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The Relationship Between Ventilatory Threshold And Repeated-Sprint Ability In Hockey Players

Matthew Robert Lowery

THE RELATIONSHIP BETWEEN VENTILATORY THRESHOLD AND REPEATED-
SPRINT ABILITY IN HOCKEY PLAYERS

by

Matthew Robert Lowery
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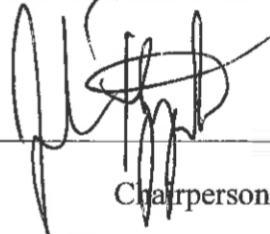
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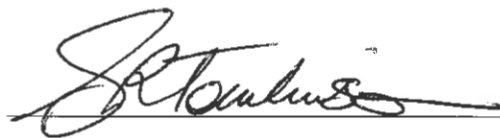
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Sandra Short

Martin Short

Grant Tomkinson

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Wayne Swisher☐

Dean of the School of Graduate Studies

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ABSTRACT

The relationship between ventilatory threshold (VT1, VT2) and repeated-sprint ability (RSA) in collegiate level male ice-hockey players was investigated. Forty-three male ice-hockey players (age 20.0 ± 1.4 years; height 182.5 ± 6.3 cm; body mass 84.8 ± 6.5 kg; percent body fat $11.8 \pm 2.8\%$; relative $\dot{V}O_{2peak}$ 55.0 ± 4.5 mL/kg/min) competing at the Division I, Division III, and Junior A level volunteered to participate in the study during the off-season. Participants first performed an incremental graded exercise test on a skate treadmill to determine their $\dot{V}O_{2peak}$, VT1, and VT2. Analysis of aerobic fitness was assessed by MedGraphics Breezesuit™ software (v-slope). After at least 48 hours, participants performed an on-ice repeated shift (RSA) test consisting of 8-maximal skating bouts lasting approximately 22.7 seconds interspersed with 90 seconds of passive recovery, to determine first gate, second gate, and total sprint decrement ($\%_{dec}$). Pearson product-moment correlations and multiple regressions were used to assess the relationship between ventilatory threshold variables (VT1, VT2, Stage at VT1, and Stage at VT2) and RSA (first gate, second gate, and total course decrement). Moderate negative correlations were detected between second gate decrement and VT1 ($r=-0.33$, $p<0.05$) and VT2 ($r=-0.30$, $p<0.05$). There were also strong negative correlations between second gate decrement and both stage at VT1 and stage at VT2 ($r=-0.55$, $p<0.001$; $r=-0.58$, $p<0.001$, respectively). Stage at VT2 was the only variable that was substantially correlated with both first gate ($r=-0.35$, $p<0.05$) and total

course decrement (r=-0.42, $p<0.05$). Multiple regressions revealed that stage at VT1 and Stage at VT2 were stronger predictors of second gate performance decrement than $\dot{V}O_{2peak}$, explaining 9% and 14% of the variance, respectively ($p<0.05$). However, stage at VT2 was not a significant independent predictor of total course decrement ($p=0.08$). The results of this study demonstrated that RSA is more strongly correlated with VT and work rates at VT than $\dot{V}O_{2peak}$. The results of this study suggest that the implementation of a training program to improve VT may lead to in improvements in RSA performance. Further longitudinal research is necessary to assess this effect.

CHAPTER I

INTRODUCTION

Hockey performance is dependent on many factors, including repeated-sprint ability (RSA) (Peterson et al., 2015), which in itself is dependent on a number of factors. Several authors have examined the relationship between RSA and aerobic fitness (the capacity to perform prolonged and exhaustive large muscle group exercise) as operationalized as $\dot{V}O_{2peak}$ (Bishop & Edge, 2006; Bishop, Edge, & Goodman, 2004; Bishop & Spencer, 2004; McMahon & Wenger, 1998; Peterson et al., 2015; Rampinini, Sassi, & Morelli, 2009; Tomlin & Wenger, 2001). Other important determinants of aerobic fitness are the lactate threshold (workload where a sustained increase in blood lactate rises above resting levels) (Gore, 2000) and economy of movement (gross oxygen cost of running per meter) (Pate & Kriska, 1994). While substantial relationships have been reported between RSA and $\dot{V}O_{2peak}$ (Bishop & Edge, 2006; Bishop et al., 2004; Bishop & Spencer, 2004; McMahon & Wenger, 1998; Peterson et al., 2015; Rampinini, Sassi, & Morelli, 2009; Tomlin & Wenger, 2001), few have examined the relationship between RSA and other components of aerobic fitness (Da Silva, Guglielmo, & Bishop, 2010).

While directly measured $\dot{V}O_{2peak}$ is considered the "gold standard" for the evaluation of aerobic fitness (Gaskill, Ruby, Walker, Sanchez, & Serfass et al., 2001; Mitchell, Sproule, & Chapman, 1958; Saltin & Astrand, 1967), the lactate threshold (LT), the highest

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sustainable work rate where lactate production and clearance is at equilibrium (Helgerud, Ingjer, & Stromme, 1990), and its associated measurement of ventilatory threshold (VT1/VT2) may be a better indicator of RSA than $\dot{V}O_{2peak}$ (Gaskill et al., 2001; Da Silva et al., 2010). In soccer players, lactate threshold was more strongly correlated with RSA than $\dot{V}O_{2peak}$ (r=-0.54 vs. r=-0.39 respectively) (Da Silva et al., 2010). This evidence indicates that it may be more important to develop a greater LT than $\dot{V}O_{2peak}$ for RSA performance among endurance athletes (Bishop, Girard, & Mendez-Villanueva, 2011).

