OXYGEN CONSUMPTION AND HEART RATE RESPONSES TO A SIMULATED ICE HOCKEY GAME IN COMPETITIVE YOUTH PLAYERS WITH TYPE 1 DIABETES MELLITUS: NO EVIDENCE FOR ANY CARDIORESPIRATORY DYSFUNCTION

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Abstract

The objective of this study was to investigate oxygen consumption (VO_2) and heart rate (HR) response in competitive youth athletes without (ND) and with type 1 diabetes mellitus (T1D) during a simulated hockey game (SHG). There were no significant differences in the nadir, mean, peak %VO2max and %HRmax values across the 3 periods of the SHG between T1D and ND (n=13) (p>0.05). Significant differences (p<0.05) for the within T1D and ND group analysis are attributed to self-selected pacing during the play shifts and active recovery components of the SHG. It is concluded that athletic adolescents with T1D achieve similar VO_2max and HRmax values plus exhibit similar VO^2 and HR responses during a SHG when compared to matched ND. Therefore adolescents with uncomplicated T1D can participate in sports; exercise and non-exercise physical activity at the same level as their ND counterparts, and not be limited by diabetes.

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SYMBOLS AND DEFINITIONS

Aerobic capacity- the maximal amount of physiologic work achieved by an individual and the ability to sustain a certain level of aerobic activity during exercise

Bench- passive recovery intervals during the hockey game simulation protocol

BG- blood glucose

bpm- beats per minute

CE- exercise controls

CS- sedentary controls

DE- exercise individuals with T1D

DS- sedentary individuals with T1D

ePARmed-X+ - Online Physical Activity Readiness Medical Examination (www.eparmedx.com)

Exercise physical activity- refers to intentional/planned, structured and repetitive physical activity and exercise that improves and maintains one or more aspects of physical plus physiological fitness

F_ECO_2- fractional concentration of expired carbon dioxide

F_EO_2- fractional concentration of expired oxygen

HITT- high-intensity intermittent training

HR- heart rate

HRmax- maximum heart rate

%HRmax- measured heart rate divided by maximum heart rate

ND- non diabetic/control

NDE- exercise individuals with neuropathy and diabetes

Non-exercise physical activity- refers to normal daily life activities including household, workplace, lifestyle, and sedentary activities

O_2 deficit- the difference between oxygen uptake of the body during the first few minutes of exercise and an equal time period after steady-state is achieved

PA- physical activity

PAR-Q+- physical activity readiness questionnaire for everyone

Physical fitness- includes a set of attributes that can be measured such as strength, flexibility, and aerobic endurance

Physiological fitness-the functional ability of the physiological responses that drive the body's response to exercise

RPM- revolutions per minute

SIT- sprint interval training

Shift- varying durations of vigorous-to-maximum cycling and active recovery repetitions during the hockey game simulation protocol

T1D- type 1 diabetes

T2D- type 2 diabetes

VO_2- oxygen consumption

VO_2max- maximal oxygen consumption

%VO_2max- measured oxygen consumption divided by maximal oxygen consumption multiple by 100 (aka oxygen consumption expressed relative to VO_2max)

WC- waist circumference

CHAPTER 1

LITERATURE REVIEW

1.0 Type 1 Diabetes Mellitus

Type I Diabetes Mellitus (T1D), formerly known as insulin-dependent diabetes or immune-mediated diabetes (Alberti & Zimmet, 1998; Weltman et al., 2009) has the highest incidence rates in youth under the age of 14 years in Canada (Canadian Diabetes Association, 2012; DIAMOND Project Group, 2006). According to the Canadian Diabetes Association, T1D affects approximately 300,000 Canadians (or 10% of diagnosed diabetes). It is a metabolic disease that requires daily vigilant medical treatment and self-management to reduce the risk of future complications associated with chronic unstable dysglycemia (Weltman, Saliba, Barrett, & Weltman, 2009). The etiology of T1D is characterized by the autoimmune destruction of beta cells of the pancreas, where the rate of cell damage is variable between individuals (Alberti & Zimmet, 1998; Maahs, West, Lawrence, & Mayer-Davis, 2010; Shugart, Jackson, & Fields, 2010). The destruction of these cells prohibits the pancreas from secreting insulin, resulting in absolute insulin deficiency and forcing individuals to rely on the injection (or infusion) of exogenous insulin to stabilize blood glucose (BG) levels (Maahs et al., 2010; Shugart et al., 2010). Exogenous insulin administration is critical for survival in persons with T1D, to prevent ketoacidosis, hypoglycemia, hyperglycemia and possibly death (Alberti & Zimmet, 1998).

It is well known that the early onset of T1D, and its long-term physiological effects, increases the risk for the development of future disorders and diseases (Chu, Hamilton, &Riddell, 2011; Jones & Poole, 2005), such as cardiovascular disease (Veves et al., 1997). T1D is

associated with a greater than 3-fold increased risk of cardiovascular disease compared to individuals without T1D (ND) (Liese, Ma, Maahs, & Trilk, 2013). For this reason the importance of habitual physical activity (PA) participation is advocated, given the evidence confirming PA to be a behavioural factor that reduces the risk of cardiovascular disease, along with many other chronic diseases and conditions (Liese et al., 2013). The Diabetes Control and Complications Trial research group showed that keeping BG levels as close to the ND range as possible reduces the risk of developing major long-term complications associated with T1D, including retinopathy, neuropathy, and nephropathy (Diabetes Control and Complications Trial, 1995; Jacobsen, Henriksen, Hother-Nielsen, Vach, & Beck-Nielson, 2009; Nathan et al., 2005). It is therefore critical for individuals to manage and sustain a healthy BG range, which can be done by partaking in a vigilant treatment plus self-management routine (Landt, Campaigne, James, & Sperling, 1985; Sideraviciute, Gailiuniene, Visagurskiene, & Vizbaraite, 2006; Weltman et al., 2009). By balancing insulin therapy and diet, most persons with T1D can manage their BG reasonably well (Canadian Diabetes Association, 2012). However, participation in non-exercise and exercise PA imposes additional and very unique challenges to the task of BG management (Canadian Diabetes Association, 2012).

1.1 Type 1 Diabetes and Physical Activity

PA plays a critical role in both the prevention and management of multiple chronic diseases and conditions, with T1D being one of them. Clinical guidelines for non-exercise and exercise PA participation for persons with T1D are still evolving (Chimen et al., 2012; Liese et al.,

2013). A large proportion of the evidence-based PA guidelines for individuals with T1D are grounded on understandings gained from studies investigating the benefits of PA interventions on type 2 diabetes (T2D) (Chimen et al., 2012; Liese et al., 2013). Presently the amount of evidence regarding the benefits of acute and chronic PA participation in T2D exceeds the information that is available for T1D (Chimen et al., 2012). Nonetheless, the limited number of studies and comprehensive reviews focusing on the health benefits of PA participation in persons with T1D clearly demonstrate its advantages in this population.

Regular non-exercise (ie. climbing the stairs, household chores) and exercise PA participation can confer the same health benefits in persons with and without T1D (Chu et al., 2011; Guelfi, Jones, & Fournier, 2005). Specifically, in studies focused on T1D, PA has been proven to: improve blood pressure (Chu et al., 2011), glucose uptake (Norris, Carroll, & Cochrane, 1990), assist with weight management (Landt et al., 1985; Norris et al., 1990), enhance cardiovascular plus peripheral vascular function (Landt et al., 1985; Mosher, Nash, Perry, LaPerriere, & Goldberg, 1998), improve musculoskeletal fitness (Chu et al., 2011; Mosher et al., 1998), improve the blood lipid profile (Campaigne, Landt, & Mellies, 1985; Laaksonen et al., 2000), as well as, reduce stress, depression and the risks for developing cardiovascular plus peripheral vascular diseases (Chu et al., 2011; Guelfi et al., 2005; Komatsu, Castro, Saraiva, Chacra, & de Barros Neto, 2005; Sideraviciute et al., 2006; Veves et al., 1997; Zinker, 1999). Additionally, and with specific reference to young persons with T1D, participation in PA including competitive sports assists with social adaptation (Sideraviciute et al., 2006; Zinker, 1999). It provides more opportunity for social interaction and allows them to integrate with their peers (Zinker, 1999). Therefore, regular non-exercise plus exercise PA is advocated and

should be a goal for youth with T1D, as it increases their sense of well-being, self-esteem, mitigates long-term complications and improves overall quality of life (Rowland, Martha Jr, Reiter, & Cunningham, 1992; Sideraviciute et al., 2006; Veves et al., 1997; Wasserman & Zinman, 1994; Zinker, 1999).

Recent epidemiological data reveal that a significant number of youth with T1D do not participate in, or meet the current global PA guidelines set-out for youth; 60 minutes of moderate-to-vigorous PA per day (ParticipACTION, 2013; Liese et al., 2013). The epidemiological data show that a large proportion of these youth spend an excessive amount of time engaging in sedentary activities such as watching TV (Liese et al., 2013). Currently, there is very little data that describe the types of activities youth with T1D participate in, and, if they differ from those of their ND counterparts (Liese et al., 2013). The limited number of studies investigating youth with T1D reveal that their overall physical plus physiological fitness levels are in the low range (Liese et al., 2013), when compared to ND individuals matched for age and levels of PA (Chimen et al., 2012).

1.2 Type 1 Diabetes and Metabolic Responses to Non-Exercise and Exercise Physical Activity

Both non-exercise and exercise PA present a unique challenge for persons with T1D, as it further complicates daily BG regulation. The regulation of insulin release during PA in ND is met by a synchronized metabolic response (Gallen, 2005; Landt et al., 1985; Wasserman &Zinman, 1994; Zinker, 1999), where the increased energy demand and BG during exercise are matched by the release of insulin from the beta cells of the pancreas and increased glucose

utilization rate by the exercising muscles (Gallen, 2005; Landt et al., 1985; Zinker, 1999). For BG levels to remain constant during exercise, glucagon is released from the pancreas to increase hepatic glucose production (Chu et al., 2011). This homeostatic response is what persons with T1D lack, and it can be particularly problematic for the athlete with T1D (Gallen, 2005; Landt et al., 1985; Wasserman & Zinman, 1994; Zinker, 1999). The inability of athletes with T1D to automatically regulate insulin delivery increases the risk for severe hyperglycemia or hypoglycemia, both during and post exercise. It is critical for athletes with T1D to pay close attention to insulin administration before, during and after PA participation. With an inadequate amount of insulin, active persons with T1D will experience a decrease in insulin-mediated glucose uptake by the muscles, along with the exercise induced release of glucose from the liver (Riddell & Perkins, 2006; Toni, Reali, Barni, Lenzi, & Festini, 2006; Tonoli et al., 2012) which leads to rising BG levels during and post exercise resulting in hyperglycemia (Riddell & Perkins, 2006; Toni et al., 2006; Tonoli et al., 2012). Hyperglycemia will persist following exercise unless insulin is administered. If the exercise intensity is high, requiring vigorous-to-maximum effort (Figure 1), individuals may experience a further increase in hyperglycaemia and ketoacidosis, especially if the individual has poor glycemic control (Jain, McVie, & Bocchini, 2006; Riddell & Perkins, 2006; Toni et al., 2006; Tonoli et al., 2012). Ketoacidosis occurs from an increased production of ketones; when glucose can no longer be used as a fuel source due to insulin deficiency, body fuel is then derived from fat releasing ketones (Jain et al., 2006; Riddell &Perkins, 2006). Hyperglycaemia and ketoacidosis may result in dehydration and decreased blood pH, which will impair athletic performance and cause severe illness (Jain et al., 2006; Riddell &Perkins, 2006). A chronically sustained hyperglycaemic

state is a major risk factor for complications leading to blindness, neuropathy, and other vascular problems (Jain et al., 2006; Riddell & Perkins, 2006; Toni et al., 2006; Tonoli et al., 2012).

Figure 1: Physical activity intensity continuum corresponding with the recruitment order of the different muscle fiber types, health benefits and physical/physiological adaptations.

Engaged Muscle Fibers:	Slow Twitch Oxidative	Fast Twitch Oxidative Glycolytic	Fast Twitch Glycolytic
Physical Activity Intensity	Sedentary	Light / Moderate / Vigorous	Maximal
Health Benefits	None	Minimum to Many Acute to Some Chronic	Many Acute and Chronic
Physical /Physiological Fitness Adaptations	None	None to Some	Enhanced

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On the other hand, the opposite can occur, with excess circulating insulin; persons with T1D experience an accelerated rate of glucose uptake by the muscle, while glucose release from the liver is decreased (Riddell & Perkins, 2006; Toni et al., 2006; Tonoli et al., 2012). With hyperinsulinemia during exercise, glucagon release is inhibited resulting in a decrease in hepatic glucose production while glucose transport out of the blood continues (Chu et al. 2011). This imbalance between glucose uptake and production lowers BG levels leading to hypoglycemia, which is a priority during and after exercise as it can cause acute life-threatening complications (Riddell & Perkins, 2006; Toni et al., 2006; Tonoli et al., 2012). Therefore, athletes with T1D must be extremely vigilant in monitoring BG levels in order to prevent the complications that

occur from poor BG control. Despite the risks that non-exercise and exercise PA pose on this population and the lack of understanding regarding the possible limitations on exercise tolerance induced by unstable BG control, the literature still supports habitual non-exercise and exercise PA participation (Gallen, 2005; Komatsu et al., 2005; Veves et al., 1997).

For many parents of children with T1D, there is a large concern around the topic of low blood glucose (i.e. hypoglycaemia) and particularly the strong link between hypoglycaemia and PA (Barnard, Thomas, Royle, Noyes, & Waugh, 2010; Frier, 2008; Robertson, Adolfsson, Scheiner, Hanas, & Riddell, 2009). Persons with T1D and their families are aware of the adverse effects of hypoglycemia and recognize that these episodes are potentially life threatening, as well as a source of social embarrassment (Barnard et al, 2010). As a result, parents and their children who have T1D may develop fear and anxiety towards the occurrence of hypoglycemic episodes and attempt to take measures to avoid hypoglycemia (Barnard et al., 2010). Di Battista et al. (2009) showed that social anxiety towards hypoglycemia is common in adolescents with T1D (mean age 15.9 ± 1.44 years), which interferes with their behaviour and quality of life. Fear of hypoglycemia is an important and highly prevalent barrier to the involvement in non-exercise and exercise PA in youth with T1D (Liese et al., 2013). While awareness can reduce the levels of fear, it is important to further investigate the physiological responses of young persons with T1D to both non-exercise and exercise PA. The outcomes of this focussed research will provide diabetes professionals, parents and persons with T1D with the information that is essential for safe and effective PA participation (Robertson et al., 2009; Wild et al., 2007).

1.3 Oxygen Uptake during Rest and Physical Activity

Skeletal muscles are responsible for facilitating movement and the capacity to carry out physical work (Jones & Poole, 2005). As such, the human body possesses a remarkable ability to alter its metabolic requirements in response to various energetic challenges, predominantly via oxidative metabolism (Poole, Kindig, Behnke, & Jones, 2004). Humans are rarely in situations of metabolic steady-state, rather, they engage in activities involving sudden transitions from one metabolic rate to another (Jones &Poole, 2005). The study of oxygen uptake (VO_2) and VO_2 kinetics encompasses the physiological mechanisms responsible for the transition between metabolic rates during non-exercise and exercise PA (Poole et al., 2004). The rate of change during transitions between energy demands is a fundamental parameter of metabolic capacity, ultimately impacting exercise performance and tolerance (Poole et al., 2004). Aerobic or cardiorespiratory fitness, defined here as the ability and the efficiency of the body to extract oxygen from the atmosphere, transport it to the working muscles, and utilize the oxygen at the muscular and cellular level during prolonged moderate-to-high-to-maximum intensity exercise, is influenced by many factors including chronic disease, and therefore has been extensively examined and described by many. It is well known that by measuring the rate of VO_2 during exercise, cardiorespiratory fitness can be evaluated (Gist, Fedewa, Dishman, & Cureton, 2014; Jones & Poole, 2005). Maximum oxygen uptake (VO_2max or VO_2peak) is considered to be the best net index of how efficiently the heart, lungs and muscles (Astrand, 1971; Komatsu et al., 2005; Veves et al., 1997) function together when subjected to varying energy demands. Physiologically, VO_2max represents the maximal rate at which the body can re-synthesize ATP

oxidatively (aka aerobically), providing an upper limit for endurance performance in athletes, sedentary, and patient populations (Jones & Poole, 2005).

In the literature, the term VO_2peak is also frequently used. This term may be defined as the highest VO_2 value achieved during an aerobic or cardiovascular fitness test. It is different from VO_2max and it is therefore important to highlight its distinction. Although VO_2peak describes the highest VO_2 value achieved during a cardiovascular fitness test, it does not necessarily define the maximum VO_2 value attainable by an individual (Whipp, n.d.). Achieving a true VO_2max depends on the determination of a particular criterion, where VO_2 no longer increases, or only increases by a trivial amount, despite a further increase in work rate (Whipp, n.d.). VO_2max tests involve high-intensity exercise and are designed to bring individuals to the limit of tolerance (Whipp, n.d.). An individual may not be able to achieve a true VO_2max as a result of the inability to perform high-intensity exercise. As a result, the VO_2peak is used as the measure of cardiovascular fitness instead of VO_2max.

1.4 Oxygen Uptake and Type 1 Diabetes

The limitations associated with the pathology of T1D on exercise performance and/or tolerance are still not well understood. The majority of research regarding T1D and exercise centres around the glucoregulatory responses (Guelfi, Ratnam, Smythe, Jones, & Fournier, 2007; Herbst, Bachran, Kapellen, & Holl, 2006; Metcaf et al., 2014; Zinman, Zuniga-Guajardo, & Kelly, 1984), whereas cardio respiratory or aerobic fitness has been inconsistently reported (Komatsu et al., 2005). As summarized in Table 1, studies evaluating aerobic fitness in children,

non-athletic and athletic youth with T1D have demonstrated lower measured VO_2max values when compared to their healthy counterparts (Austin, Warty, Janosky, & Arslanian, 1993; Komatsu et al., 2005; Nadeau et al., 2010). In the studies conducted by Arslanian, Nixon, Becker, & Drash (1990), Austin, et al. (1993), Faulkner (2010), and Faulkner, Quinn, Rimmer, & Rich (2005), VO_2peak and VO_2max were used as measures of aerobic fitness in youth with T1D. All four studies consistently reported VO_2max and or VO_2peak measures between 34-41 $mL \cdot kg^{-1} \cdot min^{-1}$, which for youth is classified as having low cardiorespiratory fitness (Liese et al, 2013).

Table 1. Summary of the studies investigating aerobic fitness measured via VO_2max values in children, non-athletic and athletic youth with and without T1D

Author	**Age**	**Sex**	**VO_2max ($mL \cdot kg^{-1} \cdot min^{-1}$)**
Austin et al. (1993)	15.6 ± 2.5 years	M/F	T1D- 33.7 ± 7.0 ND- 41.0 ± 10.4
Komatsu et al. (2005)	9-20 years	M/F	T1D- 41.57 ± 7.68 ND- 51.12 ± 9.94
Nadeau et al. (2010)	12-19 years	M/F	T1D- 31.5 ± 7.6 ND- 40.4 ± 9.9

In ND, there is a characteristic positive response in VO_2 during incremental-to-maximum exercise (Regensteiner, Reusch, Stewart, & Veves, 2009). An increase in workload during exercise requires an increase in VO_2 to meet the given energy demands, and this response describes the ability of the body's physiological mechanisms to adjust to exercise demands (Regensteiner et al., 2009). A reduction in the slope of this positive relationship could reveal abnormalities in the physiological mechanisms behind the response, including a decrease in

oxygen delivery, cardiac function, and/or an irregularity of muscle oxidative metabolism (Regensteiner et al., 2009). In cardiorespiratory and vascular disease, it has been demonstrated that a decrease in the slope of VO_2 indicates abnormalities or insufficiencies in cardiac output and gas exchange (Regensteiner et al., 2009). Specific to T1D, Austin et al. (1993) and Komatsu et al. (2005) reported that physiological fitness, as measured by VO_2peak, is associated with glycemic control, where Austin et al. (1993) concluded that a lower fitness predicted higher glycated hemoglobin (HbA1c) levels. It is still unclear in the literature how VO_2max (or VO_2peak) and HbA1c levels are related; whether a lower measured aerobic fitness elevates HbA1c levels, or higher HbA1c levels contributes to a lower fitness. Other studies investigating the mechanisms behind the impaired functional aerobic fitness observed in this population have linked it to idiopathic structural and functional changes within the cardiac and skeletal muscle (Hilsted, Galbo, & Christensen, 1979; Jones & Poole, 2005; Mildenberger et al., 1984; Nadeau et al., 2010; Vered et al., 1984; Veves et al., 1997; Williams et al., 2011). It is possible that along with the lower VO_2max or VO_2peak observed in individuals with T1D, the rate of VO_2 during exercise could also be impaired.

1.5 Oxygen Uptake Kinetics

How well an individual physiologically copes with the demands of exercise depends, in part, upon the rate of oxygen uptake, or VO_2 kinetics (Jones & Poole, 2005). The rapid increase in VO_2 at the onset of and during incremental exercise (deemed as O_2 kinetics) requires the coordination between the respiratory, cardiovascular, and muscular systems to transport

oxygen from the atmosphere, plus circulate, deliver, and utilize it at the cellular level (Jones & Poole, 2005; Whipp, 1994; Wasserman & Whipp 1972). The difference in the rate of VO_2 kinetics delineates ranges in athletic performance, where trained individuals have faster VO_2 kinetics compared to healthy untrained individuals, who sequentially have faster VO_2 kinetics than persons with chronic diseases or conditions (Jones & Poole, 2005).

1.5.1 A Brief History of Oxygen Uptake Kinetics

The pioneers of the study of VO_2 kinetics have made major contributions within the field and are inherently responsible for setting the foundation of VO_2 kinetics (Jones & Poole, 2005). Since Krogh and Lindhard (1913) first reported on the fast cardiovascular responses seen at the onset of exercise, the dynamics of the VO_2 response has been described and studied extensively (Xu & Rhodes, 1999). In the 1920s, Hill and colleagues were the first to demonstrate the exponential nature of the VO_2 response at the beginning of exercise in humans (Hill, Long, & Lupton, 1924; Jones & Poole, 2005). The energy and supply demand model proposed by Hill et al. (1924) suggested that running performance was determined by three factors: energy demand (VO_2 required), VO_2 max, and sustained aerobic capacity; and is the foundation on which recent models are built (Jones & Poole, 2005). Later on, Whipp and Wasserman had the advantage of newer technology (breath-by-breath measurements of ventilation and pulmonary gas exchange) to establish the 3-phase VO_2 response at the beginning of exercise, validate the physiological mechanisms responsible, and describe the relationship between VO_2 kinetics and exercise intensity (Jones & Poole, 2005). Lamarra and Wasserman developed the equations used to measure breath-by-breath alveolar gas exchange that are still used today (Jones &

Poole, 2005). With the advances in technology and the manufacture of reliable gas analysis systems for the measurement of VO_2, it is now possible to plot and model the kinetics of the VO_2 response.

1.5.2 Traditional Method of Measuring Oxygen Uptake Kinetics

During steady-state exercise, VO_2 increases linearly as a function of work rate (Whipp, 1994; Xu & Rhodes, 1999). This response during the transition from rest to steady-state exercise has been described by Whipp, Ward, Lamara, Davies, and Wasserman (1982) to include three phases (Barstow & Mole, 1991). The initial phase is defined as the early rapid response or "cardiodynamic stage", the second phase is characterized by the slower exponential increase of the response and described as the "fundamental stage," and the third phase refers to the "slow component" representing steady-state (Barstow & Mole, 1991; Carter et al., 2000; Jones & Poole, 2005; Whipp, 1994). All three of the phases are associated with to specific physiological mechanisms reflecting the initial increase of pulmonary VO_2 in the lungs, the increase of muscle VO_2 via mean tissue gas exchange, and the utilization of oxygen at the muscle level for the metabolic requirements of the body during steady-state exercise (Whipp, et al., 1982; Wasserman & Whipp, 1972). The three components of the kinetic behavior of VO_2 can be described by applying an exponential model:

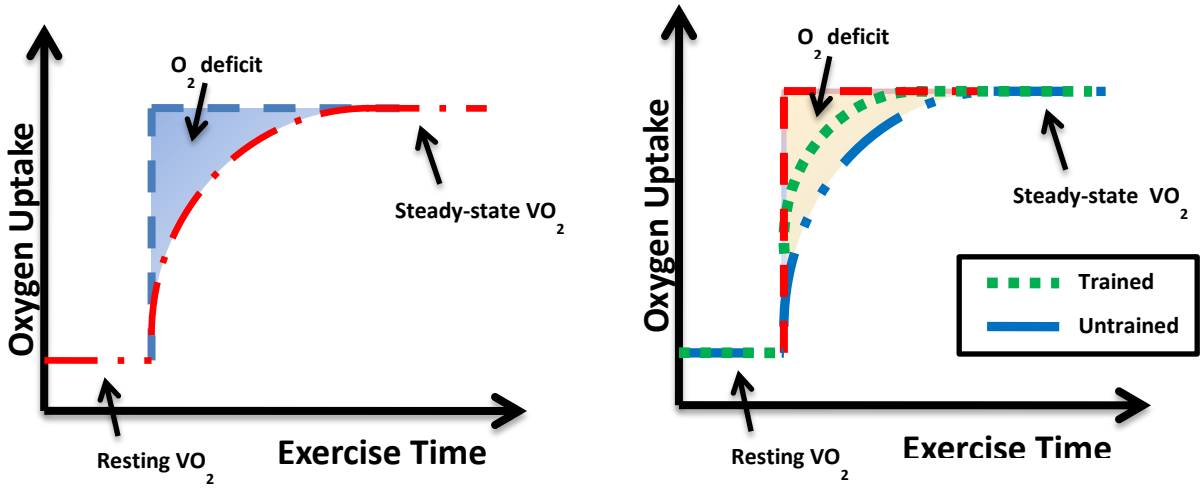
$$\mathbf{\Delta VO_2(\mathit{t}) = VO_2(b) + A(1 - e^{-\mathit{t}/T})}$$

Where $\Delta VO_2(t)$ is the increase in VO_2 at time (t); VO_2(b) is the baseline VO_2; A is the response (steady-state) amplitude; 1 – e is the base for the natural logarithm; and T is the time constant (Carter, Pringle, Barstow, & Doust, 2006; Jones & Poole, 2005; Whipp, 1994). This first-order exponential model calculates the rate of change (or slope of the line) of VO_2 at a specified time, reflecting the efficiency of the physiological mechanisms that drive VO_2, such as the adequate functioning of the O_2 transport chain (Jones & Poole, 2005; Whipp, 1994).

