# Repeated Sprint Ability:
# The Influence of Aerobic Capacity on Energy Pathway Response and Fatigue of Hockey Players

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## CHAPTER 1: Introduction

Sport specific training has been shown to improve performance in team sport athletes (Castagna et al., 2008; Girard, Mendez-Villanueava, & Bishop, 2011). An important factor for success in repeated sprint sports is a player's ability to produce power; the player that is quicker to the ball or faster off the line will often beat their opponent, giving them an advantage. Competitions for these sports, however, take place over the period of several hours and are divided into quarters, halves, or periods; each of these consisting of numerous maximal-work bouts interspersed with relatively short recovery periods (Montgomery, 2000). The ability to repeatedly produce high power outputs throughout a competition gives a competitive edge to a player over his/her opponent and is an important fitness component in repeated sprint sports (Glaister, 2005). This ability has come to be known as repeated-sprint ability (RSA).

In the repeated sprint sports of rugby and soccer, a higher $VO_{2peak}$ has been shown to correlate with improved RSA, ranging from r = -0.50 to -0.83 (Bishop & Edge, 2006; Tomlin & Wenger, 2001). This relationship is further supported by research showing that a state of hypoxia can impair RSA in athletes (Balsom, Ekblom, & Sjodin, 2004; Quistorff, Johansen, & Sahlin, 1992). In addition, creating a hyperoxic environment appears to improve RSA through increased aerobic adenosine triphosphate (ATP) contribution and phosphocreatine (PCr) resynthesis rate (Balsom et al., 2004). This evidence suggests that aerobic capacity and improved oxygen utilization may affect RSA by 1) increasing the rate of the fast and slow phase of PCr resynthesis (McMahon & Jenkins, 2002), 2) enhancing the clearance rate of metabolites created by PCr breakdown

and glycogenolysis (Bishop & Spencer, 2004), 3) improving oxygen ($VO_2$) kinetics (Dupont, Millet, Guinhouya, & Berthoin, 2005), and 4) increasing aerobic energy contribution during maximal sprint bouts (Tomlin & Wenger, 2001).

Disagreement exists regarding the strength of this association, as there is research to refute these findings. Numerous researchers have failed to find a significant association between $VO_{2peak}$ and RSA in rugby and soccer athletes (Aziz, Chia, & Teh, 2000; Bishop & Spencer, 2004; Carey, Drake, Pliego, & Raymond, 2007; Wadley & Rossignol, 1998), but it is not clear what role sample size ($n < 15$) played in calculating these statistics. The discrepancy in the research would advocate for additional studies to be conducted to better understand the possible relationship between aerobic capacity and RSA.

While there is research specific to the relationship between aerobic capacity and RSA in field-based team sports (e.g., rugby and soccer) there is very little published on the sport of ice hockey. To date, this researcher is aware of only one study to have tested this relationship, which found no significant correlation between the variables (Carey et al., 2007). A deficiency of the current research is the mode with which researchers test the $VO_{2peak}$ of hockey players, which typically uses a modified Bruce protocol on a motor-driven treadmill (Carey et al., 2007). A recent study by Durocher et al. (2010) found that there was no correlation between the on-ice and off-ice $VO_{2peak}$ values in collegiate hockey players. The researchers concluded that off-ice $VO_{2peak}$ was not an adequate predictor of on-ice $VO_{2peak}$ and suggested that hockey players must be tested in a sport-specific manner to garner reliable results. Taking the findings of Durocher et al.

(2010) into account, it is possible that the lack of a correlation in the current research could be due to poor testing protocol selection.

In addition, the majority of RSA tests employ protocols that only utilize straight ahead running or skating (Carey et al., 2007; Gaitanos et al., 1993). This can be problematic when interpreting direct correlations to performance, as athletes move in a 360° plane of motion during competition (Girard, Mendez-Villanueva, & Bishop, 2011). A study by Spencer et al. (2008) found that athletes completing a course with multiple changes in direction placed a greater energy demand on their metabolic systems when compared to straight ahead running covering the same distance (Reilly, 1996). This would indicate that RSA tests employing only straight ahead running do not simulate the stress placed on an athlete during competition. Consequently, there is a need for a study to look at the role aerobic capacity plays on RSA in hockey players, using a protocol that accounts for both the task-specificity of skating and the movement patterns performed in competition.

This series of studies aim to eliminate the shortfalls of the current research by addressing a current, population-specific void in ice hockey literature. The first study, "Aerobic Capacity is Associated with Improved Repeated Shift Performance in Hockey," establishes a foundation for future RSA research to build on by 1) accounting for task-specificity by obtaining players' $VO_{2peak}$ on a skating treadmill using a graded exercise test; and 2) evaluate RSA using an on-ice test, developed to mimic the motor patterns typically performed by hockey players during competition. The second study, "Division I Hockey Players Generate More Power than Division III Players During On- and Off-ice

Performance Tests," establishes baseline, normative data for hockey players for both on- and off-ice performance tests; stratified by level of play. Finally, the third study, "Off-Ice Anaerobic Power is Not a Good Predictor of On-ice Repeated Shift Performance in Hockey Players," challenges both the scientific and sporting community to re-evaluate the emphasis placed on anaerobic power during off-ice player testing. The results of these studies could have important implications for the training and evaluation methods used by scientists, coaches, and players to prepare for the competitive season.

The purpose of this thesis will be to investigate the relationship between aerobic capacity ($VO_{2peak}$) and RSA in Division-I, Division-III, and Junior level hockey players, while accounting for stride efficiency on sprint decrement. It is hypothesized that hockey players with a higher aerobic capacity ($VO_{2peak}$) will exhibit less fatigue during an on-ice repeated shift test than those with lower values.

## CHAPTER 2: Literature Review

### 2.1 Defining Key Concepts

*2.1a Repeated Sprint Ability*

Fatigue is defined throughout the literature as a decline in maximal sprinting speed (increased time in sprint running) or a decrease in peak power or total work (cycling) (Gastin, 2001; Bishop et al., 2011; Glaister, 2008; Spencer et al., 2005; Glaister, 2005). To accurately evaluate fatigue the word "sprint" must be clearly defined. Unfortunately this term has been used with a great deal of latitude when addressing repeated sprint ability (RSA). Research has been published evaluating RSA with experimental protocols consisting of 30-second maximal work bouts, interspersed with up to four minutes of recovery (Bogdanis, Nevill, & Boobis, 1995; Bogdanis et al., 1996). Alternatively, research has been published evaluating RSA with protocols consisting of 4-second work bouts interspersed with 20-seconds of passive recovery (Rampinini et al., 2009). The current accepted definition of a true 'sprint,' in the sense of RSA, has been defined in the research as a maximal work bout lasting ≤10 seconds (Girard, Mendez-Villanueva & Bishop, 2011). This time frame has been established as it is the approximate duration that PCr and glycogenolysis can support a maximal ATP turnover rate of approximately 14 mmol/kg/dm (Gaitanos, Boobis, & Brooks, 1993). After that point, the energy system can no longer sustain its peak ATP turnover rate, thereby voiding the work being performed as a true maximal sprint.

A researcher and/or coach must also know the duration of the sprint bouts specific to their sport of interest to make direct inferences to training. Further analysis of the

literature shows work-to-rest ratios in field-based team sports can range from 1:3 to 1:30 (Reilly & Thomas, 1976; Spencer et al., 2004; McLellan, Lovell, & Gass, 2011) As a result, further definition of the term is required to outline the rest intervals associated with RSA. Girard et al. (2011) advocates that repeated sprints should be further delineated into two categories; intermittent-sprints and repeated sprint exercise (RSE). Intermittent-sprints are defined as sprints performed with a work-to-rest ratio of >1:6, and up to 1:30. Recovery periods such as these typically will allow for complete, or near complete, recovery of the muscle, showing little to no fatigue. Repeated sprint exercise, conversely, is defined as sprints performed with a recovery ratio of <1:6, typically resulting in performance decrement, or fatigue.

*2.1b Energy Pathway*

It should be noted that the term "energy pathway" is used instead of "energy system" when referring to a specific method in which energy is supplied to muscle during RSE. Current research regarding the metabolic demands of field-based, repeated sprint sports, are unanimous in its conclusions that they place considerable demands on the entire energy system of the athlete (Mendez-Villanueava, & Bishop, 2011). The percentage of the burden being placed on each pathway, however, be it anaerobic or aerobic, is a topic of considerable debate. There is never one singular energy system being used at a given time. It should be understood that while physiologists often refer to these systems as separate entities, they all contribute to RSA ability from its onset to completion. The human body uses multiple energy pathways that form one energy system, the goal of which is to meet the energy demands of the activity.

A study by Gastin et al. (2001) found that short maximal sprints (<10 seconds) attained 6% of their energy expenditure from aerobic metabolism. This has led researchers like Glaister (2005) to view the human body's energy system as a progressive, changing metabolic environment that leads to gradual inhibition of specific energy pathways, leaving others to increase production. Similarly, a study by Gaitanos et al. (1993) found that anaerobic glycolysis, during RSA tests of 10x6 second bike sprints, contributed 44% of the total energy during the first maximal sprint, while only contributing 16% on the final, tenth sprint. Energy pathways do not turn on and off; they get turned up or down. The largest obstacle in developing RSA is learning the how, when, and why a pathway might be altered. An understanding of these pathways would enable programming that could maximize repeated power development.

*2.1c Field-based Team Sports*

It is important to avoid using the term "anaerobic" when referring to team sports like football, hockey, and soccer. The term "anaerobic sport" implies a singular form of energy synthesis and jades the true functional effect of the human metabolic system during high-intensity activity. To a greater extreme, the term can bias readers and inhibit their ability to think about the material critically. A contributing factor to the success of "anaerobic" athletes is their aerobic energy pathway. Sports, in this sense, will be referred to as "field-based team sports," thus defining, more correctly, some of the true governing aspects of these sports.

## 2.2 The Energetic Pathways of Brief Maximal Work

*2.2a Adenosine Triphosphate*

The energy that fuels muscle comes from the hydrolysis of adenosine triphosphate (ATP). Human muscle can store approximately 20-25mmol/kg dry muscle (dm) of ATP at a time (Gaitanos et al., 1993). At peak turnover rate, estimated to be approximately 14 to 15 mmol/kg/dm/sec, ATP stores are sufficient to provide 1-2 seconds of maximal work before being exhausted (Glaister, 2005). As the intracellular level of ATP decreases, it is resynthesized via the integrated response of the other metabolic pathways.

2.2b Phosphocreatine

Phosphocreatine (PCr) appears to play the largest role in providing energy during short, maximal work bouts (e.g., during RSE). It is theorized that the increased concentration of intracellular ADP, produced from ATP hydrolysis, up-regulates the PCr pathway (Brooks, Fehey, & Baldwin, 2005; McMahon & Jenkins, 2002). Phosphocreatine plays a crucial role in RSA by combining with ADP and $H^+$, a reaction catalyzed by creatine kinase, to form ATP and free creatine (Cr) (Brooks et al., 2005).

Research has found intramuscular PCr stores to be approximately 80 mmol/kg/dm, with a maximal turnover rate of 9 mmol/kg/dm/sec (Gerard, Mendez-Villanueva, and Bishop, 2011). Maximal PCr turnover rate is attained almost instantly at the start of high-intensity exercise, declining within 1.3 seconds of the onset of exercise (Maughan, Gleeson, & Greenhaff, 1997). These estimates make the PCr pathway ideal to buffer high ATP demand during high intensity, short duration activity.

*2.2c Glycogenolysis*

There are two energy substrates broken down by the glycolytic pathway in the muscle: glucose and glycogen (Brooks et al., 2004). Of the two, muscle glycogen is the preferred fuel during RSE (Spreit et al., 1987). A normal resting intracellular concentration of glycogen has been estimated to be approximately 300 mmol/kg/dm (Gaitanos et al., 1993). Glycogenolysis operates on a feed-forward loop, responding quickly to intracellular changes in the ATP/ADP+Pi flux (Brooks et al., 2005). The breakdown of glycogen begins at the onset of maximal exercise, reaching its peak turnover rate of 6-9 mmol/ATP/kg/dm/sec at approximately 5 seconds (Gastin et al., 2001; Paroline et al., 1999). When combined, glycogenolysis and PCr can maintain an ATP turnover rate of 11-14 mmol/ATP/kg/dm/sec for approximately 10 seconds (Glaister, 2011; Boobis, Williams, & Wootten, 1982). Glycogen should therefore be considered the main contributor to glycolytic flux during RSE.

*2.2d Oxidative*

The oxidative pathway is the smallest contributor in terms of direct ATP synthesis during RSE. This pathway resynthesizes ATP primarily through the oxidation of glucose. The intracellular storage form for the aerobic pathway is myoglobin ($MbO_2$). The $MbO_2$ content in human skeletal muscle is estimated to be 2 mmol $O_2$/kg/dm (Akeson, Biorck, & Simon, 1968), with this level rapidly decreasing at the onset of exercise. Research has shown that at a work rate equal to $VO_{2max}$, it takes upwards of 20 seconds to desaturate $MbO_2$ to 50% of its resting level (Mole et al., 1999; Richardson, Noyszewski, Kendrick, Leigh, & Wagner, 1995). It can therefore be assumed that work rates drastically above

$VO_{2peak}$, like those during RSE, would deplete intracellular $O_2$ stores much more quickly.

Bangsbo, Krustrup, Gonzalez-Alonso, and Saltin (2001) have estimated the aerobic pathway contributes, on average, 0.7 mmol ATP/kg/dm/sec during the first 5 seconds of exercise at a work rate equivalent to 120% of $VO_{2peak}$. Other studies, such as those by Parolin et al. (1999) have reported similar findings, with the aerobic pathway contributing approximately 1.3 mmol ATP/kg/dm/sec during the first 6 seconds of a 30-second Wingate test.

**2.3 The Physiology of Repeated Sprint Ability**

To meet the high energy demands of RSE, athletes have evolved a complex, yet well orchestrated set of energy pathways. The overarching goal of these pathways is to meet the energy demands placed on them by a much larger system − the human body. The beauty and efficiency within which the energy system of the human body functions can be elucidated by the fact that ATP concentration will remain relatively stable despite a more than a 1,000-fold increase in ATP utilization during a single, maximal sprint bout lasting less than 5 seconds (Baker et al., 2010). Some of that beauty gets lost when researchers attempt to define energy pathway interaction by confining it with labeling terms; terms like system, rate limiting, or inhibited. The language that is currently used to describe fatigue is detrimental to a person's ability to conceptually think about and understand the process of flow taking place during the up-regulation of energy flux within a cell. This stifles true understanding of a dynamic process, one that likely changes from person to person, by compartmentalizing it into a "one size fits all" model.

These terms imply that questions regarding energy system interaction should not be answered with a "yes or no," "on or off" response. If science has found anything, it is that absolute terms such as these should be completely abolished from the language used to describe energy system interaction; replaced instead with responses of "up," "down," "maybe," or "not sure." Metabolic interactions during any mode of repeated sprint exercise (RSE), regardless of duration or intensity, will show energy contribution from each of the three energy pathways (Gastin, 2001; Spencer et al., 2005; Baker et al., 2010). What follows is a summary of the parameters science has discovered regarding energy pathway interaction and RSE energy flow, expressed by two diagrams that attempt to combine those parameters to show the dynamic flow of repeated sprint metabolism.

*2.3a Phosphocreatine Pathway*

Intracellular PCr cannot meet the full demand for ATP during RSE, and begins to decline shortly after the start of maximal exercise (Maughan, Gleeson, & Greenhaff, 1997). It is estimated that PCr can supply 50% of the total ATP production during a maximal 6-second sprint (Gaitanos, Williams, & Boobis, 1993). Phosphocreatine stores have been reported to fall to 35-55% of their resting level after a 6-second sprint (Gaitanos et al., 1993; Dawson et al., 1997). Longer duration maximal exercise has been found to further deplete PCr stores in muscle. After two 30-second bouts of maximal cycling, interspersed with 3.8 minutes of recovery, Bogdanis et al. (1996) found intracellular PCr levels had been depleted to 16.9% and 15.4% of their resting values, respectively.

While the total energy production from PCr decreases during RSE, the relative

percentage of PCr contribution to total energy production appears to stay constant, or increase slightly (Nevill et al, 1997; Yoshida, 2002). During a repeated sprint protocol consisting of six 10-second sprints, Holmyard et al. (1988) found the relative percentage of energy produced from PCr increased, from 46% to 49% on the tenth sprint when compared to the first sprint. In addition, a study by Gaitanos et al. (1993) found PCr to contribute 49% of the total ATP produced during the initial 6-second bike sprint of a RSA protocol, with it contributing 80% of the total ATP used during the final, tenth sprint. This is not to say that total energy output increased by over 30%, but rather that the relative total energy contribution from the PCr pathway increased (McMahon & Jenkins, 2002). This implies PCr is a robust, fatigue resistant energy source when recovery time is below the optimal amount to return the cell to a homeostatic environment.

Studies have shown PCr resynthesis to follow a biphasic pattern of recovery after intense muscular activity (Baker et al., 2010; Gaesser & Brooks, 1984; McCann, Mole, & Canton, 1988; Bogdanis et al., 1995; Nevill et al., 1997; Harris et al., 1976). The slow phase of PCr recovery is likely affected by the pH of the muscle (McCann, Mole, & Canton, 1988; Harris et al., 1976; Sahlin et al., 1979). As the intracellular pH drops, the associated rise in $H^+$ appears to effect creatine kinase equilibrium. Sahlin et al. (1975) found that there was a significant correlation ($p < 0.01$) between muscle pH and creatine kinase equilibrium, with that correlation becoming stronger as the muscle became more acidic. The slow phase of recovery appears to have a half time of approximately >170 seconds (Harris et al., 1976), with some studies showing up to 15 to 25 minutes for full

recovery (Baker et al., 1993).

The fast phase of PCr recovery appears unaffected by the pH of the muscle, with several studies reporting high rates of PCr resynthesis when intracellular pH is low, or continuing to fall (Takahashi et al., 1995; Cooke, Peterson, & Quinney, 1997). Instead, it appears that the fast phase is dependent on $O_2$ availability (McMahon & Jenkins, 2002). Harris et al. (1976) was the first to establish this link by occluding blood flow to the thigh using a pneumatic cuff during both dynamic and isometric exercise to fatigue. During a 6-minute recovery period, the cuff would either keep the muscle completely occluded or be deflated slightly to allow a level of ischemia. The results found that PCr resynthesis was completely abolished during occlusion and drastically suppressed when ischemic. Other studies are in accord with the results of Harris et al. (1976), finding the fast phase of PCr synthesis to be dependent on $O_2$ availability (Sahlin et al., 1979; Quistorff, Johansen, & Sahlin, 1992; Taylor et al., 1983). The recovery rate of the fast phase of PCr appears to have a half time of approximately 21 seconds (Harris et al., 1976).

*2.3b Glycolytic Pathway*

Glycogen is broken down quickly during RSE by phosphorylase *a* (glycogen phosphorylase) (Kjaer et al., 1986). Activation of this enzyme is initiated by one of two mechanisms: 1) hormone-mediated response or 2) contractile/metabolite response (Brooks et al., 2005). The hormone-mediated response, while an efficient pathway, operates on a feedback loop and has been calculated to be too slow to account for the observed glycolytic flux during short, maximal exercise (Paronlin et al., 1999). The contraction/metabolite-mediated response is quicker to respond to the energy demands of

RSE and accepted by researchers as the most likely signaling pathway of glycogenolysis (Westerblad & Allen, 2002; Gaitanos et al., 1993; Brooks et al., 2005).

The up-regulation of glycogenolysis likely comes from a rise in intracellular $P_i$, a substrate required in the conversion of phosphorylase *a* and a by-product of ATP synthesis (Jost & Rickenberg, 1971). Research by Crowther et al. (2002) found that glycolytic flux was activated at approximately the same $P_i$ and ADP concentrations during repeated bouts of maximal exercise in subjects. Further argument adding weight to the importance of the muscle contraction to the up-regulation of glycogenolysis comes from studies that found no significant increase in glycolytic flux despite a direct injection of epinephrine into the muscle (Crowther et al., 2002; Brooks et al., 2005).

The relative contribution from glycogenolysis begins to fall rapidly after it reaches its peak turnover rate. This reduction in flux, however, does not appear to be the result of insufficient substrate. Gaitanos et al. (1993) found that total energy contribution from glycolysis fell from 44.1% during an initial 6-second sprint to 16.1% on the tenth sprint during a 10-bout RSA testing protocol despite only finding a 37% decrease in pre-test glycogen levels. From these findings, it can be inferred that the amount of muscle glycogen in a muscle, when within normal levels, is not the rate-limiting factor in glycogenolysis.