If LT exhibits a stronger relationship to RSA than $\dot{V}O_{2peak}$, then LT may be a more useful construct for coaches, practitioners, and scientists interested in evaluating training parameters that influence RSA. Training practices to optimize improvement in LT are different to those targeting $\dot{V}O_{2peak}$ (Christopher, 2000; Maud & Foster, 2006). Furthermore, LT appears to demonstrate greater change with training and continues to improve over a longer duration compared to $\dot{V}O_{2peak}$ (Christopher, 2000; Hurley, Hagberg, & Allen et al., 1984; Katch, Weltman, & Freedson, 1978; Maud & Foster, 2006; Sjödin, Jacobs, & Svedenhag, 1982). This makes LT an attractive candidate for evaluation and programming. To date, we are unaware of any investigations looking at LT or its associated measure of ventilatory threshold (VT) in competitive hockey players. The primary aim of this study was to assess the strength and direction of the association between VT and RSA in competitive hockey players. The secondary aim was to determine if VT was a better predictor of RSA than $\dot{V}O_{2peak}$. It was hypothesized that VT would be substantially related to RSA and more strongly associated than $\dot{V}O_{2peak}$.

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CHAPTER II

REVIEW OF LITERATURE

The ability to repeatedly produce near maximal-to-maximal sprints is termed repeated sprint ability (RSA) (Bangsbo, Norregaard, & Thorso, 1991; Bishop, Spencer, & Duffield, 2001; Faude, Meyer, & Rosenberger et al., 2007; Fitzsimons, Dawson, & Ward et al., 1993; Glaister, 2005; Manrique & Gonzalez-Badillo, 2003; Spencer, Bishop, Dawson, & Goodman, 2005). This ability is highly prevalent in the sport of ice hockey where athletes perform multiple high-intensity shifts throughout the course of a single period and game. In a recent study done by Peterson et al. (2015) looking to create and on-ice repeated-shift test that mimics that of an elite level hockey player. They determined that the average shift length of a National Hockey League (NHL) forward, from 2009-2011, was found to be 45.5 ± 3.9 seconds. With an average of 6.8 ± 1.1 shifts taken per period and a rest interval of 73.4 ± 16.6 seconds between shifts (Peterson et al., 2015). Furthermore, Montgomery (2000) estimated that a hockey player spends approximately 50% of time on the ice during a game in a state of high-intensity activity (sprinting, striding, skirmishing, etc.). Thus, it can be determined that a hockey player will spend approximately 20-25 seconds of a shift in some form of maximal to near-maximal state of play (Peterson et al, 2015).

In a sport such as ice hockey, the ability of the athlete to continuously produce these repeated maximal effort shifts with minimal fatigue over the course of the game is vital.

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Understanding and being able to train and keep these athletes performing at such a maximal and repeated state is paramount for success in the competition and throughout the competitive season (Glaister, 2005; Rampinini, Bishop, Marcora, Ferrari Bravo, Sassi, & Impellizzeri, 2007). In doing this it is important to understand the physiological factors that may impair the ability of these athletes to perform maximal effort repeated shifts.

Physiological Demands of Multiple-Sprint Sports

Current research examining the physiological demands of repeated-sprint sports contends that these repeated bouts of high intensity work place considerable demands on both the anaerobic and aerobic energy systems. However, the relative contribution of each system is uncertain during repeated bouts of performance (Elliott, Dawson, & Pyke, 1985; Maud, 1983; Mayhew & Wenger, 1985; Seliger, Ejem, Pauer, & Šafařík, 1973). In the following paragraphs, we will be reviewing the parameters of each of the metabolic energy systems. Additionally, how they all interact with each other during repeated sprint performance.

Phosphocreatine

During a single short (≤7 seconds) maximal sprint, phosphocreatine (PCr) degradation is reported to account for approximately 50% of the total anaerobic ATP production (Boobis, Williams, & Wootton, 1983; Gaitanos, Williams, Boobis, & Brooks, 1993; Parolin, Chelsey, Matsos, Spriet, Jones, & Heigenhauser, 1999). Additionally, after a single 6-second all-out effort, PCr stores can be reduced to approximately 35-55% of resting levels (Bangsbo et al., 2000; Seliger et al, 1973) and the complete recovery time could potentially take up to 5-minutes or more (Akeson, Biorck, & Simon, 1968; Elliott et al.,

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1985). With what we know about the average maximal effort sprint time of a hockey shift being much longer than 7 seconds, it can accurately be assumed that PCr stores will be much more depleted over the course of a single shift, along with an increase in contribution from other energy sources as shift time is increased.

Glycolysis

As PCr contribution begins to diminish during maximal effort sprints, the increased activation of anaerobic glycolysis begins to contribute to maintain ATP turnover until PCr stores are all but exhausted. Anaerobic glycolysis has been reported to contribute approximately 40% of the total ATP production during a single 6-second sprint, with a progressive decline in energy production contribution from glycolysis as sprint work is repeated (Boobis et al., 1983; Gaitanos et al., 1993).