At the start of moderate-intensity exercise which is 40-60% of VO_2max or 55-69% of maximal heart rate (HRmax) (Guelfi, Jones, & Fournier, 2007), VO_2 initially increases rapidly and then steady-state is achieved within 1.5-3 minutes (Zoladz et al., 2013). The time constant (defined as the characteristic transition time representing the speed at which a particular system can respond to change) of the VO_2 increase observed at the start of exercise varies between individuals (Grassi, Porcelli, Salvadego, & Zoladz, 2011; Zoladz et al., 2013). The faster the VO_2 kinetics, the greater the exercise tolerance and fitness level of the individual; therefore endurance trained athletes have a significantly shorter time constant 10-20 seconds, and deconditioned persons plus persons with chronic diseases or conditions have a substantially longer time constant (40-60 seconds) (Jones & Poole, 2005; Zoladz et al., 2013). Endurance trained athletes will have a smaller O_2 deficit (Figure 2), accompanied by less disturbance in muscle metabolic stability (ie. lower acid-based disturbance) (Grassi et al., 2011; Jones & Poole, 2005; Zoladz et al., 2013). Unfit or unhealthy individuals will have a very slow response with time constants between 20-45 seconds and 40-60 seconds, respectively (Jones & Poole, 2005; Zoladz et al., 2013). As a result, these individuals will experience a larger O_2 deficit (Jones & Poole, 2005; Poole, Barstow, McDonough, & Jones, 2008; Zoladz et al., 2013) and mandate a

greater degree of intramuscular disturbance (decreased pH, ADP_{free}, and inorganic phosphate, as well as greater depletion of phosphocreatine) (Jones & Poole, 2005; Poole et al., 2008). These factors predispose individuals to reduced exercise performance and tolerance (Jones & Poole, 2005; Zoladz et al., 2013). Thus pathological conditions, such as T1D, may alter the efficiency of the VO_2 kinetic response. Given that glycemic control and changes in cardiac muscle function affect VO_2max, these factors may also affect the VO_2 kinetics in T1D, incurring a significantly slowed VO_2 response to exercise. It is essential to characterize the behaviour of the VO_2 response to PA in persons with T1D to facilitate the investigation of the underlying causes of exercise intolerance, and to develop therapeutic strategies to improve physical plus physiological fitness and quality of life (Poole et al., 2008).

Figure 2. VO_2 during the transition from rest to steady-state exercise



A B

(Powers & Howley, 2011)

Panel A. The shaded area illustrates the O_2 deficit, representing the lag in VO_2 at the start of exercise. Panel B. Comparing the differences in the time course of VO_2 from rest to steady-state exercise between trained and untrained individuals. In untrained individuals, the time to reach steady-state VO_2 is slower compared with trained individuals; as a result, untrained individuals have a greater O_2 deficit.

1.6 Oxygen Uptake Kinetics and Type 1 Diabetes

In the healthy population, limitations in highan individual's VO_2 kinetics during exercise could be a result of either inadequate blood flow to working tissues limiting oxygen transfer, or the inertia of oxidative metabolism (Regensteiner et al., 2009). In diseased states like cardiovascular disease or perhaps diabetes, oxygen delivery is compromised and the VO_2 kinetics are restricted by oxygen delivery to working muscles (Regensteiner et al., 2009). Currently, there are limited (or no) studies conducted on the VO_2 kinetics during exercise in T1D, specifically in youth with T1D. However, there have been a handful of studies that have investigated the VO_2 kinetics in premenopausal women with T2D (Ananey et al., 2011; Bauer, Reush, Levi, & Regensteiner, 2007; Brandenburg et al., 1999, Regensteiner et al., 1998). These studies reported that VO_2peak or VO_2max are consistently reduced in premenopausal women with T2D, and that the VO_2 kinetics in this population adjust more slowly compared with healthy controls matched for age, body mass, and activity levels during the onset of submaximal cycling exercise (Ananey et al., 2011; Bauer et al., 2007; Brandenburg et al., 1999, Regensteiner et al., 1998). Although the participants were matched for activity level, the authors of the aforementioned investigations may not have matched for intensity or physical plus physiological fitness levels. The participants may have performed the same activities, but it is possible that the women with T2D and the ND worked at different intensities. The ND could have been working at higher intensities, which could account for the discrepancies found in VO_2max and VO_2peak between the investigations.

Similar to T1D, the cause of exercise intolerance in T2D is still not well understood (Regensteiner et al., 1998). Regensteiner et al. (1998) assessed the dynamics of VO_2 kinetics in response to constant-load exercise in premenopausal women with T2D, and found there was a reduction in the cardiopulmonary response at the beginning of exercise. Regensteiner et al. (1998) noted that along with a possible compromised cardiac response to exercise in the participants with T2D, their VO_2 kinetics could additionally be affected by inadequate skeletal muscle oxygen diffusion, mitochondrial density, or mitochondrial oxygen utilization. Ananey et al. (2011) investigated whether cardiac output responses to constant-load cycling at light versus moderate versus vigorous intensities were related to the VO_2 kinetics during cycling exercise in women with uncomplicated T2D. The investigators observed that there was no cardiac dysfunction within the cohort with T2D and suggested that the slowed VO_2 kinetic response is partly attributed to uneven distribution and slowed dynamic adaptations of blood flow to the exercising muscles (Ananey et al., 2011). Ananey et al. (2011) also noted that the reduced VO_2 kinetics of the participants with T2D during submaximal exercise might suggest a greater reliance on the anaerobic system causing increased fatigue and contributing to exercise intolerance. It is possible, although not highlighted in the manuscript, that the slower VO_2 kinetics observed in the cohort with T2D is a function of the lower baseline fitness levels. As previously discussed, individuals with T1D have been shown to have a lower VO_2max or VO_2peak and exercise tolerance than ND matched for age, body mass and PA level, and similar to T2D, this reduced cardiorespiratory fitness may not just be attributed to deconditioning. It is also important to note that while the PA levels may be the same between individuals with and without T2D, the intensities of the exercise may differ, which could account for differences in

VO_2max. Drawing from the evidence gained from the T2D exercise research, one could presume that a lower VO_2max or peak in T1D could also be attributed to a reduction in the dynamics of VO_2 kinetics.

1.7 Oxygen Uptake Kinetics and Exercise

Traditionally, research focussed on the dynamic behaviour of VO_2 use submaximal effort constant-load incremental exercise for their investigations. In numerous studies of VO_2 kinetics, constant-load exercise was performed at moderate-to-vigorous intensity workloads below the lactate threshold. This approach was employed because it controls the rate of progression in workloads and assures a steady-state VO_2 (Regensteiner et al., 1998). Wasserman & Whipp (1972) investigated the effect of various work intensities on the VO_2 kinetics in 6 healthy volunteer using an exercise protocol that involved participants cycling at a constant-load for 6 minutes at a given workload to achieve a steady-state VO_2. Brandenburg et al. (1999) and Regensteiner et al (1998) also used constant-load exercise on cycle ergometers in their studies of VO_2 kinetics in premenopausal women with T2D. It is important to note that not all PA encountered throughout the day is at a steady-state. Many activities incorporate brief, intermittent bouts of high intensity activity interspersed with rest periods, similar to the short sprints inherent in multiple team and field sports and spontaneous play of children (Bigger, Fleiss, Rolnitzky, & Steinman, 1993; Robertson et al., 2009). It is imperative to understand the physiological response of active youth with T1D to multiple forms of non-exercise and exercise PA to ensure safe participation and optimize sport performance.

1.8 High-Intensity Intermittent Exercise

Many sports, like hockey in particular, are defined by short, high-intensity intermittent bouts of activity (Montgomery, 1988). High-intensity intermittent training (HITT) is characterized by brief bouts of vigorous-to-maximum effort. These short periods of high-intensity activity cannot be maintained for very long, and therefore are separated by intervals of light intensity PA (Burgomaster, Heigenhauser, & Gibala, 2006; Gibala et al., 2009). High-intensity exercise occurs at 80-100% of VO_2max or greater than 75% of HRmax (Guelfi et al., 2007).

HITT has been proven in the literature to be an effective strategy for inducing skeletal muscle oxidative and metabolic adaptations similar to the changes seen with chronic moderate-to-vigorous intensity endurance training (Burgomaster et al., 2006; Burgomaster et al., 2008; Gibala et al., 2009; Harmer et al., 2008). This has been the focus of the majority of research in this area. Gibala et al. (2006) reported that sprint interval training (SIT) involving 4-6 bouts of 30 second "all out" cycling at approximately 250% of the workload that elicited the VO_2peak, induced improvements in muscle oxidative capacity (reflected by the maximum activity of cytochrome C oxidase and COX subunit protein content), muscle buffering capacity and exercise performance (measured by total cycling time and mean power output) to the same extent as did traditional continuous moderate-to-vigorous intensity training involving 90-120 minutes of continuous cycling at approximately 65% of VO_2peak. In another study, Burgomaster et al. (2008) showed that a low volume of SIT involving 4-6 30 second "all out" Wingate tests, elicited similar adaptations in specific mitochondrial markers for skeletal muscle carbohydrate and lipid

metabolism and metabolic control (reduced glycogen and phosphocreatine utilization) compared with traditional continuous moderate-to-vigorous intensity endurance training involving 40-60 minutes of continuous cycling at approximately 65% VO_2peak. Along with the myocellular adaptations, HITT and SIT are also associated with increases in VO_2max or peak and work tolerance (Burgomaster et al., 2008; Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Gibala et al., 2006). In the aforementioned studies (Gibala et al., 2006 and Burgomaster et al., 2008) the researchers included participants who had no health complications and were recreationally active or untrained, respectively. One study in particular investigated the muscle oxidative adaptations to HITT in young adults with T1D (Harmer et al., 2008). The findings demonstrate that during intense exercise, HITT increased muscle oxidative capacity and reduced muscular metabolic destabilization (reducing lactate accumulation, glycogenolytic and glycolytic rates, and attenuated ATP degradation) in T1D. The participants did not experience any adverse effects due to HITT and Harmer et al. (2008) concluded that HITT is well tolerated by persons with T1D.

1.9 High-Intensity Intermittent Exercise and Type 1 Diabetes

Recently, the glucoregulatory response to HITT in T1D has been investigated and this type of exercise has been associated with higher BG concentrations. Studies reported that brief high-intensity bouts could attenuate the typical drop in BG concentration, both during and after moderate intensity exercise in T1D (Guelfi et al., 2005; Bussau, Ferreira, Jones, & Fournier, 2006; Bussau, Ferreira, Jones, & Fournier, 2007; Robertson et al., 2009). Bussau et al. (2006)

investigated the repercussions of a 10-second maximal cycling sprint following 20 minutes of moderate-intensity cycling exercise in males with T1D aged 21 ± 3.5 years. When the investigators introduced a 10-second cycling sprint at the end of the 20-minute workout, the decline in BG was no longer apparent for 2 hours following cessation of the activity. The investigators discovered that adding a short maximal sprint counters the rapid fall in glucose associated with continuous moderate-intensity exercise, and thus, provides another means to attenuate or offset the risk of hypoglycemia in active persons with T1D independent of pre-exercise dietary or insulin adjustments. Bussau et al. (2006) proposed that the mechanisms responsible for attenuating the exercise-mediated decrease in BG concentration and the stabilization of BG after exercise is consequence of the elevated levels of counterregulatory hormones including catecholamines, growth hormone, plus cortisol and lactate levels. When persons with T1D participate in intermittent high-intensity exercise, BG levels fall less rapidly and remain more stable following the activity in comparison to sustained light-to-moderate-intensity exercise (American Diabetes Association, 2004).

Most team and individual sports have a HITT component. Guelfi et al. (2007) demonstrated the glucoregulatory behavior in response to a bike protocol simulating activity patterns of team sports. The protocol involved, 30 minutes of continuous exercise at 40% of VO_2peak, interspersed with 4 maximal sprint efforts every 2 minutes. Currently, this is one of the only studies that investigated a HITT protocol tailored to simulate a game situation in T1D. More research is required on this type of activity for persons with T1D, as it will assist in creating detailed, evidenced-based guidelines for safe participation in regular intermittent high-

intensity exercise and sports. To date, no study has evaluated the cardiorespiratory responses to such exercise in individuals with T1D.

1.10 Heart Rate Kinetics

The heart rate (HR) response during PA is closely related to the rate of change of VO_2 and like VO_2 kinetics, HR may also be described mathematically as an exponential function (Stirling, Zakynthinaki, Refoyo, &Sampedro, 2008). Measuring the HR kinetics during exercise is frequently used as a simple way of assessing the degree of adaptation to a submaximal effort exercise bout and evaluating the functional circulatory capacity (Bunc, Heller, & Leso, 1988). During constant-load submaximal effort exercise the kinetics of the HR response can be separated into distinct physiological phases, similar to what has been observed with the VO_2 kinetics (Bunc, Heller, Leso, &Sprynarova, 1986). The initial phase of the HR response during exercise is designated as the "fast" component, while the second is deemed the "slow" component (Bunc et al., 1986). In a study by Bunc et al. (1988), the investigators used a mono-exponential model to determine the behaviour in HR at the onset of constant-load sub-maximal effort exercise on a cycle ergometer. The investigators confirmed, using healthy endurance trained and untrained males, that the kinetics of HR can be characterized by applying the same type of model used to describe VO_2 kinetics (i.e. a fast and slow component) and concluded that the change in HR during submaximal steady-state exercise can help assess the functional capacity and training state of individuals (Bunc et al., 1988). Since the basic response patterns for VO_2 and HR kinetics are similar in response to incremental steady-state submaximal exercise

(Stirling et al. 2008), Ananey et al. (2011), and Regensteiner et al. (1998) adapted the mathematical models developed for VO_2 kinetics to apply to HR in their T2D studies.

Comparable to VO_2 kinetics, the HR dynamics at the start of exercise are faster with a more advanced state of training, and thus trained individuals have faster HR kinetics in comparison to untrained individuals (Bunc et al., 1988). Like VO_2 kinetics, there is also a lack of research analysing the HR kinetics in persons with T1D or T2D. Currently two studies have measured HR kinetics in T2D, and found conflicting results (Ananey et al., 2011; Regensteiner et al., 1998). Along with VO_2 kinetics, Regensteiner et al. (1998) examined the HR kinetics of premenopausal women with uncomplicated T2D and found that compared to the ND, the T2D had slower HR kinetics during constant-load cycling exercise. Regensteiner et al. (1998) attributed a possible impaired cardiac response to the slower HR kinetics observed in the women with T2D. In contrast, Ananey et al. (2011) found no differences in the HR kinetics between middle-aged women with uncomplicated T2D and ND at any time point during the constant-load cycling exercise. Thus, more research is needed to confirm if HR kinetics during exercise are impaired in diabetes, and if a reduction in HR kinetics influences the VO_2 kinetic response. Investigating both VO_2 and HR kinetics in active youth with T1D will provide a more profound understanding of the limitations to exercise performance and tolerance induced by diabetes.

1.11 Type 1 Diabetes and Hockey

The sport of ice hockey is both physically demanding and multifactorial (Burr et al., 2008), as it involves full body contact, moderate-to-vigorous and vigorous-to-maximum or high-intensity intermittent skating with frequent changes in velocity and duration (Montgomery, 1988). Research is needed for hockey athletes because of its high-intensity intermittent nature, which demands its athletes to possesses high levels of muscular strength, power, endurance, aerobic power, anaerobic power and anaerobic-aerobic power (Montgomery, 1988). A typical hockey player will spend 15-20 minutes of a 60-minute game on-ice, performing at high-intensities in shifts that last 30-80 seconds interspersed with 4-5 minute passive rest periods in between (Montgomery, 1988). During on-ice hockey shifts, peak HR exceeds 90% of HRmax, with average on-ice values of approximately 85% or more of HRmax (Montgomery, 1988). Top performance in this sport necessitates an efficient aerobic system to be able to recover quickly between shifts and to meet the energy demands throughout the duration of a game (Montgomery, 1988).

Traditionally the VO_2max of hockey players is tested using continuous, incremental to maximum protocols (Montgomery, 2006). No studies have reported on the VO_2 and HR kinetics during sport-specific exercise in T1D, leaving a large gap in this specific area. Studies need to be conducted that examine sport-specific intermittent exercise to characterize the behaviour of the aerobic (cardiovascular) system in response to this type of activity, where changes in effort and metabolic requirements frequently fluctuate, to provide insight into two basic questions: A) How the aerobic system of athletes with T1D responds across a full game has yet to be

characterized; and B) Are their cumulative VO_2 and HR kinetics delayed in comparison to their healthy counterparts, and does this in turn impair or hinder their performance in the sport? It is important to indicate the differences between T1D and their healthy counterparts in order to understand their limitations and help these athletes achieve their full athletic potential.

Due to the lack of research examining HITT in persons with diabetes, research-based guidelines specific to hockey players with T1D do not exist. This creates yet another challenge for young athletes with T1D and their families in terms of insulin therapy and BG management. It forces them to discover their own strategies for BG management through trial and error or by modifying the recommendations provided from other continuous moderate-to-vigorous intensity sports. This may have severe consequences for the athlete with T1D if proper precautions are not taken, and the possibility of life threatening situations could prematurely end their participation in ice-hockey due to uncertainty and fear. Future research is required to further understand how hockey players with T1D respond throughout an entire game. This knowledge will assist in the development of detailed, evidence-based guidelines enabling athletes with T1D to safely enjoy the benefits of regular non-exercise and exercise PA and to assist with the training of these athletes to achieve their full athletic potential and performance.

CHAPTER 2

Contributions of the authors include: Deandra Filippo- Data analysis and manuscript writing, Robert Gumieniak, Chip Rowan and Justin Sanderson- Data collection, Lisa Miadovnik- Recruitment and data collection, Michael Riddell- Secondary supervisor, Veronica Jamnik- Supervisor

2.0 PURPOSE AND OBJECTIVE

The purpose of the present study was to investigate the effects of a laboratory simulated hockey game on the VO_2 and HR response in young, competitive athletes with T1D. While the principle objective is to determine whether the VO_2 and HR response differs between T1D and healthy controls, it will be interesting to observe any general trends that may occur across the three periods of the games.

Hypothesis

We hypothesized that the game VO_2 and game HR response, and $\%VO_2max$ and %HRmax will be lower in the young hockey players with T1D compared to their healthy counterparts.

2.1 METHODS

Experimental data were collected in conjunction with another study conducted by Miadovnik (2013). All protocols were reviewed and approved by the Human Participants Review Sub-Committee at York University's Office of Research Ethics. Throughout the study there were no adverse events to report on.

2.1.1 Study Participants and Recruitment

Study participants were recruited through word of mouth techniques in the Greater Toronto Area. Recruitment targeted rep-level hockey players between the ages of 13-21 years, who were diagnosed with T1D at least 1 year prior. Recruitment flyers were distributed to endocrinologist and diabetes clinics, posted in various arenas and hockey-centered fitness facilities throughout the Toronto area, and postings were made online through social networks including Facebook and Twitter. As well, parents of youth who participated in a T1D-focused sports camp were contacted via email. Interested individuals were questioned regarding pubertal level and age of diabetes diagnosis. Based on parental or guardian report, if the study participants had not commenced puberty, were newly diagnosed with T1D, played hockey at a non-competitive level, played goalie, or were diagnosed with nervous system dysfunctions or retinopathy, they were excluded from the study. A teammate who does not have T1D was selected to function as a matched control participant. The control participants were similar in age, sex, height, body mass, and physical activity level. All study participants underwent identical measures. A schematic overview of the laboratory measures is presented in Figure 3.

A total of 18 competitive, youth hockey players participated in the investigation; 12 males and 6 females with and an age range of 13-17 (mean 14.7 ± 1.58). To screen for contraindications to completing vigorous exercise, all study participants completed evidence-based 2013 PAR-Q+ and ePAR-med-X+ PA participation questionnaires (www.eparmedx.com). Minor assent forms were sent to study participants under 16 years, along with consent forms to parents or guardians, and study participants over the age of 16 were directly supplied with

informed consent forms. All forms were emailed to the study participants before visiting the laboratory, and were collected during their initial visit. All participants had a minimum of two visits to the laboratory.

2.1.2 Visit One: Laboratory Physical and Physiological Fitness Assessments

Anthropometric Data. Wearing light clothing and no shoes, body mass was measured using a digital scale (Seca Alpha, Germany). Height was measured without footwear using a wall-mounted stadiometer. BMI was calculated by dividing body mass in kilograms by height in meters squared. Waist circumference was measured using the National Institute of Health protocol, where the measuring tape is positioned horizontally on the skin, on the top border of the iliac crest. Bioelectrical impedance analysis (Tanita scale, model TBF-612, Arlington Heights, lll) was used to determine body fat percentage. Skinfold measurements were taken in accordance with the Canadian Physical Activity, Fitness and Lifestyle Approach (Gledhill & Jamnik, 2003 and the Physical Activity and Lifestyle "R" Medicine (Gledhill & Jamnik, 2012). Markings were made at the following skinfold sites: biceps, triceps, subscapula, iliac crest and medial calf, and measurements were made using Harpenden fat callipers.

Anaerobic and Aerobic Fitness. To participate in both the anaerobic and aerobic fitness assessment, individuals with T1D were required to have a BG level between 5-15 mmol/L. BG was measured with the individual's own hand held device. Individuals were allowed one attempt to correct BG levels if they were below the threshold by administering 16 grams of carbohydrate (4 tablets of Dex4). In contrast, if participants were above the BG threshold they

were instructed to consume water and take insulin if they deem that appropriate. Participants were asked to take a subsequent BG reading 15-30 minutes after their initial check and testing proceeded only if the BG levels were corrected. If the participants did not achieve the target range within 15-30 minutes, the laboratory testing was re-scheduled for another day.

A 30-second Wingate protocol (Inbar, Bar-or, & Skinner, 1996) on a Monark Ergomedic 894E Peak cycle ergometer was conducted to assess anaerobic fitness. Individuals' seat heights were appropriately fitted allowing for a slight bend of 10-15 degrees of the knee joint during maximal extension. For each individual, resistance was calculated at 7.5% of their body mass. The ergometers were connected to a Monark software program that continuously recorded revolutions per minute (RPMs) and watts, which was used to determine peak, average and minimum power output, and fatigue index. A 5-minute warm-up on the cycle ergometer was allotted, during which the protocol was explained to the study participants by a qualified exercise professional administering the test. Participants were instructed to stay seated while pedaling as hard and as fast as possible for the full 30-seconds of the test. The verbal cues "faster, faster, go" were given prior to releasing the resistance to increase their pedalling frequency for the all-out sprint. These cues were used to ensure maximal pedaling cadence was achieved prior to applying the resistance, and verbal encouragements were given to the participants for the test duration. Immediately following the 30-second test, the resistance was removed, participants remained on the cycle ergometer to cool-down, and then were instructed to lie in a supine position for a 30-minute recovery period, where they were permitted to stretch and the athletes with T1D were directed to check their BG levels.

Aerobic fitness was assessed following the 30-minute rest period provided BG levels were stable and above 5mmol/L. Appropriate measures were taken if the BG values were below this level. A modified Astrand and Pollock (1978) incremental-to-maximum effort treadmill protocol was administered where both VO_2max and HRmax were determined. Polar heart rate monitors were worn by the participants to obtain peak or maximum exercise HR. Participants were familiarized with the testing protocol and equipment during a brief warm up of walking on the treadmill. The protocol design incorporated multiple incremental 2-minute stages, where running speed and incline were progressively adjusted to increase intensity. All participants started the first stage at 3.5 mph (5.6 kph) at 0% incline, then proceeded to 5 mph (8.1 kph) at 0% incline, and then followed by 6.0 mph (9.7 kph) at 0% incline. Following the first 3 stages the speed was adjusted based on individual running biomechanics, therefore some participants may have stayed at a speed of 6.0 mph (9.7 kph), while others were increased to 6.5 (10.5 kph), 7 (11.3 kph), or 7.5 mph (12.1 kph). Once a comfortable running speed between 6.0 (9.7 kph) and 7.5 mph (12.1 kph) was determined, the speed was kept constant and the intensity was increased by adjusting the incline for the duration of the test. The incline was increased by 2% for all of the following 2-minute stages. Verbal motivation was given to the participants to run for as many continuous stages as possible. When continuous running was no longer possible for the participant, the speed was adjusted to a walking speed for 2 minutes, marking the beginning of the discontinuous workloads to maximal (or supramaximal- see below) effort. During the discontinuous portion, participants ran at the speed determined during the continuous portion, while the incline was raised 2% higher than the previous workload. Each discontinuous stage lasted 2 minutes and was separated by 2 minutes of active

recovery (walking), this pattern continued for the duration of the test until VO_2max was attained, that is the VO_2 remained the same or dropped with increasing workloads (Gledhill, Cox, & Jamnik, 1994; Howley, Bassett, & Welch, 1995). Supramaximal testing represents high intensity exercise, specifically performed by an individual at workloads greater than that required to elicit VO_2max (Crisafulli et al., 2004). Supramaximal workloads are widely used to confirm the attainment of VO_2max during cardiovascular exercise testing (Astorino, White, & Dalleck, 2009).

Indirect calorimetry, specifically open circuit spirometry, was used for the determination of VO_2 and VO_2max or VO_2peak. Open circuit spirometry requires an individual to inhale air from the atmosphere and exhale air through a mouth-piece attached to a two-way valve (Ewald Koegal Co, S*an Antonio Texas*), while wearing a nose plug. The two-way valve was attached to a hose, which was then connected to a 120L Tissotgasometer (Warren E Collins LTD. *Braintree, Massachusetts*). During the last 30 seconds of each incremental stage the fractional concentration of expired oxygen (F_EO_2) and carbon dioxide (F_ECO_2), along with the volume of air expired was measured. Oxygen consumption (VO_2) and carbon dioxide production (VCO_2) was calculated for each workload using the recorded measurements (F_EO_2, F_ECO_2, volume of air expired). Rapid response gas analyzers (Applied Electrochemistry, Model S-3A and CD-3S, *Sunnyvale, California)* were used to analyze the oxygen and carbon dioxide concentrations of the expired air. At the end of each incremental stage Rating of Perceived Exertion on a scale of 6 to 20 (Borg, 1982) and HR were recorded. The VO_2max test was terminated only if the participant could no longer complete a workload as a result of volitional fatigue, or if the qualified exercise professionals indicated that the participant reached their VO_2max .

2.1.3 Visit Two: Laboratory Simulation Hockey Game Protocol

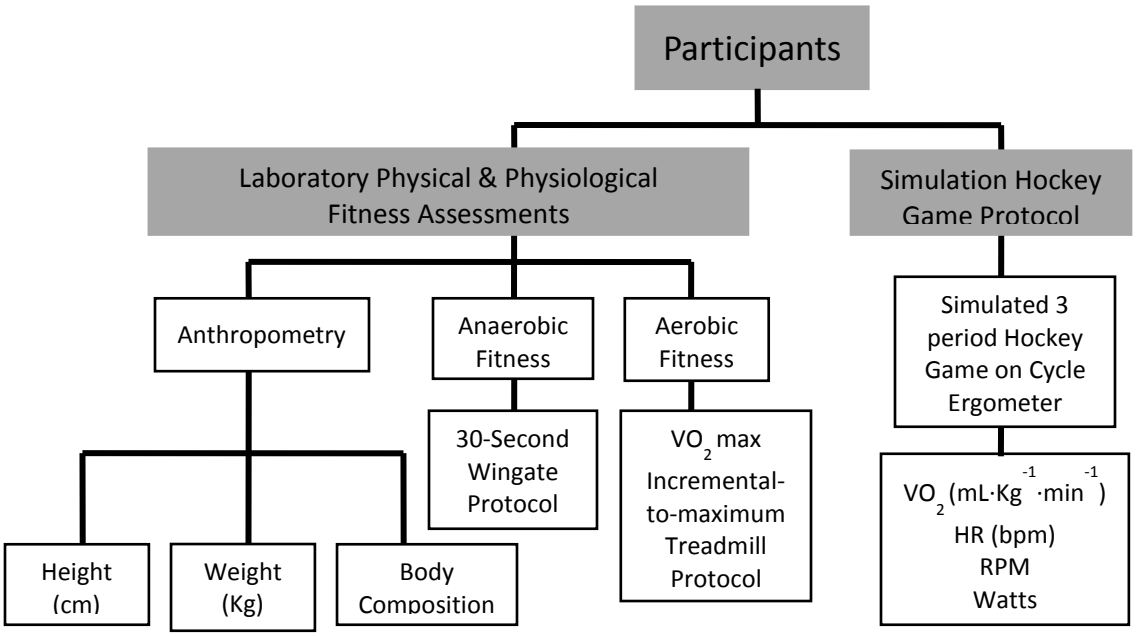
Design. The "play" intervals during the laboratory simulated hockey game were based on the duration and intensity of HR monitored during play intervals throughout regular hockey games. Individuals wore a coded polar HR monitor during a regular on-ice hockey game. The recorded HR values were then examined to identify HR fluctuations throughout the games. Each sustained increase, or dip, in HR was analyzed for duration and intensity, as the HR was assumed to indicate a hockey shift involving skating sprints and stops during play, or a bench period between shifts respectively. A standardized laboratory-exercise protocol (Appendix D) was developed from the on-ice game information.