The rate of recovery for glycolysis is slow, estimated to have a half time of approximately nine minutes, following a mono-exponential pattern of recovery that is much slower than that of PCr (Glaister, 2005). The results of Gaitanos et al. (1993) previously outlined point to a set point of maximal glycolytic usage; the decrease is likely

caused by other intracellular factors acting to limit the continued production of ATP from glycogenolysis. Many researchers have proposed that glycogenolysis is its own limiting factor during RSE (Ren, Broberg, Sahlin, & Hultman, 1990; Bangsbo, Graham, Kiens, & Saltin, 1992; Crowther et al., 2002). The breakdown of glycogen builds metabolic waste in the muscle ($H^+$) causing a fall in intracellular pH, which has been found to inhibit the two main rate limiting enzymes of glycogenolysis; phosphorylase *a* and phosphofructokinase (PFK) (Parolin et al., 1999).

It has been argued by some that, much like its effects on PCr resynthesis and creatine kinase, a high aerobic metabolism will speed up lactate clearance during recovery, raising the pH of the muscle more quickly, thus reducing the inhibitory effects of $H^+$ on the rate limiting enzymes of glycogenolysis: phosphorylase *a,* hexokinase and PFK (Spriet et al., 1987; Brooks et al., 2005; Crowther et al., 2002).

*2.3c Oxidative Pathway*

The oxidative pathway is slow to respond to RSE (Bassett & Howley, 2000). Research has found that oxidative metabolism contributes approximately 3-8% of total energy during a 6-second sprint (McGawley & Bishop, 2008; Spencer et al., 2005). That percentage, however, appears to increase when subsequent sprints are performed with minimal recovery, or the total length of the sprint is increased. Kavanagh and Jacobs (1988) calculated that aerobic metabolism accounted for 16 to 18.5% of total energy used during a 30-second Wingate test. A study by Bogdanis et al. (1996) found that oxidative metabolism contributed 29% of total energy used during a 30-second Wingate, increasing to 44% of total energy when a second Wingate was performed 4 minutes later. This

implies that the oxidative pathway, while slow to respond, plays a critical role in supplying ATP during maximal exercise.

The immediate need for oxygen during RSE is met by oxygen binding myoglobin ($MbO_2$) within the muscle cell. The $MbO_2$ content in human skeletal muscle is estimated to be 2 mmol $O_2$/kg/dm (Akeson, Biorck, & Simon, 1968), with this level rapidly decreasing at the onset of exercise. Research has shown that at a work rate equal to $VO_{2peak}$, it takes upwards of 20 seconds to desaturate $MbO_2$ to 50% of its resting (Mole, Chung, & Tran, 1999; Richardson, Noyszewski, & Kendrick, 1995). This would imply that there is sufficient oxygen within the cell to maintain maximal oxidative metabolism for one maximal sprint bout.

During RSE, however, due to the variable nature of sport and $O_2$ demand, $MbO_2$ cannot continually meet oxygen demand. To explain how the oxidative pathway attempts to do this, researchers developed a term to describe the dynamic changes that take place within an athlete's oxidative pathway during exercise: $O_2$ kinetics (Raminini et al., 2009). Oxygen kinetics describes the transition of a person's oxidative system from rest to exercise. This transition and its effect on $O_2$ kinetics can vary greatly based on the intensity and duration of exercise. For example, when exercise is performed at a intensity below an athlete's lactate threshold (50-70% $VO_{2max}$), $VO_2$ increases quickly to a steady state level to meet demand. If an athlete works out at a higher intensity (100% $VO_{2max}$), there is a shift seen in the $O_2$ kinetics after a few minutes to a slow component; this change either delays the attainment of a steady state or pushes $VO_2$ to its maximum (Gastin, 2001). In either case, the combined effect of an athlete's maximum cardiac

output (Q) and arteriovenous oxygen difference (a-$\tilde{v}O_2$ diff) dictates the effectiveness and speed with which their $O_2$ kinetics meet the demands placed on their system during RSE.

In addition to a direct increase in ATP contribution during RSE, an increased oxidative capacity would provide more energy during maximal exercise bouts, reducing the strain on the PCr and glycolytic pathways. This, in turn, would theoretically reduce the amount of metabolite accumulated during exercise, reducing the amount needing to be cleared from the muscle before the next maximal work bout (Dupont, Millet, Guinhouya, & Berthoin, 2005).

**2.4 Mechanisms of Fatigue**

Repeated sprint bouts are affected by fatigue. Fatigue, in this sense, is defined as a decline in peak or mean power over a series of repetitive sprints. The level of fatigue seen during the work periods of RSE is correlated with the duration of their associated recovery period (Balsom et al., 1992). This makes sense when compared to what is known about energy pathway integration and their main function, to return the muscle cell to homeostasis after maximal work bouts. If the system is given a long time to recover, it will do so completely. If its recovery is limited, it will be forced to function in an ever-increasing state of fatigue.

There is no single contributing factor to RSA ability. Research indicates a inverse correlation between peak power and fatigue (Girard et al., 2011). As athletes' initial peak power output (PPO) increases, their successive sprints seem to fatigue more quickly when compared to athletes with lower PPO's. This correlation is likely due to muscle

recruitment response and excessive metabolite accumulation due to slow fiber type dependent clearance mechanisms (Mendez-Villanueva, Hamer, and Bishop, 2008). Different types of muscle fiber have been reported to have "fiber-type-dependent differences in the usage of high-energy phosphates with greater phosphocreatine reduction in fast twitch fibers than in slow-twitch fibers (Girard et al., 2011)." Athletes who produce more power likely recruit more type IIAx muscle fibers. Type IIAx fibers consume more ATP than type I fibers and have a much lower mitochondrial density, limiting their ability to oxidatively clear the metabolite byproduct from those reactions. While there is considerable debate over the specific cause of fatigue and its respective effects on RSA performance, most researchers agree that fatigue is associated primarily with one of, or a combination of, four intramuscular conditions: 1) PCr availability; 2) glycogen availability; 3) inorganic phosphate accumulation; and 4) an increased level of muscle acidosis.

*2.4a Phosphocreatine Availability*

It is widely accepted that one of the main fatigue-causing factors in RSA is PCr availability (Girard et al., 2011). There is a strong parametric correlation between PCr levels and power production of a muscle during high-intensity exercise (Miller et al., 1987; McMahon & Jenkins, 2002; Bogdanis et al., 1995; Bogdanis et al., 1996;). Research has found a similar association between PCr recovery following an initial maximal work bout and the recovery of peak power in subsequent bouts (McMahon & Jenkins, 2002; Sahlin & Ren, 1989; Nevill et al., 1996; Bogdanis et al., 1996). Numerous studies, such as one by Dawson et al. (1997), have shown the resynthesis curve for PCr

tracks very closely with the time-course recovery curve for power output. Researchers found that longer rest periods between maximal 6-second sprint bouts correlated to both increased PCr resynthesis as well as increased power production in subsequent sprints. Furthermore, a study by Bogdanis et al. (1995) found strong correlations ($r = 0.71$-$0.86$; $p < 0.05$) between resynthesis of PCr and the restoration of peak power output and mean power after 90 seconds of recovery.

Hiroven et al. (1987) looked at the breakdown of PCr and its association with sprint speed in the 40, 60, 80, and 100m dash. Seven subjects performed two sprints on two separate testing days, taking muscle biopsies pre- and post-sprint for each trial. Sufficient time was given between sprint bouts to allow for complete recovery of the muscle. For analysis, the researchers divided the subjects into two groups: 1) sprinters who achieved a maximal speed of $10.07 \pm 0.13$ m/sec and 2) sprinters who achieved a maximal speed of $9.75 \pm 0.10$ m/sec. Their findings demonstrated that sprint performance correlated with the depletion of PCr stores in the muscle. In addition, the researchers concluded that severe depletion of PCr occurred between 5 to 7 seconds during maximal sprinting, at which point maximal running speed was seen to decline. The results of Hiroven et al. (1987) show two findings: 1) sprinters who can better utilize more of their intracellular PCr stores are faster, more powerful sprinters; and 2) when PCr levels are significantly depleted there is an associated drop in performance. These results imply that a diminished rate of PCr resynthesis during RSE could dramatically affect the performance by increasing the rate of fatigue.

Additional weight to the importance of PCr availability is given by research

showing that supplementing with creatine reduces the rate of fatigue between multiple sprint bouts (Kendall et al., 2009; Kreider, 2003). Creatine in the cytosol of the muscle cell is rephosphorylated by mitochondrial creatine kinase (CK) to form PCr and intramitochondrial ADP (Brooks et al., 2005). By increasing the intracellular components needed to form PCr, researchers theorize it up-regulates the rate of PCr resynthesis between work bouts.

A study by Yquel et al. (2002) found that ingesting 20g of creatine/day for six days increased power output by 5% during a RSA protocol, as well as increased PCr resynthesis during a 10-minute recovery period. Increasing free creatine concentrations within the muscle has three advantages for field-based team sport athletes performing RSE: 1) it increases the amount of intracellular PCr available for hydrolysis, thus increasing intracellular ATP (Soderlund, Balsom, & Ekblom, 1994; Greenhaff et al., 1993; Balsom, Soderlund, & Ekblom, 1994); 2) creatine acts to buffer pH by utilizing $H^+$ during the creatine kinase reaction (McArdle, Katch, & Katch, 1991); and 3) it acts as a metabolic buffer, lowering the intracellular level of inorganic phosphate ($P_i$). Lowering intracellular $P_i$ has been theorized to limit inhibition of the contraction-coupling of the muscle and possibly reduce the glycolytic rate by reduced signaling to phosphorylase *a* and PFK (Volek & Kraemer, 1996).

Peyrebrune et al. (1998) compared 14 elite level male swimmers in a double blind procedure. All swimmers performed a single 50-yard sprint and a RSA test, consisting of 8x50-yard intervals with 90 seconds rest between sprints, before and after a 5-day period of supplementing with either creatine (9g creatine + 4.5g maltodextrin + 4.5g

glucose/day) or a placebo (18g glucose). The researchers found no change in single 50-yard sprint time. During the RSA test, however, the average times increased during each trial, but overall percent decrement was improved in the experimental group. Although the percent decrement scores of both groups improved, only those of the creatine supplement group were significant (control = 12.7 ± 5.7% pre vs. 11.0 ± 5.5% post; creatine = 15.7 ± 4.3% vs. 10.0 ± 2.5%; $p < 0.05$). The researchers concluded that 9g of creatine ingestion per day for 5 days improved repeated-sprint swimming.

The correlation between creatine supplementation and improved RSA is not a universal finding. Several studies have been published that found no correlation between creatine supplementation and RSA in athletes. A study by McKenna et al. (1999) investigated the effects of creatine supplementation on intermittent sprint performance in fourteen, recreationally active college students. The subjects were split into two groups, with the experimental group receiving a creatine solution (5g creatine + 5g dextrose/day) and the control group a placebo (5g dextrose/day) for five days. Both groups performed a a pre- and post-test, consisting of 5x10-second sprints with 180, 50, and 20 seconds rest between bouts, respectively. After analysis, the researchers found that there was no improvement in intermittent sprint performance between groups, and that creatine supplementation was not correlated with fatigue resistance during RSA.

The discrepancy in these findings can be rationalized by viewing the participants as "responders" and "non-responders." It has been reported that PCr concentration varies by fiber type. Demant and Rhodes (1999) reported that PCr content at rest in type I and type II fibers was significantly different ($p < 0.05$) at 73.1 ± 9.5 and 82.7 ± 11.2 mmol/kg/dm,

respectively. In addition, type II fibers appear to rely more heavily on PCr for energy supply during maximal exercise (Gray, Soderlund, & Richardson, 2008). For example, Karatzaferi et al. (2001) found that 10 seconds of maximal bike sprint decreased the PCr content in type I, IIa, IIAx, and IIXa by 45, 53, 62, and 59%, respectively. As a result of these findings, people who have a higher concentration of type II muscle fibers have come to be known as "responders," because they respond better to increased levels of creatine compared to those with higher percentages of type I muscle fiber (Demant & Rhodes, 1999). Field-based team sport athletes, i.e. those requiring high power outputs for short periods of time (<60-seconds), have a higher propensity to type II muscle fiber (Korhonen et al., 2006). With that in mind, when comparing the studies of Peyrebrune et al. (1998) and McKenna et al. (1999), which tested a sprint-based athletic group and an untrained population, respectively, one could expect the sprint-based athletic group to show better results from creatine supplementation.

The theory that increased intracellular creatine could reduce fatigue by mediating the rate of glycolytic flux and reducing intracellular $P_i$ is still heavily debated among researchers, with substantial research supporting both sides. Some researchers have found no difference in blood lactate levels after RSE between experimental and control groups, pre- and post- test, despite a reduced rate of fatigue in the experimental group (Peyrebrune et al., 1998; Bosco et al., 1997). These findings imply that though PCr synthesis is increased, it does not inhibit the rate of glycolytic flux. On the other hand, researchers have found creatine supplementation decreases the level of intramuscular lactate, measured after a RSE protocol, implying that it somehow affects the rate of

glycolytic flux (Soderlund, Balsom, & Ekblom, 1994; Balsom, Soderlund, & Ekblom, 1994).

One large confounding factor that must be taken into account is the lack of standardization between these studies in regards to creatine supplementation and RSA testing parameters. Some studies supplemented with 20g of creatine (Balsom et al., 1994), whereas others only used 9g (Peyrebrune et al., 1998). One study had rest intervals of 30 seconds (Soderlund et al., 1993) with another allowing 60 seconds of recovery (Kendall et al., 2009). Changes in duration and intensity have drastic effects on energy system integration during RSE. Without more rigid testing guidelines, this brings in to question the construct validity of these studies. Despite these conflicting reports, the majority share of the research would support the notion that PCr availability and fatigue are more than a coincidental finding.

*2.4b Glycogen Availability*

Unlike PCr, the amount of glycogen stored within a muscle is unlikely to be a limiting factor in RSA. Studies have found muscle glycogen levels to be a limiting factor in time to exhaustion during sub-maximal exercise lasting longer than 60 minutes (<80% $VO_{2peak}$; Balsom et al., 1999; Hargreaves et al., 1995), however no studies have found this same trend when exercise was performed at high-intensities (>90% $VO_{2peak}$) (Maughan & Poole, 1981; Symons & Jacobs, 1989).

There are exceptions to glycogen storage limiting RSA, such as if extreme carbohydrate restriction takes place. One study performed an extrapolated calculation based on its findings of glycolytic rates during an RSA protocol and estimated that an

athlete would need a starting intracellular glycogen level of only 75 mmol/kg/dm to see any substantial performance decrement (Spencer & Katz, 1991); however, this is well below normal intramuscular glycogen levels, which are typically around 300 mmol/kg/dm (Glaister, 2005). A study by Spencer & Katz (1991) examined the difference in glycolytic rate based on resting muscle glycogen levels before exercise. Eight subjects performed two cycling trials at 95% of their $VO_{2peak}$, the first trial was performed to fatigue and the second trial was performed for the same duration and workload as the first trial. Before the first trial, subjects lowered their glycogen stores through a combination of exercise and dietary restrictions. The second trial was performed in a supercompensated state, with subjects drastically increasing their muscle glycogen stores, again by diet and exercise manipulation. The low glycogen (LG) trial found a decrease from a resting state of 201 ± 31 mmol/gluc/kg/dm to 105 ± 28. The high glycogen (HG) trial decreased from 583 ± 40 to 460 ± 49. While the starting values were very different, the calculated rate of glycogenolysis was not significantly different between groups (LG = 88 ± 17, HG = 106 ± 43 mmol/gluc/kg/dm; $p > 0.05$) (Spencer et al., 1991).

Bangsbo et al. (1991) found that glycogen utilization was not related to pre-exercise intracellular levels. The study found that muscle lactate production (a sign of glycolytic rate) was similar across all groups, correlating to an average glycogen depletion of 25.9 ± 0.4 mmol/kg/dm. This suggests a set point based on metabolite accumulation where glycolytic enzymes are downregulated and decrease the rate of glycogenolysis, limiting ATP production, and subsequently inducing fatigue (Parolin et

al., 1999). From these results researchers have concluded that intracellular glycogen stores are not a major limiting factor in RSE, except in extreme conditions (Ren et al., 1990).

With glycogen concentration not likely affecting RSE fatigue, researchers developed different theories, instead arguing the effects of glycolytic flux on RSE. Subjects with the highest glycolytic rates during initial sprint performance have been found to have the greatest drop-off in power output during subsequent sprints during RSE (Bogdanis et al. 1995; Bishop, Edge, & Goodman, 2004). These studies also found that the same subjects had a much higher initial peak power output (PPO), average power output (APO), and final power output (FPO) when compared to subjects with lower glycolytic rates.

The mechanism that causes the decline in glycolytic rate is unclear. It has been hypothesized that a rapid depletion of muscle glycogen occurs, limiting the continued contribution of ATP from glycogenolysis (Hargreaves, McConnell, & Proietto, 1995). A second hypothesis offered by Parolin et al. (1999) is glycolysis itself is its own limiting factor; the breakdown of glycogen builds metabolic waste in the muscle ($H^+$) causing a fall in pH and inhibiting the rate limiting enzymes of glycogenolysis, specifically phosphorylase *a* and PFK, which break down glycogen to form ATP.

A study by Gaitanos et al. (1993) examined glycolytic flux in a muscle during a RSA protocol consisting of ten, 6-second bike sprints with 30 seconds of recovery between sprints. They found glycogen breakdown to be significantly greater during the first sprint, with glucose being the main glycolytic substrate used during the final sprint.

The authors reported an 11-fold and 8-fold reduction in glycogenolysis and glycolysis, respectively, from the first to last sprint. During the first sprint, it was calculated that glycogenolysis provided 44% of the total energy pool, while contributing only 16% on the final, tenth sprint. This was surprising as they found total glycogen degradation to be only 37% of its resting level. From these findings, it can be inferred that the amount of muscle glycogen in a muscle, when within normal levels, is not the rate-limiting factor in glycogenolysis. While the study shows that the contribution of ATP from glycolytic flux decreases during RSE and limits total energy production during repeated bouts, this decrease is likely caused by other intracellular factors acting to limit the continued production of ATP from glycogenolysis, resulting in fatigue.

*2.4c Acidosis*

Muscle acidosis has been shown to be significantly correlated to a decrease in muscle force production (Vaughn-Jones, Eisner & Lederer, 1987; Cady et al., 1989; DeGroot et al., 1993; Miller et al., 1988). Similar to the findings of the association between PCr and fatigue, muscle pH has shown a non-parametric correlation to power output during RSA tests; as pH falls, power output decreases (Dawson et al., 1978). This association has been solidified through research that has found high levels of acidosis to have negative effects on both the isometric force production and the standard free energy release from ATP hydrolysis (Westerblad, Allen, and Lannergren, 2006; Chase & Kushmerick, 1988; Gote & Nosek, 1989). In addition, high $H^+$ accumulation may inhibit ATP production from the glycolytic pathway by inhibiting the rate-limiting enzyme PFK (Girard et al., 2011). Finally, acidosis has been shown to affect the function of creatine

kinase, the enzyme associated with PCr synthesis and the slow phase of PCr recovery (McMahon and Jenkins, 2002).

Studies have found low pH may cause $Ca^{2+}$ insensitivity in the muscle, leading to a decline in maximal tension and shortening velocity (Godt & Nosak, 1989; Cook et al., 1988; Miller et al., 1988). A study by Donaldson et al. (1983) used skinned mammalian muscle fiber to look at the effects of pH on force production. It found that type I, type IIa, and type IIx fibers lost 12%, 25%, and 44% of their maximal force capacity as a result of acidosis (McMahon & Jenkins, 2002). One of the most important steps in the excitation-contraction coupling cycle of muscle is the binding of $Ca^{2+}$ to troponin (Brooks et al., 2005). High $H^+$ concentrations have been theorized to negatively affect this process by displacing $Ca^{2+}$ from troponin, resulting in a decreased responsiveness of the muscle (Stackhouse, Reisman, & Stuart, 2001; Miller et al., 1988; Lannergren & Westerblad, 1990).

Further evidence supporting acidosis to be a fatigue causing factor in RSA are reports that reducing intracellular levels of $H^+$ improve performance. Studies have shown regular RSE can improve $H^+$ buffering through lactate removal and the monocarboxylate transporter (MCT1) pathway, in turn decreasing muscle pH and lowering fatigue. A study by Thomas et al. (2005) found that several weeks of RSE training led to increased MCT1 expression. There was a significant correlation ($p < 0.05$) with increased blood lactate removal ability after a 1-minute all-out test and decreased fatigue as opposed to pre-trial tests.

Acidosis may also inhibit the slow phase of PCr recovery. A study by Arnold et al.

(1984) found that the rate of PCr recovery was dependent on the pH level of the muscle. In the study, two groups squeezed a rubber bulb of a sphygmomanometer at different intensities: 100mm Hg for the light exercise group and 500mm Hg for the heavy exercise group. Each group performed contractions until they were completely fatigued, with the light group being able to squeeze the bulb for approximately 270 seconds while the heavy group only lasted for approximately 150 seconds. Analysis found that PCr levels were depleted to 55.8 ± 8 and 33 ± 4% of resting levels in the light and high exercise groups, respectively, with a coinciding drop in pH to 6.88 ± 0.02 and 6.23 ± 0.08. While the total amount of PCr resynthesized during recovery was greater in the heavy exercise group, the rate of resynthesis over time was lower than that of the light exercise group. This correlation between PCr resynthesis rate and muscle pH led the researchers to conclude that PCr recovery, in part, depends on the acidity of the muscle. Other studies have found similar associations between $H^+$ and PCr recovery ($r = 0.71$ to $0.92$, $p < 0.05$) (Harris et al., 1976; Sahlin et al. 1979, Walter et al., 1997).