At these high glycolytic rates of energy production, the concentration of metabolites such as lactic acid and hydrogen ions (H^+) concentrations have often been associated with the cause of fatigue and diminished power output in subsequent sprint bouts; this will be discussed later in further detail (Bergstrom & Hultman, 1991; Metzger & Fitts, 1987; Sahlin, 1992). During these maximal repeated sprint work efforts, the increased changes in the intracellular environment due to metabolic byproducts leads to a gradual inhibition in glycolytic ATP production (Mohr, Krustrup, & Bangsbo, 2003; Signorile, Tremblay, & Ingalls 1993; Smith & Billaut, 2010; Spencer, Dawson, Goodman, Dascombe, & Bishop, 2008). For example, Gaitanos et al. (1993), reported during 10x6-second maximal sprints with 30-second recovery periods, anaerobic glycolysis accounted for 44% of total ATP contribution during the first sprint. As the latter sprints ensued, anaerobic glycolysis only accounted for 16% of total ATP production (Gaitanos et al., 1993).

As maximal effort sprints are repeated at such great intensities, the ability of the body to produce adequate levels of ATP production though PCr and anaerobic glycolysis becomes limited. As this ensues, the contribution of aerobic oxidative metabolism to energy production increases as the number of repeat sprint bouts increases.

Oxidative Metabolism

During a single short maximal sprint, the contribution of oxidative phosphorylation to total ATP production is limited to approximately <10% of total energy expenditure (McGawley & Bishop, 2008; Parolin et al., 1999). Although, as maximal intensity sprints become repeated, the level of aerobic ATP production increases as much as 40% of total energy supply during the later sprints (McGawley & Bishop, 2008). Multiple studies have found moderate to strong correlations (r=-0.45 to -0.75) between $\dot{V}O_{2peak}$ and sprint performance fatigue indices: sprint decrement (S_{dec}) and fatigue index (FI) (Bishop & Edge, 2006; Bishop & Spencer, 2004; Brown, Hughes, & Tong, 2007; Dawson, Fitzsimons, & Ward, 1993; Rampinini et al., 2009). However, there are inconsistencies in the research on the relationship between $\dot{V}O_{2peak}$ and RSA. Additional research has also shown negligible to weak correlations between $\dot{V}O_{2peak}$ and FI during repeated-sprint exercise (RSE) (r=-0.20 to -0.30) (Aziz, Chia, & Teh, 2000; Aziz, Mukherjee, Chia, & Teh, 2007; Bishop, Lawrence, & Spencer, 2003; Castagna, Manzi, D'Ottavio, Annino, Padua, & Bishop, 2007; Lane, Wenger, & Blair, 1997; McMahon & Wenger, 1998; Wadley & Le Rossignol, 1998). Whilst $\dot{V}O_{2peak}$ limits the rate at which oxygen can be provided during exercise, it does not describe all aspects of aerobic fitness, and it is unknown whether other aspects of aerobic fitness (e.g. lactate/ventilatory threshold) may have a stronger association with RSA.

As suggested by Bassett & Howley (2000), $\dot{V}O_{2peak}$ is primarily limited by what is considered "central factors" which is the ability of the cardiorespiratory system to sufficiently deliver oxygen (O_2) to the working musculature. Whereas, RSA may be primarily limited by local muscle disturbances such as increased metabolite production, termed "peripheral factors" (Bassett & Howley, 2000).

Manifestation of Fatigue During Multiple-Sprint Work

Multiple mechanisms have been reported to account for the inhibition of glycolysis contribution to energy production during repeated sprint work, one of which being a progressive drop in intracellular pH from increased production of metabolites (Bishop et al., 2003; Bishop et al., 2004; Bishop & Edge, 2006). These parameters are important because they combine together in large to contribute to the manifestation of fatigue during repeated sprint performance (Bishop et al., 2003). In the following paragraphs, we will be reviewing how the accumulation of metabolites leads to decreased RSA and ability to determine the lactate and ventilatory thresholds.

Metabolic Accumulation

Previous studies have suggested that increases in muscle (Bishop & Edge, 2006; Edge, Bishop, & Goodman, 2006; Spencer, Fitzsimons, Dawson, Bishop, & Goodman, 2006) and blood (Bishop, Lawrence, & Spencer, 2003; Ratel, Williams, Oliver, & Armstrong, 2006) hydrogen (H^+) ion accumulation produced as a result of successive sprints may impair repeated sprint performance (Spriet, Lindinger, & Mckelvie, et al. 1989; Bishop et al., 2011). This is supported by correlations that found that muscle buffering capacity and changes in pH have been associated with sprint decrement and it appears that training to improve buffering

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capacity may improve repeated sprint ability (Bishop, Lawrence, & Spencer 2003; Bishop et al., 2004; Bishop & Spencer, 2004; Mohr, Krustrup, & Nielsen et al., 2007; Bishop et al., 2011). This would suggest that instead of looking at the capacity of athletes' aerobic fitness ($\dot{V}O_{2peak}$), perhaps it would make more sense to look at the point at which these athletes can sustain a prolonged and continuous maximal work effort with this increased amount of metabolite accumulation, determined by the lactate/ventilatory threshold (LT/VT).