The simulation hockey game protocol consisted of three 20-minute periods of intermittent high-intensity exercise separated by a 10-minute break period. Participants exercised on a Monark Ergomedic 894E Peak cycle ergometers, the same ergometer was used for the Wingate test, which were hooked up to laptop computers to measure RPMs and power output in Watts. Resistance was set at 5.25% of the participant's body mass, or 70% of their Wingate resistance. The participants were verbally prompted to accelerate and decelerate their pedalling frequency using the cues "go," "whistle" and "bench." The term "go" was used to direct the participant to sprint on the ergometer with the same exertion they would during a hockey shift. "Whistle" signified a stoppage in play and the participants were permitted to sit stationary or pedal lightly with no resistance. Lastly, "bench" was used to indicate a prolonged break between shifts, during which participants rested without pedalling on the cycle ergometer.

Laboratory Game Simulation Procedures. Upon entering the laboratory, participants were instructed to treat the simulated hockey exercise similar to how they would during a regular game. Appropriate measures were taken to ensure BG levels were in the appropriate range before commencing game simulation. Whole BG concentration was measured with the individual's own hand held device. Study participants were permitted one attempt to correct BG levels if they were below threshold by administering 16 grams of carbohydrate (4 tablets of Dex4), or consume a snack of choice with the same carbohydrate content. If participants were above the BG threshold upon entering the laboratory they were instructed to consume water and take insulin if deemed appropriate. A second BG reading was taken 15-30 minutes after their initial check. Participation in the laboratory hockey game simulation proceeded only if the individuals' BG levels were corrected, if the individuals' BG levels were not within the safe range the laboratory protocol was re-scheduled for another day.

Before commencing the game simulation protocol, the participants were weighed and fitted with a Polar heart rate monitor and Fitmate VO_2 max system (Image Monitoring). During the protocol HR, VO_2, RPMs and Watts were continuosly monitored and recorded. At the end of each period, participants were given a 10-minute break where they were permitted to get off the cycle ergometer, walk around and stretch. At this time the individual with T1D checked their BG levels, and if at any time BG levels approached unsafe levels, 4 dextrose tablets were administered orally (16 grams of carbohydrate). The hockey game simulation protocol was followed by a cool-down.

Figure 3. Summary of Laboratory Measures



2.1.4 Data and Statistical Analysis of the VO_2 and HR Response

The raw breath-by-breath VO_2 and HR data measured via the Fitmate VO_2max and Polar Heart Rate systems were time aligned to the start of exercise, with the onset of exercise defined as t = 0. The participants' data for all three periods were then averaged over consecutive 10-second intervals to reduce noise and enhance the underlying characteristic. The resulting VO_2 and HR data were then converted to be expressed as a percentage of VO_2 max (%VO_2) and HRmax (%HR) for each participant. Finally, the nadir (minimum) and peak (maximum) VO_2 and HR percentage values for each shift and bench intervals were also compiled for statistical analysis.

The SPSS statistical software Version 20, was used for all the analysis, with a threshold for statistical significance set at $p \leq 0.05$ a priori. Descriptive statistics (MSD) were calculated to

summarize the participants' anthropometric and physical plus physiological fitness characteristics. Independent samples t-tests were performed to assess any differences between the T1D and their matched controls for the various anthropometric, physical and physiologic variables where appropriate. Data collection errors in the Fitmate VO_2max system resulted in incomplete data sets for 5 participants; therefore 13 participants were included in the final statistical analysis of VO_2 and HR. An initial two-way repeated measures analysis of variance (ANOVA) was conducted using the group means of the raw %VO_2 and %HR data to determine any group differences across the three periods. Prior to the analysis, the individual participants' raw VO_2 and HR data was converted to be expressed as a percentage of VO_2 max and HRmax. Lastly, the mean differences of VO_2 and HR, expressed as the mean nadir (minimum) and mean peak (maximum) values of percent VO_2max and HRmax, were assessed using a two-way repeated measures analysis of variance (ANOVA) to determine if the VO_2 and HR response differed across the three simulated hockey periods, and between the T1D and their matched controls. Sphericity tests were evaluated, and if necessary, the Greenhouse-Geiser method was used to adjust the degrees of freedom. The Bonferroni post-hoc comparison was used to locate specific pairwise differences between the three hockey game simulation periods.

2.2 RESULTS

2.2.1 Study Participants' Characteristics

In total, 12 males and 6 females participated in laboratory physical and physiological fitness assessments and simulated hockey game. Nine of the participants had T1D and each of

the nine had a control (ND) that was matched in age, sex, and physical plus physiological fitness level. All participants played competitive ice hockey; 4 at the A- level, 6 at the AA- level, and 8 at the AAA- level, with the AAA- being the most competitive level. Descriptive statistics of the anthropometric and physical and physiological fitness profiles of the ice hockey players are reported in Table 1. According to an independent samples t-test, there were no significant differences between the T1D and ND groups in any of the anthropometric and physical plus physiological fitness measurements ($p < 0.05$). Participants' age ranged from 13 to 17 years with a mean age of 14.7 ± 1.58. The mean relative VO_2 max was 55.6 ± 5.7 $mL \cdot kg^{-1} \cdot min^{-1}$ (range 43.3-64.1) and the mean HRmax was 196.9 ± 6.6 bpm (range 186-209). Given the small number of female study participants no statistical analyses on sex differences were conducted.

Table 2. Anthropometric and Physical plus Physiological Fitness Profiles of Competitive Youth Ice Hockey Players With and Without T1D (M ± SD).

	T1D (*n = 9*)	**ND (*n = 9*)**	**P value**
Age (*yrs*)	14.7 ± 1.58	14.7 ± 1.58	1.00
Height (*cm*)	169.3 ± 7.7	170.1 ± 8.5	.841
Weight (*kg*)	67.1 ± 8.6	66.2 ± 10.4	.850
WC (*cm*)	77.0 ± 5.3	78.0 ± 6.1	.740
Skinfolds (*mm*)	64.8 ± 22.6	52.2 ± 11.9	.185
VO_2max ($mL \cdot kg^{-1} \cdot min^{-1}$)	54.6 ± 5.3	56.7 ± 6.2	.453
HRmin (*bpm*)	53.8 ± 5.7	52.4 ± 8.4	.699
HRmax (*bpm*)	196.7 ± 8.1	197.2 ± 5.2	.864
Peak Power (*Watts*)	670.1 ± 148.9	716.1 ± 192.1	.579
Peak Power (*Watts/kg*)	9.9 ± 1.4	10.7 ± 1.6	.318
Fatigue (*%*)	46.4 ± 8.9	48.4 ± 9.6	.719

*Significant difference between T1D and ND groups $p < 0.05$

Traditionally, VO_2 kinetics has been analyzed by plotting and modeling the VO_2 response using an exponential equation. In this investigation we chose to use a different approach, because of experimental conditions and technological limitations, as the traditional method was designed for investigating the VO_2 kinetic response during constant-load exercise. The specific exponential equation ($\Delta VO_2(t) = VO_2(b) + A(1 - e^{-t/T})$) could not be applied to the HITT exercise performed in the present study, as the equation assumes that from baseline, VO_2 increases exponentially until a steady-state is achieved. The simple exponential function $y = e^x$ is used to model a relationship in which a constant change in the independent variable (in this case time) gives the same proportional change in the dependent variable (VO_2). The simulation hockey game protocol was not a constant-load situation; within the 7 play shifts, the duration of the bouts of play and active rests continually varied. As well, the play shifts performed were not long enough in duration to achieve a steady-state VO_2. Moreover, it was deemed unlikely that VO_2 increased at the same rate during each play shift in each shift as there was a different amount of work being accomplished with each simulated play shift (e.g. 30s of work vs 10s vs 40s). As such, VO_2 could not be compared across the play shifts. Therefore, we chose to analyse the VO_2 and HR response simply by plotting and representing the data as a percentage of measured VO_2max and HRmax and using the nadir, mean and peak values from the play shifts to identify a possible unique response.

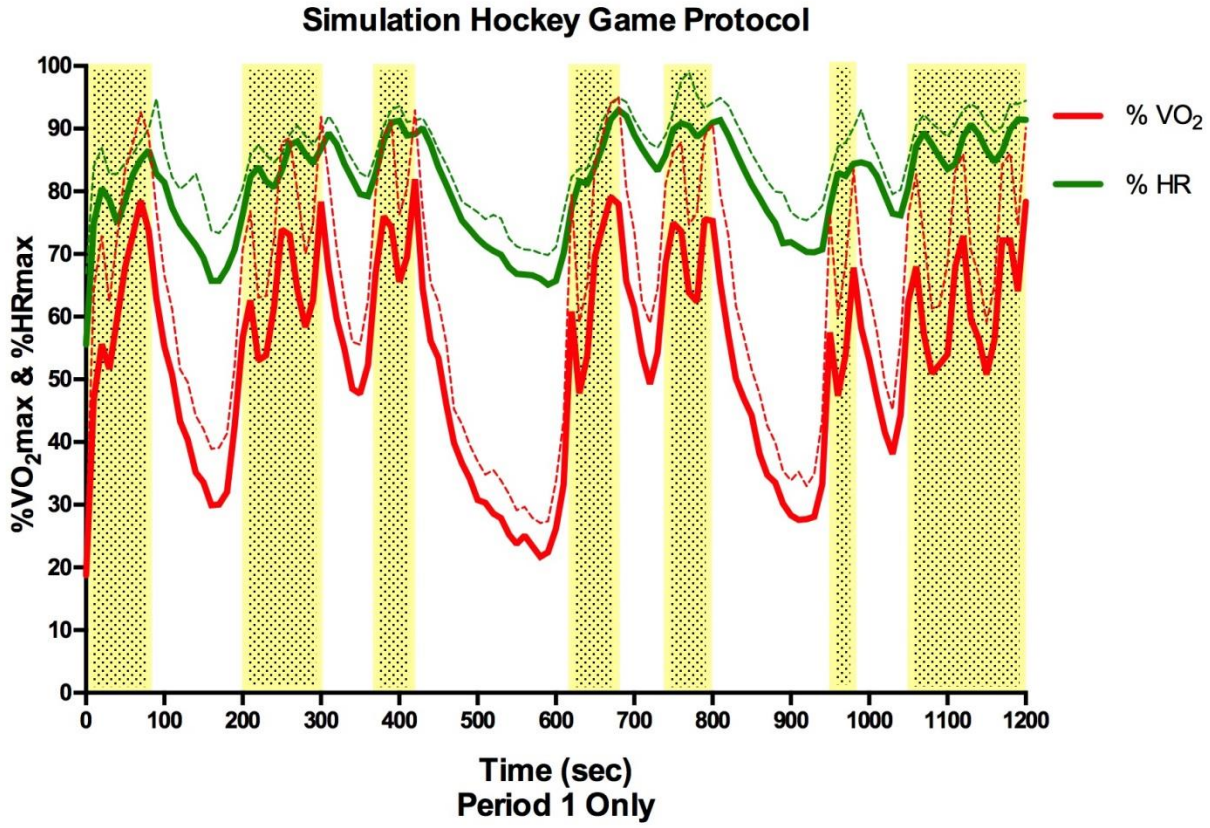
2.2.2 Laboratory Simulation Hockey Game Protocol

The study participants with T1D attended the laboratory sessions along with their paired ND teammate. The pairs completed the simulation hockey game protocol in tandem which consisted of three 20-minute periods, each separated by 10 minute of non-exercise rest. During rest, all participants were allowed to walk around. As well, the participants with T1D were directed to check their BG and consume dextrose tablets if required.

Prior to starting the simulation hockey game protocol, two participants with T1D had BG levels below the 5 mmol/L safety cut-off, and therefore were treated with either 16 grams of dextrose or a self-supplied snack (comparable to what they would consume during an ice hockey game their BG was low). The single attempt to correct BG was successful for both participants and the hockey game simulation protocol proceeded. During the simulation hockey game, one participant with T1D required dextrose supplementation because of low BG levels (<5 mmol/L). VO_2 and HR recordings were completed for 13 individuals (7 T1D, and 6 ND). One participant in the T1D group was unable to complete the first period of the simulation hockey game resulting in incomplete recordings for this individual and their paired ND control. For three other individuals, incomplete data recordings were due to limitations of the equipment, where the Fitmate VO_2 system stopped recording part way through a 20-minute period, or failed to record VO_2 and HR entirely. VO_2 and HR data from 13 completed recordings for period 1 of the simulation hockey game are represented in Figure 4. During the initial shift, it was observed that VO_2 and HR increased rapidly from approximately 20% to 80% and approximately 55% to 90% respectively during the first 80 seconds of period 1. In each of the subsequent shifts

within the period VO_2 ranged from 50% to 80% of VO_2max, and HR ranged from 75% to 90% of HRmax.

Figure 4. 10-Second averaged percentage of VO_2max and HRmax during the first 20-minute period of the simulation hockey game with playing shifts highlighted for all of the study participants (M ± SE).



The shaded areas represent the 7 shifts within the periods which consist of play sprints and active recovery. The white areas represent the 6 bench intervals within the periods that involved passive rest. The red and green dashed lines represent the standard error for VO_2 and HR. Within the first period VO_2 ranged between 20% and 80% of VO_2max and HR ranged between 55% and 90% of HRmax.

2.2.3 Raw Data

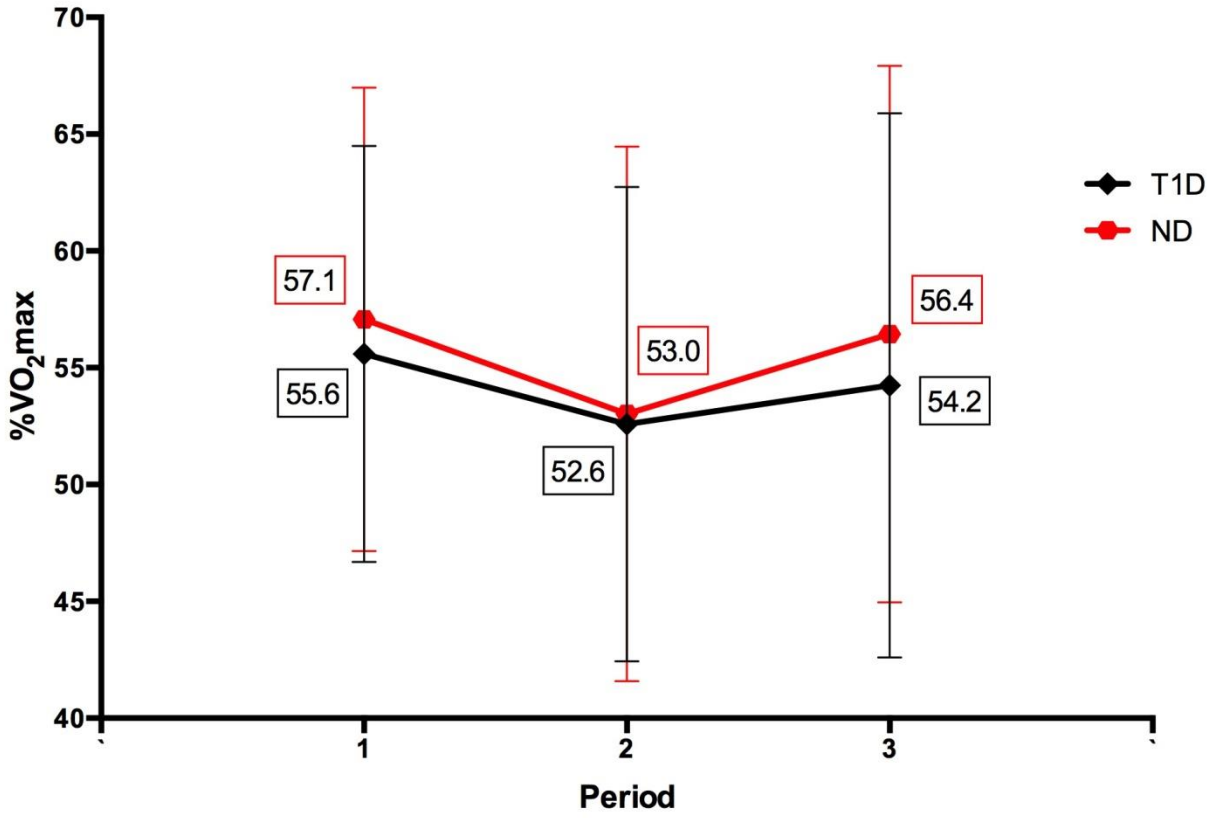
An initial two-way ANOVA with repeated measures on the mean breath-by-breath %VO_2max and %HRmax was conducted. Table 3 contains the mean breath-by-breath %VO_2max and %HRmax between the two groups for all 3 periods of the simulation hockey game. Figure 5 illustrates the comparison of %VO_2max between the individuals with and without T1D. There was no significant difference in the mean %VO_2max between the groups during the simulation hockey game ($F(1.11) = 0.06$, $p > 0.05$). Figure 6 illustrates the % HRmax comparison between the individuals with and without T1D. The analysis of %HRmax indicated that there were no significant differences in the mean %HRmax between the two groups of participants ($F(1,11) = 0.06$, $p > 0.05$).

Table 3. The mean breath-by-breath %VO_2max and %HRmax for all 3 periods of the simulation hockey game for competitive youth ice hockey players with and without T1D (M ± SE).

	% VO_2max		% HRmax	
	T1D (*n=7*)	ND (*n=6*)	T1D (*n=7*)	ND (*n=6*)
Period 1	55.6 ± 3.5	57.1 ± 3.8	82.3 ± 1.0	82.0 ± 1.1
Period 2	52.6 ± 4.1	53.0 ± 4.4	82.5 ± 1.1	82.3 ± 1.2
Period 3	54.2 ± 4.4	56.4 ± 4.7	82.2 ± 1.3	81.5 ± 1.4

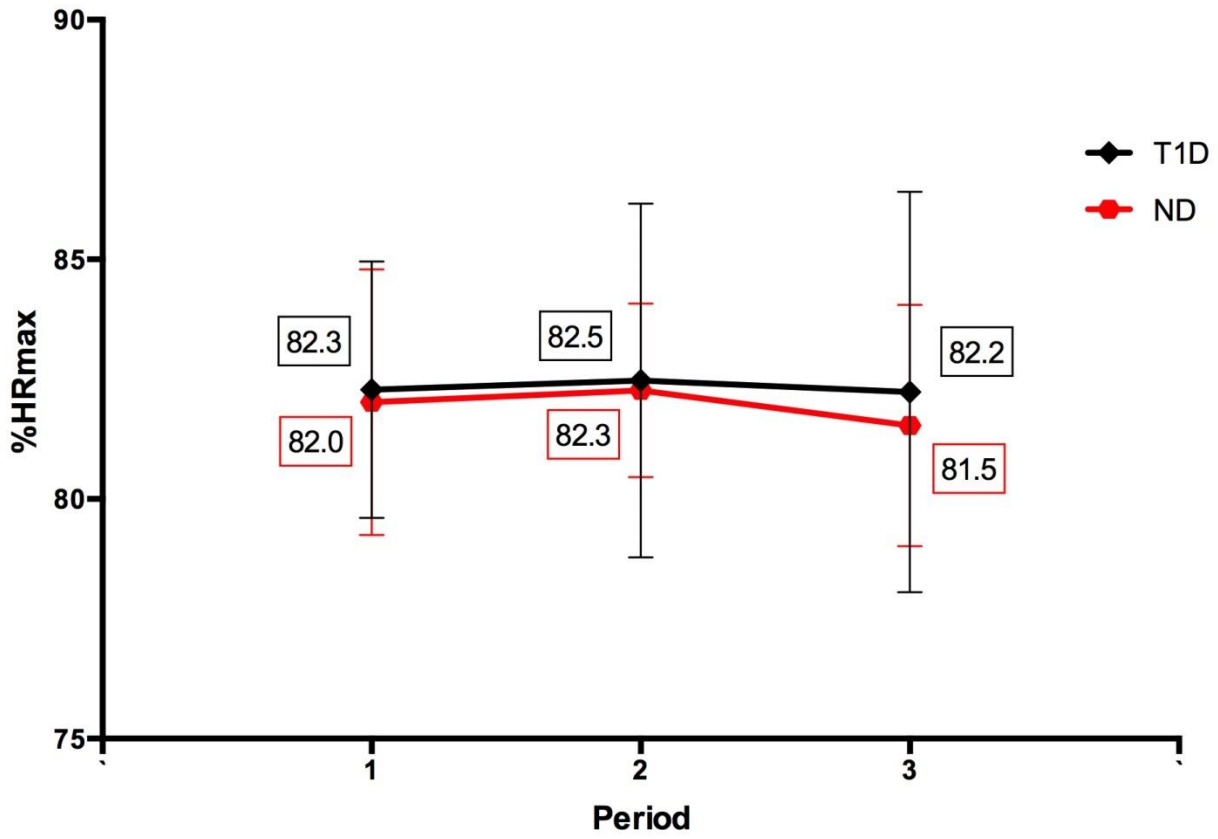
There were no significant differences between groups $p < 0.05$

Figure 5. Comparing the mean %VO_2max during 3 periods of a simulation hockey game between competitive youth ice hockey players with and without T1D (M ± SE).



There are no significant differences ($p > 0.05$) in the mean %VO_2max between the T1D and ND groups. Mean values and standard error bars are included. During the 3 periods all of the participants are working at approximately 55% of their VO_2max.

Figure 6. Comparing the mean %HRmax during periods of a simulation hockey game between competitive youth ice hockey players with and without T1D (M ± SE).



There are no significant differences ($p > 0.05$) in the mean %HRmax between the T1D and ND groups. Mean values and standard error bars are included. During the 3 periods all of the participants are working at approximately 82% of their HRmax.

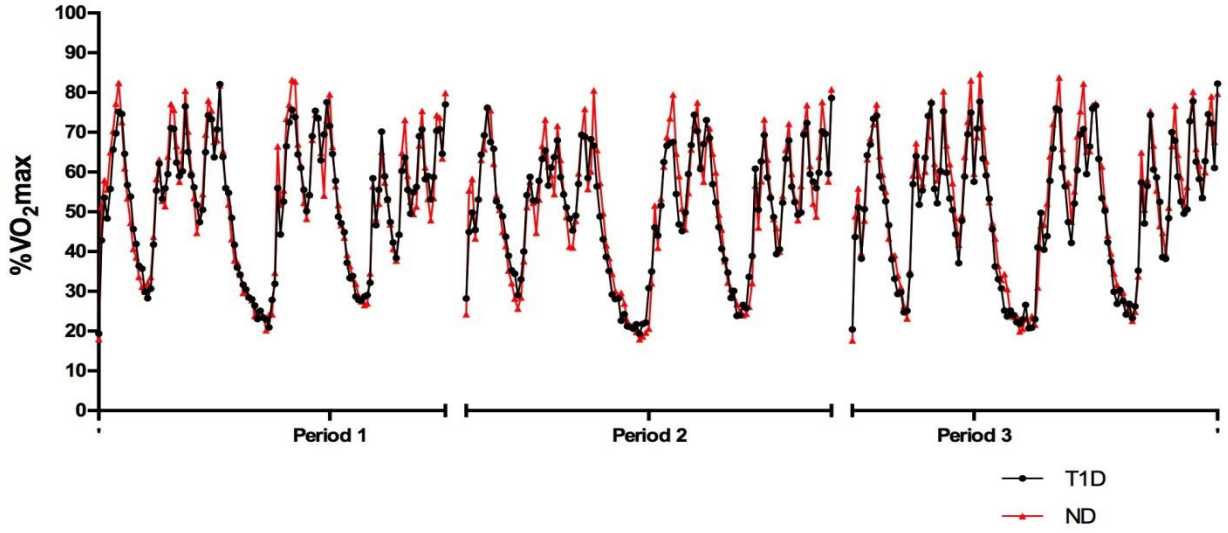
In addition to the between groups analysis, a within group between periods analysis was also conducted for the mean breath-by-breath %VO_2max and %HRmax data. Table 4 summarizes the mean and standard error for %VO_2max and %HRmax between the 3 periods for the groups individually. Figure 7 illustrates the mean %VO_2max displayed in 10-second averaged intervals across the 3 simulation hockey periods. There was no significant difference observed in the mean %VO_2max between the 3 periods for both groups ($F(2,22) = 3.29$, $p > 0.05$). Figure 8 illustrates the mean %HRmax displayed in 10-second averaged intervals across the 3 simulation hockey periods. There was no significant difference observed in the mean %HRmax between the 3 periods for both groups ($F(1.19,13.12) = 0.27$, $p > 0.05$). Both the T1D and ND groups worked at approximately 55% and 82% of their VO_2max and HRmax respectively, throughout the 60-minute simulation hockey game.

Table 4.The mean breath-by-breath %VO_2max and %HRmax between periods of a simulation hockey game for competitive youth ice hockey players with and without T1D (M ± SE).