There is considerable evidence to support an inhibitory effect of $H^+$ on PFK, a rate limiting enzyme of glycogenolysis (Bishop, Edge, & Goodmann, 2004; Sahlin, Gorski, & Edstrom, 1990; Peters & Spriet, 1995). As the rate of glycogenolysis increases, there is an associated drop in pH and an increase in citrate within the muscle cell (Dobson, Yamamoto, & Hochachka, 1986). Their combined effect has been associated with downregulating PFK and inhibiting $Ca^{2+}$ uptake by the sarcoplasmic reticulum (Sahlin, Tonkonogi, & Soderlund, 1998). In that sense, glycogenolysis would appear to be a self-regulating pathway; its metabolic byproducts affect both the rate of key enzymes used in

the breakdown of glycogen and interfere with the force production ability of the muscle (McCartney et al., 1986; Metzger & Fitts, 1987; Spriet et al., 1989). While acidosis may not directly affect the contractile efficiency of the muscle, its affect on PFK appears to interfere with energy production, and subsequently may cause fatigue during RSE.

The energy available from ATP hydrolysis is variable, depending on several regulating factors that all change in a state of muscle acidosis: pH, $H^+$, and ADP concentration (Brooks et al., 2005). The standard free energy of ATP hydrolysis ($\Delta G^o$) is -7.3 kcal/mol. Some researchers have estimated that this could drop to -11 kcal/mol in extreme acidic conditions believed to be attainable by exercising muscle (Brooks et al., 2005).

Sodium bicarbonate ($NaHCO_3$) has been shown in some studies to reduce the rate of fatigue in RSE (Lavender & Bird, 1989). A study by Naughton & Cedaro (1991) found that there was no ergogenic effect in consuming 0.5 g/kg body mass of $NaHCO_3$ in regards to fatigue during a 10 or 30-second maximal sprint. A study by Bishop et al. (2004) found that ingestion of 0.3g/kg of bicarbonate ($NaHCO_3$), a component of the MCT1 shuttle, produced significant ($p < 0.05$) improvements in total work and power output in sprints 3, 4, and 5 in a series of six 10-second sprints with 30 seconds of recovery between bouts. The results of this study would suggest that increased $NaHCO_3$ improves extracellular $H^+$ buffering, reduce muscle acidosis and improve power development in repeated-sprint bouts.

Despite these findings, several points of contention exist among researchers that bring their validity and correlation to fatigue into question. First, high power outputs have

been seen in sprints where the subject had high levels of $H^+$ accumulation, casting doubt on acidosis being the major cause of fatigue during RSA (Glaister et al., 2008). The association between intracellular pH and fatigue is further questioned by the dissociation seen in research between the recovery curves of the two parameters: force production and pH. Force output in subsequent contractions recovers more quickly than pH does, according to several studies (Bodganis et al., 1995; Holmyard et al., 1994). To further bring into question the effect of low intracellular pH on muscle function, several studies have documented high force/power outputs while subjects are in extremely acidic conditions (Hitchcock, 1984; Sahlin & Ren, 1989).

Second, the research originally done on acidosis comes from a study in which skinned rabbit psoas muscle fibers were examined at temperatures below $15^o$ C (Dawson, Gadian, & Wilkie, 1978). This low temperature confounded the results by decreasing the pH to a level that would never been seen in normal human tissue. More recent studies have re-created these experiments at close to normal body temperatures of $30^o$ C (Pate et al., 1995; Westerblad, Bruton, & Lannergren, 1997). Their findings show acidosis has little, if any, effect on the shortening speed or fatigue of muscle.

*2.4d Inorganic Phosphate Accumulation*

While earlier research focused on acidosis as the main culprit in RSE fatigue, advances in technology, through P-Magnetic Resonance Spectroscopy (P-MARS), have led researchers to a new conclusion. Inorganic phosphate ($P_i$), once thought to be a harmless byproduct, is now considered to be the leading cause of fatigue during high-intensity muscular activity (Dalstedt, Katz, & Westerblad, 2001; Duke & Steele, 2000;

Kabbara & Allen, 2001). A study by Thompson and Fitts (1992) found that recovery of peak tetanic force correlated with total $P_i$ in the semitendinosus muscle of frogs. Increased intracellular levels of $P_i$ have been shown to inhibit the ability of the sarcoplasmic reticulum to release $Ca^{2+}$ during high intensity contractions and decrease force production through the impairment of cross-bridge cycling (Westerblad & Allen, 2002).

Force decline, an associated measure of fatigue, has been shown by some researchers to be triphasic in nature (Westerblad & Allen, 1991; Steele & Duke, 2003; Lannergren & Westerblad, 1990). Of all the possible factors that could likely influence rate of force production, $P_i$ appears to best fit the model. A study by Lanergren and Westerblad (1990) found that tetanic force in the flexor brevis foot muscle of a mouse did not decline in an expected, linear fashion. Instead, the researchers found that there was an initial rapid decline in tetanic tension (phase one), followed by a long period of nearly constant tension (phase two), with a fast final tension loss (phase three). After testing 26 samples, they found the average tension fell 15.4%, 10%, and 70% during phases one, two, and three, respectively. In addition, the researchers found that caffeine (15-25 mM) caused a rapid increase in tetanic force when applied to fibers during phase three, increasing force output from 29.8 to 82.5% of resting levels. Caffeine's effect on tension was much lower in phases one and two. From these results, the researchers concluded that there are two mechanisms influencing force decline during fatigue: 1) one reacts quickly to an intracellular rise in $P_i$, likely influencing the cross-bridge function of the myofilaments within the muscle and 2) another takes longer to manifest, but has a much

larger effect on total fatigue, with the likely cause being $P_i$'s infiltration of the sarcoplasmic reticulum (SR) and inhibition of the release of $Ca^{2+}$ into the cell.

Further support for the $P_i$ fatigue model comes from evidence that populations tested that lacked the enzyme creatine kinase do not exhibit the usual rise in $P_i$ during exercise and show no impairment of early force production, $Ca^{2+}$ sensitivity, or $Ca^{2+}$ release from the SR (Allen, Kabbara, & Westerblad, 2002; Dahlstedt et al., 2001; Allen and Weserblad, 2001). A study by Dahlstedt et al. (2001) found that decreasing the rate of $P_i$ accumulation significantly slowed the rate of fatigue in muscle, measured as a decline in rate of force production. Researchers used genetically engineered mice, which had no detectable creatine kinase (CK) in their skeletal muscle, to slow the rate of $P_i$ accumulation. The prerequisite for accumulation of $P_i$ in the myoplasm is PCr breakdown, which is facilitated by the intracellular enzyme CK to form creatine and $P_i$. Without the presence of CK, the rate of $P_i$ accumulation within the intracellular space was reduced, showing an associated decline in the rate of fatigue.

Elevated levels of $P_i$ concentration may also inhibit a muscles ability to generate power by inhibiting SR function. A rise in intracellular phosphate has been associated with a decline in $Ca^{2+}$ release from the SR and is widely accepted as one of the leading causes of fatigue during RSE (Williams & Klung, 1995; Favero, 1999; Favero et al., 1997; Allen & Westerblad, 2001; Allen, Kabbara, & Westerblad, 2002). Favero et al. (1995) stated that inhibition of the ryanodine receptors within the SR reduced the release of $Ca^{2+}$ by decreasing the single-channel open probability and leading to a decline in tetanic force production.

Current research shows that high intracellular levels of $P_i$, which have been shown to occur as a result of a high rate of PCr degradation during RSE, diffuse into the SR and combine with $Ca^{2+}$ to form calcium-phosphate ($CaP_i$) (Fryer et al., 1995). This process has been termed the "calcium phosphate precipitation hypothesis." It proposes that, as phosphate in the myoplasm rises, it enters the SR and binds with $Ca^{2+}$, thus reducing free $Ca^{2+}$ within the SR and limiting its release during exercise, causing fatigue (Allen, Kabbara, & Westerblad, 2002). Support for this hypothesis can be seen in Dutka, Cole, and Lamb (2005), which found that the total amount of $Ca^{2+}$ released from the SR was reduced by 20% when intracellular $P_i$ rose to 30 mM in mechanically skinned mammalian muscle. In addition, Fryer et al. (1995) looked at mechanically skinned fast twitch (FT) and slow twitch (ST) fibers of rats to determine the effects of fatigue-like changes in the concentration $P_i$ on $Ca^{2+}$ flux of the sarcoplasmic reticulum (SR) and its resulting effect on the contractile properties of the myofilaments. They found that an increase in $P_i$ from 0 to 25, and then to 50 mM, after normalizing the results, reduced the maximal force production in both fiber types by 39 and 48%, respectively.

As was outlined earlier, several studies have found the rate of fatigue within muscle, as expressed as a decline in force production as a percentage of maximum, to be triphasic (Westerblad & Allen, 1991; Steele & Duke, 2003; Lannergren & Westerblad, 1990). In all of these studies, researchers found that the addition of caffeine to the muscle caused a rapid increase in tetanic force during the final, rapid phase of fatigue. It is widely accepted in the research that caffeine is a stimulant that increases $Ca^{2+}$ release from the SR by widening the channel operated by the ryanodine receptor (Steele & Duke,

2003). Furthermore, studies have shown that the addition of caffeine to fatiguing muscle has limited the rate of $Ca^{2+}$ clearance and force production (Lannergren and Westerblad, 1991; Westerblad and Allen, 1994). When all of the evidence is examined, it points to the inhibited release of $Ca^{2+}$ from the SR as being a major contributing factor to the decline in force production of muscle.

Further, $P_i$ may impair of the cross-bridge mechanism during muscle contraction. The correlating factors of fatigue during high-intensity exercise as they pertain to the myofibril component are reduced force production, decreased maximal shortening velocity, and slowed relaxation (Fitts, 1999; Jones, Ruiter, & Hann, 2006; Allen & Westerblad, 2001; Cook & Pate, 1985). Resting intracellular $P_i$ levels are estimated to be approximately 1-5 mM (Kushmerick et al., 1992). These levels can rise to 30-40 mM during high intensity exercise, possibly affecting all three of the mechanisms controlling force-generating ability (Cady et al., 1989). One of the original theories regarding muscle fatigue was that either $H^+$ or $P_i$ somehow affected the coupling of $Ca^{2+}$ to troponin, exposing tropomyosin and allowing cross-bridging to take place. This theory is now largely discredited for both metabolites because the rate of $Ca^{2+}$ dissociation from troponin is believed to be too fast to be a limiting factor during muscle contraction (Allen, Lannergren, & Westerblad, 1995). Researchers now believe it is not the limited availability of cross-bridge binding sites, but the energy bound state of the cross-bridge and the cycling rate of the myosin head that are likely causes of fatigue.

The decline in peak force production is explained in the research as a decline in the force produced per cross-bridge and/or the number of cross-bridges in a high-force

state at any given time (Fitts, 2008). High levels of intracellular $P_i$ may lead to a greater number of cross-bridges in a weakly bound state (ADP + $P_i$) (Stackhouse, Reisman, & Binder-Macleod, 2001). In addition, during the muscle cross-bridge cycle, myosin spends only 5% of the time in a strongly bound state (Sweeney & Houdusse, 2004). It is believed that the time it takes the cross-bridge cycle to transition from a weakly bound low-force state, back to a strongly bound high-force state is the key limiter in peak rate of force development (Fitts, 2008). This concept is known as the rate constant of tension redevelopment ($k_{tr}$). The $k_{tr}$ of type II muscle fiber has been estimated to be 7-times faster when compared to type I. In both cases, the time constant of the cycle appears to be closely related to $Ca^{2+}$ sensitivity of the fiber, with some studies showing longer $kt_r$ times with diminished $Ca^{2+}$ levels (Metzger & Ross, 1990). Some research has suggested that an increase in $P_i$ may also result in the slower dissociation of actin from myosin during fatigue, lengthening the time spent in a weakly bound state (Westerblad & Allen, 1993; Weserblad & Allen, 1994).

**2.5 The Influence of Oxygen Availability on Repeated Sprint Ability**

One of two stories is often told in physiology textbooks linking oxygen availability to repeated sprint ability (RSA). The first goes something like this -- the rate of phosphocreatine (PCr) resynthesis is significantly correlated to power output; PCr resynthesis is an oxygen dependent reaction; thus aerobic capacity must be related to the ability to repeat high levels of power output. The second story talks about recovery -- metabolite accumulates during high-intensity exercise; oxygen debt builds; more oxygen entering the muscle cell clears inhibiting byproducts more quickly; fatigue is mitigated.

While several studies have found there to be no correlation between aerobic capacity ($VO_{2peak}$) and the ability to recover from high-intensity exercise (Bishop et al., 2004; Aziz et al., 2000; Cooke et al., 1997; Hoffman, 1997; Wadley & LeRossignol, 1998), the majority support oxygen availability as a crucial component to RSA (Collander et al., 1988; McMahon & Wenger, 1998; Short & Sedlock, 1998; Tesch & Wright, 1983; Duponte et al., 2005; Buchheit & Ufland, 2010; Tomlin & Wenger, 2002; Haseler et al., 1999; Trump et al., 1996; Dupont et al., 2010; DaSilva, Guglielmo, & Bishop, 2010; Edge et al., 2005). At this time, there is no clear-cut explanation or hypothesis of how to rationalize the discrepancies of these findings, yet several explanations have been postulated as possible causes -- a minimal level of aerobic capacity required to observe effects, lack of standardization and definition of parameters of RSA tests, and relative homogeneity of subjects (Carey et al., Girard et al., 2011).

Increasing the rate of oxygen delivery during RSE appears to allow for an improved rate of recovery. Tomlin and Wenger (2002) examined the relationship between $VO_{2peak}$ and oxygen consumption during high-intensity, intermittent exercise. Subjects for the study were assigned to either a low aerobic group (LOW, $VO_{2peak} = 34.4 \pm 2.4$) or a moderate aerobic group (MOD, $VO_{2peak} = 47.6 \pm 3.8$). $VO_2$ of each subject was measured while they performed 10x6-second maximal sprints with 30 seconds of passive recovery on a cycle ergometer. The results of the study found that while both groups generated similar peak power outputs on the first sprint, the MOD group had a significantly smaller decrement score over the ten sprints than the LOW group ($8.8 \pm 3.7\%$ compared to $18.0 \pm 7.6\%$; $p < 0.02$). From these findings, the researchers concluded that the increased

aerobic capacity of the MOD group enabled them to resist fatigue during intense intermittent exercise better than the LOW group.

McMahon and Wenger (2002) came to a similar conclusion after having 20 University level rugby players perform 6x15-second maximal intensity sprints with 90 seconds of active recovery on a cycle ergometer. They found a significant relationship between $VO_{2peak}$ and percent drop-off in both mean and peak power during bouts five and six compared with bout one (mean power: $r = -0.49$, $p < 0.03$; peak power: $r = -0.62$, $p < 0.002$). Gaitanos et al. (1993) found the average decline in power output, between sprint one and ten in a 10x6-second cycle sprint RSA test, to be 27% when compared to the first sprint. This modest decline in power output occurred in spite of their finding a 64% decline in anaerobic ATP production during the final sprint from its resting level. These findings show that $VO_{2peak}$ plays an important role in both the recovery and maintenance of peak power over repeated, subsequent bouts of maximal effort exercise.

Other research to examine the oxygen/RSA relationship has found that a high $VO_{2peak}$ may not be the only factor to ensure optimal RSA (Dupont et al., 2010). Studies have demonstrated faster $O_2$ kinetics are related to better RSA when compared as a percent decrement score (% dec) of RSE (Dupont et al., 2010; DaSilva et al., 2010; Buchheit & Ufland, 2011; Dupont et al., 2005). Based on these results, researchers have concluded that enhanced $O_2$ kinetics, both on-transient and off-transient, may aid in replenishing $MbO_2$ stores, quicken the resynthesis of PCr, improve lactate clearance, as well as speed the removal of $H^+$ and $P_i$ (Hogan, Richardson, & Haseler et al., 1999).

*2.5a On- and Off-transient Oxygen Uptake Kinetics*

The term "running economy" is not one commonly used to evaluate or describe an athlete's RSA. Running economy, in a general sense, is used as a measure of oxygen uptake needed to run at a given velocity (Bassett & Howley, 2000). In the world of endurance training, running economy has been used for decades to help explain some of the variability that exists between running distance times in athletes with similar $VO_{2peak}$ values. Conley and Krahenbuhl (1980) compared the 10km race times of 12 elite distance runners with their $VO_{2peak}$ (average 71.7ml/kg/min) and three steady state running paces (241, 268, and 295 m/min) which equated to approximately 60, 70, and 80% of their $VO_{2peak}$, respectively. The results showed that the relationship between $VO_{2peak}$ and 10km time was r = 0.12. The correlations between steady state and 10km time were r = 0.83, 0.82, and 0.79 ($p < 0.01$). These results showed that 65.4% of the variance in race performance was accounted for by running economy. It stands to argue that a similar phenomena could occur within team-sport athletes, a "sprinting economy," that could help explain the variability found between $VO_{2peak}$ and fatigue during repeated sprint exercise (RSE).

The impact that $O_2$ kinetics has on RSA has been overlooked due to the general belief that it is directly associated with $VO_{2peak}$. Tomlin & Wenger (2002), for example, found a significant relationship between $VO_{2peak}$ and $O_2$ kinetics during a RSA test (r = 0.78, $p < 0.002$). Other research, however, has pointed to discrepancies between the two variables. For example, DaSilva et al. (2010) demonstrated that RSA was more strongly correlated with $O_2$ kinetics than $VO_{2peak}$. In the study, 29 national level Brazilian soccer

players performed a graded exercise test to exhaustion and a RSA test consisting of 7x34.2-meter sprints interspersed with 25 seconds of active recovery. The results of the study found negative correlations between the athletes' percent decrement score (%Dec) and $O_2$ kinetics (r = -0.49) and $VO_{2peak}$ (r = -0.39). Rampinini et al. (2009) found a significant correlations between $VO_{2peak}$ and $O_2$ kinetics to high-intensity repeated exercise, however $O_2$ kinetics explained more of the variability in the model (r = 0.65 vs. -0.45). While both $VO_{2peak}$ and $O_2$ kinetics in these examples appear to have an effect on RSA and %Dec, it would appear $O_2$ kinetics is a better predictor of performance.

There is further evidence that oxygen kinetics may be improved independent of $VO_{2peak}$ (Green, 1997). Research has shown that the human body adapts differently to high-intensity versus low-intensity stress. A study by Edge, Bishop, Goodman, & Dawson (2005) compared the effects on repeated sprint ability of HIIT (2-minute intervals at $VO_{2peak}$ with 1 minute of recovery, starting at four intervals and progressing to ten) to those of moderate-intensity training (MIT) (70% of $VO_{2peak}$, starting at 12 minutes and progressing to 30) when matched for total work. Edge and his associates found that both training groups significantly improved their $VO_{2peak}$ (10-12%, $p < 0.05$) and lactic threshold (8-10%, $p < 0.05$). They also found that only the HIIT group had a significant increase in total work during the RSA test (13%, $p < 0.05$), with no significant improvement seen in the MIT group. While the underlying mechanism is not understood, the results clearly show that HIIT training somehow enhances $O_2$ kinetics better than conventional MIT training.

Further weight can be added to this argument from studies performed with patients

having metabolic myopathies that directly affect oxidative delivery and transition. For example, Grassi (2003) points out that subjects with McArdle's disease show an enhanced level of $O_2$ delivery, yet have a slower than normal $O_2$ kinetic response to exercise, a deficiency that would be limited at the muscle level. An improved sprinting economy could encompass two underlying physiological factors that likely contribute to variability in RSA literature: 1) $O_2$ on-transient kinetics; and 2) $O_2$ off-transient kinetics; both via increased or enhanced mitochondrial function (Saunders, Telford, & Hawley, 2004).

In research that has looked at running economy in endurance athletes, trained subjects appear to reach steady state sooner than untrained subjects (Morgan, Martin, & Krahenbuhl, 1999; Bassett & Howley, 2000). If this trend, seen at sub-maximal intensities, holds true during high-intensity exercise, faster $VO_2$ on-transient kinetics could reduce the metabolic strain on the active tissue by decreasing the oxygen deficit during sprinting (Hickson, Bomez, & Holloszy, 1978), while off-transient kinetics could speed up PCr resynthesis (Dupont et al., 2010), re-oxygenate myoglobin (Dupont et al., 2005), and/or increase the clearance rate of metabolite associated with fatigue (Bailey, Wilderson, DiMenna, & Jones, 2009). This rate of adjustment in oxidative metabolism during transitions from rest to maximal exertion could hold large implications for fatigue during RSE.