Lactate Threshold

The disproportionate increase in blood lactate accumulation when exceeding a certain rate of exercise/oxygen uptake has been deemed the lactate threshold (LT) (Astrand & Rodahl, 1986; Brooks, Fahey, & Baldwin, 2005). As Brooks et al. (2005) states, the lactate threshold is a result of lactate removal failing to keep pace with lactate accumulation from the working musculature. Several mechanisms have been associated with the lactate threshold, the most common being the acid/base balance and blood lactate buffering capacity (Jones & Ehrsam, 1982; Walsh & Banister, 1988; Wyatt, 1999). As stated previously, research has shown that increases in muscle (Bishop & Edge, 2006; Edge et al., 2006; Spencer et al., 2008) and blood H^+ accumulation (Bishop et al., 2003; Ratel et al., 2006) (decreased pH) observed following sprint work may impair repeated-sprint performance (Spriet, Linderinger, Mckelvie, Heigenhauser, & Jones, 1989). In supported research, significant correlations between sprint decrement (S_{dec}), and both changes in muscle and blood pH and muscle buffer capacity (βm) have been found (Bishop et al., 2003; Bishop & Spencer, 2004). The physiological changes resulting from training can influence the lactate threshold by improving mitochondrial and capillary density volume, increased pyruvate utilization, glycolytic rate changes with increased work intensities, and increased bicarbonate

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buffering capacity (Burke, Thayer, & Belcamino, 1994; Donovan & Brooks, 1983; Pierce, Weltman, Seip, & Snead, 1990).

Ventilatory Threshold

One of the determinants of LT is the non-invasive measurement of the ventilatory threshold (VT). Through gas exchange measures during a graded exercise test, it has been suggested that there are two identifiable points—the VT1 and VT2 points. The VT1 point, which reflects the exercise intensity corresponding to the initial increase in lactate concentration in the blood, is associated with an increase in V_E/VO_2 (ventilatory equivalent for oxygen) with no increase in V_E/VCO_2 (ventilatory equivalent for carbondioxide) and the departure from linearity of V_E (ventilation) as work intensifies (Caiozzo, Davis, & Ellis et al., 1982; Fontana, Keir, Bellotti, De Roia, Murias, & Pogliaghi, 2015; Plato, McNulty, Crunk, & Tung, 2008; Wasserman, Whipp, Koyal, & Beaver, 1973; Wyatt, 1999). Whereas the second ventilatory threshold (VT2) point, or respiratory compensation point (RCP), which is related to the ventilatory response to metabolic acidosis during high intensity work (Whipp, Davis, & Wasserman, 1989) is associated with an increase in both V_E/VO_2 and V_E/VCO_2, as work continues to intensify after the initial VT1 point is reached (Chicharro, Hoyos, & Lucia, 2000). The respiratory compensation point (VT2) of exercise, as work intensity increases, marks the final point from which buffering capacity has reached maximum and exercise acidosis has ensued (Chicharro et al., 2000).

The link between the lactate and ventilatory thresholds is an increase in peripheral chemoreceptor drive as work intensity increases (Davis, Frank, Whipp, & Wasserman, 1979; Swanson, 1978). As lactate accumulates with increased work intensity, there is a decrease in blood pH. This is the result of decreased buffering capacity due to an increase in CO_2 and H^+

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ions accumulation, which both act as peripheral chemoreceptors, stimulating the increase in ventilation (Brooks, 1985; Davis, 1985; Davis et al., 1979; Jones & Ehrsam, 1982; Williamson, Raven, Foresman, & Whipp, 1993). This increase in ventilation is to clear out metabolic waste (CO_2 and H+ ions) from the blood (Astrand & Rodahl, 1986) to continue to produce sufficient ATP at a higher work intensity. A meta-analysis by Wyatt (1999) identified that no significant difference exists between LT and VT. A study by Gaskill et al. (2001), examining the validity and reliability of the correlation between methods determining VT and LT, found that they were nearly perfectly correlated when using multiple methods to determine VT (r=0.95-0.98, $p<0.001$).

Summary

While $\dot{V}O_{2peak}$ has almost exclusively been examined with respect to RSA, it does not fully describe all aspects of aerobic fitness, and it may be that other aspects relate meaningfully to RSA. It has been determined that athletes with a higher aerobic fitness and the ability to reduce fatigue throughout the subsequent repeated maximal effort sprints should be able to perform with the smallest drop off in sprint decrement. Athletes that are better able to continually match metabolite removal to production during these high intensity work bouts should have better RSA. Athletes that have a higher VO_2 at ventilatory threshold (VT1 and VT2) should be able to sustain a high level of work output while still being able to match metabolite production with removal. This leads to the question: do athletes with higher VT1 and VT2 have better repeated sprint performance?

CHAPTER III

METHOD

Experimental Approach to the Problem

A cross-sectional study design was used to determine the relationship between VT (VT1 & VT2) and RSA ($\%_{dec}$ of first gate, second gate, and total course), during an on-ice repeated shift test, in competitive ice-hockey players. As acidosis begins to rise during high intensity work two points can be identified, that have been related to physical performance, with regard to change in ventilation (Chicharro et al., 2000). The first ventilatory threshold (VT1) can be determined by the increase in ventilation in response to buffering the initial rise in metabolite accumulation (Chicharro et al., 2000). The second ventilatory threshold (VT2) is identified as hyperventilation, which occurs in response to metabolic acidosis (Chicharro et al., 2000).

At the start of the participant's offseason training schedule, athletes participated in three testing sessions that consisted of the assessment of anthropometric characteristics, aerobic fitness, and on-ice repeated shift performance. All sessions were completed within a 10-day period, ensuring at least 48-hours rest between testing sessions. Participants were instructed to refrain from caffeine, tobacco, and alcohol 12 hours prior, and to avoid heavy exercise 24 hours prior to testing sessions. Exclusion criteria included absence from on-ice skating over the previous 30 days due to injury and athletes that identified their position as goaltender.

