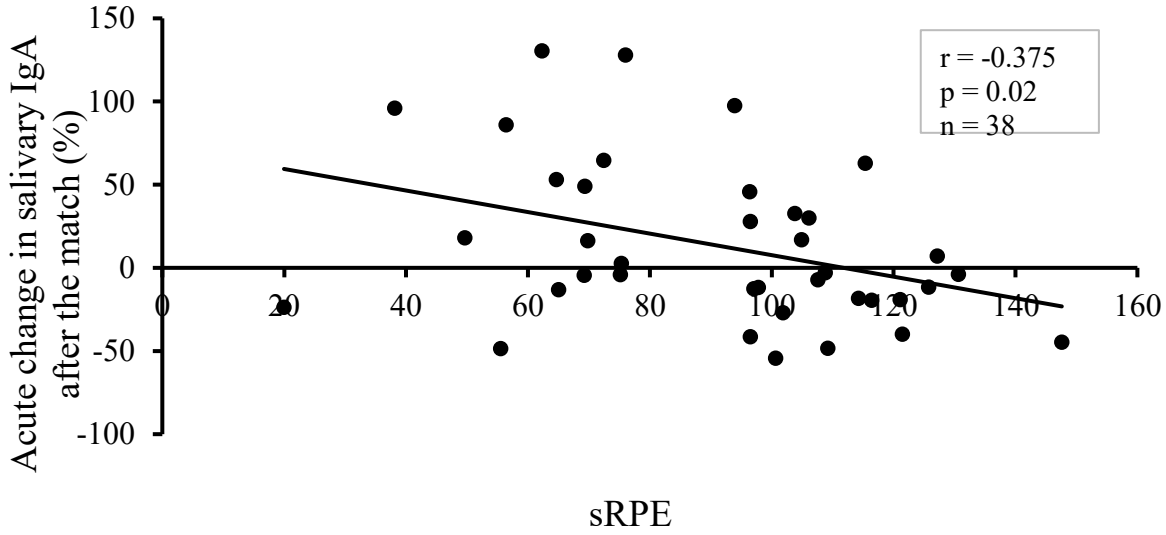



FIGURE 27. Correlation between acute change in salivary IgA and sRPE.

# 8 DISCUSSION

The main purpose of the study was to find out what are the acute physiological responses to official elite league ice hockey match and follow the recovery for 12 hours. The study focused on responses in neuromuscular performance, markers of muscle damage and in hormonal and immunological status. The study also investigated what are the associations between physiological responses and external loading during the match-play.

The main findings of the present study showed that neuromuscular performance is improved immediately after the match and then returns to baseline 12 hours after the match. Although neuromuscular performance is maintained and even improved after the match, other physiological responses indicate that the players are not fully recovered 12 hours after the match. According to the results, it seems that ice hockey match causes muscle damage as serum CK levels are increased immediately and 12 hours after the match and perceived muscle soreness is also greater at the morning after the match compared to the morning of the matchday. Ice hockey match-play also decreases salivary testosterone concentration and T/C-ratio, but these changes are recovered to baseline by the following morning. Immune function is altered after the match and these changes are not fully recovered in 12 hours. There was some individual variation in the physiological responses, but by the most part, these changes were not explained by the external loading during the match. Skating intensity during the match measured as total skating distance, skating distance/playing time -ratio or average skating speed were associated with increased anabolic hormonal activity (increased testosterone and/or DHEA-S) at the morning after the match. In addition, match sRPE correlated with amount of high-intensity skating, acute changes in testosterone, DHEA-S and IgA and changes in the morning values in cortisol and DHEA-S.

## 8.1 Differences in match characteristics in comparison to NHL matches

In this study, the mean total skating distance during the match was 3650 ± 657 m of which 1062 ± 256 m and 2202 ± 506 m were done in low and high intensity skating, respectively. The total amount of skating was remarkably lower than the 4606 ± 219 m that was reported

by Lignell et al. (2018) in the study measuring match activities in official NHL-match. However, despite the lower amount of total skating distance, the average amount of high intensity skating was greater in this study than in the study done in NHL-match. In this study, the amount of high intensity skating was 60% of the total skating distance whereas in the study by Lignell et al. (2018) the corresponding amount was 44%. At first glance, one could make conclusions that in Finnish elite ice hockey league the players cover less distance during matches, but greater proportion of the distance is covered at high intensities in comparison to matches in NHL. However, there are few major variables that could have affected the measured match activities in this study.