A study by Dupont, Millet, Guinhouya, and Berthoin (2005) found that $VO_2$ on-transient kinetics was significantly correlated to sprint times ($r = 0.80$, $p < 0.01$) during a RSA test consisting of fifteen 40-m sprints interspersed with 25 seconds of active

recovery at 50% of their maximal aerobic speed. Other studies have found instances where athletes were able to reach 90-100% of their $VO_{2peak}$ after only 30 to 60 seconds of maximal exercise; a feat requiring a fast rate of $O_2$ kinetics (Dupont et al. 2005; Kavanagh & Jacobs, 1988). A study by McKay, Peterson, and Kowalchuk (2009) looked at changes in $O_2$ on-transient kinetics during the transition from rest to moderate exercise after eight weeks of either high intensity interval training (8-12 x 1 min intervals at 120% maximal $O_2$ uptake interspersed with 1 min recovery) or continuous endurance training (90-120 min at 65% maximal $O_2$ uptake). The results found $O_2$ uptake time was reduced by 40% ($p < 0.05$) in both groups. The researchers concluded that the faster time was a quicker activation of muscle $O_2$ utilization and local adaptations in muscle blood flow.

A study by Dupont, McCall, Prieur, Millet, and Berthoin (2010) found that a decrease in %Dec score during a repeated sprint test was correlated to $O_2$ off-transient kinetic time ($r = 0.85$; $p < .0001$). Twelve male soccer players completed a graded exercise test and a RSA test consisting of seven 30-m sprints alternating with 20 seconds of active recovery. $O_2$ off-transient kinetics were modeled from two severe-intensity exercise bouts at 120% of the subject's maximal aerobic speed (MAS) until exhaustion. After each bout, subjects would sit in a chair and have their breath-by-breath $VO_2$ data analyzed for 6 minutes to evaluate recovery and off-transient $O_2$ kinetics. Other research by Buchheit & Ufland (2011) looked at the $O_2$ off-transient kinetics rate (reoxygenation, or reoxy rate) of muscle between two 15-second all-out 20-meter shuttle sprints with 15 seconds of passive recovery between bouts. Researchers used a portable near-infrared spectroscopy (NIRS) apparatus to evaluate changes in tissue oxyhemoglobin,

deoxyhemoglobin, and total hemoglobin. The Reoxy rate was found to be significantly correlated to %Dec ( r = -0.52), leaving researchers to conclude that $O_2$ kinetics play an important role in RSA.

One thing is certain regarding $O_2$ delivery to the muscle – it is dynamic. A study by Buchheit, Abbiss, Peiffer, and Laursen (2012) found that both $O_2$ on- and off-transient kinetics showed significant changes between sprints of an RSA test (6x30 second cycling sprints interspersed with 2 minutes of passive recovery). They found that muscle deoxygneation and Reoxy rate increased throughout the sprints ($p < 0.001$) and were significantly correlated to time to 90% of $VO_{2peak}$ (r = 0.68, $p < 0.05$). Further, the test found that performance on the RSA test was not significantly related to $VO_{2peak}$ ($p > 0.05$). These results show that repeated sprints elicit greater muscle $O_2$ extraction with successive repetitions. Further analysis of these findings implies that it could be possible to have inter-player variability, with certain athletes able to increase their $O_2$ kinetics earlier during repeated sprints compared to other athletes. This would give them an advantage by mitigating the onset of fatigue to a later set point compared to their peers. Despite these findings, it is still too early to state that $O_2$ kinetics accounts for all the variability seen in RSA research.

*2.5b Phosphocreatine Recovery Kinetics*

While its direct energy contribution during RSE is heavily debated, most researchers agree that aerobic metabolism is an important factor in regenerating PCr during rest periods between sprint bouts. Walter et al. (1997) found that the rate at which PCr is resynthesized after exercise is related to the oxidative capacity of the muscle.

Longer rest bouts equate to more time for $O_2$ to enter the muscle. A study by Blei, Conley & Kushmerick (1993) showed that PCr resynthesis is dictated by oxygen supplied to the muscle by electrically stimulating the flexor muscle in the finger and wrist of eight subjects during normoxic and ischemic conditions. The ischemic condition showed that after 2 minutes of post-stimulation there had been no net PCr resynthesis in the muscle compared to the normoxic group. Once oxygen was restored to the muscle, however, PCr resynthesis recovered at its normal exponential rate, thus linking the importance of oxygen to PCr resynthesis.

Further research has shown that not only is oxygen essential for PCr recovery, but that the amount of $O_2$ available also plays a large role in the rate of resynthesis. A study by Hasler et al. (1999) showed that subjects' PCr recovery, after 5 minutes of repeated maximal plantar flexions of the foot, was enhanced by inspiring hyperoxic air and inhibited when inspiring hypoxic air, when compared to normoxic conditions. Other studies, such as Quistorff et al. (1992), Sahlin, Harris, & Hultman (1979), and Taylor et al. (1983) have shown that, during ischemic conditions, the rate of PCr resynthesis is inhibited when compared to normoxic conditions. These findings, when considered with the work by Harris et al. (1976), support the theory that the ATP used to rephosphorylate creatine is derived solely from the aerobic metabolic pathway. The implication is that a better trained oxidative system would enhance repeated bouts of maximal-effort activity by increasing the rate of PCr resynthesized during the fast phase of recovery.

There is research that shows recovery modality can affect performance during RSE by reducing $O_2$ available for PCr recovery, thereby reducing PCr kinetics (Spencer et al.,

2008; Castagna et al., 2008; Spencer et al., 2006; Buchheit et al., 2008; Jougla et al., 2010). Spencer et al. (2008) had two groups of subjects; a moderate (MI) and a low-intensity (LI) active recovery group perform a RSA test consisting of 6x4-second maximal sprints with 25 seconds of recovery. Recovery intensity for the MI and LI group was 35 and 20% $VO_{2peak}$, respectively. Muscle biopsies of the vastus lateralis were taken pre- and post- trial to determine ATP, PCr, and lactate content. Compared to passive recovery controls, there was a significant decline in peak power for both the MI and LI groups (3.4-6.0% and 3.5-3.7%, respectively; $p < 0.05$). There were no differences found in ATP, PCr, and lactate between active recovery trials, however PCr trended parametrically with recovery intensity. From these results, the researchers concluded that any low-to-moderate level of muscle activation will inhibit the rate of PCr resynthesis and increase fatigue during subsequent bouts by limiting $O_2$ availability.

While there is considerable evidence supporting a relationship between oxygen availability and PCr kinetics, research has shown conflicting results when trying to prove a direct relationship between the two variables. For example, Cooke et al. (1997) found no significant correlation between the rate of PCr resynthesis and $VO_{2peak}$ among individuals grouped in either a high (mean $VO_{2peak}$ 64.4 +- 1.4 mL/kg/min) or low (mean $VO_{2peak}$ 46.6 +- 1.1 mL/kg/min) relative $VO_{2peak}$ group. Possible confounding factors of this research line are individual differences in PCr recovery due to genetic predisposition and muscle fiber variation, as well as small sample sizes seen in most of the experiments. While muscle fiber distribution between athletic populations may be a confounding factor when interpreting the effects of endurance training on RSA, some studies have shown

improvement with games players. Helgerud at el. (2001) found that 8-weeks of aerobic interval training for soccer players led to a 20% increase in total distance covered in a match, a 100% increase in number of sprints per match, as well as an increase in average work intensity from 82.7 to 85.6% of maximal heart rate.

In addition, the parameters of RSA testing may heavily influence the effects seen on PCr recovery and the associations made to aerobic capacity, making it hard to find a clear association. Dupont et al. (2004) reported a significantly shorter time to exhaustion with active, as opposed to passive, recovery ($p < 0.05$). Subjects performed 15-second sprints on a bicycle at 120% of $VO_{2peak}$, separated by either passive (sitting on the bike) or active recovery (peddling at 40% $VO_{2peak}$). The researchers theorized that active recovery restricted reoxygenation of $MbO_2$, and by association, inhibited PCr resynthesis. This finding supports that of Buchheit et al. (2008), who found that subjects who performed active recovery between 6x4-second maximal treadmill sprints resulted in a significantly higher percent decrement score (%Dec) than those who performed passive recovery ($7.2 \pm 3.7\%$ compared to $3.2 \pm 1.3\%$, respectively; $p < 0.001$). Because there is no "gold-standard" in RSA testing duration, number of sprints, and recovery times, it is difficult to rationalize the discrepancies of these investigations. While there is significant uncertainty as to the weight of the effect, the general consensus of the literature is that oxygen availability is an important factor in PCr kinetics, and thus resistance to fatigue during RSE.

*2.5c Inorganic Phosphate Kinetics*

In addition to improving PCr kinetics, oxygen availability has been shown to

influence the rate of $P_i$ accumulation and clearance during RSE (Hogan, Richardson, & Hasler et al., 1999; Idstrom et al., 1985). These results bring into question the findings of hypoxia studies and their effects on fatigue, such as Balsom et al. (1994) and Hasler et al. (1999). While these studies typically conclude that fatigue increases during hypoxic conditions due to a decreased rate of PCr resynthesis, it is just as likely that fatigue is manifested through an increase in intracellular $P_i$.

Unfortunately, there is very little research looking at oxygen availability and $P_i$ kinetics. Yoshida and Watari (1993) found that off-transient $P_i$ kinetics were significantly faster in endurance trained athletes than in untrained controls. This implies a higher aerobic capacity could influence $P_i$ accumulation; however, with so many other variables likely affecting fatigue, it is unwise to draw any conclusions without further research. While there is considerable evidence to suggest that a high aerobic capacity may help mediate the inhibitory effects of an elevated intracellular $P_i$ concentration, at this time the research is too uncertain to draw inferences.

**2.6 Conclusions**

Based on the evidence, it would appear that there is no single causative factor of fatigue limiting the RSA of a field-based sport athlete. Rather, the research points to a myriad of factors that all seem to feed off of each other, with no single factor by itself being able to cause substantial fatigue, but the sum of its parts sufficient to cause significant impairment of the muscle, its contractile efficiency, and energy substrate supply.

Perhaps the best way to reconcile the complexity of the physiologic integration that

takes place during RSE is to look at it through what Edwards (1983) has called, a "catastrophe theory." Simply put, it is a modification of the belief that one is only as strong as its weakest link. This implies that the failure of any one pathway within a larger system, made up of many adjacent pathways, will place an undue burden on those adjacent pathways; ultimately causing additional pathways to fail, in succession or simultaneously, with the eventual outcome being the failure of the entire system.

The current state of the literature regarding RSA, pertaining specifically to the sport of hockey, has found no relationship between aerobic capacity and performance. Despite these findings, there is substantial evidence, in testing other sport modalities, to argue to the contrary. This study aims to eliminate the shortfalls of earlier research by accounting for task-specificity by testing $VO_{2peak}$ on a skating treadmill and evaluating RSA using an on-ice test, developed to mimic the motor patterns typically performed by hockey players during competition.

## CHAPTER 3: Aerobic Capacity is Associated with Improved Repeated Shift Performance in Hockey

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### ABSTRACT

Current research has found conflicting results regarding the relationship between maximal oxygen uptake ($VO_{2peak}$) and repeated shift performance (RSP). Specific to hockey, recent evidence suggests there is no correlation between on-ice and off-ice $VO_{2peak}$ values in collegiate hockey players. Off-ice $VO_{2peak}$ may not be an adequate predictor of on-ice performance and should be tested in a sport-specific manner to achieve transferable results. **Purpose:** The purpose of this study was to use sport-specific testing methods to investigate the relationship between aerobic capacity ($VO_{2peak}$) and RSP in college hockey players. **Methods:** Forty-five (range: 18-24) college hockey players completed a graded exercise test on a skating treadmill to ascertain their $VO_{2peak}$. An on-ice repeated shift test was conducted to evaluate each player's susceptibility to fatigue. Course times were measured by a wireless timing system and fatigue calculated as a percent decrement score. First gate, second gate, and total test times were collected on the course and then used to calculate associated decrement scores. **Results:** Second gate decrement was significantly correlated to $VO_{2peak}$ ($r = -0.31$, $p = 0.04$). Final stage

completed during the graded exercise test was significantly correlated to second gate and total decrement ($r = -0.46$, $p = 0.001$; $r = -0.32$, $p = 0.033$). No significant correlation was found between either first gate or total decrement score and $VO_{2peak}$ ($r = -0.11$, $p = 0.46$; $r = -0.17$, $p = 0.26$). **Conclusion:** On-ice performance is associated with $VO_{2peak}$. In addition, stride efficiency plays a large role in predicting on-ice fatigue. This evidence suggests it may be advantageous for hockey players to include training methods that improve aerobic capacity and stride efficiency during off-season workouts.

## 1. INTRODUCTION

Sport specific training has been shown to improve performance in repeated sprint sport athletes (Castagna et al., 2008; Girard, Mendez-Villanueava, & Bishop, 2011). An important factor for success in repeated sprint sports is a player's ability to produce power; the player that is quicker to the ball or faster off the line will often beat their opponent, giving them an advantage. Competitions for these sports, however, take place over the period of several hours and are divided into quarters, halves, or periods; each of these consisting of numerous maximal-work bouts interspersed with relatively short recovery periods (Montgomery, 2000). The ability to repeatedly produce high power outputs throughout a competition gives a competitive edge to a player over his/her opponent and is an important fitness component in repeated sprint sports (Glaister, 2005). This ability has come to be known as repeated-sprint ability (RSA).

In the repeated sprint sports of rugby and soccer, $VO_{2peak}$ has been shown to correlate with RSA, ranging from r = -0.50 to -0.83 (Bishop & Edge, 2006; Tomlin & Wenger, 2001). This relationship is further supported by research showing that a state of hypoxia can impair RSA in athletes (Balsom, Ekblom, & Sjodin, 2004; Quistorff, Johansen, & Sahlin, 1992). In addition, creating a hyperoxic environment appears to improve RSA through increased aerobic adenosine triphosphate (ATP) contribution and phosphocreatine (PCr) resynthesis rate (Balsom et al., 2004). This evidence suggests that aerobic capacity and improved oxygen utilization may affect RSA by 1) increasing the rate of the fast and slow phase of PCr resynthesis (McMahon & Jenkins, 2002), 2) enhancing the clearance rate of metabolites created by PCr breakdown and

glycogenolysis (Bishop & Spencer, 2004), 3) improving oxygen ($VO_2$) kinetics (Dupont, Millet, Guinhouya, & Berthoin, 2005), and 4) increasing aerobic energy contribution during maximal sprint bouts (Tomlin & Wenger, 2001).

Disagreement exists regarding the strength of this association, as there is research to refute these findings. Numerous researchers have failed to find a significant association ($-0.35 < r < -0.46$) between $VO_{2peak}$ and RSA in rugby and soccer athletes (Aziz, Chia, & Teh, 2000; Bishop & Spencer, 2004; Carey, Drake, Pliego, & Raymond, 2007; Wadley & Rossignol, 1998), but it is not clear what role sample size ($n < 15$) played in calculating these statistics. The discrepancy in the research would advocate for additional studies to be conducted to better understand the possible relationship between aerobic capacity and RSA.

This study aims to address the current limitations specific to ice hockey and RSA in three ways: 1) address a current population-specific void in ice-hockey RSA research; 2) account for task-specificity by obtaining players' $VO_{2peak}$ on a skating treadmill using a graded exercise test; and 3) evaluate RSA using an on-ice test that mimics the motor patterns typically performed by ice-hockey players during competition. The results of this study could have important implications for the training methods used to prepare ice-hockey players for their competitive season. Therefore, the purpose of this study was to investigate the relationship between $VO_{2peak}$ and RSA in hockey players. It is hypothesized that hockey players with a higher aerobic $VO_{2peak}$ will exhibit less fatigue during an on-ice repeated shift test than those with lower values.

# 2. METHODS

**2.1 Participants.** All testing procedures were approved by the Institutional Review Board (IRB) at the University of Minnesota prior to participant recruitment and data collection. Written, informed consent was obtained from all participants prior to the start of the study. Participants for this study were male hockey players (ages 18-24) playing Division I, Division III, or Junior level hockey in the Minneapolis, Minnesota area. All testing took place at the start of their summer training schedule. Exclusion criteria for the study included absence from on-ice skating over the previous 30 days due to prior or current injury and players self-reporting their position of goaltender.

After enrolling in the study, participants were signed up for three testing sessions. Each session took approximately one hour and was held on the University of Minnesota campus. All sessions were completed within a ten-day period, ensuring at least two days rest between sessions. In addition, all testing took place between 7 a.m. and 3 p.m. to ensure consistency. Participants were told prior to each session to refrain from heavy exercise 24 hours prior to their testing sessions. All participants were told to eat a light meal two hours prior to their testing session. In addition, they were asked to refrain from caffeine, tobacco, and alcohol 12 hours prior to testing. Aside from these guidelines, participants were asked to maintain a normal diet and exercise regimen during testing.

**2.2 Determination of Physical Characteristics.** Session One took place in the Laboratory of Physical Hygiene and Exercise Science (LPHES). It consisted of anthropometric, vertical jump, Wingate, and grip strength testing. Standing height was recorded using the Frankfort Plane criterion (Heymsfield, Lohmna, Wang & Going,

2010) and weight using a Detecto Mechanical Doctor's scale (Model #439). Body composition was assessed via hydrostatic weighing using Exertech Body Densitometry Systems software (Dresbach, Minnesota). The method is considered to be a valid and reliable method for measurement of body composition (Heymsfield et al., 2010). The participants underwater weight was recorded eight times to account for both a learning effect, as well as to ensure consistency of the measure and accuracy of the reported weight (Heymsfield et al., 2010). The heaviest duplicated weight was recorded as the participant's underwater weight. If the participants were unable to duplicate their first or second heaviest weight, their third heaviest weight was recorded as the official underwater weight. Percent body fat was calculated using the Brozak equation (Heymsfield et al., 2010). Residual lung volume was also calculated (Maud & Foster, 2006).

**2.3 Determination of aerobic capacity**. Aerobic capacity was assessed on both a Frappier™ (Acceleration, Minneapolis, MN, n = 30) and The Blade™ (Woodway, Waukesha, WI, n = 15) skating treadmills to ascertain the $VO_{2peak}$ of each participant. Breath-by-breath analysis was performed by an Ultima CPX™ (Medgraphics, St. Paul, MN). The skating treadmill protocol used has been previously validated as a reliable means for participants to reach volitional exhaustion and accurately measure $VO_{2peak}$ (Koepp & Janot, 2008). The protocol began with participants skating at a speed of 6.5 miles-per-hour (mph) and a 2% grade. Every minute, the speed of the treadmill was increased 0.5 mph until a maximal speed of 10 mph was reached; this occurred eight minutes into the test. Once the participants had reached maximal speed, the grade was

increased by 1% every minute until they reached volitional exhaustion. Criteria for reaching maximal aerobic capacity was determined by achieving two of the three following criteria: 1) maximal heart rate (220 - age ± 10); 2) RER value > 1.10; and 3) rate of perceived exertion > 18 (Bassett & Howley).

**2.4 On-Ice Repeated Shift Test.** Session Three took place at the hockey arena and consisted of the on-ice repeated shift test. To our knowledge, there is no precedence in the literature for an on-ice repeated shift test. In addition, it has been shown that off-ice tests do not predict the on-ice performance of hockey players (Vescovi, Murray, Fiala, & VanHeest, 2006). To address this shortfall, an on-ice repeated shift test was created for this study that mimics the intensity, duration, and movement patters performed by a hockey player during a shift to evaluate the participant's susceptibility to fatigue during maximal intensity skating.

To create the test, data was collected from the National Hockey League (NHL) database concerning the shift length, number of shifts played per period, and rest intervals between shifts by forwards in the NHL from 2009-2011. It was found the average shift length by an NHL forward to be 45.5 ± 3.9 seconds, with average shifts completed per period calculated at 6.8 ± 1.1 (excluding power plays). Other research has estimated that hockey players spend approximately 50% of their time on the ice in some form of high-intensity activity (sprinting, striding, skirmishing, etc) (Montgomery, 2000). When this percentage is applied to the calculated parameters of a hockey shift, then it can be inferred that a hockey player will spend approximately 22.7 seconds of a shift in a maximal, or near-maximal, skating state.

When the participants arrived at the rink, they were told to change into full gear and go out on the ice. Participants were instructed to have their skates sharpened to game specifications prior to testing. When the participants stepped onto the ice, they were told to go through their typical pre-game warm-up. When the participants said they were ready, they joined the researchers at the starting line. The participants were told the basic parameters of the test. In addition, they were told the two goals of the test: 1) skate the course as fast as possible, and 2) have the lowest drop-off in time from your best course time as possible.

The participants then watched an instructor glide through the course to understand its layout. Each participant was then asked to skate the course at 50% of his best effort to gain familiarization. Upon completion of the familiarization trial, each participant was asked if he felt comfortable with the course. If he said, "no," he was instructed to skate the course one additional time. The participants were given three minutes to rest following the familiarization trial to ensure they were fully recovered before commencement of testing.