%VO_2max		Mean ± SE	
T1D(***n=7***)	Period 1	55.6 ± 3.5	
	Period 2	52.6 ± 4.1	
	Period 3	54.2 ± 4.4	
ND (***n=6***)	Period 1	57.1 ± 3.8	
	Period 2	53.0 ± 4.4	
	Period 3	56.4 ± 4.7	
%HRmax			
T1D(***n=7***)	Period 1	82.3 ± 1.0	
	Period 2	82.5 ± 1.1	
	Period 3	82.2 ± 1.3	
ND (***n=6***)	Period 1	82.0 ± 1.1	
	Period 2	82.3 ± 1.2	
	Period 3	81.5 ± 1.4	

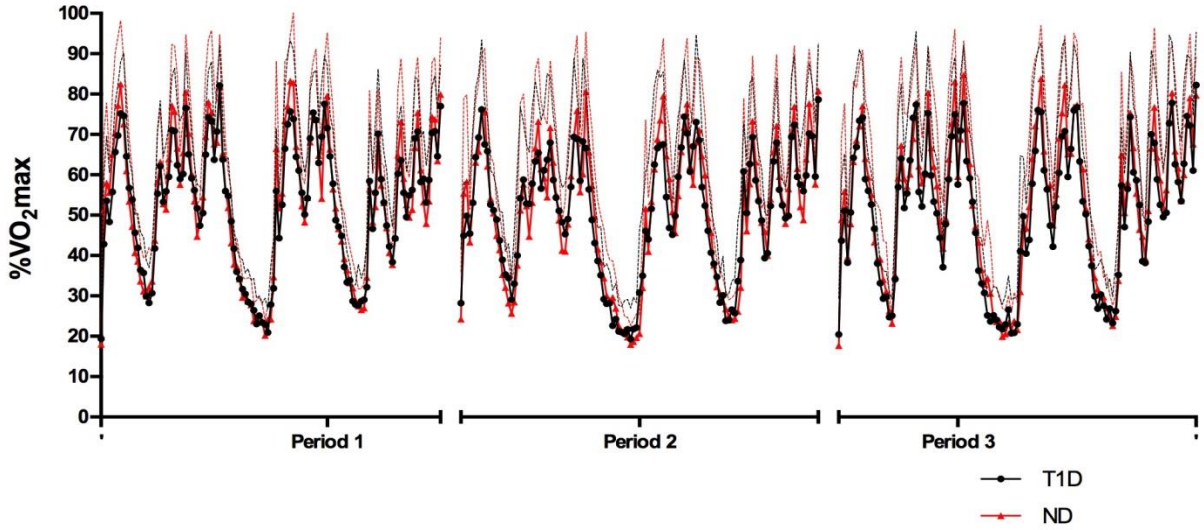
There were no significant differences between periods $p > 0.05$

Figure 7a. 10-Second average oxygen consumption expressed relative to VO_2max across all 3 periods of a 60-minute simulation hockey game for competitive youth ice hockey players with and without T1D. Plotted without the standard error.



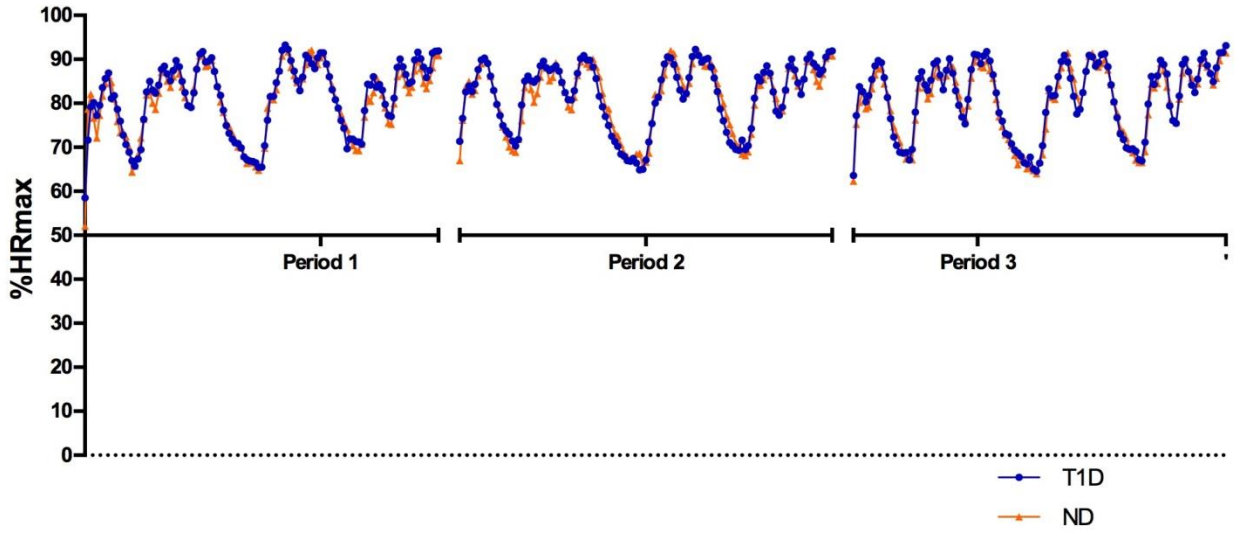
There are no differences in %VO_2max between periods 1, 2 and 3 for the T1D and ND groups ($p > 0.05$). The %VO_2max is fairly consistent between periods 1, 2 and, and ranges between 20% and 90% for both groups. It is evident that the individuals with and without T1D are able to perform a longer duration of HITT without fatiguing.

Figure 7b. 10-Second average oxygen consumption expressed relative to VO_2max across all 3 periods of a 60-minute simulation hockey game for competitive youth ice hockey players with and without T1D. Plotted with the standard error (M ± SE).



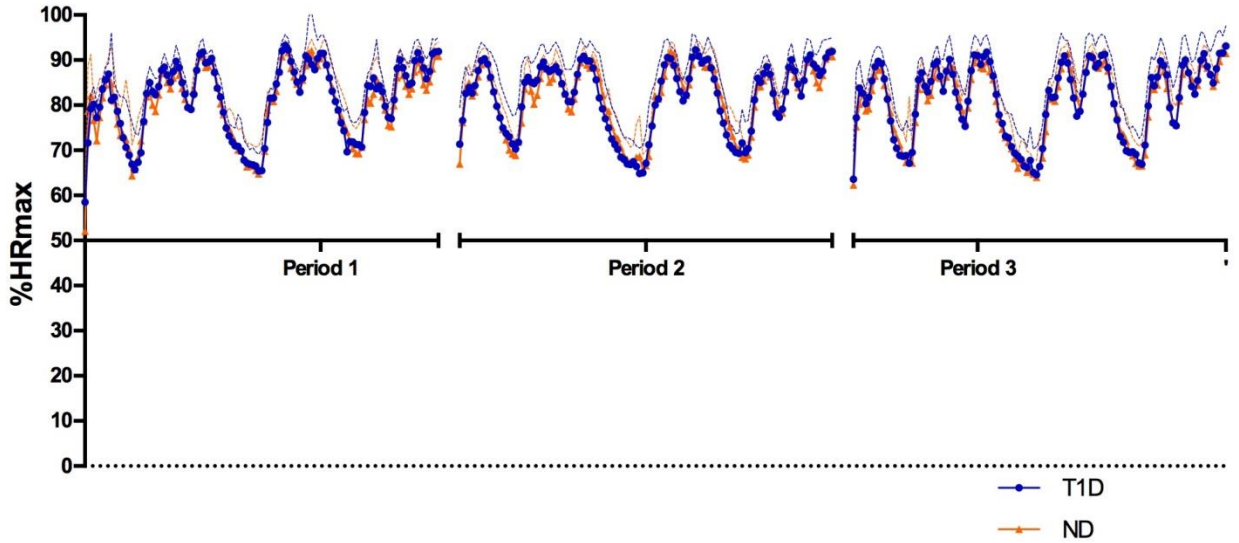
The standard error is represented by the dashed black and red lines for the T1D and ND groups respectively. There are no differences in %VO_2max between periods 1, 2 and 3 for the T1D and ND groups ($p > 0.05$). The %VO_2max is fairly consistent between periods 1, 2 and, and ranges between 20% and 90% for both groups. It is evident that the individuals with and without T1D are able to perform a longer duration of HITT without fatiguing.

Figure 8a. 10-Second average heart rate expressed relative to HRmax across all 3 periods of a 60-minute simulation hockey game for competitive youth ice hockey players with and without T1D. Plotted without the standard error.



There are no differences in %HRmax between periods 1, 2 and 3 for the T1D and ND groups. It is illustrated that the %HRmax remains consistent between periods 1, 2 and 3. %HRmax ranges between 60% and 90% for both groups of participants.

Figure 8b. 10-Second average heart rate expressed relative to HRmax across all 3 periods of a 60-minute simulation hockey game for competitive youth ice hockey players with and without T1D. Plotted with the error bars (M ± SE).



The standard error is represented by the dashed blue and orange lines for the T1D and ND groups respectively. There are no differences in %HRmax between periods 1, 2 and 3 for the T1D and ND groups. It is illustrated that the %HRmax remains consistent between periods 1, 2 and 3. %HRmax ranges between 60% and 90% for both groups of participants.

2.2.4 Averaged Nadir and Peak %VO_2max and %HRmax During the Play Shift Intervals

Further analyses were conducted on the means of the nadir and peak %VO_2max and %HRmax values for each play shift and bench intervals using the 10-second averaged data. A two-way ANOVA with repeated measures was used to determine if significant mean differences in the resting (resting VO_2 expressed as a percentage of VO_2max), average nadir and the average peak values of %VO_2max and %HRmax were present between the T1D and ND groups at rest and during play shift and bench intervals. In addition, the two-way ANOVA with repeated measures was used to reveal any significant mean differences in the rest, average nadir and the average peak values of %VO_2max and %HRmax across the 3 periods for the T1D and ND groups individually.

Table 5 summarizes the means and standard errors for the average nadir and the average peak %VO_2max from the pairwise comparison between the T1D and ND groups. There are no significant differences in either of the average nadir and the average peak %VO_2max between the T1D and ND individuals at rest, or during any of the 7 play shift and 6 bench intervals during the 3 periods (Figure 9). The individuals with T1D are able to achieve similar high range %VO_2max values, 70-90% of VO_2max, compared with the ND during the 7 play shift intervals across all 3 periods.

Table 6 summarizes the means and standard errors for the average nadir and the average peak %HRmax from the pairwise comparison between the T1D and ND groups. The analyses indicated that there were no significant differences in both the average nadir and the average peak %HRmax between the T1D and ND groups at rest, or during any of the 7 play shift and 6 bench intervals within the 3 periods (Figure 10). Across the 3 periods similar %HRmax

high values, 80-95% of HRmax, between the T1D and ND individuals were reached during the 7 play shift intervals.

Table 5. Comparison of the average oxygen consumption expressed relative to VO_2max at the beginning and end of each play shift and bench interval during a 60-minute simulation hockey game between competitive youth ice hockey players with and without T1D (M ± SE).

		Period 1		Period 2		Period 3	
		T1D (*n=7*)	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**
Rest		0.19 ± 0.027	0.18 ± 0.181	0.30 ± 0.049	0.24 ± 0.243	0.20 ± 0.028	0.18 ± 0.177
Shift 1	**High**	0.80 ± 0.052	0.85 ± 0.057	0.76 ± 0.055	0.78 ± 0.059	0.78 ± 0.055	0.77 ± 0.059
	Low	0.41 ± 0.050	0.47 ± 0.054	0.40 ± 0.037	0.42 ± 0.040	0.37 ± 0.038	0.38 ± 0.041
Bench 1	**High**	0.66 ± 0.052	0.62 ± 0.056	0.67 ± 0.038	0.62 ± 0.042	0.60 ± 0.048	0.65 ± 0.052
	Low	0.26 ± 0.029	0.28 ± 0.031	0.27 ± 0.030	0.25 ± 0.033	0.23 ± 0.015	0.23 ± 0.016
Shift 2	**High**	0.77 ± 0.050	0.83 ± 0.054	0.74 ± 0.059	0.76 ± 0.064	0.80 ± 0.050	0.83 ± 0.054
	Low	0.48 ± 0.029	0.48 ± 0.031	0.48 ± 0.030	0.40 ± 0.033	0.45 ± 0.015	0.51 ± 0.016
Bench 2	**High**	0.66 ± 0.053	0.72 ± 0.057	0.62 ± 0.053	0.64 ± 0.057	0.63 ± 0.061	0.68 ± 0.065
	Low	0.45 ± 0.027	0.40 ± 0.029	0.38 ± 0.035	0.38 ± 0.038	0.37 ± 0.037	0.41 ± 0.040
Shift 3	**High**	0.83 ± 0.038	0.87 ± 0.041	0.78 ± 0.062	0.81 ± 0.067	0.82 ± 0.041	0.86 ± 0.044
	Low	0.60 ± 0.037	0.61 ± 0.040	0.47 ± 0.040	0.55 ± 0.043	0.54 ± 0.040	0.59 ± 0.043
Bench 3	**High**	0.65 ± 0.049	0.66 ± 0.053	0.56 ± 0.049	0.66 ± 0.053	0.64 ± 0.056	0.72 ± 0.061
	Low	0.19 ± 0.017	0.18 ± 0.019	0.17 ± 0.021	0.17 ± 0.023	0.18 ± 0.019	0.17 ± 0.021
Shift 4	**High**	0.77 ± 0.052	0.89 ± 0.056	0.74 ± 0.060	0.80 ± 0.065	0.79 ± 0.056	0.84 ± 0.061
	Low	0.44 ± 0.038	0.49 ± 0.041	0.36 ± 0.052	0.40 ± 0.056	0.39 ± 0.044	0.40 ± 0.048
Bench 4	**High**	0.68 ± 0.049	0.69 ± 0.052	0.64 ± 0.050	0.66 ± 0.053	0.65 ± 0.059	0.69 ± 0.063
	Low	0.48 ± 0.039	0.46 ± 0.043	0.41 ± 0.021	0.45 ± 0.023	0.38 ± 0.026	0.46 ± 0.028
Shift 5	**High**	0.81 ± 0.038	0.84 ± 0.041	0.83 ± 0.051	0.79 ± 0.055	0.81 ± 0.040	0.87 ± 0.043
	Low	0.63 ± 0.038	0.54 ± 0.041	0.55 ± 0.048	0.53 ± 0.052	0.57 ± 0.061	0.60 ± 0.066
Bench 5	**High**	0.64 ± 0.055	0.66 ± 0.060	0.59 ± 0.060	0.65 ± 0.065	0.64 ± 0.054	0.66 ± 0.059
	Low	0.23 ± 0.022	0.25 ± 0.024	0.20 ± 0.019	0.22 ± 0.020	0.21 ± 0.022	0.21 ± 0.024
Shift 6	**High**	0.71 ± 0.065	0.67 ± 0.070	0.76 ± 0.062	0.77 ± 0.067	0.74 ± 0.057	0.76 ± 0.061
	Low	0.37 ± 0.048	0.46 ± 0.052	0.50 ± 0.042	0.44 ± 0.045	0.45 ± 0.048	0.49 ± 0.052
Bench 6	**High**	0.61 ± 0.041	0.58 ± 0.044	0.62 ± 0.043	0.65 ± 0.047	0.65 ± 0.064	0.67 ± 0.069
	Low	0.35 ± 0.032	0.36 ± 0.034	0.37 ± 0.034	0.39 ± 0.037	0.35 ± 0.036	0.38 ± 0.038
Shift 7	**High**	0.80 ± 0.039	0.84 ± 0.043	0.81 ± 0.045	0.86 ± 0.049	0.85 ± 0.049	0.87 ± 0.053
	Low	0.47 ± 0.032	0.43 ± 0.035	0.46 ± 0.034	0.46 ± 0.037	0.45 ± 0.044	0.47 ± 0.048

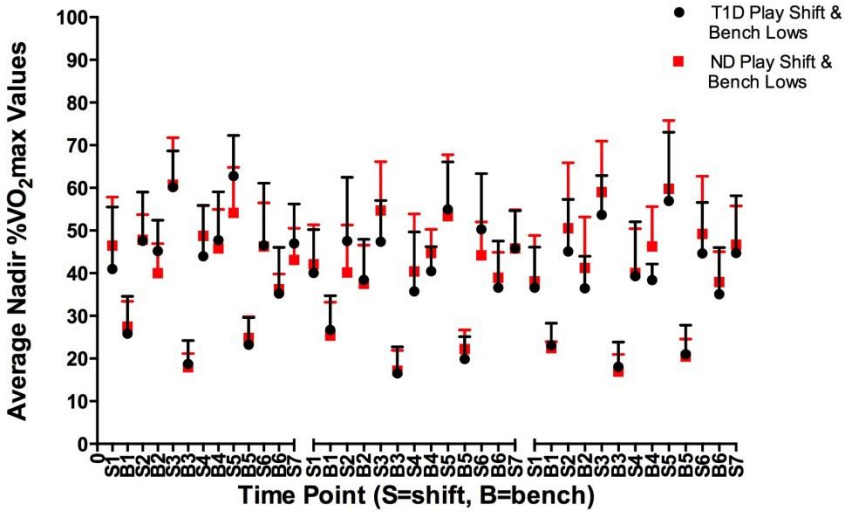
There were no significant differences between groups $p > 0.05$

Table 6. Comparison of the average heart rate expressed relative to HRmax at the beginning and end of each play shift and bench interval during a 60-minute simulation hockey game between competitive youth ice hockey players with and without T1D (M ± SE).

		Period 1		Period 2		Period 3	
		T1D (*n=7*)	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**
REST		0.59 ± 0.043	0.52 ± 0.521	0.72 ± 0.026	0.67 ± 0.670	0.64 ± 0.020	0.62 ± 0.624
Shift 1	**High**	0.87 ± 0.018	0.87 ± 0.020	0.91 ± 0.012	0.90 ± 0.013	0.91 ± 0.016	0.89 ± 0.018
	Low	0.71 ± 0.030	0.69 ± 0.033	0.76 ± 0.024	0.76 ± 0.026	0.75 ± 0.020	0.73 ± 0.021
Bench 1	**High**	0.86 ± 0.023	0.85 ± 0.025	0.89 ± 0.011	0.90 ± 0.012	0.89 ± 0.012	0.89 ± 0.012
	Low	0.62 ± 0.034	0.63 ± 0.036	0.69 ± 0.023	0.68 ± 0.025	0.64 ± 0.020	0.65 ± 0.022
Shift 2	**High**	0.86 ± 0.025	0.88 ± 0.027	0.91 ± 0.012	0.89 ± 0.013	0.90 ± 0.010	0.89 ± 0.011
	Low	0.72 ± 0.035	0.76 ± 0.038	0.78 ± 0.024	0.76 ± 0.025	0.78 ± 0.018	0.76 ± 0.019
Bench 2	**High**	0.90 ± 0.011	0.89 ± 0.011	0.90 ± 0.014	0.89 ± 0.015	0.90 ± 0.015	0.89 ± 0.016
	Low	0.78 ± 0.011	0.79 ± 0.012	0.78 ± 0.020	0.78 ± 0.022	0.75 ± 0.025	0.76 ± 0.027
Shift 3	**High**	0.92 ± 0.008	0.91 ± 0.009	0.92 ± 0.011	0.91 ± 0.012	0.92 ± 0.012	0.91 ± 0.013
	Low	0.82 ± 0.011	0.83 ± 0.012	0.82 ± 0.018	0.81 ± 0.020	0.81 ± 0.019	0.79 ± 0.021
Bench 3	**High**	0.90 ± 0.006	0.90 ± 0.007	0.88 ± 0.015	0.90 ± 0.016	0.92 ± 0.011	0.90 ± 0.012
	Low	0.63 ± 0.018	0.63 ± 0.020	0.63 ± 0.018	0.65 ± 0.020	0.64 ± 0.018	0.62 ± 0.020
Shift 4	**High**	0.93 ± 0.032	0.88 ± 0.034	0.92 ± 0.011	0.92 ± 0.011	0.91 ± 0.013	0.91 ± 0.014
	Low	0.76 ± 0.038	0.71 ± 0.041	0.75 ± 0.023	0.75 ± 0.025	0.77 ± 0.016	0.74 ± 0.018
Bench 4	**High**	0.92 ± 0.009	0.92 ± 0.009	0.89 ± 0.012	0.91 ± 0.013	0.90 ± 0.015	0.92 ± 0.016
	Low	0.83 ± 0.014	0.84 ± 0.015	0.79 ± 0.016	0.82 ± 0.018	0.76 ± 0.019	0.79 ± 0.021
Shift 5	**High**	0.93 ± 0.011	0.93 ± 0.012	0.93 ± 0.011	0.92 ± 0.012	0.92 ± 0.014	0.92 ± 0.015
	Low	0.83 ± 0.027	0.85 ± 0.029	0.86 ± 0.014	0.84 ± 0.015	0.82 ± 0.020	0.82 ± 0.022
Bench 5	**High**	0.92 ± 0.015	0.92 ± 0.016	0.88 ± 0.017	0.90 ± 0.018	0.91 ± 0.013	0.90 ± 0.014
	Low	0.66 ± 0.031	0.68 ± 0.033	0.67 ± 0.016	0.67 ± 0.018	0.65 ± 0.020	0.66 ± 0.021
Shift 6	**High**	0.87 ± 0.019	0.83 ± 0.020	0.88 ± 0.011	0.87 ± 0.011	0.87 ± 0.015	0.86 ± 0.017
	Low	0.78 ± 0.019	0.77 ± 0.021	0.80 ± 0.020	0.76 ± 0.022	0.80 ± 0.019	0.78 ± 0.021
Bench 6	**High**	0.87 ± 0.016	0.86 ± 0.017	0.89 ± 0.010	0.88 ± 0.010	0.90 ± 0.016	0.89 ± 0.018
	Low	0.75 ± 0.022	0.75 ± 0.024	0.76 ± 0.016	0.77 ± 0.018	0.74 ± 0.029	0.75 ± 0.031
Shift 7	**High**	0.93 ± 0.012	0.91 ± 0.012	0.91 ± 0.018	0.92 ± 0.019	0.93 ± 0.013	0.92 ± 0.014
	Low	0.80 ± 0.019	0.79 ± 0.020	0.82 ± 0.015	0.83 ± 0.016	0.81 ± 0.024	0.81 ± 0.026

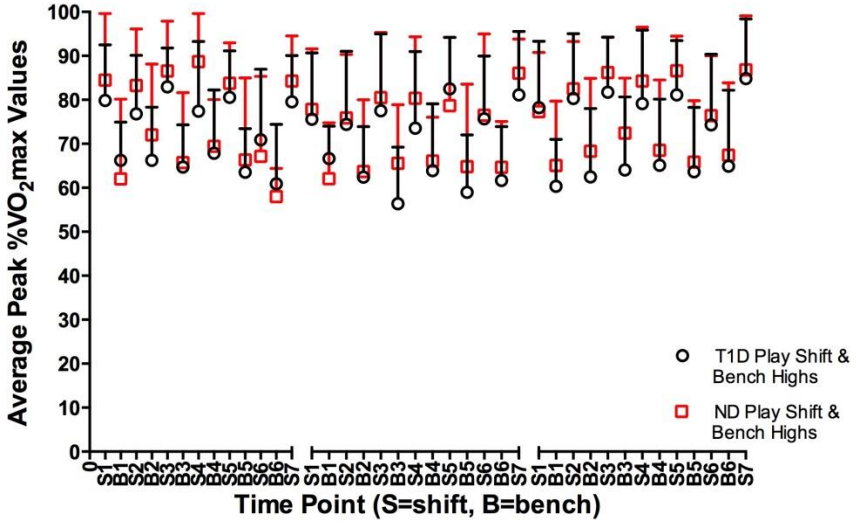
There were no significant differences between groups $p > 0.05$

Figure 9a. Comparison of the nadir oxygen consumption rate expressed relative to VO_2max, at the beginning and end of each play shift and bench interval during a 60-minute simulation hockey game between individuals with and without T1D (M ± SE).



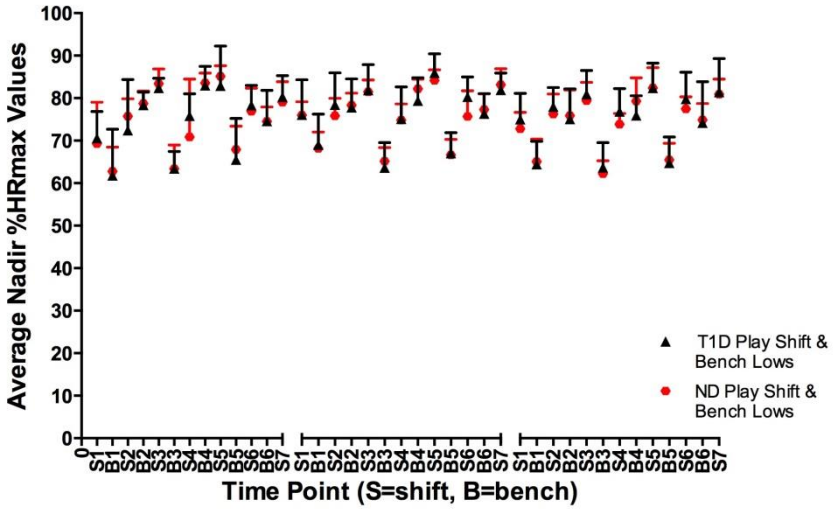
The filled circles and squares represent the nadir %VO_2max for the T1D and ND groups respectively. There are no significant differences in %VO_2max between the T1D and ND group observed in the 7 play shift and 6 bench intervals during any of the 3 periods ($p > 0.05$). The nadir %VO_2max is similar between individuals with and without T1D. Nadirs range from 30-60% of VO_2max for the individuals with and without T1D during the 7 play shifts for the 3 periods.

Figure 9b. Comparison of the peak oxygen consumption rate expressed relative to VO_2max, at the beginning and end of each play shift and bench interval during a 60-minute simulation hockey game between individuals with and without T1D (M ± SE).



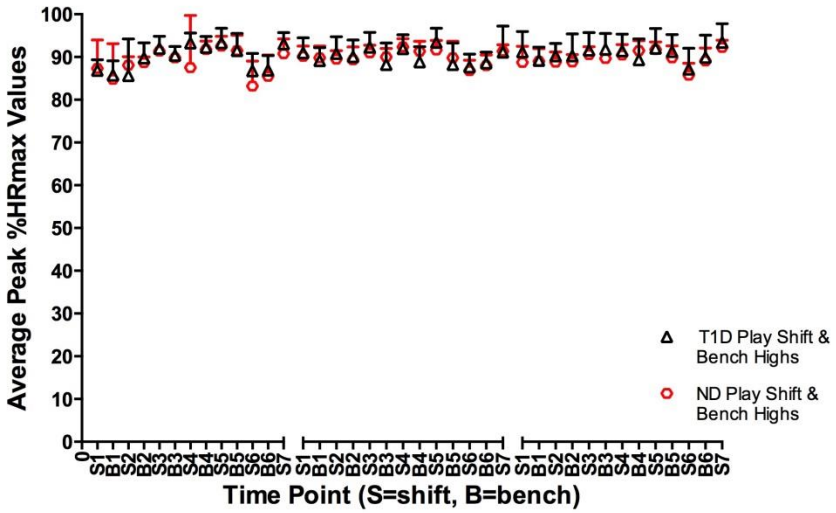
The open circles and squares represent the peak %VO2max for the T1D and ND groups respectively. There are no significant differences in %VO2max between the T1D and ND group observed in the 7 play shift and 6 bench intervals during any of the 3 periods ($p > 0.05$). The peak %VO2max is similar between individuals with and without T1D. Peaks range from 70-90% of VO2max for the individuals with and without T1D during the 7 play shifts for the 3 periods.

Figure 10a. Comparison of the nadir heart rate expressed relative to HRmax, at the beginning and end of each play shift and bench interval during a 60-minute simulation hockey game between individuals with and without T1D (M ± SE).



The filled triangles and circles represent the nadir %HRmax for the T1D and ND groups respectively. There are no significant differences in %HRmax between the T1D and ND group observed in the 7 play shift and 6 bench intervals during any of the 3 periods ($p > 0.05$). The nadir %HRmax is similar between individuals with and without T1D. Nadirs range from 70-85% of HRmax for the individuals with and without T1D during the 7 play shifts for the 3 periods.