Firstly, in the study by Lignell et al. (2018), they used different speed thresholds to categorize skating into different intensity zones. The intensity zones used in that study were originally made for soccer, whereas in this study the used zones that were made especially for ice hockey. According to their selected speed thresholds, Lignell et al. (2018) described low intensity skating as skating done at speeds 1 – 16.9 km/h and high intensity skating was all the skating done faster than 17 km/h. In the present study, low intensity skating was described as skating done at speeds 0 – <15 km/h and high intensity skating was skating done at speeds 15 km/h and faster. Therefore, the selected different speed thresholds most likely explain the different portions of high intensity skating. However, both selected speed thresholds are justifiable. Lignell et al. (2018) used multiple-camera computerized tracking system for match analysis and they reported that their selected speed thresholds were valid for ice hockey according to their pilot measurements. In this study, match analysis was done with LPS and the used speed zones were especially made for this method with the data gathered from multiple ice hockey matches by the software provider. Therefore, the difference in the used methods makes the comparison between these results complicated.

One very important factor that explain the large difference in the amount of total skating between these studies is the fact that in the measured NHL-match, the score was tied at the end of the regular playing time and therefore the match continued to overtime. Unfortunately, in that study, they did not report the skating distances separately for each period and thus comparisons cannot be made only for the skating distance during regular playing time. Also, the lesser proportion of high intensity skating in the NHL-match could be explained by the

smaller rink size in NHL (NHL 2018) in comparison to European ice hockey leagues (IIHF 2018). Due to the smaller rink size, the players have less space to accelerate into high speed skating velocities.

However, there was also some similarities between the match-play analyses of these two studies. Lignell et al. (2018) reported that the average sprint skating speed was significantly lower during the third period in comparison to first and second periods. In this study, similar observations were made, as the skating distance/playing time -ratio and average skating speeds were significantly lower in the third period than in the two previous periods. Lignell et al. (2018) hypothesized that the observed decrease in the skating speed was due to neuromuscular fatigue generated by the match-play. That may very well be the case in that study, since the score was tied at the end of the third period and therefore the player's effort was probably near maximal throughout the match. However, in this study that is most likely not the case, since the measured responses in neuromuscular performance after the match does not support that. More likely explanation is that the effort of the players started to decrease during the third period, because at that point, the score was very much uneven between the teams. To conclude, when taken all these confounding factors into considerations, it is advisable to not make any strict comparisons about match-play analyses between these two studies.

## 8.2 Possible factors explaining the improved neuromuscular performance

Arguably, the most interesting finding in the present study was that neuromuscular performance was actually improved immediately after the match with all the different variables measured from CMJ and this was in disagreement with the hypothesis. The hypothesis was that neuromuscular performance would be impaired immediately after the match and remain below baseline levels 12 hours after the match. It was also hypothesized that external loading during the match would correlate with the decrease in neuromuscular performance. In fact, there was even a moderate correlation between total playing time and increase in CMJ height immediately after the match. These results differ greatly from what has been reported in responses to neuromuscular performance after matches in other

intermittent team sports. For example, when neuromuscular performance has been measured with CMJ immediately after match in soccer and rugby, significant reductions in jump height have been observed (Duffield et al. 2012; Romagnoli et al. 2016; Twist & Sykes 2011). Also, the recovery of the performance in CMJ typically takes up to 48 hours in other team ball games (Doeven et al. 2018).