The test itself consisted of 8-maximal skating bouts, performed in full gear (including their stick), lasting approximately 22.7 seconds with 90 seconds of passive recovery between bouts. The course protocol can be seen in Figure 1. Course times were measured by a TC Speed Trap-II wireless timing system (E38720, Gill Athletics, Champaign, IL). There were three separate timing gates, used to evaluate first half, second half, and total fatigue decrement. Fatigue was calculated as a percent decrement score (% Decrement Score = (100 x (Total Sprint Time ÷ Ideal Sprint Time) – 100)

(Glaister, Howatson, Pattison, & McInnes, 2008).

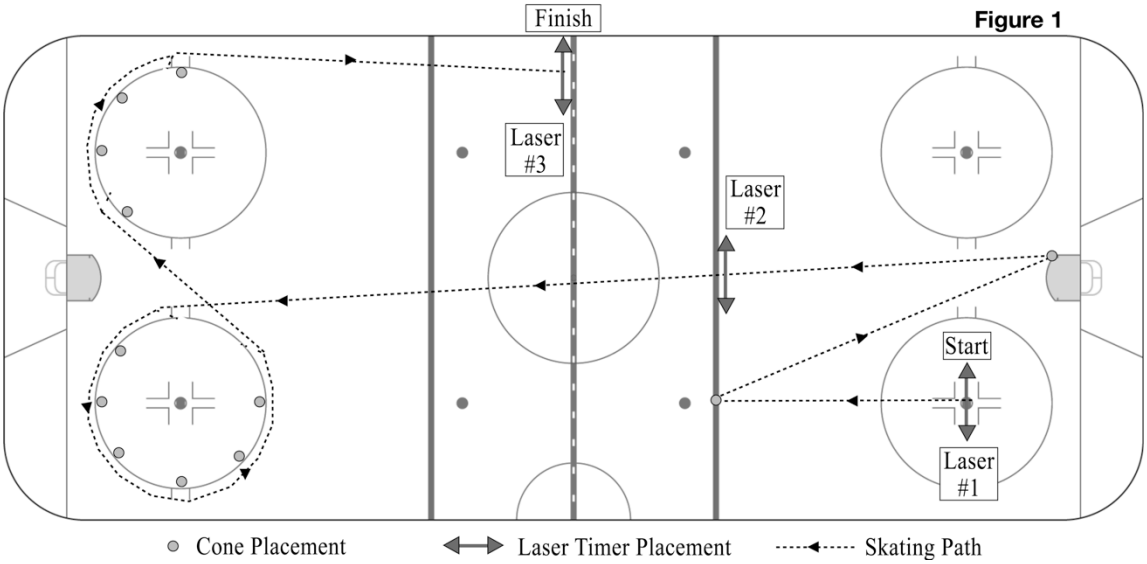


**Figure 1:** The on-ice repeated shift test consists of 8-maximal skating bouts, with 90 seconds of passive recovery between bouts. The skater starts with their skate at the face-off line; stick behind the laser timer. When given the "start" command, the skater sprints to the blue line, making a hard cut, pushing off their left leg (post leg), and sprinting to the far side of the goalie crease. The skater then makes a hard cut, pushing off their right leg (post leg), and sprints the length of the ice. The skater makes hard crossover steps to their left, going around the circle. After reaching the top of the circle the skater sprints to the base of the right circle, making hard crossover steps to their right. When the skater had completed the turn, they sprinted along the boards through the red line (finish line).

The 90-second recovery time started immediately after the participants crossed the finish line. Participants were told that during their recovery time they could stand, take a knee, or lay on the ice; they could not skate around. The participants were informed when they had 30 seconds and 10 seconds of rest time remaining, with a 5-second count down to the start. This sequence was completed eight times, with times being recorded after each sprint bout. Participants were not allowed to drink any sports drinks or mixes during testing, however water was provided without restriction to the participants.

**2.5 Data Analysis.** This cross-sectional study is powered for a two-sided test with

an effect size of 0.5 and a power of 0.80. The sample size required to show significance within these parameters was calculated to be n=36 (Hully, Cummings, Browner, Grady, & Newman, 2007). Mean and standard deviations (SD) were calculated for all variables. Analysis of $VO_{2peak}$ data was performed by the Breezesuite software package (Medgraphics, St. Paul, MN). Statistical analysis of the collated data was performed by SPSS software (IBM, vr. 21.0). Data was analyzed using either a Pearson's or Spearman's correlation test, depending on the distribution of the data. Correlational coefficients (r) were used to detect associations between the independent and dependent variable. Multiple regression was used to determine the combined influence of $VO_{2peak}$ and end stage completed during the graded exercise test on measures of fatigue while controlling for the use of two skating treadmills. The on-ice repeated shift test had one participant with a problematic point that was significantly deviated from the mean for all three timing gates. Because this subject's data point was not a statistical outlier, this point was winsorized to one unit above the next highest data point to meet criteria for normal distribution. For all statistical tests, an alpha level of $p < 0.05$ was operationally defined as statistical significance.

**3. RESULTS**

Forty-five male hockey players participated in this study (Table 1). All participants met criteria for normalcy in each tested variable. The mean $VO_{2peak}$ for participants was $54.9 \pm 4.4$ ml/kg/min. Times and decrement scores for first gate, second gate, and total are described in Table 2. Skate times continued to increase from sprint one to sprint 8, indicating fatigue, which was the main objective of the course design. No significant

correlation was found between either first gate or total decrement score and $VO_{2peak}$ (r = -0.11, p = 0.46; r = -0.17, p = 0.26; Figure 2). Second gate decrement, however, was significantly correlated to $VO_{2peak}$ (r = -0.31, p = 0.04; Figure 2), indicating that 9.6% of the variance in fatigue of the participants can be explained by their aerobic capacity ($VO_{2peak}$).

**Table 1.** Descriptive Characteristics of subjects

| Variable | Mean | ± | SD |
|---|---|---|---|
| Sample Size (n) | 45 | | |
| Age (years) | 20 | ± | 2.0 |
| Height (cm) | 181 | ± | 9.0 |
| Weight (kg) | 84 | ± | 12.0 |
| Body Fat (%) | 12.5 | ± | 4.0 |
| Relative $VO_{2peak}$ (ml/kg/min) | 54.9 | ± | 4.4 |
| Absolute $VO_{2peak}$ (ml/min) | 4649 | ± | 415 |
| Final Stage Completed (Treadmill Test) | 9.67 | ± | 2.0 |

*$VO_{2peak}$*: Maximal Oxygen Consumption
*Final Stage Completed*: Furthest stage reached during the skating treadmill test

**Table 2.** On-Ice Repeated Shift Test Results

| Variable (seconds) | Mean | ± | SD |
|---|---|---|---|
| Fastest Course Time | 23.02 | ± | 0.61 |
| Slowest Course Time | 26.47 | ± | 1.53 |
| Average Course Time | 25.05 | ± | 1.02 |
| Average First Gate Time | 9.90 | ± | 0.86 |
| Average Second Gate Time | 15.30 | ± | 1.17 |
| First Gate Decrement (%) | 8.12 | ± | 3.47 |
| Second Gate Decrement (%) | 9.08 | ± | 2.95 |
| Total Course Decrement (%) | 8.90 | ± | 3.30 |

Figure 2

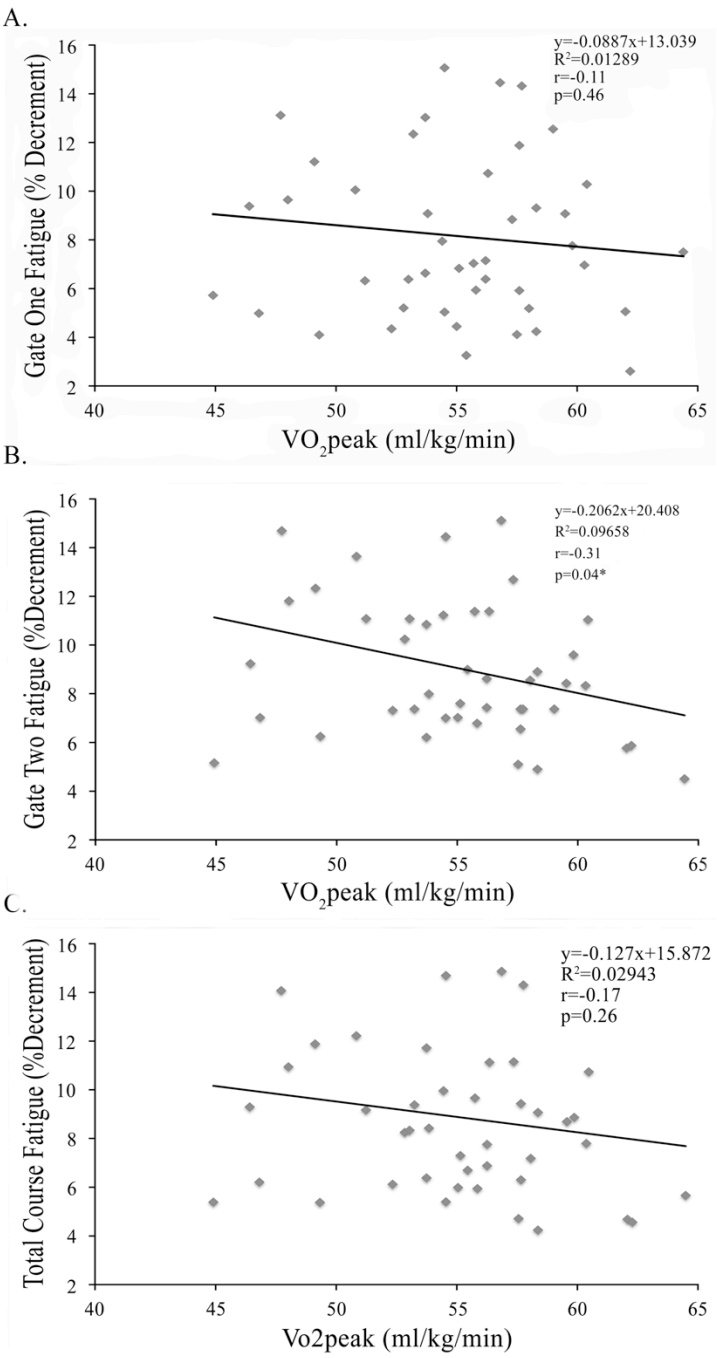


**Figure 2:** The relationship between $VO_{2peak}$ and fatigue (percentage decrement score) during the on-ice repeated shift test; broken down into (A) Gate One Fatigue, (B) Gate Two Fatigue, and (C) Total Course Fatigue. *Significant correlation between variables ($p < 0.05$).

Final stage completed during the graded skating treadmill test was significantly correlated to second gate and total decrement found during the on-ice repeated shift test when controlled for treadmill type ($r = -0.49$, $p = 0.001$; $r = -0.32$, $p = 0.03$; Figure 3). Final stage completed accounted for 21% and 10% of the variance, respectively. These correlation coefficients indicate that a larger portion of the variance in on-ice fatigue can be explained by end stage completed than $VO_{2peak}$. There was no significant correlation between final stage completed and first gate decrement ($r = -0.21$, $p = 0.17$; Figure 3).

Using multiple regression analysis final stage completed and $VO_{2peak}$, combined, were significant predictors of second gate fatigue when accounting for the use of two treadmills ($r^2 = 28.7$, $p < 0.05$). Independently, final stage completed accounted for 23.0% of the variance ($p < 0.05$) of gate two fatigue, with an additional 4.2% of the model being accounted for by $VO_{2peak}$ ($p = ns$). Final stage completed was also significantly related to total decrement ($r^2 = 16.8$, $p < 0.05$), however the addition of $VO_{2peak}$ did not improve the predictive ability of the model, as indicated by a non-significant F-value ($r^2 = 18.8$, $p = ns$).

Figure 3

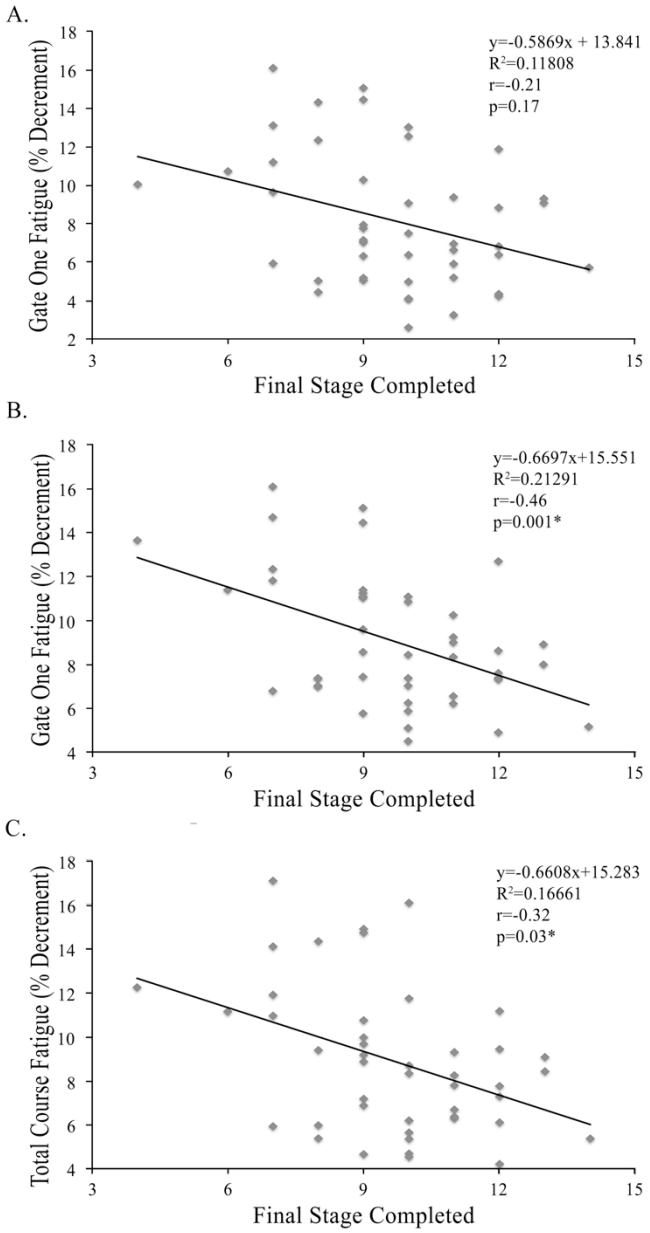


**Figure 3:** The relationship between Final Stage Completed during the aerobic capacity test on the skating treadmill and Fatigue (percentage decrement score) during the on-ice repeated shift test; broken down into (A) Gate One Fatigue, (B) Gate Two Fatigue, and (C) Total Course Fatigue. *Significant correlation between variables ($p < 0.05$).

# 4. DISCUSSION

There are two key findings of this study. First, maximal oxygen uptake ($VO_{2peak}$), assessed on a skating treadmill, was significantly correlated to fatigue, expressed as a decrement score, during the second half of a mock hockey shift in competitive college hockey players. This study is the first to show this significant correlation exists in this population of athletes. Second, end stage completed during the graded exercise test was significantly correlated to second gate and total course fatigue, accounting for a greater portion of the variance than $VO_{2peak}$, and likely being a better predictor of on-ice performance than aerobic capacity alone. In addition, the manner in which data was collected during the on-ice repeated shift test was unique to this study and allowed for a more thorough understanding of the physical performance and possible energy pathway contributions of hockey players during competition.

Earlier research has found $VO_{2peak}$, assessed using running protocols, to be significantly correlated to RSA in field based team sports ($r = -0.50$ to $-0.83$; $p < 0.05$) (Bishop & Edge, 2006; McMahon & Jenkings, 2002). These researchers have theorized that fatigue is associated primarily with one, or a combination of, four intramuscular conditions; all of which are affected by oxygen availability: 1) diminished rate of PCr resynthesis; 2) slow the rate of inorganic phosphate accumulation; 3) decreased rate of glycolytic flux; and 4) increase the level of muscle acidosis. This research would imply that hockey players with higher maximal oxygen capacities would be better able to mitigate the effects of these fatigue causing factors and maintain their performance on the ice better than players with lower levels. Additionally, players that are more efficient

skaters would place less strain on their metabolic system while skating, limiting the accumulation or affect of fatigue causing factors. Simply put, efficient skaters do not have to work as hard as their inefficient peers and thus perform better on the ice. Despite ice-hockey having similar energy demands as other team-sports, these associations have not been found in hockey players.

A recent study found that there was no correlation between on-ice and off-ice $VO_{2peak}$ values in collegiate hockey players (Durocher, Guisfredi, Leetun, & Carter, 2010). The researchers concluded that running $VO_{2peak}$ was not an adequate predictor of on-ice $VO_{2peak}$ and suggested that hockey players be tested in a sport-specific manner to garner reliable results. Taking these findings into account, it is possible that the lack of an association in the earlier research was due to testing protocol selection and specificity of task. The present study is the first to compare a sport-specific, skating $VO_{2peak}$ testing protocol to an RSA test in ice-hockey.

Most RSA tests employ protocols that only utilize linear movement (Gaitanos, Williams, & Boobis, 1993). This can be problematic when interpreting direct correlations to performance, as athletes move in a 360° plane of motion during competition (Girard et al., 2011). Athletes completing a course with multiple changes in direction, when compared to straight ahead running, covering the same distance, place a greater energy demand on their metabolic system (Reilly, 1997). This would indicate that RSA tests employing only linear movement might not adequately simulate the stress placed on an athlete during competition. The present study addressed this deficiency by creating an on-ice shift test that mimicked the movement patterns a hockey player would regularly

perform during competition.

In addition, the parameters of an RSA test may heavily influence the relationship between fatigue and $VO_{2peak}$. The results of an RSA test can vary greatly between studies due to the fact that there is not a "gold-standard" RSA testing protocol. The rate of fatigue can be influenced by the number of sprint bouts, the duration of the bouts, or the recovery period between bouts (Balsom, Seger, Sjodin, & Ekblom, 1992; Buchheit et al., 2008). The recovery period can further be examined in terms of total time, intensity, and mode (active or passive); having a significant effect on a studies results (Spencer, Dawson, Goodman, Dascomb, & Bishop, 2008). Therefore, a primary goal of the present study was to create an on-ice skating test that mimicked the intensity, skating time, movement patterns, and recovery that a hockey player would be subject to during a hockey shift. Each aspect of the course was chosen to require the skater to perform a movement pattern typically completed during competition; 1) straight ahead sprint; 2) hard cutting change of direction (rt/lt); 3) short acceleration; 4) long acceleration; and 5) crossover (rt/lt). These movement parameters were established from conversations with both NHL and collegiate hockey coaches, as well as from published research (Pearsall, Turcotte, & Murphy, 2000; Quinney et al., 2008).

Gate two fatigue was significantly correlated to $VO_{2peak}$. This time point captured the fatigue rate of each subject during the second half of the course; a time frame of approximately 10 to 25 seconds of a maximal effort sprint. During this time, the glycolytic pathway is likely the main contributor to ATP production (Gastin, 2001). One of the main byproducts of the glycolytic pathway is hydrogen ions ($H^+$); causing an

associated drop in intracellular pH (Brooks, Fahey, & Baldwin, 2005; Metzger & Fitts, 1987), which inhibits the rate limiting enzymes of glycogenolysis (phosphorylase *a* and phosphofructokinase (PFK)), and is associated with fatigue (Girard et al., 2011; Westerblad & Allen, 2002). As a result, fatigue could increase in subsequent bouts of maximal exercise if the muscle is not given sufficient time to clear the $H^+$ from the intracellular space. The rate of recovery for glycolysis is slow, estimated to have a half time of approximately nine minutes (Glaister, 2005). With the recovery time between sprints during the on-ice test being only 90 seconds, it is unlikely that the glycolytic pathway would be able to return to a homeostatic balance between sprint bouts (Spreit, Lindinger, McKelvie, Heigenhouser, & Jones, 1989). As a result, hockey players with a higher $VO_{2peak}$ may be better at buffering $H^+$.

Gate two and total course fatigue were also significantly correlated to end stage completed during the skating treadmill test. This association implies that skating mechanics (stride efficiency) are an important aspect of on-ice performance. End stage completed can be interpreted as a composite measure of a player's aerobic capacity and stride efficiency (Pearsall et al., 2000). A player with great stride efficiency but a low aerobic capacity has the potential to reach the same end stage as a player with poor stride efficiency and a high aerobic capacity. This is due to the fact that an efficient skater does not require the same amount of energy output to meet a specific workload. Similar phenomena have been seen in endurance athletes and the effect of running economy on marathon times (Saunders, Pyne, Telford, & Hawley, 2004). This may also explain why $VO_{2peak}$ was not significantly correlated with total course fatigue. $VO_{2peak}$ appears to be

confounded by hockey player's stride efficiency, as a factor of end stage completed, in that it only accounted for 1.4% of the variance found in total course fatigue. While this study did not directly measure stride efficiency, our findings of a significant association between end stage completed and on-ice fatigue argue for future research to look more closely at this relationship.