Figure 10b. Comparison of the peak heart rate expressed relative to HRmax, at the beginning and end of each play shift and bench interval during a 60-minute simulation hockey game between individuals with and without T1D (M ± SE).



The open triangles and circles represent the peak %HRmax for the T1D and ND groups respectively. There are no significant differences in %HRmax between the T1D and ND group observed in the 7 play shift and 6 bench intervals during any of the 3 periods ($p > 0.05$). The peak %HRmax is similar between individuals with and without T1D. Peaks range from 80-95% of HRmax for the individuals with and without T1D during the 7 play shifts for the 3 periods.

Looking at the T1D and ND groups separately, there were some significant differences in the average nadir and the average peak %VO_2max and %HRmax values between the three periods for both groups. The Bonferroni post hoc analysis revealed that there were no significant differences between the periods in the mean resting %VO_2max (resting VO_2 expressed as a percentage of VO_2max) for the T1D and ND groups, however the mean resting %HRmax (resting HR expressed as a percentage of HRmax) was shown to be significantly different in both of the groups (Table 7). For the T1D group the mean resting expressed as a %HRmax, was significantly lower in period 1 compared with period 2 ($p < 0.01$), was significantly higher in period 2 compared to period 3 ($p < 0.01$), but no differences were observed between periods 1 and 3 ($p = 0.756$). In the ND group the mean resting %HRmax during period 1 was significantly lower from period 2 ($p < 0.01$), and no differences were revealed between periods 2 and 3 ($p = 0.137$), as well as periods 1 and 3 ($p = 0.135$).

Table 7. Mean resting oxygen consumption expressed relative to VO_2max and mean resting heart expressed relative to HRmax between periods for competitive youth ice hockey players with and without T1D (M ± SE).

Resting %VO_2max		Mean ± SE	
T1D(***n=7***)	Period 1	19.4 ± 2.698	
	Period 2	29.9 ± 4.883	
	Period 3	20.4 ± 2.795	
ND (***n=6***)	Period 1	18.1 ± 2.915	
	Period 2	24.3 ± 5.274	
	Period 3	17.7 ± 3.019	
Resting %HRmax			
T1D(***n=7***)	Period 1	58.5 ± 4.307 +	
	Period 2	71.7 ± 2.574 +‡	
	Period 3	63.6 ± 2.020 ‡	
ND (***n=6***)	Period 1	52.1 ± 4.652 +	
	Period 2	67.0 ± 2.780 +	
	Period 3	62.4 ± 2.181	

+ $p < 0.01$Significant difference between periods 1 and 2

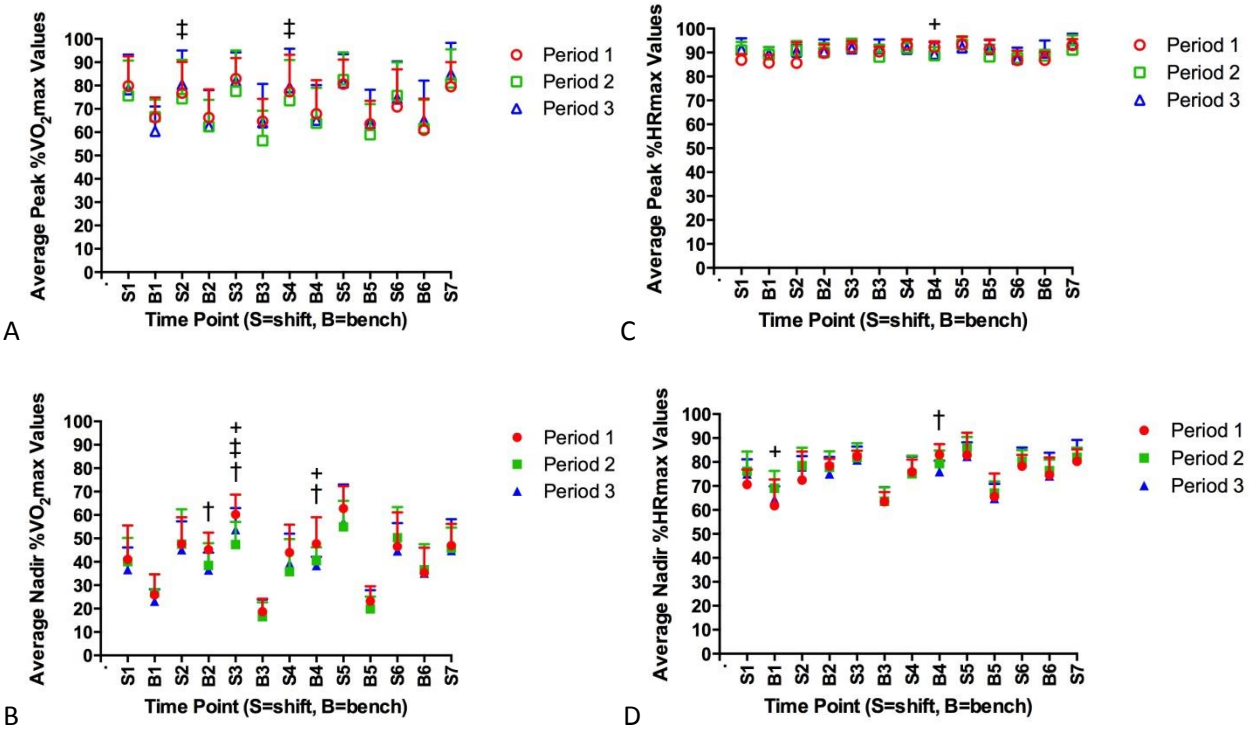
‡ $p < 0.01$ Significant difference between periods 2 and 3

† $p < 0.01$ Significant difference between periods 1 and 3

Figures 11 and 12 depict the differences in the average nadir and average peak %VO_2max values during each play shift and bench intervals for the T1D and ND groups respectively between the three periods. During the simulation hockey game protocol there were significant differences in average nadir %VO_2max values between the periods for the T1D group during the 3rd play shift and bench intervals 2 and 4 respectively ($p < 0.05$). For this same group, significant differences in the average peak %VO_2max values were noted between periods in the 2nd and 4th play shifts, plus the 3rd bench interval ($p < 0.05$). In the ND group significant differences in the average peak %VO_2max values were noted during the 2nd play shift ($p < 0.05$). The differences observed in the average nadir and the average peak %VO_2max values for both the T1D and ND groups are likely due to the participants' self-selected pacing throughout the play shifts and active rest intervals throughout the simulation hockey game.

Specific group differences in the average nadir and the average peak %HRmax values for each play shift and bench interval between the 3 periods are illustrated in Figures 11 and 12. The analysis revealed that the average nadir %HRmax values were significantly different between the periods in the T1D group during the 1st and 4th bench intervals, and the average peak %HRmax values were only different between periods during the 4th bench interval ($p < 0.05$). In the ND group, the average nadir %HRmax values were shown to be significantly different during the 3rd bench interval, however there were no significant differences in the average peak %HRmax values between any of the periods in this group ($p < 0.05$). Similar to the %VO_2max, the differences observed in the average nadir and the average peak %HRmax values for both the T1D and ND groups are likely due to individuals' self-selected pacing during the play shifts and active rest intervals throughout the simulation hockey game.

Figure 11. T1D group differences in the average nadir and peak oxygen consumption rate and the average nadir and peak heart rate, expressed relative to VO_2max and HRmax respectively, between periods in a 60-minute simulation hockey game (M ± SE).



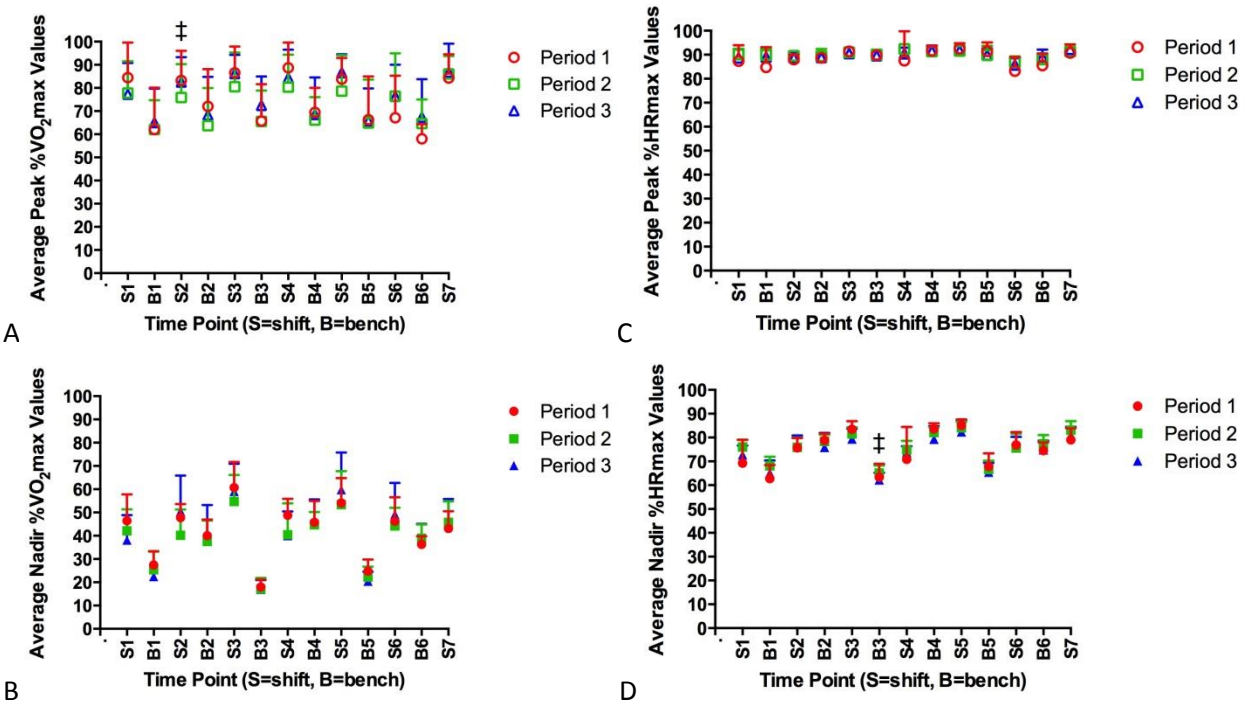
+ $p < 0.05$ Significant difference between periods 1 and 2

‡ $p < 0.05$ Significant difference between periods 2 and 3

† $p < 0.05$ Significant difference between periods 1 and 3

Average nadir and peak %VO_2max and % HRmax values for all participants with T1D (n=7) for a 60 minute hockey game simulation protocol between periods. Some significant differences were shown in some of the play shift and bench intervals. Panel A. Shows the differences in average peak %VO_2 values for all play shift and bench intervals between periods. Panel B. Shows the differences in average nadir %VO_2max values for all play shift and bench intervals between periods. Panel C. Shows the differences in average peak %HRmax values for all play shift and bench intervals between periods. Panel D. Shows the differences in average nadir %HRmax values for all play shift and bench intervals between periods. The differences observed in the average nadir and peak %VO_2max and %HRmax values for the participants with T1D are most likely due to the individuals' self-selected pacing during the play shifts and active intervals rest during each period.

Figure 12. ND group differences in the average nadir and peak oxygen consumption rate and the average nadir and peak heart rate, expressed relative to VO_2max and HRmax respectively, between periods in a 60-minute simulation hockey game (M ± SE).



+ $p < 0.05$ Significant difference between periods 1 and 2

‡ $p < 0.05$ Significant difference between periods 2 and 3

† $p < 0.05$ Significant difference between periods 1 and 3

Average nadir and peak %VO2max and %HRmax values for all ND participants (n=6) for a 60-minute hockey game simulation protocol between periods. Some significant differences were shown in some of the play shift and bench intervals. Panel A. Shows the differences in average peak %VO2max values for all play shift and bench intervals between periods. Panel B. Shows the differences in average nadir %VO2max values for all play shift and bench intervals between periods. Panel C. Shows the differences in average peak %HRmax values for all play shift and bench intervals between periods. Panel D. Shows the differences in average nadir %HRmax values for all play shift and bench intervals between periods. The differences observed in the average nadir and peak %VO2max and %HRmax values for the ND are most likely due to the individuals' self-selected pacing during the play shifts and active rest intervals during each period.

2.3 DISCUSSION

The primary purpose of this study was to examine and compare the VO_2 and HR responses in a simulation hockey game protocol between a population of young, competitive athletes with and without T1D. The study outcomes revealed that highly active youth with uncomplicated T1D whose glycaemia is well controlled have similar physical plus physiological fitness levels and exercise tolerances to their matched ND controls in response to HITT. Remarkably, there were no significant differences in the VO_2max, or in the average nadir, mean and average peak %VO_2max and %HRmax responses between the T1D and ND groups during the simulation hockey game. Some significant differences were noted in the average nadir and average peak %VO_2max and %HRmax responses between the three periods for both the T1D and ND groups individually. These observed differences during the simulation hockey game were not consistent between the periods and were observed in both groups. Therefore, we can postulate that the random differences observed during the simulation hockey game (HITT) were not due to diabetes related influence on the response, but more likely due to self-selected pacing differences between the participants during the play shift intervals.

The results of this study build on those previously published supporting no differences in the maximal aerobic capacity between T1D and ND populations. Mosher et al. 1998, reported that physically inactive adolescents with T1D (17.2 ± 1.2yrs) have similar VO_2max values when compared with physically inactive ND adolescents (19.4 ± 1.3yrs). The investigators measured VO_2 before and after implementing a 12-week exercise training program, and found that both groups improved their cardiovascular endurance with no differences in VO_2max between individuals with and without T1D, pre and post training (T1D: Pre- 40.5 ± 5.8, Post- 44.8 ± 5.2

mL·kg^{-1}·min^{-1} compared with ND: Pre- 41.6 ± 6.7, Post- 46.6 ± 7.2 mL·kg^{-1}·min^{-1}). Mosher et al. (1998) found that adolescents with T1D respond similarly to an aerobic exercise training program in comparison with ND, which may suggest that the VO_2 kinetics would respond to training in a similar way. Unfortunately the investigators did not measure the VO_2 kinetics and HR responses so the influence of training on the rate of change for VO_2 was not determined.

Veves et al. (1997) investigated groups of participants with complicated and uncomplicated T1D. Five groups of participants aged 30-40 years were included in the study. Three exercise groups: a control group (CE), a group with T1D (DE), and a neuropathy group with T1D (NDE), who all performed greater than 10 hrs/wk of endurance exercise. Two sedentary groups one control group without T1D(CS) and one group with T1D (DS). The investigators found that the participants with T1D had similar aerobic capacities to ND individuals with similar exercise histories (CE- 56.8 ± 9.1 vs. DE- 54.0 ± 8.1 mL·kg^{-1}·min^{-1}, CS- 38.7 ± 6.0 vs. DS- 36.7 ± 9.6 mL·kg^{-1}·min^{-1}). The NDE group had a significantly lower VO_2max (42.0 ± 11.6 mL·kg^{-1}·min^{-1}) in comparison with the CE and DE groups. HRmax was also measured and there were no differences between CE- 184 ± 12 vs. DE- 186 ± 3 bpm and CS- 187 ± 11 vs. DS- 181 ± 3 bpm. The authors concluded that in the absence of neuropathy, individuals with uncomplicated T1D are able to reach the same VO_2max and HRmax as similarly trained ND. Our results are in line with this since none of our participants with T1D had neuropathy.

In the present investigation, the measured VO_2max values for the ND and T1D groups were 56.7 ± 8.4 mL·kg^{-1}·min^{-1}and 54.6 ± 5.3 mL·kg^{-1}·min^{-1}respectively. These VO_2max values in this investigation are similar to the athletic groups CE- 56.8 ± 9.1 and DE- 54.0 ± 8.1 mL·kg^{-1}·min^{-}

[1] found in the investigation by Veves et al. (1997) despite the age difference in the study participant cohorts. The HRmax of the athletic groups in Veves at al. (1997) study are substantially lower CE- 184 ± 12 and DE- 186 ± 3 bpm, than those found in the present investigation ND- 197.2 ± 5.2 and T1D- 196.7 ± 8.1 bpm, but this could be partially attributed to the protocol that Veves et al. employed to attain the VO_2max values. It is generally assumed that the T1D population have significantly lower aerobic fitness levels compared to the healthy ND population. However, it seems that with equivalent exercise histories persons with uncomplicated T1D are able to achieve similar high range VO_2max and HRmax values as their ND counterparts, indicating comparable exercise capacities and trainability responses.

It is important to note that studies have reported more consistently that individuals with T1D have lower exercise tolerances in comparison with ND (Chu et al., 2011; Komatsu et al., 2005; Masuda & Sato, 1989; Metcaf et al., 2014; Nadeau et al., 2010; Poortmans et al., 1986; Riddell & Perkins, 2006; Robertson et al., 2009; Wanke et al., 1992; Williams et al., 2011). A possible explanation for this inconsistency in the literature may be due to of the select population investigators recruited. Many fail to assess the habitual non-exercise plus exercise physical activity patterns in young persons with T1D prior to study commencement, as well as focus on non-athletic populations. Consequently, their conclusions regarding lower aerobic endurance and exercise tolerance in individuals with T1D may not be related to diabetes per se, but in fact due to a lower physical plus physiological fitness level to begin with (Brandenburg et al., 1999). Mentioned previously, the investigators Arslanian et al., (1990), Austin et al., (1993), Faulkner (2010), Komatsu et al., (2005) and Wanke et al., (1992) reported low VO_2 measures ranging from 34- 41 $mL \cdot kg^{-1} \cdot min^{-1}$ in youth with T1D. (Arslanian: T1D- 34.9 ± 8.6 vs ND- 38.6±

9.9 mL·kg^{-1}·min^{-1}; Austin: T1D- 33.7 ± 7.0 vs ND- 41.0 ± 10.4 mL·kg^{-1}·min^{-1} [matched for age, BMI, and Tanner stage]; Faulkner: T1D- 34.7 ± 8.9 vs T2D- 25.4 ± 5.9 mL·kg^{-1}·min^{-1}; Komatsu: T1D- 41.57 ± 7.68 vs ND- 51.12 ± 9.94 mL·kg^{-1}·min^{-1}; Wanke: T1D- 2.56 ± 0.71 vs ND- 3.17 ± 0.77 L·min^{-1}). With one exception, there was no mention of PA histories or matching participants for PA levels in any of these studies, therefore it is assumed that these investigators failed to assess habitual exercise and non-exercise PA patterns in their participants. Faulkner (2010) was the only investigator to report previous PA levels for the participants, but did not include a control group and compared VO_2max between youth with T1D and T2D. Faulkner (2010) employed the Physical Activity Recall, which obtains information on participants' recent PA participation for the previous 7 days. Using a 7-day recall towards assessing exercise history is insufficient to understand a participants long-term non-exercise plus exercise physical activity habits and properly categorize their physical plus physiological fitness levels. The athletes with T1D who participated in the present investigation were matched for physical plus physiological fitness levels, with the ND recruited from the same hockey team. Therefore, the PA levels did not differ between pairs in terms of frequency and intensity of training and game schedule. This may be one reason why a difference in VO_2max was not observed between the two groups.

To avoid some of the inherent weaknesses in the previous investigations, the present investigation employed the following:

1) Matched controls for age, sex, height, body mass, and non-exercise plus exercise PA levels. We recruited the participants from the same ice-hockey team to ensure no difference in habitual exercise. This was essential, as if it was found that the participants

with T1D had lower VO_2max, VO_2 and HR responses it would undoubtedly be diabetes related. Previous investigators may not have matched for physical plus physiological fitness levels, and consequently, would not have been founded on a select, athletic population.

2) Directly measured VO_2 and HR and documented the responses during the simulation hockey game (HITT) to identify any abnormalities in the rate of the response of individuals with T1D at any time point during exercise.
3) Used elite athletic participants who already possessed a high aerobic capacity and were familiar with strenuous, high-intensity exercise. Therefore, fatigue or exercise incapability due to deconditioning or low exercise tolerance possibly confounding the results was avoided.
4) Carefully considered the protocol design, which simulated a 60 minute ice-hockey game. Recording HR during an on-ice game allowed for a more accurate/comprehensive idea of the intensity and energy demand required by the athletes during a real-life game. This approach enabled us to design a high-intensity intermittent laboratory protocol incorporating the appropriate frequency, intensity and duration of hockey shifts (sprints vs. active recovery) and bench times. One limitation of the laboratory protocol was that even though the participants were instructed to approach the simulation hockey game as they would a real-life ice-hockey game, the protocol could not simulate the same motivation and adrenaline that the individuals would experience during an actual on-ice game.

Another relevant finding reported by Veves et al. (1997) was that the VO_2max of the NDE group was lower (42.0 ± 11.6 $mL \cdot kg^{-1} \cdot min^{-1}$) compared with the CE and DE groups, and similar to those of the sedentary CS (38.7 ± 6.0 $mL \cdot kg^{-1} \cdot min^{-1}$) and DS (36.7 ± 9.6 $mL \cdot kg^{-1} \cdot min^{-1}$) groups. These findings indicate that with additional diabetic related complications exercise capacity is decreased. While this is true, many studies have proven that PA and exercise can reduce diabetes related comorbidities and improve quality of life (Chu et al., 2011; Komatsu et al., 2005; Mosher et al., 1998; Rowland et al., 1992; Sideraviciute et al., 2006; Veves et al., 1997; Wasserman & Zinman, 1994; Zinker, 1999). In addition, studies that have investigated the effects of exercise programs on the VO_2max of children and adolescents with T1D, have reported significant increases in aerobic fitness (Campaigne, et al., 1984; Mosher et al., 1998). Some studies have reported increases in peak aerobic fitness (VO_2peak) as much as 27% in individuals with T1D (Chimen et al., 2012), which indicates that individuals with T1D can improve cardiorespiratory function similar to ND with exercise training. These findings reaffirm the significance of PA and exercise as a beneficial component for long-term physical plus physiological fitness and health for individuals with T1D.

This is the first study to characterize the VO_2 and HR responses in youths with uncomplicated T1D in response to a HITT protocol. We chose a simulated hockey game because of the sport's unique high-intensity and intermittent nature and its popularity among Canadian youth. The high-intensity bursts of activity, the prolonged duration of a full-length game, and the need for rapid recovery between shifts demands strength, power, and high anaerobic and aerobic fitness. Regensteiner et al. (1998), Brandenburg et al. (1999), Bauer et al. (2007), and Ananey et al. (2011) are the only authors who have published data on VO_2 and HR kinetics of a

diabetic population in response to constant-load exercise. These studies involved measuring the VO_2 and HR kinetics of persons with T2D during constant-load exercise above and below the lactate threshold. Regensteiner et al. (1998) characterized the VO_2 and HR kinetic response in premenopausal women with T2D during constant-load exercise and found that they had a reduced response compared to both their lean and overweight controls. The authors concluded that the impaired cardiac response observed at the onset of exercise compromised oxygen delivery attributing to the limited response rate, but Regensteiner et al. (1998) did not exclude the possibility of an additional defect in skeletal muscle oxygen diffusion or mitochondrial oxygen utilization. Brandenburg et al. (1999), in response to Regensteiner's findings, examined if VO_2max and VO_2 kinetics would improve with exercise training in untrained premenopausal women with T2D before and after 3 months of supervised training. Before training the women with T2D had the lowest VO_2max and slowest VO_2 kinetics. Brandenburg et al. (1999) found that after the exercise program the women with T2D benefited more from the exercise training than the controls. Ananey et al. (2011) investigated whether an abnormal cardiac output response was related to VO_2 kinetics in middle-aged women with uncomplicated T2D. All the participants were sedentary (< 1 hr/wk of moderate-intensity exercise) for at least 3 months before the study commenced, which was determined via the Low-Level PA Recall questionnaire. The results showed that the VO_2peak was significantly lower in the participants with T2D compared with both their overweight and lean controls. The participants with T2D had a slowed dynamic response in VO_2 during light and moderate intensity exercise, but this occurred in the absence of a reduced cardiac output response during exercise; HR kinetics had a similar response. To explain the slowed VO_2 kinetics observed in the T2D group, the

investigators proposed that it was attributable to blood flow and the arteriovenous oxygen difference (a-v O_2). Blood flow could be limited by poor cardiac output and/or impaired vasodilation, whereas a-v O_2 reflects the interaction between blood flow and myocyte VO_2. Consequently, due to the slow rise in oxygen consumption by the contracting muscles, there is a slower increase in a-v O_2, subsequently impairing the VO_2 kinetic response.

The present investigation was the first to examine the dynamic response of VO_2 and HR during HITT, specifically a simulation hockey game. Since the exponential equations for calculating kinetics cannot be applied to HITT, we characterized the VO_2 and HR response in athletic youths with and without T1D by calculating the average nadir, mean, and average peak %VO_2max and %HRmax for every play shift and bench intervals. Some differences were observed between the 3 periods in both the T1D and ND groups. Looking at Figures 11 and 12, the differences between the play shifts and bench intervals appear to be random and inconsistent across periods 1, 2 and 3. Since the differences were observed in both groups, it is doubtful that the response is attributed to diabetes related exercise intolerance. Therefore, the differences in the average nadir and average peak %VO_2max and %HRmax values are most likely attributed to the athletes' self-selected pacing during the simulation hockey game.

The T1D group had similar average nadir and average peak %VO_2max and %HRmax responses in comparison with the ND group throughout the simulation hockey game; no differences between the responses were evident. This is a select population given that they were highly active individuals with well-controlled glycemia, and did not possess any diabetes related comorbidities. It seems that within this elite-athletic population, the VO_2 response is

not hindered. During the simulation hockey game, the individuals with T1D were able to reach similar peaks in %VO_2max and %HRmax during the exercise play shifts (Figures 9 and 10) as the ND participants. This suggests that individuals with T1D are not physiologically limited by diabetes during HITT and are able to uptake, deliver, and utilize oxygen efficiently. The paralleled HR response between the groups with and without T1D gives no indication of cardiovascular impairment during this type of exercise. These findings also suggest, there is no sign of diabetes related fatigue, or reduced aerobic endurance due to abnormalities in the function and rate of oxygen transportation or diffusion. On the basis of the Fick equation for VO_2, VO_2 is influenced by a combination of cardiovascular and peripheral factors. In the absence of physiological data, a limitation of the current study, the effect of T1D on the VO_2 and HR response to HITT is difficult to explain. Understanding the physiological mechanisms that control the dynamic response of VO_2 in T1D is important, as a reduction in this response may be linked to increased fatigue during exercise.

In addition to understanding the physiological significance of the VO_2 response in individuals with T1D, it is also essential to recognize the clinical significance (Brandenburg et al., 1999). If youth with T1D are discouraged from participating in sport and exercise due to lack of knowledge and apprehension, they are more likely to avoid PA leading to low exercise tolerance. Sequentially this may contribute to an individual's perception of their ability to initiate or to perform exercise and non-exercise PA as being more difficult (Brandenburg et al., 1999). Ultimately this could dissuade individuals from adopting a more physically active lifestyle. Increasing the awareness on the positive effects of exercise on T1D and improving the initial experience to exercise are necessary to encourage participation and reduce the

unwillingness to engage in PA and exercise. Understanding the magnitude and causes of any exercise impairment is crucial to improving performance and preventing increasing disability in this population.