There are several reasons that might explain the observed increase in neuromuscular performance. For one, the external loading during ice hockey match is lesser than in other more studied team sports. In this study the total playing time was 15.5 ± 0.4 min and the total skating distance was 3650 ± 107 m, whereas in soccer and rugby league the mean total covered distances and playing times are 10274 ± 946 m and 95.3 ± 1.8 min for soccer and 6276 ± 1950 m and 64.9 ± 18.8 min for rugby league, respectively (Varley et al. 2014). Even though the intensity of locomotion is much greater in ice hockey than in soccer and rugby league (235 ± 19 m/min in ice hockey vs. 104 ± 10 m/min in soccer and 97 ± 16 m/min in rugby league) it seems that the volume is not high enough to cause reductions in neuromuscular performance, at least in elite league ice hockey players (Varley et al. 2014). It is good to bear in mind that this might not be the case with players that are physically not as developed as elite league ice hockey players, since it has been observed that reductions in neuromuscular performance are not as severe after rugby match in players with higher fitness level in comparison to players with lower fitness level (Johnston et al. 2015).

One important factor that might explain the differences in CMJ performance after match between ice hockey and other team ball games is the differences between kinematics in running and on-ice skating. The main differences in skating compared to running are greater emphasis on lateral and rotational movement in the hips, longer ground contact times (330 ms in skating vs. 100-80 ms in running) and consistent forward lean of the trunk during skating (Nagahara et al. 2014; Robert-Lachaine et al. 2012). These factors contribute to less vertical force absorption which might explain the lesser reductions in vertical power production after ice hockey match. When taken into consideration that impairments in neuromuscular performance seems to be sport specific, it could be so that measuring lateral power production from lower limbs would better describe responses in neuromuscular performance after ice

hockey match than CMJ. For example, in basketball, which includes high number of vertical jumps, vertical power production is impaired even more than after soccer match, even though more distance is covered during soccer matches (Chatzinikolaou et al. 2015; Pliauga et al. 2015).

A plausible explanation for improved neuromuscular performance immediately after the match could be the time of the day when the measurements took place. Due to the fact that this study was designed to interfere the players' preparation to the official match as little as possible, a decision was made to do the pre-measurements in the morning at 9:00 a.m. instead of immediately before the match. This might have affected the results as significant circadian rhythm have been observed for neuromuscular performance (Mora-Rodriquez et al. 2012; Souissi et al. 2007; Teo et al. 2011). For example, rate of force development, peak force and peak power in CMJ have been reported to be significantly lower at 8:00 a.m. compared to 4:00 p.m. (Teo et al. 2011). Also, higher body temperature after the match might have contributed to better performance in CMJ since increased body temperature is associated with improved neuromuscular performance (Racinais et al. 2005). However, the circadian rhythm and body temperature does not explain why neuromuscular performance was at the baseline values at the following morning after the match, because the measurements were done at the same time of the day and after same controlled warm-up as at the morning of the match day.

Even though the mean values measured in CMJ indicated improved performance, there was some individual variation in neuromuscular performance after the match. There were five players whose jump height was reduced by more than 5% immediately after the match. This individual variation in responses in neuromuscular performance did not correlate with any measures of external loading during the match. Thus, it is possible that this individual variation is rather explained by differences in physical qualities. As stated previously, at least after rugby match, it has been shown that players with better aerobic capacity cover more distance and yet recover faster after the match (Johnston et al. 2015).

## 8.3 Markers of muscle damage in comparison to other team sports

In this study, muscle damage was assessed by measuring serum CK concentrations and subjective muscle soreness with VAS. These markers indicated that ice hockey match-play causes muscle damage as significant increases were observed with both variables and this finding was in agreement with the hypothesis. It was also hypothesized that changes in serum CK would correlate with external loading during the match but that was not observed in this study. Serum CK peaked at 12 hours after the match and it is possible that the values kept increasing from there as peak values are usually reached at 24 hours post-match in other team ball games (Doeven et al. 2018). One previous study (Lignell et all. 2018) has measured CK concentrations after ice hockey match, but they only reported values at 24 hours after match with no baseline values pre-match. In that study, the CK values were 338 ± 45 U/L, whereas peak values in the present study were 520 ± 423 U/L. (Lignell et all. 2018) Hence, it might be that peak in serum CK concentration is reached earlier after ice hockey match than after other team ball games. That would make sense, since the overall loading during match in these other team sports (e.g. soccer, rugby, basketball) also seems to be greater. (Doeven et al. 2017) However, to be sure, CK concentrations should be monitored for at least 48 hours to find out when the peak values are actually reached after ice hockey match.