Gate one fatigue was not significantly correlated to $VO_{2peak}$. With an average time to completion of 9.9 seconds, gate one captured the section of the course most reliant on ATP production from the PCr energy pathway (Bogdanis, Nevill, Boobis, & Lakomy, 1996). Studies have shown PCr synthesis to follow a biphasic pattern of recovery after intense muscular activity (Baker, McCormick, & Robergs, 2010). The rate of the fast phase of PCr recovery appears to have a half time of only 21 seconds, in which 50% of used PCr is resynthesized (Harris, Edwards, & Hultman, 1976). This appears to be unaffected by the pH of the muscle and is a very robust, fatigue resistant pathway (McMahon & Jenkins, 2002). Keeping these facts in mind when examining the parameters of the on-ice repeated shift test gives a possible, justifiable, explanation as to why gate one was not significantly correlated to $VO_{2peak}$. The recovery time between sprint bouts was over four times longer than PCr requires to recover half of its pre-exercise level after maximal exercise. With the primary energy system contributing to gate one performance being given sufficient time to recover, the reductions in PCr during repeated bouts are not great enough to elicit measurable fatigue in our sample population of athletes.

Total course time was also not significantly correlated to $VO_{2peak}$, however that could be a factor of the fatigue resistant nature of the PCr pathway. The significance of total course time, as it relates to $VO_{2peak}$, is the sum effect of the energy system taking place during gates one and two. With the recovery of PCr being robust against fatigue it is plausible that gate one times skew the results, reducing the net fatigue effect seen in total sprint time when compared to the athletes $VO_{2peak}$. It would stand to reason that using a larger sample size, reducing the recovery time, or lengthening the duration of the sprint would have reduced the skew of the data by placing less statistical weight on the fatigue resistant PCr pathway and increase the effect of oxidative contribution to energy during sprint bouts.

## 5. CONCLUSIONS

The results of this study indicate that a hockey player's maximal aerobic capacity is associated with fatigue during a mock hockey shift, and that end stage completed during a graded skating treadmill test is a good predictor of on-ice performance. We hypothesize that this is likely due to the fact that end stage completed takes into account the stride efficiency of the hockey player, in addition to their aerobic capacity. This evidence suggests that it may be prudent for hockey players to emphasize training methods that improve skating efficiency, as well as incorporate $VO_{2peak}$ specific training during off-season workouts. These findings could be beneficial for coaches as they advocate for testing hockey players in a sport specific manner, via a skating treadmill or on-ice skating test, as the best means of evaluating physical performance on the ice.

**CHAPTER 4: Division I hockey players generate more power than Division III players during on- and off-ice performance tests**

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## ABSTRACT

Current research has found anthropometric and physiologic characteristics of hockey players that are correlated to performance. These characteristics, however, have never been examined to see if significant differences exist between on- and off-ice performance markers at different levels of play (e.g., Division I, Elite Junior, and Division III). The purpose of this study was to examine the differences that may exist between these characteristics in Division I (24), Elite Junior (10), and Division III hockey (11) players. Forty-five (age 18-24) male hockey players completed anthropometric, on-ice, and off-ice tests to ascertain average measures for each division of play. On-ice testing was conducted in full hockey gear and consisted of acceleration, top speed, and on-ice repeated shift test (RST). Off-ice tests included vertical jump, Wingate, grip strength, and a graded exercise test performed on a skating treadmill to ascertain their $VO_{2peak}$. Division I players had significantly lower body fat than their Division III peers ($p = 0.004$). Division I players also scored significantly better than their Division III

counterparts on measures of anaerobic power; vertical jump ($p = .001$), Wingate peak power ($p = 0.05$), grip strength ($p = 0.008$), top speed ($p = .001$), and fastest RST course time ($p = .001$). There was no significant difference between Division I and Elite Junior players for any on- or off-ice performance variable. The results of this study indicate that performance differences between Division I and Division III hockey players appears to be primarily due to rate of force production.

**Key Words:** Rate of Force Development, Skating, Anaerobic, Specificity

## 1. INTRODUCTION

Team sports have varying physiologic demands. Soccer, for example, tends to require its athletes to have a high aerobic capacity, while sports like football place a emphasis on strength and power (Spencer, Bishop, Dawson, and Goodman, 2005). Teams with athletes that are better suited to meet their sports demands often outperform their competition via superior sport-specific athleticism. To that end, the ability to evaluate and recruit players in possession of these physical qualities becomes imperative for a team's success. Anthropometric and physiologic characteristics have been used by coaches in many team sports to identify and recruit elite players, classify starters verses non-starters, and stratify athletes by level of play (Fitzgerald et al., 2013; Leone, Lariviere, & Comtois, 2002; Keogh, 1999, Houston et al.). For example, Rampanini et al. (2009) found that $VO_{2peak}$ was a physiologic characteristic that differentiated professional from elite junior soccer players. According to Rampinini, regardless of their skill, an elite junior player that had a $VO_{2peak}$ below a given threshold would not succeed at the professional level due to an inadequate ability to perform high levels of aerobic work.

To be effective, decision makers (coaches, general managers, and scouts) need a quantifiable list of characteristics that have been shown to be associated with aspects of performance in their respective sport. The creation of a sport-specific physiological profile would aid coaches in selecting athletes from a much larger pool who have the physiologic attributes to compete at their respective level, identify athlete strengths and weaknesses, and allow for the creation of athlete-specific training programs that address their limitations.

Ice-hockey requires a unique set of physiologic qualities to be successful; speed, power, endurance, and skill (Noonen et al, 2002; Buchheit, Lefebvre, Laursen, & Ahmaidi, 2011; Potteiger et al., 2010). Previous literature has found on- and off-ice tests that correlate with performance; including anthropometry, aerobic capacity, power, and skating ability (Peterson et al., 2014; Fitzgerald et al., 2013; Agre et al., 2000; Potteiger et al., 2010; Bracko, 2001; Montgomery, 2000; Behm et al., 2005). However, this research has focused solely on evaluating the National Hockey League (NHL) (Vescovi, Murray, Fiala, & VanHeest, 2006; Quinney et al., 2008; Pearsall, Turcotte, & Murphy, 2000). Little to no research has investigated differences that may exist at the colligate and Elite Junior level. This has lead to a void in the literature that prevents coaches at the collegiate level from making informed, scientifically based decisions regarding the recruiting training methods employed with their teams.

This study aims to address the current limitations specific to ice-hockey by 1) evaluating common physiologic performance characteristics of collegiate hockey players and 2) stratifying these results by division of play to look for differences that may exist between the Division I, Elite Junior, and Division III level. The results of this study could have important implications for the training and recruiting practices used by coaches at different levels of collegiate hockey. It is hypothesized that Division I hockey players will have higher on- and off-ice performance scores compared to their peers playing at the Elite Junior or Division III level.

## 2. METHODS

**2.1 Experimental Approach to the Problem**. Subjects were signed up for three testing sessions; 1) anthropometric and off-ice performance; 2) aerobic capacity; and 3) on-ice performance testing. Each session took approximately one hour and was held on the University of Minnesota campus. All subjects were tested in early June at the start of their summer training schedules. All three sessions were completed within a ten-day period, ensuring at least two days rest between sessions. Subjects were told prior to each session to refrain from heavy exercise for 24 hours prior to their testing sessions. All subjects were told to eat a light meal two hours prior to testing and to refrain from caffeine, tobacco, and alcohol 12 hours prior to testing. Aside from these guidelines, subjects were asked to maintain a normal diet and exercise regimen during testing. Exclusion criteria for the study included absence from on-ice skating over the previous 30 days due to prior or current injury and players self-reporting their position as goaltender.

**2.2 Subjects.** Forty-five male hockey players, ages 18-24 and playing Division I, Division III, or Elite Junior level hockey in the Minneapolis, Minnesota area volunteered for this study. All testing procedures were approved by the Institutional Review Board (IRB) at the University of Minnesota prior to participant recruitment and data collection. Written, informed consent was obtained from all participants prior to the start of the study.

**2.3 Session One - Anthropometric and Off-ice Performance Testing**. Session one consisted of anthropometric, vertical jump, Wingate, and grip strength testing.

Standing height was recorded using the Frankfort Plane criterion (Heymsfield, Lohmna, Wang & Going, 2010) and weight using a Detecto Mechanical Doctor's scale (Model #439). Body composition was assessed via hydrostatic weighing using Exertech Body Densitometry Systems software (Dresbach, Minnesota). The method is considered to be a valid and reliable method for measurement of body composition (Heymsfield et al., 2010). The participants weight was recorded eight times to account for both a learning effect, as well as to ensure consistency of the measure and accuracy of the reported weight (Heymsfield et al., 2010). The heaviest duplicated weight was recorded as the participant's underwater weight. If the participants were unable to duplicate their first or second heaviest weight, their third heaviest weight was recorded as the official underwater weight. Percent body fat was calculated using the Brozak equation (Heymsfield et al., 2010). Residual lung volume was also estimated (Maud & Foster, 2006).

Vertical jump was tested using a Vertec™ Vertical Jump Trainer (Huntington Beach, CA). Each participant performed a five-minute treadmill warm-up, starting at 5 mph and increasing 0.5 mph per minute to a maximum of 7 mph, before performing the vertical jump protocol to standardize the measure. The vertical jump protocol consisted of two submaximal practice jumps to gain familiarization, followed by three to five maximal jumps, allowing a minimum of 30 seconds of rest between jumps. For each jump, the participant was instructed to squat to a self-selected depth (approximately 90 degrees) and hold that position for three seconds. The participant was also instructed to place one arm on their hip with the other above their head in a reaching position. After a

3-second count, the instructor would give an audible "jump" command, and the participant would jump, touching the highest vane possible on the Vertec™ Vertical Jump Trainer. These instructions ensured a standardized jump protocol for each subject, and help reduce confounding of arm swing during jumping (Ferreira, Schilling, Weiss, Fry, & Chiu, 2010). Jump height was assessed by counting the highest vane contacted by the participant. The test was terminated when the participant failed to increase jump height in two consecutive trials, or after the fifth trial.

The Wingate Anaerobic Test (WAnT), performed on a Monarch cycle ergometer (Langley, WA), was used to assess lower-body power production. The testing protocol for the WAnT has been standardized in earlier literature (Maud et al., 2006). After the seat and bar height were properly adjusted for the participant, they were told warm-up by lightly pedaling for 30 seconds. During the last five seconds of the warm-up time, the participant was told to increase their pedal velocity to its maximum and maintain it for the duration of the test. When the warm-up time expired, a flywheel resistance load equaling 0.075 kg/kg body weight was applied, with the participant continuing to pedal maximally for 30 seconds. Participants' peak (PP) and mean (MP) power were calculated as the highest average power attained during the first five seconds and full 30 seconds of the test (Maud et al., 2006). Power values were recorded in watts and normalized for body weight. The reliability of these measures, PP and MP, have been established as robust in previous literature ($r = .92$) when using the 0.075 kg/kg loading parameter (Patton, Murphy, & Frederick, 1985).

Grip strength was assessed using a Jamar Dynamometer. Grip strength in the

participant's dominant hand was measured in kilograms using the standard testing protocol outlined by the American Society of Hand Therapists (Innes, 1999). The participant sat on the edge of a chair, holding the dynamometer with the grip diameter set at 3.8cm, with his elbow at his side in 90 degrees of flexion. The participant was given one submaximal attempt to account for a learning affect, followed by a one-minute rest period. The participant was then instructed to maximally grip the dynamometer for three seconds. This protocol was repeated two additional times, with the scores from all three attempts being averaged into a composite score used for data analysis.

**2.4 Session Two - Determination of Aerobic Capacity.** Aerobic capacity was assessed on both a Frappier™ (Acceleration, Minneapolis, MN, n = 30) and The Blade™ (Woodway, Waukesha, WI, n = 15) skating treadmills to ascertain the $VO_{2peak}$ of each participant. Breath-by-breath analysis was performed by an Ultima CPX™ (Medgraphics, St. Paul, MN). The skating treadmill protocol used has been previously validated as a reliable means for participants to reach volitional exhaustion and accurately measure $VO_{2peak}$ (Koepp & Janot, 2008). The protocol began with participants skating at a speed of 6.5 miles-per-hour (mph) and a 2% grade. Every minute, the speed of the treadmill was increased 0.5 mph until a maximal speed of 10 mph was reached; this occurred eight minutes into the test. Once the participants had reached maximal speed, the grade was increased by 1% every minute until they reached volitional exhaustion. Criteria for reaching maximal aerobic capacity was determined by achieving two of the three following criteria: 1) maximal heart rate (220 - age ± 10); 2) RER value > 1.10; and 3) rate of perceived exertion > 18 (Bassett & Howley).

**2.5 Session Three - On-ice Performance Testing.** Session three took place at the hockey arena and consisted of an acceleration, top speed, and on-ice repeated shift test. All on-ice testing was performed in full gear with skates sharpened to game specifications. Acceleration was assessed by having the participant sprint, from a stationary start, blue line to blue line (Distance = 15.24m). Similar protocols have been implemented in earlier research and the method is considered a valid way to measure acceleration (Gilenstam, Thorsen, & Henriksson-Larsen, 2011). The participant started by standing with his front skate directly behind the blue line (starting line), stick in hand. When the participant felt he was ready, he would accelerate as fast as possible through the second blue line (finish line). Time was recorded by a TC Speed Trap-II wireless timing system (E38720, Gill Athletics, Champaign, IL). The photo cells of both timing gates were placed at waist level of the participant to ensure that the laser timed his body crossing the line, not his stick. In addition, participants were told to keep their sticks on the ice to ensure they did not prematurely trip the laser timer. Once the participant crossed the finish line they coasted back to the starting line and were given two minutes to recover. The recovery time started when the participant returned to the starting line. Similar tests have been reported to have test-retest values of r = 0.8 (Bracko, 2001). Each participant performed two trials, with the fastest time being used for data analysis.

Top speed was assessed after completion of the acceleration test and a two-minute recovery period. Top speed was measured by the time it took the participant to cover the distance between blue lines (15.24m) with a skating start (Brako, 2001; Gilenstam et al., 2011). The participant was instructed to take a lap around the rink, starting at the blue

line (start line), increasing their speed as they re-approach the start line. When the participant reached the start line, they were instructed to be moving as fast as possible, and to maintain that speed through the finish line. Times were again recorded by a TC Speed Trap-II wireless timing system (E38720, Gill Athletics, Champaign, IL) with the photocells placed at waist level. Once the participant crossed the finish line they coasted back to the starting line and were given two minutes to recover. The recovery time started when the participant returned to the starting line. This test has been reported to have test-retest values of r = 0.84 (Bracko, 2001). Each participant performed two trials, with the fastest time being used for data analysis.

Fatigue resistance was assessed after the top-speed test; again giving the participant a two minute recovery period. Fatigue was measured as a percent decrement score during an on-ice repeated shift test (Peterson et al. 2014). Participants were not allowed to drink any sports drinks or mixes during testing, however water was provided without restriction to the participants.

**2.6 Data Analysis**. Statistical analysis of the data was performed using SPSS software (IBM, vr. 21.0). Mean and standard deviations (SD) were calculated for all variables. One-way, multivariate analysis (ANOVA) was performed to examine the relationship between level of play and off-ice, on-ice, and anthropometric characteristics of the participants. To quantify the strength and direction of the multivariate relationships, the Tukey method was applied to find means that were significantly different from each other. Standard error (SE) and alpha values were calculated for each variable. All participants met criteria for normality in each tested variable. The on-ice

repeated shift test had one subject with a problematic point that was significantly deviated from the mean for all three timing gates. Because this subject's data point was not a statistical outlier, this point was winsorized to one unit above the next highest data point to meet criteria for normal distribution. For all statistical tests, an alpha level of $p < 0.05$ was operationally defined as statistical significance.

**3. RESULTS**

Anthropometric characteristics of the forty-five male hockey players (Division I = 24, Junior = 10, Division III = 11) are shown in Table 1. Division I players were significantly taller, heavier, and had a lower percent body fat than Division III players ($p = 0.01$; $p = 0.04$; and $p = 0.004$, respectively). Body fat percentage was also significantly different between Division I and Elite Junior players ($p = 0.04$), as well as Elite Junior and Division III players ($p = 0.001$).

Division I and Elite Junior players had significantly higher values for most variables compared to Division III players (Table 2). Division I players scored significantly better in the vertical jump ($p = .001$), Wingate PP ($p = 0.04$), grip strength ($p = 0.008$), and maximum heart rate ($p = 0.04$). Junior level players scored significantly better than Division III players in vertical jump ($p = 0.003$) and Wingate fatigue index ($p = 0.03$).

Division I and Junior players were also significantly better in on-ice performance variables compared to Division III players (Table 3). Both groups scored significantly better in top speed ($p = .001$), fastest course time ($p = .001$), slowest course time ($p = 0.001$), and average course time ($p = 0.001$). Junior players additionally scored

significantly better than Division III players in gate two and total course fatigue ($p = 0.04$, and $p = 0.04$, respectively).

**Table 1.** Physical characteristics of the subjects

| Variable | Division of Play | N | Mean | ± | SD |
|---|---|---|---|---|---|
| Height (cm) | D I | 24 | 184.92 | ± | 5.99* |
| | Junior | 10 | 180.70 | ± | 6.78 |
| | D III | 11 | 178.55 | ± | 3.50 |
| Weight (kg) | D I | | 86.88 | ± | 6.69* |
| | Junior | | 82.30 | ± | 5.62 |
| | D III | | 81.45 | ± | 4.95 |
| Body Fat (%) | D I | | 11.46 | ± | 2.43*+ |
| | Junior | | 9.40 | ± | 2.01* |
| | D III | | 14.36 | ± | 2.29 |

D I = Division One Player
Junior = Junior Level Player
D III = Division Three Player
*Significantly different from Division III ($p < 0.05$)
+Significantly different from Junior ($p < 0.05$)

**Table 2.** Off-ice performance characteristics

| Variable | Division of Play | N | Mean | ± | SD |
|---|---|---|---|---|---|
| Vertical Jump (in) | D I | 24 | 20.96 | ± | 2.27* |
| | Junior | 10 | 21.30 | ± | 2.79* |
| | D III | 11 | 17.64 | ± | 2.20 |
| Wingate - Peak Power (w) | D I | | 1112.29 | ± | 121.67* |
| | Junior | | 1037.40 | ± | 132.71 |
| | D III | | 1024.27 | ± | 51.24 |
| Wingate - Mean Power (w) | D I | | 786.08 | ± | 75.08 |
| | Junior | | 762.80 | ± | 79.85 |
| | D III | | 725.00 | ± | 79.36 |
| Wingate - Fatigue Index (%) | D I | | 49.75 | ± | 7.41 |
| | Junior | | 45.00 | ± | 6.98* |
| | D III | | 53.09 | ± | 5.61 |
| Grip Strength (kg) | D I | | 66.75 | ± | 8.37* |
| | Junior | | 62.70 | ± | 10.39 |
| | D III | | 56.55 | ± | 8.65 |
| $VO_{2peak}$ (ml/kg/min) | D I | | 54.92 | ± | 3.90 |
| | Junior | | 57.20 | ± | 5.35 |
| | D III | | 52.82 | ± | 4.02 |
| Max Heart Rate (b/min) | D I | | 201.43 | ± | 9.16* |
| | Junior | | 203.50 | ± | 10.78* |
| | D III | | 193.91 | ± | 7.90 |

$VO_{2peak}$ = Maximal Oxygen Consumption
D I = Division One Player
Junior = Junior Level Player
D III = Division Three Player
*Significantly different from Division III ($p < 0.05$)

**Table 3.** On-ice performance characteristics

| Variable | Division of Play | N | Mean | ± | SD |
|---|---|---|---|---|---|
| Acceleration (sec) | D I | 24 | 2.62 | ± | 0.10 |
| | Junior | 10 | 2.55 | ± | 0.17 |
| | D III | 11 | 2.63 | ± | 0.12 |
| Top Speed (sec) | D I | | 1.57 | ± | 0.06* |
| | Junior | | 1.56 | ± | 0.08* |
| | D III | | 1.66 | ± | 0.06 |
| Fastest Course Time (sec) | D I | | 22.82 | ± | 0.47* |
| | Junior | | 22.75 | ± | 0.59* |
| | D III | | 23.68 | ± | 0.41 |
| Slowest Course Time (sec) | D I | | 26.03 | ± | 1.14* |
| | Junior | | 25.72 | ± | 1.00* |
| | D III | | 28.18 | ± | 2.02 |
| Average Course Time (sec) | D I | | 24.75 | ± | 0.76* |
| | Junior | | 24.41 | ± | 0.54* |
| | D III | | 26.30 | ± | 1.24 |
| Gate One Fatigue (%) | D I | | 7.80 | ± | 3.53 |
| | Junior | | 7.71 | ± | 2.67 |
| | D III | | 9.36 | ± | 3.99 |
| Gate Two Fatigue (%) | D I | | 9.25 | ± | 2.91 |
| | Junior | | 7.35 | ± | 2.49* |
| | D III | | 10.29 | ± | 3.03 |
| Total Course Fatigue (%) | D I | | 8.60 | ± | 3.08 |
| | Junior | | 7.39 | ± | 2.33* |
| | D III | | 10.89 | ± | 3.75 |

D I = Division One Player
Junior = Junior Level Player
D III = Division Three Player
*Significantly different from Division III ($p < 0.05$)

## 4. DISCUSSION

The primary aim of this study was to discern differences that exist in anthropometric and physiologic variables previously shown in the literature to correlate to the performance of collegiate hockey players. This study had two main findings. First, there were significant differences in anaerobic power indices between Division I and Division III players. Division I players had greater vertical jump heights, Wingate PP, grip strength, top speed, and fastest RST course time. While these differences were assumed to exist, this study is the first to objectively test and quantify the differences. Second, there was no significant difference in aerobic capacity between levels of play; $VO_{2peak}$, Wingate fatigue index, RST gate one fatigue, RST gate two fatigue, and RST total course fatigue.