The focus of VO_2 and HR responses to HITT in persons with T1D is relatively new, and greatly revolves around the glucoregulatory response (Bussau, Ferreira, Jones, & Fournier, 2006; Bussau, Ferreira, Jones, & Fournier, 2007; Guelfi et al., 2005; Guelfi et al., 2007; Robertson et al., 2009). These investigations have consistently shown that HITT is well tolerated by individuals with uncomplicated T1D. Guelfi et al. (2007) revealed that males and females with T1D (22.6 ± 5.7 years) performed a superior amount of total work during HITT compared with continuous moderate-intensity exercise. This was reflected by the greater response of VO_2 and HR during HITT; the average rate of VO_2 consumption during HITT corresponded to 55% of VO_2peak in comparison with 40% during moderate-intensity exercise. In the current study the participants with T1D frequently reached 60-100% of their VO_2max and HRmax during the simulation hockey game protocol. This shows that there is a significant amount of aerobic work being done during HITT and that this type of exercise is not solely reliant on the anaerobic system.

In the same investigation conducted by Guelfi et al. (2007), the participants acted as their own controls. Guelfi et al. (2007) created a 30-minute HITT protocol (cycling at 40% VO_2peak, interspersed with 4-second maximal sprint efforts performed every 2 minutes) based on other researchers investigations on time-motion analyses of various intermittent sports (soccer, hockey, gaelic football) and spontaneous play in children. The present investigation is

the first to show T1D in a full-length simulation hockey game protocol based on HR response collected during an on-ice hockey game. Building on the previous research regarding T1D and HITT, this study now shows that the VO_2 and HR response of athletic youth with T1D are not limited by diabetes while performing HITT. Like their healthy counterparts, they have the aerobic fitness level to perform long durations of intermittent high-intensity activity. This is very important, as it proves that youth with uncomplicated T1D have the ability to be just as competitive in sports as healthy ND individuals.

The results of the current investigation suggest that similar training status and exercise histories play a great role in the determination of aerobic fitness in youth with uncomplicated T1D. It is concluded that athletic adolescents with T1D achieve similar VO_2max and HRmax values, plus exhibit similar VO_2 and HR responses during a simulated hockey game when compared to their ND counterparts. The results indicate that adolescents with uncomplicated T1D can participate in sports, exercise and non-exercise PA at the same level as their ND counterparts, and not be limited by diabetes. Along with previous studies, this investigation reaffirms the benefits of being an active individual with T1D; as it increases physical and physiological fitness, and may prevent further disability, and ultimately influences quality of life. This investigation offers a more optimistic view as it suggests that diabetes itself does not affect the exercising capacity in trained individuals, and underscores that athletic youth with uncomplicated T1D have the exercise capacity and potential to participate safely in competitive sport, exercise and non-exercise PA.

2.4 LIMITATIONS

Currently there are no investigations that have focused on the VO_2 and HR response of youth with T1D during HITT protocol. This is the first study of its kind and consequently it is difficult to draw comparisons with previous investigations and findings as there is no access to detailed literature in the area. As well, research focused in the area of T1D, HITT and VO_2 kinetics is still novel. It is necessary to establish a method that investigators can employ for analysing VO_2 kinetics during HITT. This would be extremely advantageous for future research in intermittent PA and exercise, not only in persons with T1D, but also in persons with various chronic diseases and conditions.

The participants involved in the present investigation were recruited from a select population. The individuals represented a male and female population ranging from 13-17 years with a high aerobic fitness levels (mean VO_2max = 55.6 $mL \cdot kg^{-1} \cdot min^{-1}$), who all competed in high level competitive hockey. Therefore, generalizations to other active T1D populations may not be appropriate. It would be beneficial to conduct the study with a larger sample size, as well as include an equal number of males and females in the sample to compare any possible sex-related differences. It also would be advantageous to include a sedentary control and sedentary diabetic group for a more comprehensive understanding of the VO_2 and HR response during HITT.

Additionally, the small number of study participants in this study may not allow for generalizations to be made to all individuals with T1D. For instance, these findings cannot be generalized to individuals with T1D with poor glycemic control and/or diabetes related

comorbidities. Not only would these individuals possess a lower exercise tolerance, but the nature of HITT may not be well tolerated by individuals with complicated T1D. It has been recommended that individuals with diabetes related conditions, like proliferative retinopathy or nephropathy, avoid PA that can result in high arterial blood pressure such as lifting heavy weights and performing high intensity sprints (Robertson et al., 2009). A separate study looking at individuals with T1D with poor glycemic control and/or diabetes related comorbidities would be meaningful to identify the types of exercise that would be well tolerated and beneficial to their health.

A final limitation is that all performance measurements were conducted in a laboratory setting, and as such, generalizations to on-ice game situations may be limited. Despite employing the Cosmed Fitmate (a reliable and accurate VO_2 system) to measure and record VO_2 and HR, the device could have predicted some erroneous values. As well, due to failure of the Fitmate system, some data were lost. There were incidences where the Fitmate started recording the data late, stopped recording before the protocol was completed, or failed to record the data entirely. As a result, differences in the VO_2 and HR responses may have been missed. It would have been ideal for the participants whose data were lost to be scheduled to perform the laboratory protocol on a subsequent day so that their data could have been included in the analysis.

2.5 IMPLICATIONS

Exploring this relatively novel area of research has enhanced the understanding of the VO_2 and HR responses during HITT in highly active persons with uncomplicated T1D. The behaviour of the VO_2 and HR response have been characterized in the present investigation and have demonstrated that active youth with uncomplicated T1D and well controlled glycemia have a similar VO_2 and HR response to healthy active ND youths. The results of this study provide individuals with T1D, parents, and clinicians with T1D with more insight regarding the cardiovascular response and the limitations associated with PA participation. With an in-depth, comprehensive understanding of persons with T1D and their response to various types of PA and exercise, perceived barriers like fear or apprehension can be assuaged, thereby encouraging youth with T1D to increase their participation in both recreational and high-level competitive sports. It is not unheard of to see great athletes with T1D making a successful career for themselves in sport like: Jay Cutler, quarterback for the NFL's Chicago Bears, Brandon Morrow and Dustin McGowan for MLB's Toronto Blue Jays, and Bobby Clarke, Captain of the NHL's Philadelphia Flyers. Clarke had a 15-year career with many accomplishments, playing in over 1,000 regular season games, scoring 358 goals, and winning 2 Stanley cups (Macleod, 2015). Clarke has been an inspiration to many young hockey players with T1D, specifically Max Domi, who is an up-incoming NHL star (Macleod, 2015). Domi was a first-round draft pick in 2014, is a top prospect for the NHL's Arizona Coyotes, and participated in the World Junior Championship on Team Canada, finishing as the tournaments best forward. It is important to have respected figures paving the way for individuals with T1D in highly competitive and elite domains.

Further research is essential within this subject area to amalgamate highly accurate, evidence-based guidelines for persons with complicated and uncomplicated T1D who engage in exercise and sports characterized by intermittent high-intensity activity. It would be valuable to investigate the VO_2 and HR response to HITT in a population of middle age athletes with complicated and uncomplicated T1D. As well, conducting a longitudinal study following highly active youth with T1D would provide insight into understanding the possible repercussions of age and long-term T1D on the physical plus physiological fitness levels in these individuals. The more knowledge acquired on athletes with T1D and their response to exercise, the better coaches and exercise trainers can assist and properly train athletic youth with T1D. With an increase in available information, young athletes with T1D will no longer have to be apprehensive of exercise and non-exercise PA, showing them that with the proper regimen, discipline and care, their bodies can perform at the same level as their normal healthy counterparts. Ultimately research in this domain could assist in attenuating the apprehension associated with T1D and exercise plus non-exercise PA, and may foster greater participation of youth with T1D in competitive sport. Athletic figures like Clarke and Domi prove that individuals with T1D are not only able to live normal lives, but may also live extraordinary ones.

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2.7 APPENDICES

APPENDIX A : RECRUITMENT POSTER

YORK UNIVERSITÉ UNIVERSITY

Looking for Hockey Players Aged 13-21 with Type 1 Diabetes to Participate in a Study

Do you have trouble managing your glucose levels during or after games?

Are you interested in learning more about how hockey influences your body?

In an area of many unknowns, we want to help.

What we're studying: 1) glucose levels during and after hockey games
2) post-exercise heart rate variability

What is required: Must visit an exercise lab at York University on 2 occasions

- ☆ Participants will be fitted with a glucose monitor (Medtronic iPro2) and Polar heart rate monitor to wear for 1 week, including 1 hockey game
- ☆ Lab Visit 1: maximum aerobic threshold test (VO2 max) on a treadmill
- ☆ Lab Visit 2: 60-minute exercise bout on a stationary bike
- ☆ Must wear a non-invasive Holter monitor on 3 nights to assess the effect of hockey & hockey-type activity on glucose levels and heart rate variability.

If interested, please contact

YORK UNIVERSITÉ UNIVERSITY
redefine THE POSSIBLE

APPENDIX B: 2015 PAR-Q+

2015 PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

The health benefits of regular physical activity are clear; more people should engage in physical activity every day of the week. Participating in physical activity is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

GENERAL HEALTH QUESTIONS

Please read the 7 questions below carefully and answer each one honestly: check YES or NO.	YES	NO
1) Has your doctor ever said that you have a heart condition ☐ OR high blood pressure ☐?	☐	☐
2) Do you feel pain in your chest at rest, during your daily activities of living, **OR** when you do physical activity?	☐	☐
3) Do you lose balance because of dizziness **OR** have you lost consciousness in the last 12 months? **Please answer NO if your dizziness was associated with over-breathing (including during vigorous exercise).**	☐	☐
4) Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)? **PLEASE LIST CONDITION(S) HERE:** _____	☐	☐
5) Are you currently taking prescribed medications for a chronic medical condition? **PLEASE LIST CONDITION(S) AND MEDICATIONS HERE:** _____	☐	☐
6) Do you currently have (or have had within the past 12 months) a bone, joint, or soft tissue (muscle, ligament, or tendon) problem that could be made worse by becoming more physically active? **Please answer NO if you had a problem in the past, but it *does not limit your current ability* to be physically active.** **PLEASE LIST CONDITION(S) HERE:** _____	☐	☐
7) Has your doctor ever said that you should only do medically supervised physical activity?	☐	☐

If you answered NO to all of the questions above, you are cleared for physical activity.
Go to Page 4 to sign the PARTICIPANT DECLARATION. You do not need to complete Pages 2 and 3.

- Start becoming much more physically active – start slowly and build up gradually.
- Follow International Physical Activity Guidelines for your age (www.who.int/dietphysicalactivity/en/).
- You may take part in a health and fitness appraisal.
- If you are over the age of 45 yr and **NOT** accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional before engaging in this intensity of exercise.
- If you have any further questions, contact a qualified exercise professional.

If you answered YES to one or more of the questions above, COMPLETE PAGES 2 AND 3.

Delay becoming more active if:

- You have a temporary illness such as a cold or fever; it is best to wait until you feel better.
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X+ at **www.eparmedx.com** before becoming more physically active.
- Your health changes - answer the questions on Pages 2 and 3 of this document and/or talk to your doctor or a qualified exercise professional before continuing with any physical activity program.

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FOLLOW-UP QUESTIONS ABOUT YOUR MEDICAL CONDITION(S)

1. Do you have Arthritis, Osteoporosis, or Back Problems?

If the above condition(s) is/are present, answer questions 1a-1c — If **NO** ☐ go to question 2

1a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
1b.	Do you have joint problems causing pain, a recent fracture or fracture caused by osteoporosis or cancer, displaced vertebra (e.g., spondylolisthesis), and/or spondylolysis/pars defect (a crack in the bony ring on the back of the spinal column)?	YES ☐ NO ☐
1c.	Have you had steroid injections or taken steroid tablets regularly for more than 3 months?	YES ☐ NO ☐

2. Do you have Cancer of any kind?

If the above condition(s) is/are present, answer questions 2a-2b — If **NO** ☐ go to question 3

2a.	Does your cancer diagnosis include any of the following types: lung/bronchogenic, multiple myeloma (cancer of plasma cells), head, and neck?	YES ☐ NO ☐
2b.	Are you currently receiving cancer therapy (such as chemotheraphy or radiotherapy)?	YES ☐ NO ☐

3. Do you have a Heart or Cardiovascular Condition? *This includes Coronary Artery Disease, Heart Failure, Diagnosed Abnormality of Heart Rhythm*

If the above condition(s) is/are present, answer questions 3a-3d — If **NO** ☐ go to question 4

3a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
3b.	Do you have an irregular heart beat that requires medical management? (e.g., atrial fibrillation, premature ventricular contraction)	YES ☐ NO ☐
3c.	Do you have chronic heart failure?	YES ☐ NO ☐
3d.	Do you have diagnosed coronary artery (cardiovascular) disease and have not participated in regular physical activity in the last 2 months?	YES ☐ NO ☐

4. Do you have High Blood Pressure?

If the above condition(s) is/are present, answer questions 4a-4b — If **NO** ☐ go to question 5

4a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
4b.	Do you have a resting blood pressure equal to or greater than 160/90 mmHg with or without medication? (Answer **YES** if you do not know your resting blood pressure)	YES ☐ NO ☐

5. Do you have any Metabolic Conditions? *This includes Type 1 Diabetes, Type 2 Diabetes, Pre-Diabetes*

If the above condition(s) is/are present, answer questions 5a-5e — If **NO** ☐ go to question 6

5a.	Do you often have difficulty controlling your blood sugar levels with foods, medications, or other physician-prescribed therapies?	YES ☐ NO ☐
5b.	Do you often suffer from signs and symptoms of low blood sugar (hypoglycemia) following exercise and/or during activities of daily living? Signs of hypoglycemia may include shakiness, nervousness, unusual irritability, abnormal sweating, dizziness or light-headedness, mental confusion, difficulty speaking, weakness, or sleepiness.	YES ☐ NO ☐
5c.	Do you have any signs or symptoms of diabetes complications such as heart or vascular disease and/or complications affecting your eyes, kidneys, **OR** the sensation in your toes and feet?	YES ☐ NO ☐
5d.	Do you have other metabolic conditions (such as current pregnancy-related diabetes, chronic kidney disease, or liver problems)?	YES ☐ NO ☐
5e.	Are you planning to engage in what for you is unusually high (or vigorous) intensity exercise in the near future?	YES ☐ NO ☐

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6.	**Do you have any Mental Health Problems or Learning Difficulties?** *This includes Alzheimer's, Dementia, Depression, Anxiety Disorder, Eating Disorder, Psychotic Disorder, Intellectual Disability, Down Syndrome* If the above condition(s) is/are present, answer questions 6a-6b	If **NO** ☐ go to question 7
6a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
6b.	Do you **ALSO** have back problems affecting nerves or muscles?	YES ☐ NO ☐
7.	**Do you have a Respiratory Disease?** *This includes Chronic Obstructive Pulmonary Disease, Asthma, Pulmonary High Blood Pressure* If the above condition(s) is/are present, answer questions 7a-7d	If **NO** ☐ go to question 8
7a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
7b.	Has your doctor ever said your blood oxygen level is low at rest or during exercise and/or that you require supplemental oxygen therapy?	YES ☐ NO ☐
7c.	If asthmatic, do you currently have symptoms of chest tightness, wheezing, laboured breathing, consistent cough (more than 2 days/week), or have you used your rescue medication more than twice in the last week?	YES ☐ NO ☐
7d.	Has your doctor ever said you have high blood pressure in the blood vessels of your lungs?	YES ☐ NO ☐
8.	**Do you have a Spinal Cord Injury?** *This includes Tetraplegia and Paraplegia* If the above condition(s) is/are present, answer questions 8a-8c	If **NO** ☐ go to question 9
8a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
8b.	Do you commonly exhibit low resting blood pressure significant enough to cause dizziness, light-headedness, and/or fainting?	YES ☐ NO ☐
8c.	Has your physician indicated that you exhibit sudden bouts of high blood pressure (known as Autonomic Dysreflexia)?	YES ☐ NO ☐
9.	**Have you had a Stroke?** *This includes Transient Ischemic Attack (TIA) or Cerebrovascular Event* If the above condition(s) is/are present, answer questions 9a-9c	If **NO** ☐ go to question 10
9a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
9b.	Do you have any impairment in walking or mobility?	YES ☐ NO ☐
9c.	Have you experienced a stroke or impairment in nerves or muscles in the past 6 months?	YES ☐ NO ☐
10.	**Do you have any other medical condition not listed above or do you have two or more medical conditions?** If you have other medical conditions, answer questions 10a-10c	If **NO** ☐ read the Page 4 recommendations
10a.	Have you experienced a blackout, fainted, or lost consciousness as a result of a head injury within the last 12 months **OR** have you had a diagnosed concussion within the last 12 months?	YES ☐ NO ☐
10b.	Do you have a medical condition that is not listed (such as epilepsy, neurological conditions, kidney problems)?	YES ☐ NO ☐
10c.	Do you currently live with two or more medical conditions?	YES ☐ NO ☐
	PLEASE LIST YOUR MEDICAL CONDITION(S) AND ANY RELATED MEDICATIONS HERE:	

GO to Page 4 for recommendations about your current medical condition(s) and sign the PARTICIPANT DECLARATION.

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If you answered NO to all of the follow-up questions about your medical condition, you are ready to become more physically active - sign the PARTICIPANT DECLARATION below:

- It is advised that you consult a qualified exercise professional to help you develop a safe and effective physical activity plan to meet your health needs.
- You are encouraged to start slowly and build up gradually - 20 to 60 minutes of low to moderate intensity exercise, 3-5 days per week including aerobic and muscle strengthening exercises.
- As you progress, you should aim to accumulate 150 minutes or more of moderate intensity physical activity per week.
- If you are over the age of 45 yr and **NOT** accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional before engaging in this intensity of exercise.

If you answered **YES** to **one or more of the follow-up questions** about your medical condition:

You should seek further information before becoming more physically active or engaging in a fitness appraisal. You should complete the specially designed online screening and exercise recommendations program - the ePARmed-X+ at www.eparmedx.com and/or visit a qualified exercise professional to work through the ePARmed-X+ and for further information.

Delay becoming more active if:

- You have a temporary illness such as a cold or fever; it is best to wait until you feel better.
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X+ at www.eparmedx.com before becoming more physically active.
- Your health changes - talk to your doctor or qualified exercise professional before continuing with any physical activity program.

- You are encouraged to photocopy the PAR-Q+. You must use the entire questionnaire and NO changes are permitted.
- The authors, the PAR-Q+ Collaboration, partner organizations, and their agents assume no liability for persons who undertake physical activity and/or make use of the PAR-Q+ or ePARmed-X+. If in doubt after completing the questionnaire, consult your doctor prior to physical activity.

PARTICIPANT DECLARATION

- All persons who have completed the PAR-Q+ please read and sign the declaration below.
- If you are less than the legal age required for consent or require the assent of a care provider, your parent, guardian or care provider must also sign this form.

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that a Trustee (such as my employer, community/fitness centre, health care provider, or other designate) may retain a copy of this form for their records. In these instances, the Trustee will be required to adhere to local, national, and international guidelines regarding the storage of personal health information ensuring that the Trustee maintains the privacy of the information and does not misuse or wrongfully disclose such information.

NAME _____ DATE _____

SIGNATURE _____ WITNESS _____

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER _____

For more information, please contact
www.eparmedx.com
Email: eparmedx@gmail.com

citation for par-q+
Warburton DER, Jamnik VK, Bredin SSD, and Gledhill N on behalf of the PAR-Q+ Collaboration.
The Physical Activity Readiness Questionnaire for Everyone (PAR-Q+) and Electronic Physical Activity Readiness Medical Examination (ePARmed-X+). Health & Fitness Journal of Canada 4(2):3-23, 2011.

par-q+ references
1. Jamnik VK, Warburton DER, Makarski J, McKenzie DC, Shephard RJ, Stone J and Gledhill N. Enhancing the effectiveness of clearance for physical activity participation: background and overall process. APNM 36(3):3-13, 2011.
2. Warburton DER, Gledhill N, Jamnik VK, Bredin SSD, McKenzie DC, Stone J, Charlesworth S, and Shephard RJ. Evidence-based risk assessment and recommendations for physical activity clearance: Consensus Document. APNM 36(S1):S266-s298, 2011.

The PAR-Q+ was created using the evidence-based AGREE process [1] by the PAR-Q+ Collaboration chaired by Dr. Darren E. R. Warburton with Dr. Norman Gledhill, Dr. Veronica Jamnik, and Dr. Donald C. McKenzie [2]. Production of this document has been made possible through financial contributions from the Public Health Agency of Canada and the BC Ministry of Health Services. The views expressed herein do not necessarily represent the views of the Public Health Agency of Canada or the BC Ministry of Health Services.

01-01-2015

APPENDIX C: CONSENT FORMS

Minor Assent Form

Study Name: Assessing the impact of intermittent high-intensity exercise on subsequent glucose management and autonomic function in elite-level youth athletes with type 1 diabetes

Researchers: Dr. Michael Riddell, Dr. Veronica Jamnik
Lisa Miadovnik, MSc Candidate, Kinesiology & Health Science, York University

Purpose of the Research: This will be the first study completed and published on type 1 diabetic hockey players. Because of the unique glycemic response reported in hockey players, we want to see how this type of exercise impacts later glucose management. We also want to see how hockey-type exercise impacts overnight autonomic function. Autonomic function refers to how the nervous system functions and can be measured by looking at heart rate variability. A Holter monitor is used to assess heart rate variability, which is basically the variation in the time interval between heartbeats. Since a healthy nervous system constantly changes how it stimulates the heart, a healthy person will have high heart rate variability. Our overall goal is to determine the effects that hockey and hockey-type exercise have on your blood sugar management and heart rate variability afterwards. We also want to see if blood sugar levels impact heart rate variability. This research will be presented to graduate students and professors at a seminar held at York University. We also aim to have it published in an academic journal, and presented at various professional conferences on diabetes.

What You Will Be Asked to Do in the Research: This study will require 2 trips to the Human Performance Laboratory at York University. All participants will complete an initial fitness assessment in the laboratory which will take roughly 1.5 hours. This assessment will include measurement of height, weight, skinfolds/body fat, and 2 maximal exertion cardiorespiratory tests. On this visit, each participant will be required to complete a 30-second Wingate anaerobic cycling test, and a 15-20 minute aerobic (VO_2 max) test performed on a motorized treadmill.

Participants will also be required to visit the lab on a 2nd occasion which will last 2.5-3 hours. During this visit, the participants will complete an exercise protocol on a cycle ergometer which mimics the work done by the legs during a hockey game. This protocol will involve 45 minutes of intermittent cycling, which will take place over a 60 minute time frame (to simulate a game). Participants will wear a polar heart rate strap, and a Fitmate™ VO_2 device which will continuously measure the amount of oxygen you're using – or how hard you're working. Following the completion of this protocol, all equipment will be removed and a Holter Monitor ™ will be placed on the participant. This monitor will be worn for 30 minutes in exercise recovery, while lying down in a dark, quiet room.

The Holter Monitor ™ is a small electronic device, worn on the hip. Connected to it are 5 wires with gel pads that are stuck to the front of the chest cavity. These gel pads pick up electrical activity of the heart, and transmit the information to the monitor, where the information is recorded. This monitor is to be worn on 3 occasions, from 12:00 AM or earlier, until 6:00 AM or later. One occasion will be after completing the lab exercise protocol as mentioned above. A second occasion will be after a hockey game, and a third occasion will be after a low-activity day. Individuals will be asked to record the activities they partake in on each of these 3 days. It is important that minimal physical activity is performed on the low-activity day.

All of the above requirements apply to both participants with diabetes, and their control (non-diabetic) participants. All procedures listed above are non-invasive.

Participants with diabetes will have the additional requirement of wearing a Medtronic iPro2, blinded continuous glucose monitor (CGM), for the 3 days when they assess their nocturnal heart rate variability. Scheduling will be designed so that all 3 data collection nights (ie after the hockey game, after the lab exercise protocol, and after the low-activity day) occur within a 5-day span. While wearing the CGM, participants will also be asked to manually check their blood sugar using their own glucose monitoring device at least once every 9 hours, and record all carbohydrate consumption and insulin administrations on the provided sheets.

Risks and Discomforts: Because this strenuous exercise is known to induce hyperglycemia during exercise, participants may suffer post-exercise hypoglycemia afterward. Participants will receive coaching on how to reduce

the incidence of hypoglycemia by exercise specialists such as Dr. Riddell - for example, reduce onboard insulin to zero during exercise, restrict insulin after exercise, and monitor blood glucose levels closely after exercise so that if levels start to drop, glucose can be consumed. Participants are also exposed to the typical risk of injury that is associated with exercise, including fatigue, lightheadedness, loss of consciousness, abnormal blood pressure, chest discomfort, leg gramps, nausea, and in rare cases, heart rhythm disturbances or heart attacks. Because all participants are elite athletes who are accustomed to intense exericse, the likelihood of experiencing the above side effects are quite small. Additionally, exercise will be supervised by qualified professionals to limit the risks to the participants, and a consent form will be read and signed by the dependent's parent or guardian prior to initiating the study. PAR-Q+ and if necessary, ePAR-medX evaluations will also be completed prior to exercise to insure participants are all eligible to partake in exercise, and understand the associated risks.

Benefits of the Research and Benefits to You: You will have the opportunity to participate in unique fitness testing procedures that are common to many elite and national-level athletes. You will also have access to your CGM data upon study completion, to observe the impact of hockey on your blood sugar management.

Voluntary Participation: Your participation in the study is completely voluntary and you may choose to stop participating at any time. Your decision not to volunteer will not influence the nature of the relationship you may have with the researchers or study staff, or the nature of your relationship with York University either now, or in the future.

Withdrawal from the Study: You can stop participating in the study at any time, for any reason, if you so decide. Your decision to stop participating, or to refuse to answer particular questions, will not affect your relationship with the researchers, York University, or any other group associated with this project. If you choose to withdraw from the study, all associated data collected will be immediately destroyed if you choose.

Confidentiality: All information you supply during the research will be held in confidence and unless you specifically indicate your consent, your name will not appear in any report or publication of the research. Data will be collected via hand-written notes, and converted into electronic documents where possible. All collected information will be coded to preserve your anonymity. Your data will be safely stored in a password-protected computer in a locked facility and only research staff will have access to this information. Confidentiality will be provided to the fullest extent possible by law. Collected data will be kept for a maximum of 5 years, after which time it will be destroyed.

Questions About the Research? If you have questions about the research in general or about your role in the study, please feel free to contact Lisa Miadovnik or the Graduate Supervisor - Dr. Michael Riddell either by telephone (listed above) or by e-mail. This research has received approval by the Human Participants Review Sub-Committee, York University's Ethics Review Board and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines. If you have any questions about this process or about your rights as a participant in the study, please contact the Sr. Manager & Policy Advisor for the Office of Research Ethics, 5th Floor, York Research Tower, York University.

Legal Rights and Signatures:
I_____, consent to participate in "Assessing the impact of intermittent high-intensity exercise on subsequent glucose management and autonomic function in elite-level youth with type 1 diabetes" conducted by Lisa Miadovnik, Supervised by Dr. Michael Riddell & Dr. Veronica Jamnik. I have understood the nature of this project and wish to participate. I am not waiving any of my legal rights by signing this form. My signature below indicates my consent.