One interesting finding in this study was that even though there was a significant increase in serum CK concentrations after the match, the percentual increase from baseline values were relatively modest. In this study the peak percentual increase from baseline in CK was 37%, whereas after soccer and rugby matches the reported increases have been as high as 600-700% (Djaoui et al. 2016; Fatouros et al. 2010; Ispirlidis et al. 2008; Twist & Sykes 2011). However, the difference in the actual peak values is not that big, as peak values in this study were 520 ± 423 U/L in comparison to 671 to 1411 U/L after rugby and soccer matches (Doeven et al. 2018). Hence the modest percentual increase in serum CK is rather explained by higher baseline values in this study. The higher baseline values are probably explained by training sessions by the players prior to participating in this study and possibly by differences in muscle mass between ice hockey and soccer players (Sutton et al. 2009).

There was a lot of individual variation in serum CK values in this study. For example, the values at 12 hours post-match varied from 181 U/L to 2796 U/L and also the percentual changes at that time point varied from -35% to 131%. Again, this variation was not explained by external loading during the match. However, the amount of high impact collisions and tackles during the match, which could have explained these large differences, were not counted in this study. For example, the amount of high impact collisions during rugby match have been found to correlate with the changes in CK after the match. In that same study, they also found out that the total distance covered during the match correlated with the increase in plasma CK. (Gastin et al. 2019) The reason why this present study did not find that correlation between total skating distance and changes in CK might be the lesser impact during skating in comparison to running. As the impact in skating is not as great as in running, the eccentric strain imposed to the working muscles is not as severe and thus muscle cell membranes might not be as damaged.

A plausible explanation for the individual variation in CK responses might again be in the differences in the physical qualities of the players. In the study by Lignell et al. (2018) they found a correlation between higher post-match CK concentrations and greater cardiovascular loading during submaximal Yo-Yo Intermittent Recovery Ice-hockey test level 1. This suggests that the players with higher fitness level seem to experience less muscle damage after the match and thus be able to recover faster after matches. However, this current study did not address this question and thus more studies in this area are needed.

Even though some studies (Smart et al. 2008; Takarada 2003) have found correlations between match activities, such as number of tackles and impacts, and increased CK concentrations, it should be noted that changes in serum CK concentrations is a poor indicator for the magnitude of the muscle damage. It has been found in muscle biopsy studies, that elevations in muscle enzymes released into circulation are not related with the magnitude of detected histological muscle damage. (Magal et al. 2009; Van der Meulen et al. 1991) Therefore strict comparisons between individual CK responses and match activities should be evaluated critically and rather use increased CK concentrations as a general marker that indicates that muscle damage has occurred.

## 8.4 Possible explaining factors behind conflicting hormonal responses

In this study, ice hockey match-play resulted in alterations in salivary hormone concentrations. Immediately after the match salivary testosterone was decreased and cortisol remained unchanged which led to a decreased T/C -ratio after the match. Contradictory to testosterone, salivary DHEA-S increased acutely after the match. On average, these changes were no longer evident by the next morning. The acute decrease in T/C -ratio was in agreement with the hypothesis, but the fact that cortisol remained unchanged and the decrease in T/C -ratio was solely due to decreased testosterone was in disagreement with the hypothesis. The hypothesis was that cortisol would decrease and testosterone would increase immediately after the match.

When cortisol responses have been measured after matches in other intermittent team ball games, increased concentrations have fairly been consistent finding. For example, peak cortisol concentrations have been measured immediately after soccer, handball and rugby matches (Chatzinikolaou et al. 2014; Cuniffe et al. 2010; Elloumi et al. 2003; Ispirldis et al. 2008; McLellan et al. 2010; McLellan et al. 2011; Romagnoli et al. 2016) Also, in the studies that have measured salivary cortisol concentrations after matches, the peak values have ranged from 16.3 to 80 nmol/l (Doeven et al. 2018). These values are a lot higher than the 7.8 nmol/l that was measured in this study immediately after the match. At this point it is uncertain what is the reason for the lack of changes in cortisol concentration in this study. An intensity threshold of 60% of $VO_{2max}$ has been proposed for cortisol response to exercise and after that point, large increases in cortisol concentrations can be observed (Papacosta & Nassis 2011). The intensity during the shifts in this study were certainly above that threshold, since the players peak heart rates during the match ranged from 91 to 100% of their maximal heart rates. However, the total time spent at high intensity skating zones during the entire match was only 6.5 minutes and thus it might be that it was not enough to provoke large increases in cortisol concentrations. Also, the high amount of passive recovery between the shifts and during intermissions might have prevented the possible increases in cortisol concentrations. Lastly, it was not controlled whether the players were consuming sport beverages that contained carbohydrates during the match. That might have affected the