Research has shown an athlete's muscle fiber distribution can have a dramatic impact on their ability to produce force (Wilmore, Costill, & Kenney, 2008). Slow twitch muscle fibers have a high level of aerobic endurance, meaning they are efficient at producing ATP from oxidative metabolism but poorly suited to generate high levels of sustained power (Wilmore et al., 2008). Fast twitch fibers rely on anaerobic mechanisms, mainly PCr metabolism, which allow them to produce high levels of force quickly (Brooks et al., 2005). Several studies have shown this discrepancy exists in the muscle fiber distribution within athletes in the same sport (Costill et al., 1976; Fink, Constill, & Pollock, 1977; Saltin, Henriksson, Nygaard, & Anderson, 1977). For example, while hockey is generally played by a homogenous group of athletes, performance discrepancies exist between players of different positions (Montgomery, 2000). A study

by Green et al. (1976) found that defensemen skate longer shifts, and at a lower average velocity, reporting a 61.6% decrease compared to forwards. To deal with the increased skating time and decreased speed, defensemen may have adapted a higher percentage of slow switch fibers compared to forwards (Montgomery, 2000).

Discrepancies in fiber distribution would have drastic effects on the rate of force production of a hockey player. Demant & Rhodes (1999) reported that PCr content at rest in slow twitch and fast twitch fibers was significantly different ($p < 0.05$), at $73.1 \pm 9.5$ and $82.7 \pm 11.2$ mmol/kg/dm, respectively. In addition, fast twitch fibers appear to rely more heavily on PCr for energy supply during maximal exercise (Gray, Soderlund, & Richardson, 2008). Another study, by Karatzaferi et al. (2001), analyzed muscle biopsies of individuals after 10 seconds of maximal dynamic exercise. The results found that PCr levels in type I, IIa, IIAx, and IIXa fibers had been depleted to 46, 53, 62, and 59% of their resting levels, respectively. These findings were supported by Gray, Soderlund, and Ferguson (2007), who found that fast twitch fibers had a greater decline in PCr content after a 6-second maximal bike sprint than slow twitch fibers. This discrepancy could be further increased between players at the Division I and Division III level, resulting in the significant difference seen in the anaerobic measures of this study.

There was no significant difference between aerobic capacity or rate of fatigue between the levels of play. This lack of variation could be explained by the homogeneity of the participants in terms of anthropometric characteristics and training history (Quinney et al., 2008). In addition, aerobic capacity has been found to be closely tied to genetics, with heritability accounting for upward of 70% of a person's $VO_{2peak}$ (Brearley

& Zhou, 2001). With such a small portion of the variance in $VO_{2peak}$ coming from a training effect it is possible that hockey players, as a population, do not have enough variance in the measure to find significant differences.

The lack of a significant difference in aerobic capacity between levels of play begs the question, "what differentiates players at these levels?" There are two possible factors to help answer this question. First, research has demonstrated that team-sport performance is more strongly correlated with $O_2$ kinetics than $VO_{2peak}$. Rampinini et al. (2009) found a significant correlation between both $VO_{2peak}$ and $O_2$ kinetics to high-intensity repeated exercise; however, $O_2$ kinetics explained more of the variability in the model ($r = 0.65$ vs. $-0.45$). While both $VO_{2peak}$ and $O_2$ kinetics appear to have an effect on performance, it would appear $O_2$ kinetics is a better predictor of performance. Second, skating mechanics (stride efficiency) are an important aspect of on-ice performance (Pearsall et al., 2000). A player with great stride efficiency has the potential to outperform a player with poor stride efficiency but the same aerobic capacity. This is due to the fact that an efficient skater does not require the same amount of energy output to meet a specific workload. Similar phenomena have been seen in endurance athletes and the effect of running economy on marathon times (Saunders, Pyne, Telford, & Hawley, 2004). While this study did not directly measure $O_2$ kinetics or stride efficiency, future research should look more closely at these relationships and their differences between levels of play in collegiate hockey players.

## 5. PRACTICAL APPLICATIONS

The results of this study indicate that Division I hockey players, when compared to Division III, have a significantly higher rate of force production on tests measuring anaerobic performance. Significant differences did not exist between any level of play, however, when comparing scores of aerobic performance. In regards to the first finding, we hypothesize that this is likely due to muscle fiber distribution differences that exist between players at these levels. In reference to the second finding, $O_2$ kinetics and stride efficiency likely contribute to performance discrepancies between levels of play despite players possessing similar aerobic capacities. This evidence suggests that it may be prudent for hockey players to emphasize training methods that focus on rate of force production and skating efficiency. These findings could be beneficial to coaches by providing normative data of performance markers for different levels of collegiate hockey. Coaches can use this data to evaluate future players during recruiting and target the ones that posses the anthropometric and physiologic traits that best fit their system. In addition, coaches can use these markers to implement training practices with their current players to maximize performance.

**CHAPTER 5: Off-Ice Anaerobic Power is Not a Good Predictor of On-Ice Repeated Shift Performance in Hockey Players**

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## ABSTRACT

Research indicates that anaerobic power is a significant predictor of acceleration and top speed for team sport athletes. While these findings have historically been believed to apply to ice-hockey, recent research has brought their validity into question. In addition, it is unknown if single bout acceleration and top speed are important performance markers for ice hockey, given the high intensity, repeated bout nature of the sport. With game demands emphasizing the ability to repeatedly produce power, current, single bout anaerobic power tests should be examined to see if they are good predictors of on-ice performance. **Purpose:** To evaluate if conventional off-ice anaerobic power tests are good predictors of on-ice acceleration, top speed, and repeated shift performance. **Methods:** Forty-five (age 18-24) hockey players completed anthropometric, off-ice, and on-ice tests. Anthropometric and off-ice testing included height, weight, body composition, vertical jump, and Wingate tests. On-ice testing consisted of acceleration, top speed, and repeated shift tests. **Results:** On-ice acceleration and velocity were significantly correlated to vertical jump (VJ) ($r = -0.42$; $p = 0.004$; $r =$

-0.58; p = 0.001), Wingate relative peak power (WRPP) (r = -0.32; p = 0.03; r = -0.43; p = 0.003), and relative mean power (WRMP) (r = -0.34; p = 0.02; r = -0.48; p = .001). Dissimilarly, none of the off-ice tests were predictors of on-ice repeated shift performance, as measured by first gate, second gate, or total course fatigue; VJ (r = 0.06; p = 0.06; r = 0.13; p = 0.71; r = 0.09; p = 0.37), WRPP (r = 0.06; p = 0.71; r = 0.14; p = 0.35; r = 0.36; p = 0.50), or WRMP (r = -0.10; p = 0.92; r = -0.01; p = 0.53; r = -0.01; p = 0.94). **Conclusion:** While conventional off-ice anaerobic power tests predict single bout, on-ice acceleration at top speed, they do not predict the repeated shift ability of the player, and thus are not good markers for performance.

# 1. INTRODUCTION

An important factor of success in team sports is a player's ability to accelerate. The player that is quicker to the ball or faster off the line will often beat their opponent; giving them an advantage. Researchers have identified anaerobic power as a key factor in predicting team sport athletes' velocity and acceleration on the field (Pienaar & Coetzee, 2013). Due to the difficulty in quantifying these variables during competition, researchers have historically relied on laboratory tests to measure anaerobic power. These measures have included the standing long jump, triple jump, vertical jump, 10-yd sprint, 20-yd sprint, and Wingate tests (Krause et al., 2012; Farlinger, Kruisselbrink, & Fowels, 2007; Behm et al., 2005; Mascaro et al., 1992; Watson & Seargent, 1986). While this list provides a plethora of options for researchers and coaches to test their athletes, these measures have not been shown to correlate to performance equally across sports ($r = 0.71$ to 0.10) (Vescovi et al., 2006; Montgomery et al., 2000) As such, each individual sport requires its own sport-specific research to establish which tests have the highest transferal rate from the lab to the field.

In the sport of ice hockey, anaerobic power development has typically been assessed by the vertical jump and 30-second Wingate tests (Behm et al., 2005; Vescovi et al., 2005; Potteiger, Smith, Maier, & Foster, 2010; Driss & Vandewalle, 2013). Recent research, however, has brought into question the validity of the Wingate test as a predictor of on-ice velocity and acceleration (Watson & Sargeant, 1986; Driss & Vandewalle, 2013). In addition, while previous literature has shown these tests correlate to on-ice acceleration and velocity, performed as a single bout (Behm et al., 2005;

mascara et al., 1992), neither have been evaluated to see if they are predictors of performance during an on-ice repeated shift test, which more closely mimics the demands placed on players during competition. This has lead to a void in the literature that prevents coaches at all levels from making informed, scientifically based decisions regarding the evaluation and training methods of their players.

This study aims to address the current limitations specific to ice-hockey by 1) verifying the correlations that have been previously shown to exist in the literature between off-ice, anaerobic power tests and on-ice acceleration and velocity and 2) determining if these tests correlate to performance during an on-ice repeated shift test. It is hypothesized that high anaerobic power outputs in the vertical jump and Wingate tests will be correlated to both on-ice velocity and acceleration, however they will not play a significant role in predicting repeated shift ability of players.

## 2. METHODS

**2.1 Experimental Approach to the Problem**. Subjects completed two testing sessions; 1) anthropometric and off-ice performance and 2) on-ice performance testing. Each session took approximately one hour and was held on the University of Minnesota campus. All subjects were tested in early June, at the start of their summer training schedules, with both sessions completed within a ten-day period, ensuring at least two days rest between sessions. Subjects were told prior to each session to refrain from heavy exercise 24 hours prior to each testing session. All subjects were told to eat a light meal two hours prior to their testing session. In addition, they were asked to refrain from caffeine, tobacco, and alcohol 12 hours prior to testing. Aside from these guidelines,

subjects were asked to maintain a normal diet and exercise regimen during testing. Exclusion criteria for the study included absence from on-ice skating over the previous 30 days due to prior or current injury and players self-reporting their position of goaltender.

**2.2 Subjects.** Forty-five male hockey players, ages 18-24 and playing Division I, Division III, or Junior level hockey in the Minneapolis, Minnesota area volunteered for this study. All testing procedures were approved by the Institutional Review Board (IRB) at the University of Minnesota prior to participant recruitment and data collection. Written, informed consent was obtained from all participants prior to the start of the study.

**2.3 Session One - Anthropometric and off-ice performance testing.** Standing height was recorded using the Frankfort Plane criterion (Heymsfield et al., 2010) and weight using a Detecto Mechanical Doctor's scale (Model #439). Body composition was assessed via hydrostatic weighing using Exertech Body Densitometry Systems software (Dresbach, Minnesota). The method is considered to be a valid and reliable method for measurement of body composition (Heymsfield et al., 2010). The participants weight was recorded eight times to account for both a learning effect, as well as to ensure consistency of the measure and accuracy of the reported weight. Percent body fat was calculated using the Brozak equation (Heymsfield et al., 2010). Residual lung volume was also estimated (Maud & Foster, 2006).

Vertical jump was tested using a Vertec™ Vertical Jump Trainer (Huntington Beach, CA). Each participant performed a five-minute treadmill warm-up, starting at 5

mph and increasing 0.5 mph per minute to a maximum of 7 mph, before performing the vertical jump protocol to standardize the measure. The vertical jump protocol consisted of two submaximal practice jumps to gain familiarization, followed by three to five maximal jumps, allowing a minimum of 30 seconds of rest between jumps. For each jump, the participant was instructed to squat to a self-selected depth (approximately 90 degrees) and hold that position for three seconds. The participant was also instructed to place one arm on their hip with the other above their head in a reaching position. After a 3-second count, the instructor would give an audible "jump" command, and the participant would jump, touching the highest vane possible on the Vertec™ Vertical Jump Trainer. These instructions ensured a standardized jump protocol for each subject, and help reduce confounding of arm swing during jumping (Ferreira et al., 2010). Jump height was assessed by counting the highest vane contacted by the participant. The test was terminated when the participant failed to increase jump height in two consecutive trials, or after the fifth trial.

The Wingate Anaerobic Test (WAnT), performed on a Monarch cycle ergometer (Langley, WA), was used to assess lower-body power production. The testing protocol for the WAnT has been standardized in earlier literature (Maud & Foster, 2006). After the seat and bar height were properly adjusted for the participant, they were instructed to warm-up by lightly pedaling for 30 seconds. During the last five seconds of the warm-up time, the participant was told to increase their pedal velocity to its maximum and maintain it for the duration of the test. When the warm-up time expired, a flywheel resistance load equaling 0.075 kg/kg body weight was applied, with the participant

continuing to pedal maximally for 30 seconds. Participants' peak (PP) and mean (MP) power were calculated as the highest average power attained during the first five seconds and full 30 seconds of the test, respectively (Maud & Foster, 2006). Power values were recorded in watts and normalized for body weight. The reliability of these measures, PP and MP, have been established as robust in previous literature (r = .92) when using the 0.075 kg/kg loading parameter (Patton et al., 1985).

**2.4 Session Two - On-ice performance testing**. Session two took place at the hockey arena and consisted of an acceleration and top speed test. All on-ice testing was performed in full gear with skates sharpened to game specifications. Acceleration was assessed by having the participant sprint, from a stationary start, blue line to blue line (Distance = 15.24m) (Gilenstam et al., 2011). The participant started by standing with his front skate directly behind the blue line (starting line), stick in hand. When the participant felt he was ready, he would accelerate as fast as possible through the second blue line (finish line). Time was recorded by a TC Speed Trap-II wireless timing system (E38720, Gill Athletics, Champaign, IL). The photocells of both timing gates were placed at waist level of the participant to ensure that the laser timed his body crossing the line, not his stick. In addition, participants were told to keep their sticks on the ice to ensure they did not prematurely trip the laser timer. Once the participant crossed the finish line they coasted back to the starting line and were given two minutes to recover. The recovery time started when the participant returned to the starting line. Similar tests have been reported to have test-retest values of r = 0.8 (Bracko, 2001). Each participant performed two trials, with the fastest time being used for data analysis.

Maximal velocity was assessed after completion of the acceleration test and a two-minute recovery period. Velocity was measured by the time it took the participant to cover the distance between blue lines (15.24m) with a skating start (Gilenstam et al., 2011; Bracko, 2001). The participant was instructed to take a lap around the rink, starting at the blue line (start line), increasing their speed as they re-approach the start line. When the participant reached the start line, they were instructed to be moving as fast as possible, and to maintain their velocity through the finish line. Times were again recorded by a TC Speed Trap-II wireless timing system with the photocells placed at waist level. Once the participant crossed the finish line they coasted back to the starting line and were given two minutes to recover. The recovery time started when the participant returned to the starting line. This test has been reported to have test-retest values of $r = 0.84$ (Gilenstam et al., 2011). Each participant performed two trials, with the fastest time being used for data analysis.

Repeated shift ability was assessed after the velocity test, again giving the participant a two-minute recovery period before starting. Fatigue was measured as a percent decrement score during an on-ice repeated shift test (Peterson et al., 2014). Before the test began, participants were told the basic parameters of the test. In addition, they were told the two goals of the test: 1) skate the course as fast as possible, and 2) have the lowest drop-off in time from the best course time as possible. The participants then watched an instructor glide through the course to understand its layout. Each participant was then asked to skate the course at 50% of his best effort to gain familiarization. Upon completion of the familiarization trial, each participant was asked if

he felt comfortable with the course. If he said "no," he was instructed to skate the course one additional time. The participants were given three minutes to rest following the familiarization trial to ensure they were fully recovered before commencement of testing.

The test itself consisted of eight maximal skating bouts, performed in full gear (including their stick), with 90 seconds of passive recovery between bouts. Course times were measured by TC Speed Trap-II wireless timing system. There were three separate timing gates, used to evaluate first half, second half, and total fatigue decrement.

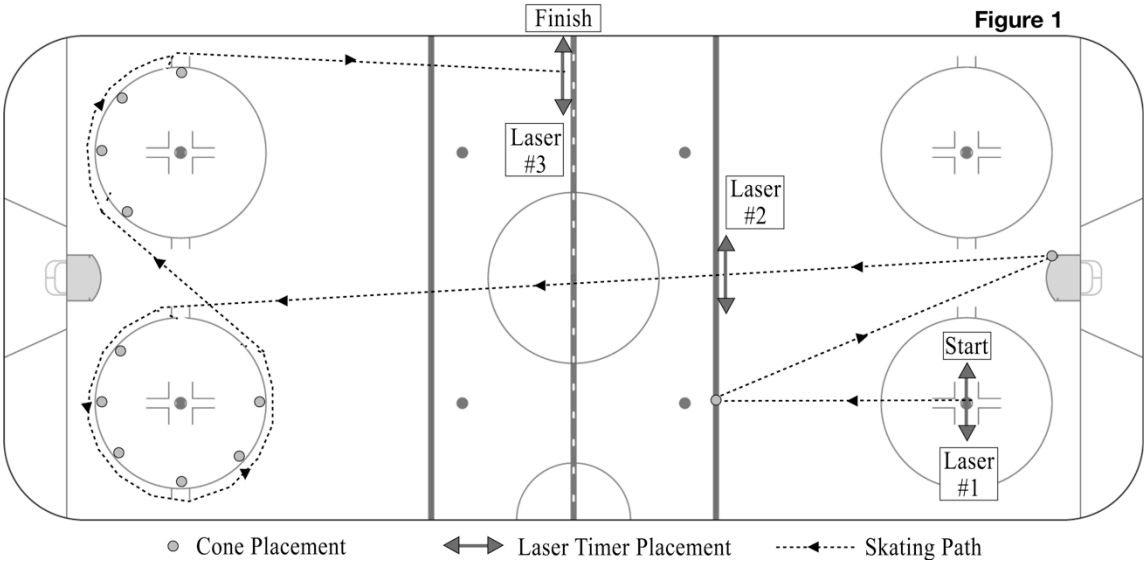


**Figure 1:** The on-ice repeated shift test consists of 8-maximal skating bouts, with 90 seconds of passive recovery between bouts. The skater starts with their skate at the face-off line; stick behind the laser timer. When given the "start" command, the skater sprints to the blue line, making a hard cut, pushing off their left leg (post leg), and sprinting to the far side of the goalie crease. The skater then makes a hard cut, pushing off their right leg (post leg), and sprints the length of the ice. The skater makes hard crossover steps to their left, going around the circle. After reaching the top of the circle the skater sprints to the base of the right circle, making hard crossover steps to their right. When the skater had completed the turn, they sprinted along the boards through the red line (finish line).

The 90-second recovery time started immediately after the participants crossed the finish line. Participants were told that during their recovery time they could stand, take a knee, or lay on the ice; they could not skate around. The participants were

informed when they had 30 seconds and 10 seconds of rest time remaining, with a 5-second countdown to the start. This sequence was completed eight times, with times being recorded after each sprint bout. Participants were not allowed to drink any sports drinks or mixes during testing, however water was provided without restriction to the participants.

**2.5 Data Analysis**. This study is powered for a two-sided test with an effect size of 0.5 and a power of 0.80. The sample size required to show significance within these parameters was calculated to be n=36 (Hulley et al., 2007). All participants met criteria for normalcy in each tested variable. Mean and standard deviations (SD) were calculated for all variables. Statistical analysis of the collated data was performed by SPSS software (IBM, vr. 21.0). Data was analyzed using either a Pearson's or Spearman's correlation test, depending on the distribution of the data. Correlational coefficients (r) were used to detect associations between the independent and dependent variable. For all statistical tests, an alpha level of $p < 0.05$ was operationally defined as statistical significance.