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Participants

Forty-three, well-trained, male ice-hockey players between the ages of 18 and 23 years (mean ± SD: age 20.0 ± 1.4 years; height 182.5 ± 6.3 cm; body mass 84.8 ± 6.5 kg; percent body fat 11.8 ± 2.8%; relative $\dot{V}O_{2peak}$ 55.0 ± 4.5 mL/kg/min) competing at the Division I, Division III, and Junior A level participated in this study. All participants were recruited via convenience sample from the Minneapolis, Minnesota area. Written, informed consent was obtained from all participants prior to the start of the study. The Institutional Review Board (IRB) at both the University of Minnesota (IRB-1203M1103) and University of North Dakota (IRB-201510-089) approved this study.

Procedure

Anthropometric Assessment

Body mass (kg) and height (cm) were recorded using a stadiometer and the Detecto Mechanical weight scale (Webb City, MO, USA), respectively. Hydrostatic weighing was used to assess body density and determine percent body fat using Exertech Body Densitometry Systems software (Dresbach, MN, USA). The procedure used for hydrostatic weighing was as described by Graves, Kanaley, Garzarella, and Pollock (2006).

Determination of Aerobic Fitness

A graded exercise test was performed on a synthetic ice skate treadmill (Frappier™ Acceleration, Minneapolis, MN. USA; The Blade™ Woodway, Waukesha, WI. USA) to determine the $\dot{V}O_{2peak}$ and VT of the athletes. The Ultima CPX (Medgraphics, St. Paul, MN. USA) was used to measure breath-by-breath respiratory gas volume and concentration using

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Breezesuite™ software (Medgraphics, St. Paul, MN). The Ultima CPX has been determined as a valid and reliable means of oxygen up-take measurement (Miodownik, Carlon, Ferri, Burda, & Melendez, 2000; Prieur et al., 1998). End stage completed during a GXT has previously been used as a work rate predictor associated with $\dot{V}O_{2peak}$ (Peterson et al., 2015). The protocol used in the assessment of VT and $\dot{V}O_{2peak}$ began with participants skating at a 2% grade and a speed of 6.5 mph (Koepp & Janot, 2008). After every minute, treadmill speed was increased by 0.5 mph until a maximal speed of 10 mph was achieved at the eight-minute mark. Once maximal speed was achieved, a 1% grade increase occurred every minute until the participant reached volitional exhaustion. This protocol has been previously deemed to be a valid means to reach volitional exhaustion and accurately determine $\dot{V}O_{2peak}$ (Koepp & Janot, 2008).

Determination of Ventilatory Threshold

Both VT1 and VT2 were determined by Breezesuite™ software (Medgraphics, St. Paul, MN, USA) without manual adjustment. Breezesuite™ software's VT detection program utilizes an iterative regression and analysis of the slope VCO_2 vs. VO_2 to determine where CO_2 production begins to increase disproportionately to the O_2 consumption (V-Slope method). A study by Dickstein, Barvik, Aarsland, Snapinn, and Millerhagen (1990), determined that this method correlates very strongly with the lactic acidosis threshold, and is a reproducible and useful non-invasive measure for the determination of VT (r=0.86; p<0.001).

On-Ice Repeated Shift Test

The on-ice repeated shift used in this study was described in Peterson et al. (2015),

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which is a test created to simulate on-ice demands that are experienced during a competition. The protocol was primarily designed based on data collected from the National Hockey League (NHL) (Peterson et al., 2015). The intent of the protocol was to mimic the same work demands during a period of play. The test consists of eight maximal skating bouts, in full hockey gear, lasting approximately 25 seconds with 90 seconds of passive rest between work bouts. The on-ice repeated shift protocol is shown in Figure 1. Three separate timing gates were used to assess first gate (first half), second gate (second half), and total fatigue decrement (total course). Time to complete the course was measured using a TC Speed Trap-II wireless timing system (E38720, Gill Athletics, Champaign, IL). Fatigue was calculated as a percent decrement score (%dec score = (100 x [Total Sprint Time ÷ Fastest Sprint Time] – 100) for gate 1, gate 2, and total course (Glaister, Howatson, Pattison, & McInnes, 2008; Peterson et al., 2015).
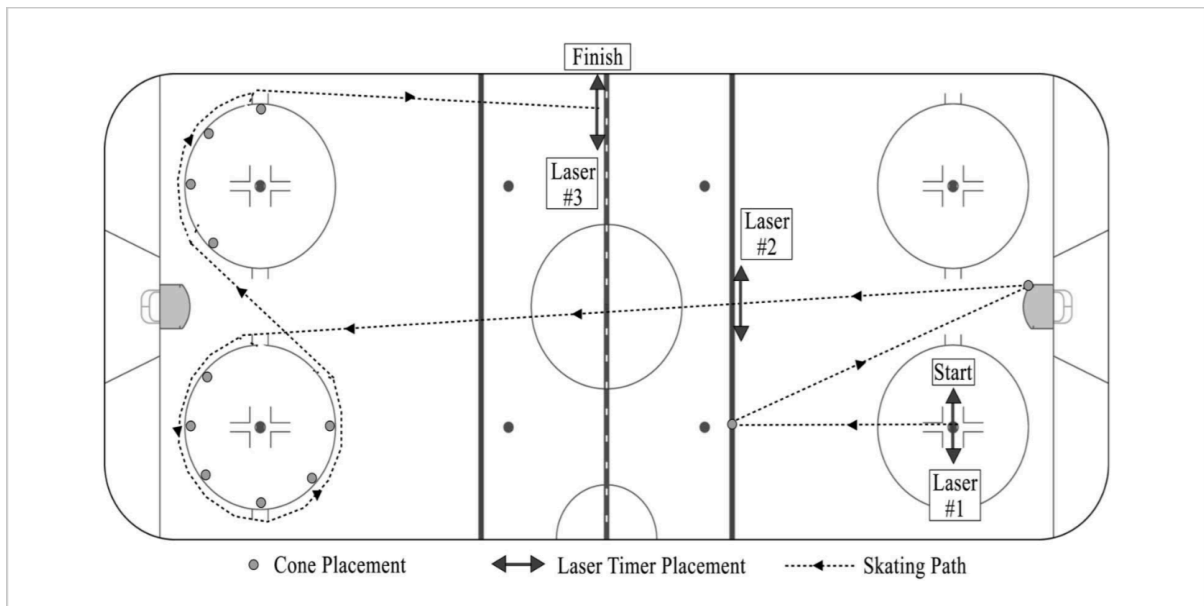
Figure 1. The on-ice repeated shift protocol described by Peterson et al. (2015) [Permission #3834891412639]