results, since carbohydrate ingestion prior and during exercise have been shown to attenuate cortisol responses to exercise (McAllister et al. 2016; Smith et al. 2018).

However, it is possible that some increase in cortisol levels did in fact occur, but it was masked by the natural circadian rhythm of the cortisol secretion. Also, the acute decrease in circulating lymphocytes after the match would suggest that cortisol levels were increased, since post-exercise decrease in lymphocytes is mediated by the HPA axis (Cupps & Fauci 1982; Nieman 1994; Nieman 1997). To be sure, what is the true magnitude of the cortisol response after ice hockey match, the post-match values should be compared to non-exercise control samples that are taken at the same exact time of the day as the post-match samples.

As stated previously, testosterone responses to match-play in different intermittent team sports does not seem to have a consistent pattern. In four other studies testosterone levels have been reported to decrease immediately after matches in Australian football, soccer and rugby (Cormack et al. 2008; Cuniffe et al. 2010; Romagnoli et al. 2016; West et al. 2014). On the opposite, two studies have found increases in testosterone levels after matches (Gravina et al. 2011; McLellan et al. 2010), whereas in four studies no changes have been found (Silva et al. 2013; Chatzinikolaou et al. 2014; Ispirlidis et al. 2008; Kreamer et al. 2009). These results in testosterone responses after matches are somewhat contradictory to testosterone responses to exercise in general, since recent meta-analysis by D'Andrea et al. (2020) shows that moderate and high intensity exercise typically increases testosterone levels acutely. Sample timing can be one explaining factor for not finding increased testosterone levels after match in most studies, since increases are typically not observed in samples that are taken over 30 minutes after the end of an exercise sessions (D'Andrea et al. 2020). However, in this study, all the samples were collected within 40 minutes after the match had ended, so sample timing probably has a minimal effect on the observed testosterone response, because majority of the samples were collected in less than 30 minutes and still a significant decrease in testosterone levels was observed.

As testosterone is characterized as an anabolic hormone, due to its ability to upregulate glycogen and muscle protein synthesis and downregulate muscle protein breakdown (Spiering

et al. 2008), it is possible that testosterone was decreased due to bodies attempt to spare energy sources in a state of increased energy expenditure. However, because there was no change in cortisol levels and cortisol acts in an opposite way by breaking down energy stores when energy expenditure is increased (Papacosta & Nassis 2011), it could be possible that the observed decrease in testosterone levels were solely due to the normal circadian rhythm of the hormone. Due to the warm-up schedules of the teams, the pre-match samples had to be taken at 4:00 p.m., whereas the post-match samples were taken at approximately 9:00 p.m. This might be on factor that explains the decrease in testosterone levels.

DHEA-S responses in this study are in agreement with the previous studies examining acute DHEA-S responses to exercise. On average DHEA-S increased immediately after the match and returned back to baseline after 12 hours. Acute increases in DHEA-S have also been observed after marathon, triathlon, swimming and soccer (Aizawa et al. 2006; Bonen & Keizer 1987; Malarkey et al. 1993; Velardo et al. 1991). According to the available literature, it seems that high intensity exercise and long durations of low intensity exercise increase DHEA-S concentrations in dose-response manner (Enea et al. 2009; Tremblay et al. 2005). This study failed to find correlations between acute increase in DHEA-S and the magnitude of external loading during the match. However, various measures of high intensity skating correlated with increased DHEA-S concentrations at the following morning. There was also a correlation with higher skating distance/playing time -ratio and higher average skating speed during the match and increased testosterone concentrations at the following morning. This would suggest that the amount of high intensity skating is associated with increased anabolic hormonal activity 12 hours after the match. This rebound in anabolic activity might be due to the need for restoring depleted energy stores and repairing of damaged tissues.