Signature_____ **Date**_____
Participant

Signature_____ **Date**_____
Principal Investigator

Parental Consent Form

Study Name: Assessing the impact of intermittent high-intensity exercise on subsequent glucose management and autonomic function in elite-level youth athletes with type 1 diabetes

Researchers: Dr. Michael Riddell, Dr. Veronica Jamnik
Lisa Miadovnik, MSc Candidate, Kinesiology & Health Science, York University,

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Signature_____ **Date**_____
Participant

Signature_____ **Date**_____
Principal Investigator

Informed Consent Form

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Researchers: Dr. Michael Riddell, Dr. Veronica Jamnik
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Risks and Discomforts: Because exercise of this intensity is known to induce hyperglycemia during exercise, participants may suffer post-exercise hypoglycemia afterward. Participants will receive coaching on how to reduce the incidence of hypoglycemia by exercise specialists such as Dr. Riddell - for example, reduce onboard insulin to zero during exercise, restrict insulin after exercise, and monitor blood glucose levels closely after exercise so that if levels start to drop, glucose can be consumed. Participants are also exposed to the typical risk of injury that is

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Benefits of the Research and Benefits to You: Participants will have the opportunity to experience unique fitness testing procedures that are common to many elite and national-level athletes. They will also have access to their CGM data upon study completion, to observe the impact of hockey on their blood sugar management. This information will be available as a result of their participation in this study.

Voluntary Participation: Participation in the study is completely voluntary and individuals may choose to stop participating at any time. The decision not to volunteer will not influence the nature of the ongoing relationship you may have with the researchers or study staff, or the nature of your relationship with York University either now, or in the future.

Withdrawal from the Study: You can stop participating in the study at any time, for any reason, if you so decide. Your decision to stop participating, or to refuse to answer particular questions, will not affect your relationship with the researchers, York University, or any other group associated with this project. In the event you withdraw from the study, all associated data collected will be immediately destroyed if you choose.

Confidentiality: All information you supply during the research will be held in confidence and unless you specifically indicate your consent, your name will not appear in any report or publication of the research. Data will be collected via hand-written notes, and converted into electronic documents where possible. All collected information will be coded to preserve your anonymity. Your data will be safely stored in a password-protected computer in a locked facility and only research staff will have access to this information. Confidentiality will be provided to the fullest extent possible by law. Collected data will be kept for a maximum of 5 years, after which time it will be destroyed.

Questions About the Research? If you have questions about the research in general or about your role in the study, please feel free to contact Lisa Miadovnik or the Graduate Supervisor - Dr. Michael Riddell either by telephone (listed above) or by e-mail. This research has received approval by the Human Participants Review Sub-Committee, York University's Ethics Review Board and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines. If you have any questions about this process, or about your rights as a participant in the study, please contact the Sr. Manager & Policy Advisor for the Office of Research Ethics, 5th Floor, York Research Tower, York University.

Legal Rights and Signatures:
I_____, provide my consent to participate in "Assessing the impact of intermittent high-intensity exercise on subsequent glucose management and autonomic function in elite-level youth with type 1 diabetes" conducted by Lisa Miadovnik, Supervised by Dr. Michael Riddell & Dr. Veronica Jamnik. I have understood the nature of this project and wish to participate. I am not waiving any of my legal rights by signing this form. My signature below indicates my consent.

Signature_____ **Date**_____
Participant

Signature_____ **Date**_____
Principal Investigator

APPENDIX D: Simulation Hockey Game Protocol (Miadovnik, 2013)

Name:
Date:
Resistance: **Pre-Weight:** **Post-Weight:** **Pre-BG:**

Shift	Cosmed Time	On Ice	Play – Whistle – Play			Off Ice/ Bench	BG Check
1		0:00	20 –20– 40			1:20	
Period 1		RPM/HR/VO2					
Period 2		RPM/HR/VO2					
Period 3		RPM/HR/VO2					
2		3:10	20 – 30 – 20 – 30 – 15			5:05	
Period 1		RPM/HR/VO2					
Period 2		RPM/HR/VO2					
Period 3		RPM/HR/VO2					
3		6:00	30 – 20 – 10			7:00	
Period 1		RPM/HR/VO2					
Period 2		RPM/HR/VO2					
Period 3		RPM/HR/VO2					
4		10:10	10 – 20 – 40			11:20	
Period 1		RPM/HR/VO2					
Period 2		RPM/HR/VO2					
Period 3		RPM/HR/VO2					
5		12:10	30 – 20 – 20			13:20	
Period 1		RPM/HR/VO2					
Period 2		RPM/HR/VO2					
Period 3		RPM/HR/VO2					
6		15:40	10 – 20 – 10			16:20	
Period 1		RPM/HR/VO2					
Period 2		RPM/HR/VO2					
Period 3		RPM/HR/VO2					
7		17:20	20 – 40– 20 – 40 – 20 – 10– 10			20:00	

Period 1		RPM/HR/VO2						
Period 2		RPM/HR/VO2						
Period 3		RPM/HR/VO2						

APPENDIX E: ADDITIONAL TABLES AND GRAPHS

Table A1. Anthropometric and physical plus physiological fitness profiles of competitive youth ice hockey players with T1D

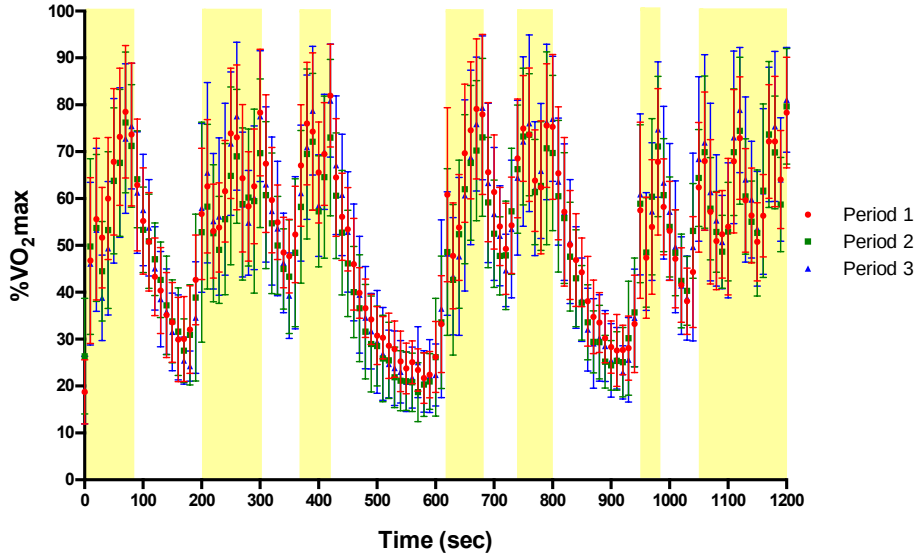
	Age (yrs)	Sex	Height (cm)	Weight (Kg)	WC (cm)	Skinfolds (mm)	Hockey Level	VO_2 max *(*mL·kg^{-1}·min^{-1}*)*	HR min (bpm)	HR max (bpm)	Peak Power (Watts)	Peak Power (Watts/kg)	Fatigue (%)
T1D 1	13	F	158	53.3	71	53.8	AA	56.2	67	202	471.9	8.9	46.4
T1D 2	17	M	175	73	79	56.6	Jr. A	49.8	51	192	735.3	10.1	51.7
T1D 3	14	M	167	58.7	74	57.7	AAA	59	51	190	647.4	11.0	48.0
T1D 4	17	F	167	64.4	74	95.3	A	51.8	57	209	567.0	8.8	39.3
T1D 5	13	F	157	64.1	74	102.7	AA	43.3	48	186	460.0	7.2	43.1
T1D 6	15	M	178	76.6	84	63	A	58.9	54	197	747.4	9.8	39.0
T1D 7	15	M	176	80.6	86.7	53.2	AAA	59.3	51	188	910.1	11.3	65.0
T1D 8	13	M	171	67.7	77		AAA	56.9	50	202	786.9	11.6	50.1
T1D 9	15	M	175	65.1	73.1	35.9	AAA	56	55	204	705.1	10.8	38.6
Mean ± SD	14.7 ± 1.58		169.3 ± 7.7	67.1 ± 8.6	77.0 ± 5.3	64.8 ± 22.6		54.6 ± 5.3	53.8 ± 5.7	196.7 ± 8.1	670.1 ± 148.9	9.9 ± 1.4	46.4 ± 8.9

Table A2. Anthropometric and physical plus physiological fitness profiles of competitive youth ice hockey players without T1D

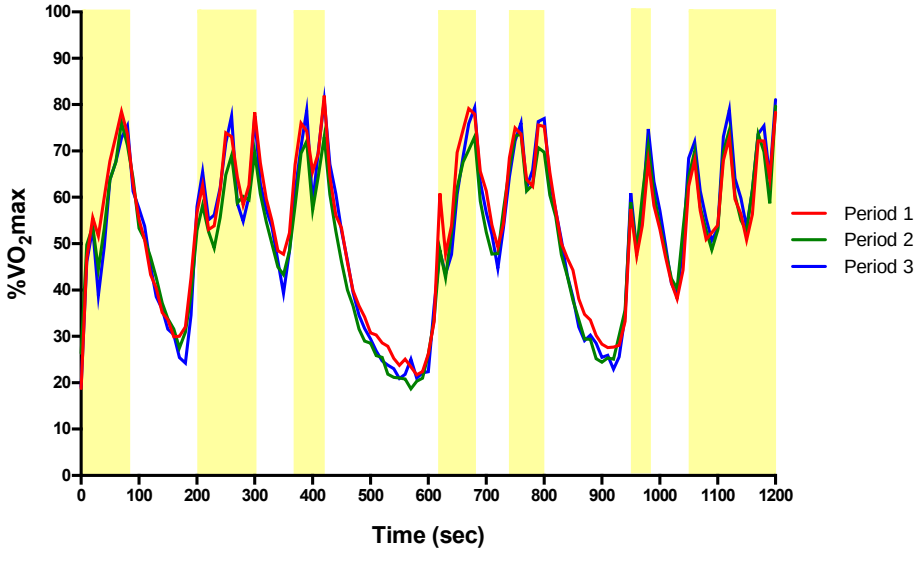
	Age (yrs)	Sex	Height (cm)	Weight (Kg)	WC (cm)	Skinfolds (mm)	Hockey Level	VO_2 max *(*mL·kg^{-1}·min^{-1}*)*	HR min (bpm)	HR max (bpm)	Peak Power (Watts)	Peak Power (Watts/kg)	Fatigue (%)
ND 1	13	F	163	55.5	74	55.8	AA	52.7	65	204	484.8	8.7	38.1
ND 2	17	M	174	75.6	83	62.4	AA	62.4	44	194	886.1	11.7	56.3
ND 3	14	M	170	68.5	77	70.8	AAA	63.3	52	193	770.9	11.3	54.5
ND 4	17	F	170	57.3	73	49.9	A	54.8	59	201	528.5	9.2	33.8
ND 5	13	F	154	51.2	72	52.8	AA	46.5	63	189	487.0	9.5	39.0
ND 6	15	M	167	71.4	83	53.2	AA	52.2	43	201	635.4	8.9	54.5
ND 7	15	M	184	82.2	89	39.2	AAA	61.1	53	195	1019.0	12.4	54.3
ND 8	13	M	176	73.1	79		AAA	64.1	50	195	860.5	11.8	
ND 9	15	M	173	61.3	71	33.5	AAA	53	43	203	772.3	12.6	56.7
Mean ± SD	14.7 ±1.58		170.1 ±8.5	66.2 ±10.4	78.0 ±6.1	52.2 ±11.9		56.7 ±6.2	52.4 ±8.4	197.2 ±5.2	716.1 ±192.1	10.7 ± 1.6	48.4 ±9.6

ND represents the non-diabetic hockey players with corresponding numbers to their T1D counterpart

Figure A1. %VO2max for all study participants combined across all 3 periods of a simulation hockey game. No differentiation between T1D and ND groups.



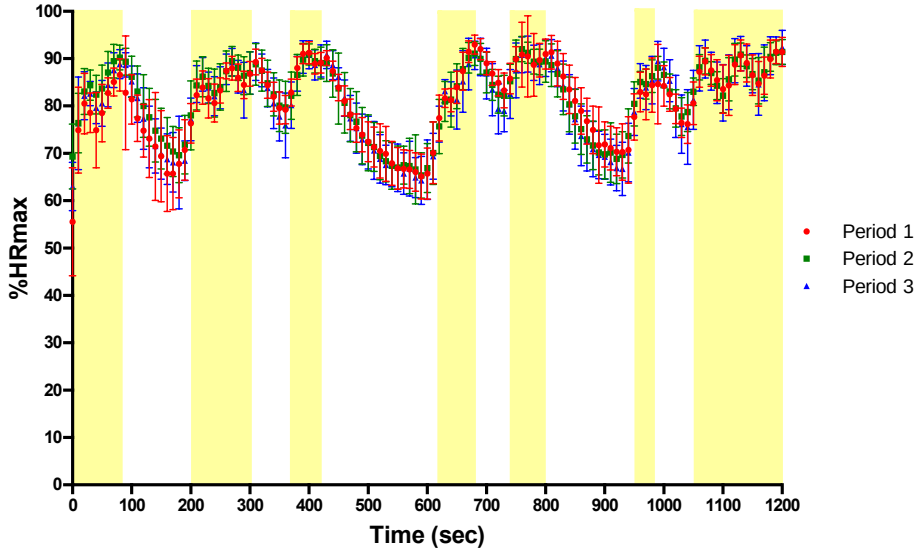
A



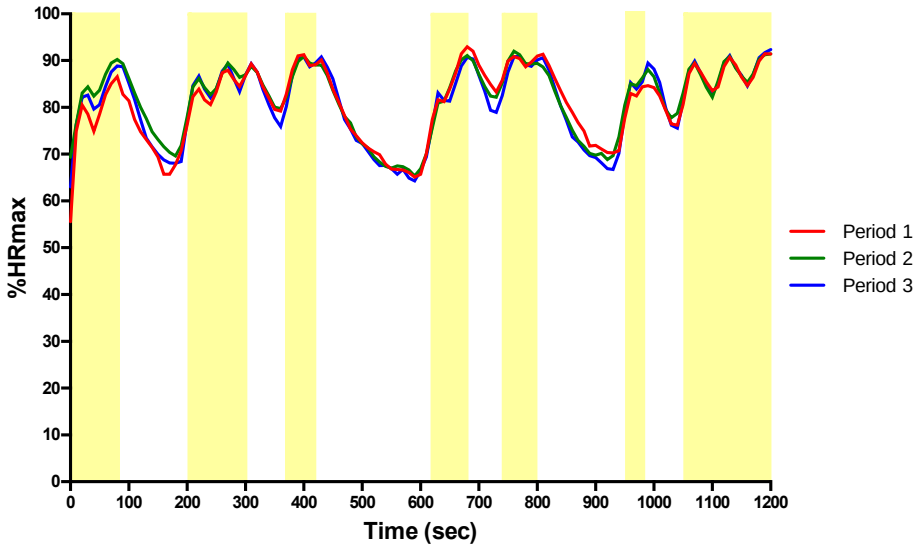
B

The above graphs illustrate the mean VO_2 response expressed as a percentage of $\%VO_2max$ for all study participants during each shift and bench interval between the 3 periods. The shaded areas represent the shift intervals and the white areas represent the bench intervals. The VO_2 response across the 3 periods is consistent, and during the shifts the participants achieved peak VO_2 values ranging between 50-80% of VO_2max. Panel A. Includes the mean $\%VO_2max$ along with the standard errors for all participants (M ± SE).

Figure A2. %HRmax for all study participants with and without T1D across all 3 periods of a simulation hockey game. No differentiation between T1D and ND groups.



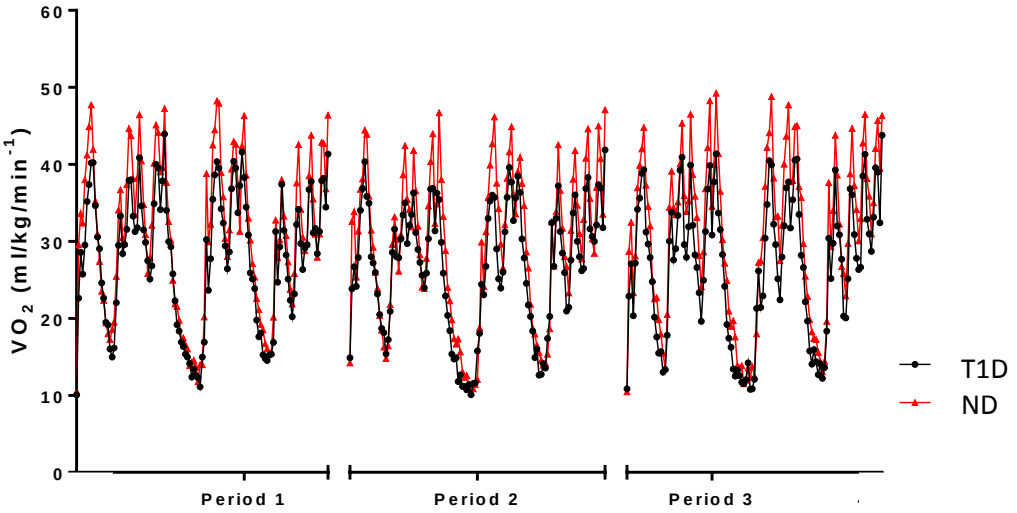
A



B

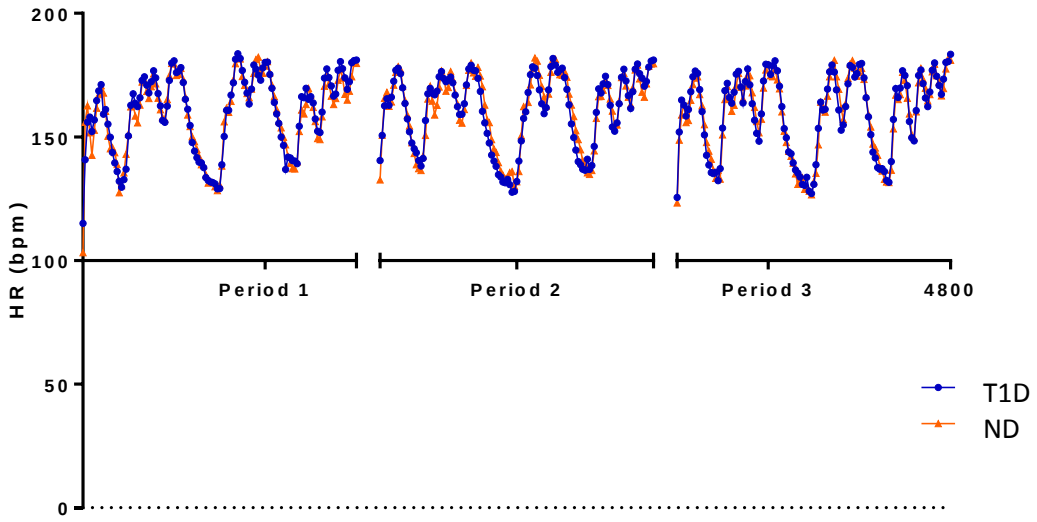
The above graphs illustrate the mean HR response expressed as a percentage of HR for all study participants during each shift and bench interval between the 3 periods. The shaded areas represent the shift intervals and the white areas represent the bench intervals. The HR response across the 3 periods is consistent, and during the shifts the participants achieved peak HR values ranging between 80-90% HRmax. Panel A. Includes the mean %HRmax along with the standard errors for all participants (M ± SE).

Figure A3. The 10-second averaged VO_2 ($ml \cdot Kg^{-1} \cdot min^{-1}$) response across 3 periods of a simulation hockey game comparing individuals with and without T1D



The VO_2 response between the T1D and ND group are similar. It seems that the ND individuals were able to reach greater VO_2 values during the shifts, but these differences were not significant. During the shifts the participants' VO_2 ranged between 30-50 $ml \cdot Kg^{-1} \cdot min^{-1}$.

Figure A4. The 10-second averaged HR (bpm) response across 3 periods of a simulation hockey game comparing individuals with and without T1D



The HR response is paralleled between the T1D and ND groups during all 3 periods. During the shifts the HR for both groups ranged between 150-190 bpm.

Table A3. Comparison of the average nadir and peak oxygen consumption rate expressed relative to VO_2max, in response to a simulation hockey game between individuals with and without T1D (M ± SD).

		Period 1		Period 2		Period 3	
		T1D (*n=7*)	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**
Rest		0.19 ± 0.08	0.18 ± 0.07	0.30 ± 0.14	0.24 ± 0.12	0.20 ± 0.09	0.18 ± 0.05
Shift 1	**High**	0.80 ± 0.13	0.85 ± 0.15	0.76 ± 0.15	0.78 ± 0.14	0.78 ± 0.15	0.77 ± 0.14
	Low	0.41 ± 0.15	0.47 ± 0.11	0.40 ± 0.10	0.42 ± 0.09	0.37 ± 0.10	0.38 ± 0.11
Bench 1	**High**	0.66 ± 0.09	0.62 ± 0.18	0.67 ± 0.07	0.62 ± 0.13	0.60 ± 0.11	0.65 ± 0.13
	Low	0.26 ± 0.09	0.28 ± 0.06	0.27 ± 0.08	0.25 ± 0.08	0.23 ± 0.05	0.23 ± 0.02
Shift 2	**High**	0.77 ± 0.13	0.83 ± 0.13	0.74 ± 0.17	0.76 ± 0.14	0.80 ± 0.15	0.83 ± 0.11
	Low	0.48 ± 0.12	0.48 ± 0.06	0.48 ± 0.15	0.40 ± 0.11	0.45 ± 0.12	0.51 ± 0.15
Bench 2	**High**	0.66 ± 0.12	0.72 ± 0.16	0.62 ± 0.12	0.64 ± 0.16	0.63 ± 0.16	0.68 ± 0.16
	Low	0.45 ± 0.07	0.40 ± 0.07	0.38 ± 0.10	0.38 ± 0.09	0.37 ± 0.08	0.41 ± 0.12
Shift 3	**High**	0.83 ± 0.09	0.87 ± 0.11	0.78 ± 0.18	0.81 ± 0.15	0.82 ± 0.13	0.86 ± 0.08
	Low	0.60 ± 0.09	0.61 ± 0.11	0.47 ± 0.10	0.55 ± 0.11	0.54 ± 0.09	0.59 ± 0.12
Bench 3	**High**	0.65 ± 0.10	0.66 ± 0.16	0.56 ± 0.13	0.66 ± 0.13	0.64 ± 0.17	0.72 ± 0.13
	Low	0.19 ± 0.06	0.18 ± 0.03	0.17 ± 0.06	0.17 ± 0.05	0.18 ± 0.06	0.17 ± 0.04
Shift 4	**High**	0.77 ± 0.16	0.89 ± 0.11	0.74 ± 0.18	0.80 ± 0.14	0.79 ± 0.17	0.84 ± 0.12
	Low	0.44 ± 0.12	0.49 ± 0.07	0.36 ± 0.14	0.40 ± 0.14	0.39 ± 0.13	0.40 ± 0.10
Bench 4	**High**	0.68 ± 0.14	0.69 ± 0.11	0.64 ± 0.15	0.66 ± 0.10	0.65 ± 0.15	0.69 ± 0.16
	Low	0.48 ± 0.11	0.46 ± 0.09	0.41 ± 0.06	0.45 ± 0.06	0.38 ± 0.04	0.46 ± 0.09
Shift 5	**High**	0.81 ± 0.11	0.84 ± 0.09	0.83 ± 0.12	0.79 ± 0.16	0.81 ± 0.12	0.87 ± 0.08
	Low	0.63 ± 0.10	0.54 ± 0.11	0.55 ± 0.11	0.53 ± 0.14	0.57 ± 0.16	0.60 ± 0.16
Bench 5	**High**	0.64 ± 0.10	0.66 ± 0.19	0.59 ± 0.13	0.65 ± 0.19	0.64 ± 0.15	0.66 ± 0.14
	Low	0.23 ± 0.06	0.25 ± 0.05	0.20 ± 0.05	0.22 ± 0.05	0.21 ± 0.07	0.21 ± 0.04
Shift 6	**High**	0.71 ± 0.16	0.67 ± 0.18	0.76 ± 0.14	0.77 ± 0.19	0.74 ± 0.16	0.76 ± 0.14
	Low	0.37 ± 0.15	0.46 ± 0.10	0.50 ± 0.13	0.44 ± 0.08	0.45 ± 0.12	0.49 ± 0.14
Bench 6	**High**	0.61 ± 0.14	0.58 ± 0.06	0.62 ± 0.12	0.65 ± 0.11	0.65 ± 0.17	0.67 ± 0.17
	Low	0.35 ± 0.11	0.36 ± 0.04	0.37 ± 0.11	0.39 ± 0.06	0.35 ± 0.11	0.38 ± 0.07
Shift 7	**High**	0.80 ± 0.11	0.84 ± 0.10	0.81 ± 0.14	0.86 ± 0.08	0.85 ± 0.14	0.87 ± 0.12
	Low	0.47 ± 0.09	0.43 ± 0.07	0.46 ± 0.09	0.46 ± 0.09	0.45 ± 0.13	0.47 ± 0.09

No differences between T1D and ND groups

Table A4. Comparison of the average nadir and peak heart rate expressed relative to HRmax, in response to a simulation hockey game between individuals with and without T1D (M ± SD).