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Statistical Analysis

The Statistical Package for Social Sciences (SPSS 23 for Mac; IBM, Armonk, NY. USA) was used to perform all statistical analyses. Descriptive characteristics are presented as means and standard deviations. Pearson product-moment correlation coefficients (r) were used to evaluate the association between ventilatory thresholds and $\%_{dec}$ scores. When significant correlations were found between variables, sequential linear regression models were run. Sequential linear regression was used to determine the strength of prediction of VT1, VT2 and $\dot{V}O_{2peak}$ gas values and associated work rates on decrement (%) during the repeated shift test. Treadmill type was entered into control block during sequential linear regression to control for influence of treadmill type. All variables met criteria for normal distributions. Statistical significance was determined using 2-sided p-values and an alpha level of 0.05. Multi-collinearity was not indicated in any of the multiple regression models as evidenced by tolerance levels being >0.1 and variance inflation factors (VIF) being <10 (Bowerman & O'Connell, 1990; Myers, 1990).

RESULTS

Complete data from 43 male hockey players were available for analysis in this study. Of the 45 athletes that initially participated in all assessment sessions, ventilatory threshold data for two athletes were not retrievable due to a software malfunction. Descriptive statistics for participants' ventilatory thresholds and $\dot{V}O_{2peak}$ are presented in Table 1.

Table 1. Descriptive statistics for VT1, VT2, and $\dot{V}O_{2peak}$ of participants (*n*=43)

	Mean (SD)	Min-Max
VT1 (mL/kg/min)	45.3 (6.2)	34.1–57.4
VT1 Stage (Treadmill Test)	6.2 (3.3)	1–12
VT2 (mL/kg/min)	50.2 (5.5)	37.8–59.9
VT2 Stage (Treadmill Test)	8.8 (2.6)	3–13
$\dot{V}O_{2peak}$ (mL/kg/min)	54.9 (4.5)	44.9–64.6
Final Stage completed	9.6 (2.0)	4-14

Correlations between ventilatory threshold and $\dot{V}O_{2peak}$ measures and decrement during the repeated shift test are presented in Table 2. Ventilatory threshold (VT1 and VT2) were not significantly correlated with first gate or total percent decrement (*p*>0.05).

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However, a moderate negative correlation was detected between second gate decrement and VT1 ($r=-0.33$, $p<0.05$) and VT2 ($r=-0.30$, $p<0.05$). Likewise, stage at VT1 and stage at VT2 were also negatively associated with RSA, but more strongly than observed for second gate decrement ($r=-0.55$, $p<0.001$; $r=-0.58$, $p<0.001$, respectively). Additionally, stage at VT2 was a moderate negative correlate of first gate decrement ($r=-0.35$, $p<0.05$) and total course decrement ($r=-0.42$, $p<0.05$).

Table 2. Correlations between graded exercise test measures and performance decrement during the repeated-shift test.

	First Gate Decrement (%)	Second Gate Decrement (%)	Total Decrement (%)
VT1 (ml/kg/min)[a]	-0.10	-0.39*	-0.18
VT1 Stage (treadmill test)[b]	-0.14	-0.55***	-0.28
VT2 (ml/kg/min)[a]	-0.13	-0.31*	-0.16
VT2 Stage (treadmill test)[b]	-0.35*	-0.58***	-0.42*
$\dot{V}O_{2peak}$ (ml/kg/min)[a]	-0.12	-0.31*	-0.17
Final Stage Completed (treadmill test)[b]	-0.22	-0.47**	-0.32*

Note: Pearson correlation test, n=43 *significance at the 0.05 level (2-tailed). **significance at the 0.01 level (2-tailed). ***significance at the 0.001 level (2-tailed). [a] Bivariate correlations. [b] Partial correlations when controlling for treadmill type.

Stage at VT1 was a significant independent predictor of second gate decrement, whereas final stage completed was not (Table 3). The squared partial correlation indicated that 9% of the variance was explained by stage at VT1. Additionally, as shown in Table 4, stage at VT2 was a significant independent predictor of second gate decrement, explaining

14% percent of the variance ($p<0.05$). Final stage completed was not a significant predictor of second gate performance ($p>0.05$). Neither stage at VT2 nor final stage completed were significant predictors of total course fatigue, (Table 5).

Table 3. Sequential linear regression with VT1 stage and final stage completed predicting second gate decrement.