### 8.5 Ice hockey match seems to cause typical immunological responses to exercise

Immunological responses observed in this study were by some parts in agreement with the hypothesis. It was hypothesized that the number of circulating leukocytes and lymphocytes would increase, and salivary IgA concentrations would decrease after match, and these changes would correlate with external loading. Indeed, significant increases were found in

circulating leukocyte count, but lymphocytes decreased after the match, with no changes observed in salivary IgA. No direct measures of external load correlated with these changes which was in disagreement with the hypothesis, but significant negative correlation was observed with sRPE and change in salivary IgA immediately after the match.

The fact that there were not any significant increases in cortisol concentrations after the match would suggest that the responses in immune function was mediated by sympathetic nervous system response and not by HPA axis. In response to exercise the activation of sympathetic nervous system results in a release of catecholamines (norepinephrine and epinephrine) which in turn mediates an increase in neutrophils and natural killer cells (Pyne 1994), whereas cortisol response to exercise is responsible for decreased lymphocyte count which usually occurs during the recovery from high-intensity exercise (Cupps & Fauci 1982; Nieman 1994; Nieman 1997). However, in extremely intense or long duration exercise, the circulating lymphocyte count can already begin to decrease during the exercise session (Gleeson 2007). In this study, it is unlikely that this was the case in the decreased lymphocyte count immediately after match, since all the other measured markers indicated that the overall loading during the match was modest rather than extremely high. More plausible explanation is that in this study the cortisol concentration had already peaked before the third period and the observed decrease in lymphocytes followed the normal recovery pattern after exercise. The fact that the covered distance and the amount of high intensity skating was lesser in the third period in comparison to the first and second periods indicates that the effort of the players was probably not maximal in the last period, since the other team already had a clear advantage at the beginning of the third period. If it is considered that the decrease in lymphocyte count was a part of normal recovery response to exercise, it can be concluded that the observed changes were expected and in agreement with previous literature. Usually total leukocyte count increase by 50-100% immediately after exercise and lymphocytes decrease by 30-50% 30 minutes after exercise (Nieman 1994), whereas decreases in this study were 86% and 20% for leukocytes and lymphocytes respectively.

In this study, no significant changes were observed in salivary IgA concentrations after the match. According to the literature mucosal immunological function might be impaired after exercise and the intensity and volume are the main factors that determine the magnitude of the

impairment (Mackinnon et al. 1993; Tomasi et al. 1982). For example, in Australian football greater external workloads during matches have been associated with suppressed salivary IgA for 36 hours post-match (Coad et al. 2015). Even though no direct measure of external workload was associated with changes in IgA in this study, a weak correlation was found with decreased post-match IgA and greater sRPE during the match. This correlation was no longer significant after 12 hours, which again indicates that the recovery after ice hockey match is probably faster than recovery after similar high-intensity intermittent team ball games played on dry land.

## 8.6 Strengths and weaknesses of the study

Arguably, the biggest strengths in this study was that this was the first time that physiological responses to ice hockey match was measured in a comprehensive manner. This gives new information about what are the different physiological changes in the human body after ice hockey match-play. The fact that this study measured official elite league in-season match is definitely also a strength of this study, even though some compromises to the study design had to be made due to that. Unlike in many other match-load studies, this study used force plate to measure CMJ. Force plate is considered to be the "gold standard" method for measuring jump height in CMJ as it has better validity and reliability than other field test methods for measuring CMJ (Vincenzo et al. 2018). This study also measured several different variables from the CMJ to assess neuromuscular performance to decrease bias in the results.