## 3. RESULTS

The mean and standard deviations for the subjects' physical characteristics, as well as their on- and off-ice performance scores can be seen in Tables 1 and 2; respectively. All participants met criteria for normalcy in each tested variable. Similar to previous research, this study reinforces the use of vertical jump (VJ) and Wingate testing as valid measures of on-ice acceleration and top speed. VJ was significantly correlated with acceleration, velocity, and fastest course time ($r = -0.42$; $p = 0.004$; $r = -0.58$; $p = 0.001$; $r = -0.59$; $p = 0.001$). In addition, Wingate relative peak power (WRPP) ($r = -0.32$; $p =$

0.03; r = -0.43; p = 0.003; r = -0.33; p = 0.01), and relative mean power (WRMP) (r = -0.34; p = 0.02; r = -0.48; p = .001; r = -0.38; p = 0.01) were significantly correlated to on-ice acceleration, velocity and fastest course time, respectively. This suggests that players capable of producing high power outputs during a single bout possess an advantage over other skaters in being able to accelerate more quickly and reach higher top end speeds on the ice.

**Table 1.** Physical characteristics of the subjects (n = 45)

| Variable | Mean | ± | SD |
|---|---|---|---|
| Age (years) | 20 | ± | 2 |
| Height (cm) | 181 | ± | 9 |
| Weight (kg) | 84 | ± | 12 |
| Body Fat (%) | 12.5 | ± | 4 |

Conversely, off-ice measures of anaerobic power did not predict on-ice repeated shift performance, as measured by a fatigue decrement score. VJ was not significantly correlated with performance when compared to first gate (r = 0.06; p = 0.06), second gate (r = 0.13; p = 0.71) or total course decrement (r = 0.09; p = 0.37). The data shows a similar trend when comparing WRPP (r = 0.06; p = 0.71; r = 0.14; p = 0.35; r = 0.36; p = 0.50) and WRMP (r = -0.10; p = 0.92; r = -0.01; p = 0.53; r = -0.01; p = 0.94) to first, second, and total course decrement. This suggests that power, while apparently advantageous during single bouts, is not a contributing factor to a player's ability to repeatedly perform shifts on the ice.

**Table 2.** On- and off-ice performance tests (n = 45)

| Variable | Mean | ± | SD |
|---|---|---|---|
| Vertical Jump (in) | 20.3 | ± | 2.7 |
| Wingate Peak Power (w) | 1074.1 | ± | 116.9 |
| Wingate Relative Peak Power (w/kg) | 12.7 | ± | 1.1 |
| Wingate Relative Mean Power (w/kg) | 9.04 | ± | 0.6 |
| Wingate Fatigue Index (%) | 49.5 | ± | 7.3 |
| Acceleration (sec) | 2.6 | ± | 0.1 |
| Top Speed (sec) | 1.6 | ± | 0.1 |
| Fastest Course Time (sec) | 23.0 | ± | 0.6 |
| Gate One Fatigue (%) | 8.1 | ± | 3.4 |
| Gate Two Fatigue (%) | 9.1 | ± | 3.0 |
| Total Course Fatigue (%) | 8.9 | ± | 3.3 |

## 4. DISCUSSION

The primary goal of this study was to determine if off-ice, anaerobic power tests typically used to predict on-ice acceleration and velocity are also good predictors of repeated shift performance. While this study found that vertical jump and Wingate tests are good predictors of acceleration and velocity, performed as a single bout with total recovery, they do not predict a player's performance during an on-ice repeated shift test. Fastest course time was significantly correlated to off-ice anaerobic power; however, these times were recorded on either the first or second bouts during the repeated shift test and were not correlated to total decrement. This implies that anaerobic power does not help players maintain their speed or mitigate fatigue during subsequent, high intensity shift bouts. This study is the first to show that the conventional measures of anaerobic

power may not be good measures to discern if hockey players are able to perform at a high level when stressed under game-like conditions. In addition, this study advocates for further analysis on the viability and transferability of off-ice tests used to assess the on-ice performance of hockey players.

Previous research has shown hockey players who generate high levels of anaerobic power in off-ice laboratory tests exhibit higher acceleration and velocity speeds on the ice compared to players with lower levels (Twist & Rhodes, 1993; Gamble & Montgomery, 1990; Cox, Rhodes, Thomas, & Quinney, 1981). From these results, it has been assumed that on-ice acceleration and velocity are important factors contributing to performance. This has lead coaches and players to believe that these parameters should be the focus of their off-season training, as well as one of the main criterion for coaches when selecting players for competitive teams (Cox et al., 1981).

While maximal acceleration and velocity represent a component of on-ice performance, they do not appear to be as important of a factor during competition as previously thought (Vescovi et al., 2006; Montgomery, 2000). A hockey shift has been shown in the research to consist of five to seven high intensity accelerations, each lasting approximately 2.0 to 3.5 seconds, and interspersed with moderate intensity skating, frequent turning, gliding, and shooting (Montgomery, 2000). The majority of hockey research performing maximal acceleration and velocity testing employ protocols that require a 6 to 7 second maximal bout; much longer than those seen during competition (Mascaro et al., 1992; Potteiger et al., 2010). In addition, these bouts are separated by long rest periods, allowing for the full recovery of the player between bouts. Protocols

used to test these variables typically employ one of two protocols: 1) the player is given a "running start" by skating a lap around the rink to build up speed before they reach the starting line (Behm et al., 2005) or 2) the player skates the length of the ice, from goal line to goal line as fast as they can (Bracko, 2001). In either case, these tests do not mimic an event that would occur during competition, nor would a player have that much time to reach maximal velocity during competition given the go-stop-change-direction nature of hockey (Pearsall et al., 2000).

Anaerobic power likely predicts how well a player performs during his first shift of each period. This study, and others, have found that high rates of anaerobic power are associated with players who accelerating faster and reaching higher velocities than peers with lower power rates, however this advantage appears to be mitigated as multiple shifts are performed. Muscle biopsy research has found the main energy substrate contributing to anaerobic power development to be phoschocreatine (PCr). Studies have shown PCr synthesis is slow to fully recover after intense muscular activity (Baker et al., 2010). Complete recovery of PCr after a maximal bout lasting longer than 10 seconds appears to take upwards of three to five minutes to fully replenish stores to pre-bout levels (Harris et al., 1976). Keeping these facts in mind when examining the parameters of the on-ice repeated shift test could explain why players' performance in this test was not significantly correlated with off-ice measures of anaerobic power. The recovery time given between sprint bouts was only 33% of the total time estimated to fully replenish PCr stores to their pre-exercise level. With the primary energy system contributing to anaerobic power not being given sufficient time to recover, it is unlikely the athlete

would be able to continue to perform at the same level during subsequent bouts.

The lack of a correlation between the laboratory tests and on-ice repeated shift performance could be the result of the discrepancy in the mechanics of the tasks being performed. Pearsall et al. (2000) has shown that mechanics are different between skating and running sports. Hockey is played on a crushable surface (ice) of low friction, requiring a different mechanical movement pattern to generate force into the surface than a conventional, ground-based sport (Pearsall et al., 2000). Skating mechanics require a sinusoidal pattern in which the player displaces a reactive force laterally, compared to displacing force posteriorly in running sports (Marino, 1977). The biphasic movement pattern exhibited while skating also requires a different posture and altered activity patterns of the leg muscles compared to field-based sports (Pearsall, 2000). While research has found the muscle recruitment patterns in cycling to be similar to those used in skating, these tests were performed on marathon speed skaters. Speed skaters have been found to have different posture, mechanics, and physiologic demands than those of hockey players, and are thus not a good population from which to draw comparisons (Geisel, 1980; Geisel, 1979).

One possible conclusion from the lack of correlation between off-ice anaerobic power and the on-ice repeated shift test is that off-ice tests with dissimilar movement patterns to skating will have a low transference to on-ice performance. This was shown in a recent study, which found there was no correlation between the on-ice and off-ice $VO_{2max}$ values in collegiate hockey players (Durocher et al., 2010). They concluded that off-ice movement modalities are significantly different from skating, altering stride

efficiency and motor unit recruitment, and should not be used to predict on-ice performance. This would imply that hockey players' need to be tested in a sport-specific manner to garner reliable and transferable results.

This is an important finding, as the use of the Wingate and vertical jump tests are prevalent throughout the sport of hockey. Both tests are used by the National Hockey League (NHL) to assess prospective players during the NHL combine each year, without any testing done to evaluate repeatable power generation (Vescovi et al., 2006). A study found that 82% of NHL teams use the Wingate and 43% use vertical jump to measure the anaerobic power capabilities of their players (Ebben et al., 2004). Many of these coaches stated that they use this information to make decisions regarding the on-ice performance capabilities of their players. Given the findings of this study, it could be argued that coaches are currently making decisions using data that does not encapsulate the parameters and demands of on-ice performance.

## 5. PRACTICAL APPLICATIONS

The results of this study indicate that conventional off-ice, anaerobic power tests are not good predictors of on-ice repeated shift performance. Coaches, scouts, and general managers should use other sport-specific tests to evaluate this variable in current or prospective players. We hypothesize this is due to a lack of specificity between variables, resulting from poor on-ice testing parameters. Despite the lack of a correlation between these variables, coaches and players should not discredit the importance of anaerobic power to on-ice performance. Instead, this study advocates for a paradigm shift, moving away from the belief that maximal anaerobic power is the most important

factor to on-ice performance, and instead emphasizing a player's ability to repeatedly produce power as a key factor to success.

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# CHAPTER 7: Appendices

## *7.1 Informed Consent*

**CONSENT FORM**
**Vitamin D and Measures Related to Physical Performance Consent Form**
**University of Minnesota**

You are invited to participate in a research study assessing vitamin D levels, physical performance, and body composition. You were selected as a possible participant because you are an NCAA Division I or Division III hockey player. We ask that you read this form and ask any questions you may have before agreeing to be in the study.
The principal investigator (person in charge) of this study in Stacy Ingraham, Ph.D. The study co-investigators are John Fitzgerald, B.S., and Ben Peterson, M.Ed. The researchers work in the Department of Kinesiology at the University of Minnesota. The study is funded by the Human and Sport Performance Laboratory.

**Study Purpose**
The purpose of the study is to determine if vitamin D levels influence physical performance, aerobic capacity, and body composition in hockey players.

**Study Procedures**
If you agree to participate in this study, we would ask you to participate in two research visits to the Laboratory of Physiological Hygiene and Exercise Science, located in 27 University Recreation Center on the campus of the University of Minnesota. One of the two visits will also include a skating treadmill exercise test that will be conducted at Mariucci arena on the University of Minnesota campus. Total testing time will be approximately 120 minutes or less per session. The two testing sessions will include the following:

**Laboratory of Physiological Hygiene and Exercise Science:**

Session 1 (approximately 120 minutes)

- Standard measurements of height and weight will be taken.
- Body composition (fat mass and fat free mass) will be taken using underwater weighing. You will be asked sit on a chair that is under water. While seated on the chair, the water level will be at approximately shoulder level. You will be asked to take a deep breath and then maximally exhale, and then submerse your head below the water for a brief period.
- Basic background history, physical activity, dietary habits, and sun exposure will be assessed. You will be asked to fill out a brief questionnaire for each topic.
- Jumping ability will be assessed. You will be asked to jump as high as possible using two different jumping techniques.
- Hand grip strength will be assessed. You will be asked to maximally squeeze a gripping device for approximately two seconds.
- Power production while cycling will be measured. You will be asked to pedal as fast as possible on a cycle ergometer (exercise bike) against a resistance for 30 seconds.
- A skating treadmill familiarization session will be preformed after testing. The skating treadmill familiarization session will be performed on a Frappier skating treadmill. This oversized treadmill has a surface that enables you to skate naturally. The familiarization

session will require you to skate on the treadmill at different speeds and grades that you will experience in the skating treadmill GXT during session 2. The purpose of the familiarization session is to allow you to become comfortable with skating on the treadmill.

Session 2 (approximately 60 minutes)

- Vitamin D levels will be assessed. Your finger will be pricked and a small blood sample will be taken. The amount of blood needed for the test is approximately 13 drops or 1/5 of a teaspoon.
- Aerobic capacity will be assessed during a skating treadmill graded exercise test (GXT). The skating GXT will be performed on a Frappier skating treadmill. This oversized treadmill has a surface that enables you to skate naturally. The exercise intensity (skating speed and treadmill grade) will begin at a low level and will be advanced in stages depending on your fitness level. During the test you will wear a facemask and breathing valve that allows for exhaled air to be analyzed. We may stop the test at any time because of signs of fatigue or changes in your heart rate or symptoms you may experience. It is important for you to realize that you may stop when you wish because of feelings of fatigue or any other discomfort. For your safety, you will be wearing a safety harness that is attached to a steel structure on the celling. If you where to lose your balance while skating, the harness will prevent you from falling.

If you agree to participate in a secondary study, further testing will involve two additional visits to Mariucci and Ridder Arena on the University of Minnesota campus and include the following:

Session 3 (approximately 35 minutes)

- At the start of the session, you will complete a 10-minute general warm-up. This includes a 5-minute bike warm-up and a 5-minute dynamic warm-up of the lower extremities. During the dynamic warm-up, each participant will be taken through a series of motions that work to activate and warm-up the muscles that will be used during the testing exercise. The dynamic warm-up will involve 6 low-intensity bodyweight exercises performed in series.
- Lower-body power will be assessed. You will be asked to perform two attempts in a broad jump test. A piece of tape will be laid on the floor, marking where you will place your toes. You will be instructed to jump as far as you can.
- Lower-body strength will be assessed. You will be asked to perform the barbell back squat exercise. You will be asked to perform multiple sets of the exercise, starting with a low weight and gradually progressing to a maximal weight that you can perform while maintaining correct exercise form. The last successful attempt will be recorded as your one repetition maximum (1RM). Your barbell back squat 1RM is the maximal amount of weight that you can lift during this exercise while maintaining correct exercise form. We may stop the test at any time, because of signs of fatigue or changes in your exercise form or symptoms you may experience. It is important for you to realize that you may stop when you wish because of feelings of fatigue or any other discomfort. A National Strength and Conditioning Association certified strength and conditioning specialist (CSCS) will conduct all 1RM testing. In addition, a 3-point person spot will be provided on every maximal attempt for your safety.

Session 4 (approximately 50 minutes)

- You will be required to wear full hockey gear to the rink. Before testing begins, you will be instructed to go through your normal team, pre-game warm-up to ensure you are loose and prepared for high intensity activity.
- Skating acceleration and speed will be assessed on ice. You will be asked to skate as fast as possible from a standing position to a cone 15 meters away. You will then coast around the perimeter of the ice rink three times, building in speed on the last lap as you re-approach the starting line. You will be instructed to skate as fast as possible and maintain your speed for 15 meters after you reach the starting line. You will be required to wear all of your hockey equipment during the assessment. We may stop the test at any time, because of signs of fatigue or changes in your heart rate or symptoms you may experience. It is important for you to realize that you may stop when you wish because of feelings of fatigue or any other discomfort.
- Repeated sprinting ability will be assessed on-ice. The assessment involves skating around a series of cones as fast as possible for approximately 26 seconds. After a rest period (110 seconds), you will be asked to complete the course again. You will be asked to repeat the course eight times. You will be required to wear all of your hockey equipment during the assessment. We may stop the test at any time, because of signs of fatigue or changes in your heart rate or symptoms you may experience. It is important for you to realize that you may stop when you wish because of feelings of fatigue or any other discomfort.

**Risks of Study Participation**

The study has the following risks. The physical performance testing in this study involves maximal effort. There exists the possibility of certain changes during this testing. These include abnormal blood pressure, fainting, irregular, fast, or slow heart rhythm, and in rare instances, heart attack, stroke, or death. In addition, there is a maximal lower-body strength test if you participate in the secondary study, which will load the spine during a period of maximal exertion. While very rare, the possibility of serious bodily injury can occur in the form of a pulled or strained muscle, disc herniation, sports hernia, or broken bone. Every effort will be made to minimize these risks by evaluation of preliminary information related to your health and fitness and by careful observation during testing. We may stop the test at any time because of signs of fatigue or changes in your heart rate or symptoms you may experience. It is important for you to realize that you may stop when you wish because of feelings of fatigue or any other discomfort. This study involves a capillary blood test in which a small quantity of blood is taken. This procedure involves minimal discomfort. You may experience slight discomfort during the finger prick and you may experience pain, tenderness at the puncture site after the procedure and a small, but possible chance of infection. Every effort will be made to minimize the risks associated with this procedure by properly selecting the puncture site, sterilizing the selected site, and using sterilized equipment. After the procedure is completed, the puncture site will be cleaned and appropriately bandaged. This study also involves underwater weighing. You may not be comfortable maximally exhaling and submerging yourself underwater for a brief period. Every effort will be made to clearly explain the procedure and minimize any anxiety associated with the procedure. Emergency equipment and trained personnel are available to deal with unusual situations that may arise.

**Benefits of Study Participation**
There may be no direct benefit to you for participating in this study. The benefit to study participation is that you will receive physiological testing results, relevant to your sport, which may allow you to evaluate or modify your current training practices.

**Study Costs/Compensation**
You will not be compensated for your participation in this study. You may incur costs due to participation in this study. The cost of travel will vary depending on the distance you live from the Laboratory of Physiological Hygiene and Exercise Science. If you will be traveling by automobile, you will have to pay to park. Parking costs will range from $4 to $9 per visit. You will not be reimbursed for either travel or parking costs associated with participation in the study.

**Research Related Injury**
In the event that this research activity results in an injury, treatment will be available, including first aid, emergency treatment and follow-up care as needed. Care for such injuries will be billed in the ordinary manner to you or your insurance company. If you think that you have suffered a research related injury, let the study investigators know right away.

**Confidentiality**
The records of this study will be kept private. In any publications or presentations, we will not include any information that will make it possible to identify you as a subject. Your record for the study may, however, be reviewed by research staff and by departments at the University with appropriate regulatory oversight. To these extents, confidentiality is not absolute. Study information will not be recorded in your medical record. Study data will be encrypted according to current University policy for protection of confidentiality. Only the previously stated research personnel will have access to the data. Neither coaches nor anyone else will be granted access to any information unless permission is given by the participant. The data/records will be destroyed after a five year time period.

**Voluntary Nature of the Study**
Participation in this study is voluntary. Your decision whether or not to participate in this study will not affect your current or future relations with the University of Minnesota. If you decide to participate, you are free to withdraw at any time without affecting those relationships.

**Contacts and Questions**
The researchers conducting this study are Stacy Ingraham, John Fitzgerald, and Ben Peterson. You may ask any questions you have now, or if you have questions later, **you are encouraged to** contact them at:

Stacy Ingraham, PhD
University of Minnesota School of Kinesiology
(612) 626-0067

John Fitzgerald
University of Minnesota School of Kinesiology
(612) 210-5599
Advisor: Stacy Ingraham, PhD

## *7.2 Preparticipation Screening Questionnaire*

# UNIVERSITY OF MINNESOTA

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming more physically active.

Please read carefully and check all that apply to you at this time.

**AHA/ACSM Health/Fitness Facility Preparticipation Screening Questionnaire**

Assess your health needs by marking all *true* statements.

***History***

You have had:

___ A heart attack
___ Heart Surgery
___ Cardiac catheterization
___ Coronary angioplasty (PTCA)
___ Pacemaker/implantable cardiac defibrillator/rhythm disturbance
___ Heart valve disease
___ Heart failure
___ Heart transplantation
___ Congenital heart disease

***Symptoms***

___ You experience chest discomfort with exertion.
___ You experience unreasonable breathlessness.
___ You experience dizziness, fainting, blackouts.
___ You take heart medications.

If you marked any of the statements in this section, consult your physician or other appropriate healthcare provider before engaging in exercise. You may need to use a facility with a **medically qualified staff**.

***Other health issues***

___ You have diabetes
___ You have asthma or other lung disease
___ You have burning or cramping in your lower legs when walking short distances
___ You have musculoskeletal problems that limit your physical activity
___ You have concerns about the safety of exercise
___ You take prescription medication (s)
___ You are pregnant

***Cardiovascular risk factors***

___ You are a man older than 45 years
___ You are a woman older than 55 years, you have had a hysterectomy, or you are postmenopausal
___ You smoke or quit within the previous 6 mo.
___ Your BP is greater than 140/90
___ You don't know your BP
___ You take BP medication
___ Your blood cholesterol level is >200 mg/dL
___ You don't know your cholesterol level
___ You have a close blood relative who had a heart attack before age 55 (father or brother) or age 65 (mother or sister)
___ You are physically inactive (i.e., you get less than 30 min. of physical activity on at least 3 days per week)
___ You are more than 20 pounds overweight

If you marked two or more of the statements in this section, you should consult your physician or other appropriate healthcare provider before engaging in exercise. You might benefit by using a facility with a **professionally qualified exercise staff** to guide your exercise program.

___ None of the above is true

You should be able to exercise safely without consulting your physician or other healthcare provider in a self-guided program or almost any facility that meets your exercise program needs.

Balady et al. (1998). AHA/ACSM Joint Statement. Recommendations for Cardiovascular Screening, Staffing, and Emergency Policies at Health/Fitness Facilities. *Medicine & Science in Sports & Exercise, 30(6).* (Also in: ACSM's Guidelines for Exercise Testing and Prescription, 7th Edition, 2005. Lippincott Williams and Wilkins http://www.lww.com)

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