Predictors	Regression Coefficient	Standard Error	Standardized Regression Coefficient	p-value	ΔR^2 (adjusted)
Step 1[a]					0.01
Treadmill Type	-1.22	0.99	-0.19	0.22	
Step 2[b]					0.28
VT1 Stage	-0.56	0.24	-0.63	0.03	
Final Stage Completed	-0.33	0.37	-0.23	0.37	

Note. Dependent Variable: Second Gate Decrement (%). [a] Predictors: (Constant) Treadmill Type. [b] Predictors: VT1 Stage (Treadmill Test), Final Stage Completed (Treadmill Test).

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Table 4. Sequential linear regression with VT2 stage and final stage completed predicting
second gate decrement.

Predictors	Regression Coefficient	Standard Error	Standardized Regression Coefficient	*p*-value	ΔR^2 (adjusted)
Step 1[a]					0.01
Treadmill Type	-1.22	0.99	-0.19	0.22	
Step 2[b]					0.30
VT2 Stage	-0.75	0.29	-0.65	0.01	
Final Stage Completed	-0.11	0.40	-0.08	0.78	

Note. Dependent Variable: Second Gate Decrement (%). [a] Predictors: (Constant) Treadmill Type. [b] Predictors: VT2 Stage (Treadmill Test), Final Stage Completed (Treadmill Test).

Table 5. Sequential linear regression with VT2 stage and final stage completed predicting
total course decrement.

Predictors	Regression Coefficient	Standard Error	Standardized Regression Coefficient	*p*-value	ΔR^2 (adjusted)
Step 1[a]					0.01
Treadmill Type	-1.22	0.99	-0.19	0.22	
Step 2[b]					0.17
VT2 Stage	-0.65	0.36	-0.50	0.08	
Final Stage Completed	0.01	0.45	0.003	0.99	

Note. Dependent Variable: Total Course Decrement (%). [a] Predictors: (Constant) Treadmill Type. [b] Predictors: VT2 Stage (Treadmill Test), Final Stage Completed (Treadmill Test).

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CHAPTER V

DISCUSSION

The main purpose of this study was to investigate the relationship between VT and RSA in collegiate level male ice-hockey players. The major finding was that both VT1 and VT2 were moderately and statistically significant correlates of second gate fatigue decrement ($\%_{dec}$) assessed from an on-ice repeated shift test. Additionally, the stage corresponding to VT1 and VT2 showed the strongest correlations with second gate decrement, with stage at VT2 being the only variable that was statistically significant across all three aspects of on-ice repeated shift performance. The secondary purpose of this study was to determine if VT was a stronger predictor of RSA compared to $\dot{V}O_{2peak}$. The results showed that both stage at VT1 and stage at VT2 were stronger predictors of second gate decrement than final stage completed during the GXT, which is a work rate predictor associated with $\dot{V}O_{2peak}$. As indicated by the strength of the bivariate associations and the trending regression results, stage at VT2 appears to be a better predictor of RSA than $\dot{V}O_{2peak}$.

To our knowledge, this study is the first to report VT for competitive ice-hockey players as it relates to RSA. Consistent with the results from this study, Da Silva et al. (2010) reported similar findings for soccer players. They found that RSA was more strongly associated with the work rate during a GXT at which the onset of blood lactate accumulation occurred (vOBLA) ($r=-0.54$, $p<0.01$) than $\dot{V}O_{2peak}$ ($r=-0.39$, $p<0.05$). Furthermore, Da Silva et al. (2010) also suggested that ventilatory threshold may better reflect the peripheral aspects

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associated with fatigue during RSA and has been associated with an increased ability to buffer metabolites (Billat, Sirvent, Py, Koralsztein, & Mercier, 2003; Thomas, Sirvent, Perrey, Raynaud, & Mercier, 2004). Buffering metabolites appears to be one of the primary limiting factors inducing fatigue during RSA (Bishop et al., 2011; Girard et al., 2011).

Stage at VT2 was also significantly correlated to first gate and total decrement; although it is unclear as to the underlying relationship between work rate at VT2 and first gate decrement performance during a repeated sprint test. To our knowledge, there has been no research looking into this association. It is possible that the underlying mechanism for this finding is that work rate at VT2 may be more strongly related to buffering capacity associated with major pH disturbances that reduce the rate of PCr resynthesis. Future studies are needed to confirm this hypothesis. However, the athletes skating economy at increased work rates may partially explain the strength of correlation between stage at VT2 and total course decrement. A recent study by Peterson et al. (2015) found similar correlations between end stage completed, a work rate predictor associated with $\dot{V}O_{2peak}$, and total course decrement during a repeated shift test (r=-0.32, p<0.05). We speculate that this is likely due to the fact that stage at VT2 suggests an increased capacity to buffer acidosis at a higher work rate. These associations suggest that athletes with greater skating mechanics reaching greater sustainable work rates may have increased RSA.

This study has its limitations. Such as, the measurement of VT by Medgraphics™ software, although unbiased by human error, may in effect be a limitation due to it being the only method used for this variables determination. It is possible that other methods may yield slightly different VT results. The results of this study are not generalizable beyond the reach of competative hockey players. Additionally, recruitment of participants was based on a

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convenience sample where the athletes were in different stages of their training cycle and competed at different levels of play may lead pose limitations. On the other hand, this study has several strengths. It was the first study, to our knowledge, that has examined these relationships in competitive ice hockey players.. This study also used a validated and reliable laboratory measures for the determination of the outcome variables (e.g., aerobic fitness variables) and utilized sports specific testing paramaters (e.g., synthetic skate treadmill).