However, the fact that in this study only one match was measured, might have caused some bias to the results. Unfortunately, the other team had a clear advantage in this particular match, and it could have affected the effort and overall loading of the players. Also, if more official matches between different teams had been measured, the statistical power in the study would have been greater. Other limitation of this study was the fact that the recovery of the players was only measured for 12 hours after the match due to the busy in-season schedule of the teams. That time period was not enough for all the measured physiological markers to fully recover, so this study failed to identify the full recovery time needed after official ice

hockey match. Also, the time at which the measurements took place was not optimal. The circadian rhythm of neuromuscular performance could have affected the measured parameters in CMJ. Also, it was practically impossible to measure all the players at the same time immediately after the match and so the time window between the first and last player that was measured was 40 minutes. This difference could have affected some results. Lastly, since professional ice hockey players were measured during official in-season match, it was not possible to control the nutritional status and participation to tactical practices by the players before and during the study without interfering the players' preparation to the match.

## 8.7 Practical applications

This study highlighted the need for measuring external and internal loading in combination to evaluate the true overall loading during matches. In this study the different measured markers of external load were poorly associated with the internal loading after the match. That is important to note, so that decisions according to individual training loads are not made solely based on external measures (e.g. total playing time, distance covered). However, many markers of internal load are often impractical since they are costly, time consuming and require special skills to obtain and analyze (Cardinale & Varley 2017). However, sRPE is practical and easy-to-use tool to combine external load (total playing time) to internal load (subjective perceived exertion). Also, the practical value of sRPE stood out in the results of this study, as sRPE was associated with total playing time, total skating distance and amount of high intensity skating during the match and also with acute changes in anabolic hormones and salivary IgA after the match. In addition, sRPE was also associated with increased cortisol and DHEA-S at the following morning. However, these correlations were by no means perfect, so therefore caution should be taken when interpreting these results. Nevertheless, sRPE seems to be a comprehensive and very cost-efficient tool to evaluate physical loading after ice hockey matches and it can be used by any coach regardless of level of competition and budget.

The results of this study may also be applied to planning training schedules and microcycles during ice hockey in-seasons. It has been found that neuromuscular performance measured as

jump height in CMJ and SJ decrease significantly during 18 weeklong ice hockey season (Whitehead et al. 2019). However, according to the results of this study, it seems that the ice hockey match-play per se does not cause short term reductions in neuromuscular performance. Therefore, it could be that the observed reductions in power production during the in-season is rather a result of decreased amount of physical training. That is understandable due to the busy schedule of matches and increased demand for tactical on-ice practices. That arises the question, when would be appropriate time for specific physical training during in-season to maintain neuromuscular performance while making sure that the players are getting enough time to recover between training sessions and matches. The results of this study might provide some solutions for this problem. Since neuromuscular performance was actually improved after the match, but markers of muscle damage indicated that muscle damage still exists after 12 hours, short sessions of specific power or strength training could be implemented immediately after matches while the day after the match could be dedicated for active recovery. This kind of microdosing of power and strength training, at least after home matches, would make physical training a regular part of the team's weekly schedule also during in-season. Also, the volume of the training sessions after matches could be individualized by using the match sRPE, which could help to make the overall loading of the match days more equal between the players. However, it should be noted that this study only represented the responses to one match that was played at the early phase of the in-season. Therefore, the effect of accumulation of loading from several matches on neuromuscular performance and overall loading are yet to be identified, and thus caution should be taken when increasing the amount of physical training during the in-season.

## 8.8 Conclusions

This was the first study to measure changes in neuromuscular performance, markers of muscle damage and hormonal and immunological status after official ice hockey match in professional ice hockey players. According to the results, ice hockey match is not as demanding as one might expect due to the fast-paced nature of the sport. Neuromuscular performance in CMJ is improved immediately after the match and then returns back to baseline after 12 hours. However, even though neuromuscular performance is at baseline 12 hours after ice hockey match, biochemical markers of muscle damage and immune function

indicates that physiological recovery is still in progress. This highlights the need for using several markers to monitor match loads and recovery. There was some individual variability in the physiological responses to the match, but no direct measures of external load explained these differences. However, sRPE seemed to be the most useful tool to evaluate the player's individual match loads, since it correlated with the amount of high-intensity skating during the match and with hormonal and immunological responses. In the future, more research with longer measuring periods should be done to reveal the true recovery period needed after ice hockey match. Also, future studies should examine whether physical qualities of the players explain the differences in physiological responses to ice hockey match and if there is differences in match loading between the matches that are played at the beginning of the season compared to the matches played at the end of the season.

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