		Period 1		Period 2		Period 3	
		T1D (*n=7*)	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**	**T1D (*n=7*)**	**ND (*n=6*)**
REST		0.59 ± 0.13	0.52 ± 0.09	0.72 ± 0.08	0.67 ± 0.05	0.64 ± 0.06	0.62 ± 0.04
Shift 1	**High**	0.87 ± 0.03	0.87 ± 0.07	0.91 ± 0.04	0.90 ± 0.02	0.91 ± 0.05	0.89 ± 0.04
	Low	0.71 ± 0.06	0.69 ± 0.10	0.76 ± 0.08	0.76 ± 0.03	0.75 ± 0.06	0.73 ± 0.04
Bench 1	**High**	0.86 ± 0.03	0.85 ± 0.08	0.89 ± 0.03	0.90 ± 0.03	0.89 ± 0.03	0.89 ± 0.03
	Low	0.62 ± 0.11	0.63 ± 0.06	0.69 ± 0.07	0.68 ± 0.04	0.64 ± 0.05	0.65 ± 0.05
Shift 2	**High**	0.86 ± 0.09	0.88 ± 0.02	0.91 ± 0.04	0.89 ± 0.02	0.90 ± 0.03	0.89 ± 0.02
	Low	0.72 ± 0.12	0.76 ± 0.04	0.78 ± 0.08	0.76 ± 0.04	0.78 ± 0.05	0.76 ± 0.05
Bench 2	**High**	0.90 ± 0.04	0.89 ± 0.01	0.90 ± 0.04	0.89 ± 0.03	0.90 ± 0.05	0.89 ± 0.02
	Low	0.78 ± 0.03	0.79 ± 0.03	0.78 ± 0.05	0.78 ± 0.03	0.75 ± 0.07	0.76 ± 0.06
Shift 3	**High**	0.92 ± 0.03	0.91 ± 0.01	0.92 ± 0.04	0.91 ± 0.02	0.92 ± 0.04	0.91 ± 0.02
	Low	0.82 ± 0.02	0.83 ± 0.04	0.82 ± 0.06	0.81 ± 0.03	0.81 ± 0.06	0.79 ± 0.04
Bench 3	**High**	0.90 ± 0.02	0.90 ± 0.01	0.88 ± 0.05	0.90 ± 0.02	0.92 ± 0.04	0.90 ± 0.01
	Low	0.63 ± 0.04	0.63 ± 0.06	0.63 ± 0.06	0.65 ± 0.03	0.64 ± 0.06	0.62 ± 0.03
Shift 4	**High**	0.93 ± 0.02	0.88 ± 0.12	0.92 ± 0.03	0.92 ± 0.02	0.91 ± 0.03	0.91 ± 0.03
	Low	0.76 ± 0.05	0.71 ± 0.14	0.75 ± 0.08	0.75 ± 0.04	0.77 ± 0.05	0.74 ± 0.03
Bench 4	**High**	0.92 ± 0.03	0.92 ± 0.02	0.89 ± 0.04	0.91 ± 0.02	0.90 ± 0.05	0.92 ± 0.02
	Low	0.83 ± 0.05	0.84 ± 0.02	0.79 ± 0.05	0.82 ± 0.02	0.76 ± 0.05	0.79 ± 0.06
Shift 5	**High**	0.93 ± 0.03	0.93 ± 0.02	0.93 ± 0.03	0.92 ± 0.02	0.92 ± 0.05	0.92 ± 0.02
	Low	0.83 ± 0.09	0.85 ± 0.03	0.86 ± 0.05	0.84 ± 0.03	0.82 ± 0.06	0.82 ± 0.05
Bench 5	**High**	0.92 ± 0.04	0.92 ± 0.04	0.88 ± 0.05	0.90 ± 0.04	0.91 ± 0.04	0.90 ± 0.03
	Low	0.66 ± 0.10	0.68 ± 0.06	0.67 ± 0.05	0.67 ± 0.04	0.65 ± 0.06	0.66 ± 0.04
Shift 6	**High**	0.87 ± 0.04	0.83 ± 0.06	0.88 ± 0.03	0.87 ± 0.03	0.87 ± 0.05	0.86 ± 0.03
	Low	0.78 ± 0.05	0.77 ± 0.06	0.80 ± 0.05	0.76 ± 0.06	0.80 ± 0.06	0.78 ± 0.03
Bench 6	**High**	0.87 ± 0.03	0.86 ± 0.05	0.89 ± 0.03	0.88 ± 0.03	0.90 ± 0.05	0.89 ± 0.03
	Low	0.75 ± 0.07	0.75 ± 0.04	0.76 ± 0.05	0.77 ± 0.04	0.74 ± 0.10	0.75 ± 0.04
Shift 7	**High**	0.93 ± 0.03	0.91 ± 0.04	0.91 ± 0.06	0.92 ± 0.02	0.93 ± 0.04	0.92 ± 0.02
	Low	0.80 ± 0.05	0.79 ± 0.05	0.82 ± 0.04	0.83 ± 0.04	0.81 ± 0.08	0.81 ± 0.04

No differences between T1D and ND groups

Table A5. Comparison of the combined average nadir and peak oxygen consumption rate, expressed relative to VO_2max, between the 3 periods of a simulation hockey game (M ± SD).

		COMBINED (*n=13*)		
		Period 1	**Period 2**	**Period 3**
Rest		0.19 ± 0.07	0.27 ± 0.13	0.19 ± 0.07
Shift 1	**High**	0.82 ± 0.14	0.77 ± 0.14	0.78 ± 0.14
	Low	0.44 ± 0.13	0.41 ± 0.09‡	0.37 ± 0.10‡
Bench 1	**High**	0.64 ± 0.13	0.65 ± 0.10	0.63 ± 0.12
	Low	0.27 ± 0.07	0.26 ± 0.08	0.23 ± 0.04
Shift 2	**High**	0.80 ± 0.13	0.75 ± 0.15‡	0.81 ± 0.13‡
	Low	0.48 ± 0.09	0.44 ± 0.13	0.48 ± 0.14
Bench 2	**High**	0.69 ± 0.14	0.63 ± 0.13	0.65 ± 0.16
	Low	0.43 ± 0.07	0.38 ± 0.09	0.39 ± 0.10
Shift 3	**High**	0.85 ± 0.10	0.79 ± 0.16	0.84 ± 0.11
	Low	0.60 ± 0.09+	0.51 ± 0.11+‡	0.56 ± 0.11‡
Bench 3	**High**	0.65 ± 0.12	0.61 ± 0.13	0.68 ± 0.15
	Low	0.18 ± 0.04	0.17 ± 0.05	0.18 ± 0.05
Shift 4	**High**	0.83 ± 0.15	0.77 ± 0.16‡	0.82 ± 0.15‡
	Low	0.46 ± 0.10	0.38 ± 0.13	0.40 ± 0.11
Bench 4	**High**	0.69 ± 0.12	0.65 ± 0.13	0.67 ± 0.15
	Low	0.47 ± 0.10	0.42 ± 0.06	0.42 ± 0.08
Shift 5	**High**	0.82 ± 0.10	0.81 ± 0.13	0.84 ± 0.11
	Low	0.59 ± 0.11	0.54 ± 0.12	0.58 ± 0.16
Bench 5	**High**	0.65 ± 0.14	0.62 ± 0.16	0.65 ± 0.14
	Low	0.24 ± 0.21	0.21 ± 0.06	0.05 ± 0.06
Shift 6	**High**	0.69 ± 0.17	0.76 ± 0.16	0.75 ± 0.14
	Low	0.46 ± 0.12	0.48 ± 0.11	0.47 ± 0.12
Bench 6	**High**	0.60 ± 0.11	0.63 ± 0.11	0.66 ± 0.16
	Low	0.36 ± 0.08	0.38 ± 0.09	0.36 ± 0.09
Shift 7	**High**	0.82 ± 0.10	0.83 ± 0.12	0.86 ± 0.13
	Low	0.45 ± 0.08	0.46 ± 0.09	0.46 ± 0.11

+ $p < 0.05$ Significant difference between periods 1 and 2
‡ $p < 0.05$ Significant difference between periods 2 and 3
† $p < 0.05$ Significant difference between periods 1 and 3

Some significant differences were observed in the combined average %VO_2max values during some of the play shift and bench intervals. During play shift 1, the average %VO_2max low was significantly higher in period 2 compared with period 3. During play shift 2, the average %VO_2max high was significantly lower in period 2 compared with period 3. During play shift 3, the average %VO_2max low was significantly greater in period 1 compared to period 3, period 2 was significantly lower than period 3, and no significant differences were observed between periods 1 and 3. Lastly, during play shift 4 the average %VO_2max high was significantly lower in period 2 compared with period 3.

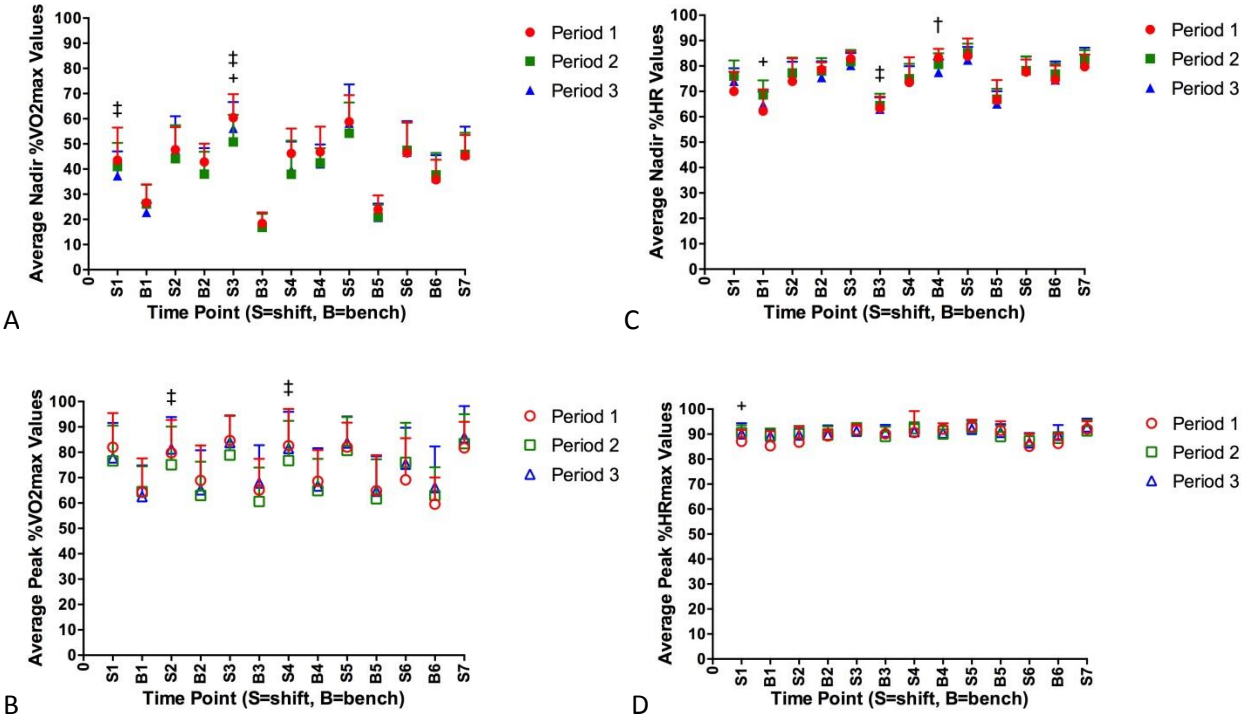
Table A6. Comparison of the combined average nadir and peak heart rate, expressed relative to HRmax, between the 3 periods of a simulation hockey game (M ± SD).

		COMBINED (*n=13*)		
		P1	**P2**	**P3**
REST		0.56 ± 0.11+	0.70 ± 0.07+‡	0.63 ± 0.05‡
Shift 1	**High**	0.87 ± 0.05+	0.91 ± 0.03	0.90 ± 0.04
	Low	0.70 ± 0.08	0.76 ± 0.06	0.74 ± 0.05
Bench 1	**High**	0.85 ± 0.06	0.89 ± 0.03	0.89 ± 0.03
	Low	0.62 ± 0.09	0.69 ± 0.06	0.65 ± 0.05
Shift 2	**High**	0.87 ± 0.06	0.90 ± 0.03	0.90 ± 0.03
	Low	0.74 ± 0.09	0.77 ± 0.06	0.77 ± 0.05
Bench 2	**High**	0.89 ± 0.03	0.90 ± 0.04	0.90 ± 0.04
	Low	0.79 ± 0.03	0.78 ± 0.05	0.75 ± 0.06
Shift 3	**High**	0.92 ± 0.02	0.92 ± 0.03	0.91 ± 0.03
	Low	0.83 ± 0.03	0.82 ± 0.05	0.80 ± 0.05
Bench 3	**High**	0.90 ± 0.02	0.89 ± 0.04	0.91 ± 0.03
	Low	0.63 ± 0.05	0.64 ± 0.05‡	0.63 ± 0.05‡
Shift 4	**High**	0.91 ± 0.09	0.92 ± 0.03	0.91 ± 0.03
	Low	0.74 ± 0.10	0.75 ± 0.06	0.76 ± 0.04
Bench 4	**High**	0.92 ± 0.02	0.90 ± 0.03	0.90 ± 0.04
	Low	0.83 ± 0.04†	0.81 ± 0.04	0.78 ± 0.05†
Shift 5	**High**	0.93 ± 0.03	0.93 ± 0.03	0.92 ± 0.04
	Low	0.84 ± 0.07	0.85 ± 0.04	0.82 ± 0.05
Bench 5	**High**	0.92 ± 0.04	0.89 ± 0.04	0.91 ± 0.03
	Low	0.67 ± 0.08	0.67 ± 0.04	0.65 ± 0.05
Shift 6	**High**	0.85 ± 0.05	0.87 ± 0.03	0.87 ± 0.04
	Low	0.78 ± 0.05	0.78 ± 0.06	0.79 ± 0.05
Bench 6	**High**	0.86 ± 0.04	0.88 ± 0.03	0.90 ± 0.04
	Low	0.75 ± 0.06	0.77 ± 0.04	0.75 ± 0.07
Shift 7	**High**	0.92 ± 0.03	0.91 ± 0.05	0.93 ± 0.03
	Low	0.80 ± 0.05	0.83 ± 0.04	0.81 ± 0.06

+ $p < 0.05$ Significant difference between periods 1 and 2
‡ $p < 0.05$ Significant difference between periods 2 and 3
† $p < 0.05$ Significant difference between periods 1 and 3

Some significant differences were observed in the combined average %HRmax values during some of the play shift and bench intervals. At rest, %HRmax was significantly lower in period 1 compared to period 2, period 2 was significantly greater than period 3, and no differences were observed between period 1 and period 3. During play shift 1, the average %HRmax low was significantly lower in period 1 than period 2. During bench 3, the average %HRmax low was significantly greater in period 2 then period 3. Lastly, during bench 4 the average %HRmax low was significantly greater in period 1 compared with period 3.

Figure A5. The average nadir and peak %VO_2max and %HRmax for all study participants during the 3 period of a simulation hockey game (M ± SE).



+ $p < 0.05$ Significant difference between periods 1 and 2

‡ $p < 0.05$ Significant difference between periods 2 and 3

† $p < 0.05$ Significant difference between periods 1 and 3

Some significant differences were observed during some of the play shift and bench intervals. Panel A. Shows the differences in the average nadir %VO_2max values for all play shift and bench intervals between periods. Panel B. Shows the differences in the average peak %VO_2max values for all play shift and bench intervals between periods. Panel C. Shows the differences in the average nadir %HRmax values for all play shift and bench intervals between periods. Panel D. Shows the differences in the average peak %HRmax values for all play shift and bench intervals between periods.

Table A7. Results of the average nadir and peak %VO_2max and %HRmax data analysis for all study participants: Two-way repeated measures ANOVA

%VO_2max Highs	Type lll Sum of Squares	df	Mean Square	F	Sig.
Rest					
Period	579.214	2	289.607	4.247	.028
Period*Diabetes	31.747	2	15.873	.233	.794
Error(Period)	1500.100	22	68.186		
Diabetes Between-Subject Effect	102.525	1	102.525	.753	.404
S1					
Period	217.336	1.072	202.725	.870	.377
Period*Diabetes	50.158	1.072	46.786	.201	.679
Error(Period)	2746.719	11.793	232.14		
Diabetes Between-Subject Effect	37.306	1	37.306	.103	.754
B1					
Period	20.782	2	10.393	.266	.769
Period*Diabetes	177.716	2	88.858	2.278	.126
Error(Period)	858.337	22	39.015		
Diabetes Between-Subject Effect	17.540	11	17.540	.047	.833
S2					
Period	278.616	2	139.308	3.475	.049
Period*Diabetes	45.804	2	22.902	.571	.573
Error(Period)	882.056	22	40.093		
Diabetes Between-Subject Effect	111.260	1	111.260	.218	.650
B2					
Period	243.013	2	121.506	3.217	.059
Period*Diabetes	43.808	2	21.904	.580	.568
Error(Period)	830.987	22	37.772		
Diabetes Between-Subject Effect	178.550	1	178.550	.312	.588
S3					
Period	250.528	2	125.264	1.987	.161
Period*Diabetes	3.679	2	1.839	.029	.971
Error(Period)	1386.681	22	63.031		
Diabetes Between-Subject	132.005	1	132.005	.321	.555

Effect					
B3					
Period	346.140	2	173.070	3.549	.046
Period*Diabetes	131.203	2	65.601	1.345	.281
Error(Period)	1072.744	22	48.761		
Diabetes Between-Subject Effect	374.387	1	374.387	.810	.387
S4					
Period	264.138	1.239	213.154	2.211	.158
Period*Diabetes	65.490	2	32.745	.548	.508
Error(Period)	1314.296	13.631	96.419		
Diabetes Between-Subject Effect	581.906	1	581.906	1.062	.325
B4					
Period	85.915	2	42.958	1.359	.278
Period*Diabetes	5.719	2	2.859	.090	.914
Error(Period)	695.641	22	31.620		
Diabetes Between-Subject Effect	55.939	1	55.939	.109	.747
S5					
Period	69.634	2	34.817	.684	.515
Period*Diabetes	152.480	2	76.240	1.498	.245
Error(Period)	1119.463	22	50.885		
Diabetes Between-Subject Effect	25.158	1	25.158	.086	.775
B5					
Period	73.890	2	36.945	.471	.630
Period*Diabetes	25.230	2	12.615	.161	.852
Error(Period)	1725.470	22	78.431		
Diabetes Between-Subject Effect	126.167	1	126.167	.245	.630
S6					
Period	389.281	2	194.640	1.961	.165
Period*Diabetes	59.908	2	29.954	.302	.742
Error(Period)	2183.545	22	99.252		
Diabetes Between-Subject Effect	.782	1	.782	.001	.971
B6					
Period	291.690	2	145.845	2.237	.131
Period*Diabetes	69.108	2	34.554	.530	.596

Error(Period)	1434.437	22	65.202		
Diabetes Between-Subject Effect	7.240	1	7.240	.018	.896
S7					
Period	100.619	2	50.309	1.017	.378
Period*Diabetes	17.243	2	8.622	.174	.841
Error(Period)	1088.730	22	49.488		
Diabetes Between-Subject Effect	146.521	1	146.521	.459	.512

%VO_2max Lows	Type lll Sum of Squares	df	Mean Square	F	Sig.
S1					
Period	265.294	1.219	132.647	3.308	.085
Period*Diabetes	30.11	1.219	24.626	.374	.692
Error(Period)	882.166	13.405	65.808		
Diabetes Between-Subject Effect	87.185	1	87.185	.299	.596
B1					
Period	112.733	2	56.366	2.595	.097
Period*Diabetes	14.961	2	7.480	.344	.712
Error(Period)	477.952	22	21.725		
Diabetes Between-Subject Effect	.115	1	.115	.001	.973
S2					
Period	132.388	2	66.194	1.115	.346
Period*Diabetes	267.557	2	133.778	2.253	.129
Error(Period)	1306.218	22	59.374		
Diabetes Between-Subject Effect	2.940	1	2.940	.009	.927
B2					
Period	156.986	2	78.493	2.755	.086
Period*Diabetes	161.833	2	80.916	2.840	.080
Error(Period)	626.830	22	28.492		
Diabetes Between-Subject Effect	1.908	1	1.908	.011	.919
S3					
Period	5757.152	1.350	287.576	14.076	.001
Period*Diabetes	77.732	1.350	57.570	1.902	.189
Error(Period)	449.475	14.852	30.263		

Diabetes Between-Subject Effect	186.157	1	186.157	.675	.429
B3					
Period	14.550	1.169	12.448	.557	.496
Period*Diabetes	5.709	1.169	4.884	.218	.686
Error(Period)	287.526	12.858	22.362		
Diabetes Between-Subject Effect	1.670	11	52.332	.032	.861
S4					
Period	497.907	2	248.954	3.783	.039
Period*Diabetes	33.846	2	16.923	.257	.776
Error(Period)	1447.883	22	65.813		
Diabetes Between-Subject Effect	111.521	1	111.521	.377	.552
B4					
Period	160.548	2	80.274	3.794	.038
Period*Diabetes	158.564	2	79.282	3.747	.040
Error(Period)	465.536	22	21.161		
Diabetes Between-Subject Effect	112.095	1	112.095	.770	.399
S5					
Period	154.798	2	77.399	1.534	.238
Period*Diabetes	215.386	2	107.693	2.134	.142
Error(Period)	1110.168	22	50.462		
Diabetes Between-Subject Effect	58.027	1	58.027	.138	.717
B5					
Period	84.662	2	42.332	2.767	.085
Period*Diabetes	14.561	2	7.280	.476	.628
Error(Period)	336.599	22	15.300		
Diabetes Between-Subject Effect	11.694	1	11.694	.194	.668
S6					
Period	4.573	2	2.286	.034	.966
Period*Diabetes	185.611	2	92.805	1.394	269
Error(Period)	1464.463	22	66.566		
Diabetes Between-Subject Effect	3.398	1	3.398	.011	.919
B6					

Period	27.337	2	13.668	.357	.704
Period*Diabetes	5.831	2	2.916	.076	.927
Error(Period)	842.054	22	38.275		
Diabetes Between-Subject Effect	41.365	1	41.365	.254	.624
S7					
Period	4.780	2	2.390	.103	.903
Period*Diabetes	55.712	2	27.856	1.198	.321
Error(Period)	511.608	22	23.255		
Diabetes Between-Subject Effect	3.927	1	3.927	.016	.901

%HRmax Highs	Type lll Sum of Squares	df	Mean Square	F	Sig.
Rest					
Period	1278.130	1.308	976.903	16.221	.001
Period*Diabetes	44.899	1.308	34.318	.570	.507
Error(Period)	866.723	14.392	60.223		
Diabetes Between-Subject Effect	165.842	1	165.842	1.316	.276
S1					
Period	88.943	2	44.471	5.023	.016
Period*Diabetes	13.660	2	6.830	.771	.474
Error(Period)	194.776	22	8.853		
Diabetes Between-Subject Effect	8.314	1	8.314	.244	.631
B1					
Period	138.718	1.286	107.851	5.362	.029
Period*Diabetes	3.954	1.286	3.074	.153	.764
Error(Period)	284.586	14.148	20.115		
Diabetes Between-Subject Effect	.331	1	.331	.011	.918
S2					
Period	78.558	1.080	72.757	2.674	.127
Period*Diabetes	32.302	1.080	29.917	1.099	.321
Error(Period)	323.195	11.877	27.212		
Diabetes Between-Subject Effect	.242	1	.242	.008	.930
B2					
Period	1.520	2	.760	.121	.886

Period*Diabetes	.940	2	.470	.075	.928
Error(Period)	137.922	22	6.269		
Diabetes Between-Subject Effect	10.913	1	10.913	.456	.514
S3					
Period	2.836	2	1.418	.429	.656
Period*Diabetes	.704	2	.352	.107	.899
Error(Period)	72.659	22	3.303		
Diabetes Between-Subject Effect	9.478	1	9.478	.546	.514
B3					
Period	15.791	2	7.895	1.384	.272
Period*Diabetes	26.444	22	13.222	2.318	.122
Error(Period)	125.492	2	5.704		
Diabetes Between-Subject Effect	.669	1	.669	.045	.836
S4					
Period	17.873	1.055	16.943	.323	.592
Period*Diabetes	63.027	1.055	59.749	1.140	.311
Error(Period)	608.256	11.604	52.420		
Diabetes Between-Subject Effect	45.500	1	45.500	1.313	.276
B4					
Period	27.250	1.349	20.198	1.416	.264
Period*Diabetes	16.955	1.349	12.567	.881	.395
Error(Period)	211.707	14.841	14.265		
Diabetes Between-Subject Effect	17.727	1	17.727	1.565	.237
S5					
Period	6.981	2	3.491	1.066	.361
Period*Diabetes	3.576	2	1.788	.546	.587
Error(Period)	72.026	22	3.274		
Diabetes Between-Subject Effect	9.221	1	9.221	.387	.546
B5					
Period	40.675	2	20.337	1.489	.248
Period*Diabetes	13.856	2	6.928	.507	.609
Error(Period)	300.525	22	13.660		
Diabetes Between-Subject	2.442E-005	1	2.442E-005	.000	.999

Effect					
S6					
Period	36.018	2	18.009	2.413	.113
Period*Diabetes	13.919	22	6.960	.932	.409
Error(Period)	164.197	2	7.464		
Diabetes Between-Subject Effect	35.783	1	35.783	1.047	.328
B6					
Period	71.756	2	35.878	3.369	.053
Period*Diabetes	.925	2	.463	.043	.958
Error(Period)	234.276	22	10.649		
Diabetes Between-Subject Effect	9.146	1	9.146	.420	.530
S7					
Period	15.860	2	7.930	.857	.438
Period*Diabetes	10.822	2	5.411	.585	.566
Error(Period)	203.535	22	9.252		
Diabetes Between-Subject Effect	10.621	1	10.621	.440	.521

%HRmax Lows	Type lll Sum of Squares	df	Mean Square	F	Sig.
S1					
Period	247.048	2	123.524	4.614	.021
Period*Diabetes	7.296	2	3.648	.136	.873
Error(Period)	588.995	22	26.772		
Diabetes Between-Subject Effect	14.441	1	14.441	.180	.679
B1					
Period	266.600	1.325	201.202	5.218	.030
Period*Diabetes	5.547	1.325	4.186	.109	.815
Error(Period)	562.024	14.575	38.560		
Diabetes Between-Subject Effect	.744	1	.744	.008	.930
S2					
Period	78.526	1.126	69.765	.983	.352
Period*Diabetes	66.645	1.126	59.210	.834	.392
Error(Period)	879.000	12.381	70.994		
Diabetes Between-Subject Effect	1.395	1	1.395	.021	.887

B2					
Period	69.418	2	34.709	2.968	.072
Period*Diabetes	.460	2	.230	.020	.981
Error(Period)	257.284	20.498	12.552		
Diabetes Between-Subject Effect	2.773	1	2.773	.048	.831
S3					
Period	49.126	2	24.563	3.080	.066
Period*Diabetes	11.012	2	5.506	.690	.512
Error(Period)	175.432	22	7.974		
Diabetes Between-Subject Effect	1.011	1	1.011	.024	.879
B3					
Period	14.839	1.051	14.126	.564	.476
Period*Diabetes	14.675	1.051	13.970	.558	.478
Error(Period)	289.387	11.556	25.043		
Diabetes Between-Subject Effect	.000	1	.000	.000	.994
S4					
Period	29.645	2	14.823	.395	.678
Period*Diabetes	36.401	2	18.201	.485	.622
Error(Period)	825.559	22	37.525		
Diabetes Between-Subject Effect	73.256	1	73.256	.905	.362
B4					
Period	207.945	2	103.973	9.847	.001
Period*Diabetes	13.806	2	6.903	.654	.530
Error(Period)	232.297	22	10.559		
Diabetes Between-Subject Effect	48.206	1	48.206	1.335	.272
S5					
Period	45.477	1.344	33.827	1.096	.334
Period*Diabetes	27.132	1.344	20.181	.654	.476
Error(Period)	456.507	14.788	30.869		
Diabetes Between-Subject Effect	.162	1	.162	.003	.957
B5					
Period	24.297	2	12.148	.491	.619
Period*Diabetes	12.120	2	6.060	.245	.785
Error(Period)	544.809	22	24.764		

Diabetes Between-Subject Effect	7.740	1	7.740	.126	.729
S6					
Period	6.949	2	3.475	.274	.763
Period*Diabetes	17.936	2	8.969	.707	.504
Error(Period)	279.188	22	12.690		
Diabetes Between-Subject Effect	77.590	1	77.590	1.432	.257
B6					
Period	46.074	2	23.037	.702	.506
Period*Diabetes	2.160	2	1.080	.033	.968
Error(Period)	722.244	22	32.829		
Diabetes Between-Subject Effect	2.684	1	2.684	.060	.811
S7		2			
Period	53.277	2	1.212	1.212	.317
Period*Diabetes	9.902	22	.225	.225	.800
Error(Period)	483.353	1	.010		
Diabetes Between-Subject Effect	.372			.010	.920