The results from this study, although cross-sectional, indicate that athletes, coaches, and practitioners should consider implementing a training program to increase VT, which may lead to an improvement in RSA. Although not alone, work rates at given aerobic measurements seem to present the best predictive power when assessing RSA. Additionally, stage at VT2 was a moderate correlate of all three aspects of the on-ice repeated shift test, suggesting that training to increase buffering capacity at higher work rates may benefit RSA. We recommend implementing longer duration high intensity interval training at intensities at or above VT for possible increased improvements in RSA. When assessing RSA among competitive athletes, work rate intensity should be considered in conjunction with maximal aerobic capacity for the assessment of RSA. We recommend further research looking into the implementation of different training regimens to improve VT and assess its outcomes on repeated sprint performance.

In conclusion, the results of this study suggest that RSA is more strongly correlated to VT than $\dot{V}O_{2peak}$ and that stage at VT2 appears to be the best predictor of RSA among collegiate-level hockey players. We speculate that this result is likely due to the fact that stage at VT2 suggests an increased capacity to buffer acidosis at a higher work rate, associated with the sustainable work rate during later repetitions during a repeated sprint test.

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Practical Applications

The strongest predictor of RSA was stage at VT2, suggesting that training to improve work rate at VT2 may lead to improvements in RSA performance. Although, it must be noted that this study is cross-section, not longitudinal, and it is not known whether changes in VT are meaningfully related to changes in RSA. However, the findings of this study suggest a pace/tempo aerobic endurance-training program with intensity at or above VT may lead to improvements in RSA (Baechle & Earle, 2008), seeing as LT/VT may improve during training without changes in $\dot{V}O_{2peak}$ (Allen, Seals, Hurley, Ehsani, & Hagberg, 1985; Bishop, Jenkins, & Mackinnon, 1998). It is important for athletes to perform sports-specific training and to increase skating efficiency to improve work rate performance at greater intensities which may lead to a greater competitive advantage (Peterson et al., 2015).

APPENDICES

UND UNIVERSITY OF NORTH DAKOTA

DIVISION OF RESEARCH & ECONOMIC DEVELOPMENT

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Institutional Review Board
Twamley Hall, Room 106
264 Centennial Dr Stop 7134
Grand Forks, ND 58202-7134
Phone: 701.777.4279
Fax: 701.777.6708

October 13, 2015

Principal Investigator(s):	Matthew Lowery
Project Title:	The Relationship between Ventilatory Threshold and Repeated-Sprint Ability of Ice Hockey Players
IRB Project Number:	IRB-201510-089
Project Review Level:	Exempt 4
Date of IRB Approval:	10/13/2015
Expiration Date of This Approval:	10/12/2018

The application form and all included documentation for the above-referenced project have been reviewed and approved via the procedures of the University of North Dakota Institutional Review Board.

If you need to make changes to your research, you must submit a Protocol Change Request Form to the IRB for approval. No changes to approved research may take place without prior IRB approval.

This project has been approved for 3 years, as permitted by UND IRB policies for exempt research. You have approval for this project through the above-listed expiration date. When this research is completed, please submit a Termination Form to the IRB.

The forms to assist you in filing your project termination, adverse event/unanticipated problem, protocol change, etc. may be accessed on the IRB website: http://und.edu/research/resources/human-subjects/

Sincerely,

Michelle L. Bowles, M.P.A., CIP
IRB Coordinator

MLB/jle

Cc: John Fitzgerald, Ph.D.

APPENDIX B

University of Minnesota IRB Approval

UNIVERSITY OF MINNESOTA

Twin Cities Campus	***Human Research Protection Program*** *Office of the Vice President for Research*	*D528 Mayo Memorial Building* *420 Delaware Street S.E.* *MMC 820* *Minneapolis, MN 55455* *Office: 612-626-5654* *Fax: 612-626-6061* *E-mail: irb@umn.edu or ibc@umn.edu* *Website: http://research.umn.edu/subjects/*

May 7, 2012

Stacy J. Ingraham
Cooke Hall Room 221
1900 University Avenue S E
Minneapolis, MN 55455

RE: "Associations Between Vitamin D and Measures Related to Physical Performance in Collegiate Hockey Players"
       IRB Code Number: **1203M11103**

Dear Dr. Ingraham:

The Institutional Review Board (IRB) received your response to its stipulations. Since this information satisfies the federal criteria for approval at 45CFR46.111 and the requirements set by the IRB, final approval for the project (version number 1, protocol date December 1, 2011) is noted in our files. Upon receipt of this letter, you may begin your research.

IRB approval of this study includes the consent form received May 4, 2012.

The IRB would like to stress that subjects who go through the consent process are considered enrolled participants and are counted toward the total number of subjects, even if they have no further participation in the study. Please keep this in mind when calculating the number of subjects you request. This study is currently approved for 110 subjects. If you desire an increase in the number of approved subjects, you will need to make a formal request to the IRB.

For your records and for grant certification purposes, the approval date for the referenced project is April 4, 2012 and the Assurance of Compliance number is FWA00000312 (Fairview Health Systems Research FWA00000325, Gillette Children's Specialty Healthcare FWA00004003). Research projects are subject to continuing review and renewal; approval will expire one year from that date. You will receive a report form two months before the expiration date. If you would like us to send certification of approval to a funding agency, please tell us the name and address of your contact person at the agency.

As Principal Investigator of this project, you are required by federal regulations to:
*Inform the IRB of any proposed changes in your research that will affect human subjects, changes should not be initiated until written IRB approval is received.
*Report to the IRB subject complaints and unanticipated problems involving risks to subjects or others as they occur.

Driven to Discover℠

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