Physiological and Performance Responses of Mild Dehydration in Ice Hockey Goaltenders During an On-Ice Scrimmage and Drills.

by

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ABSTRACT

PHYSIOLOGICAL AND PERFORMANCE RESPONSES OF MILD DEHYDRATION IN ICE HOCKEY GOALTENDERS DURING AN ON-ICE SCRIMMAGE AND DRILLS.

Devin G McCarthy
University of Guelph, 2018

Advisor: Dr. Lawrence L Spriet

This study tested the physiological, thermoregulatory, fatigue and performance responses to mild dehydration (DEH) in ice hockey goaltenders during an on-ice scrimmage, a shootout and two drills. Goaltenders drank no fluid (NF) to induce mild DEH (2.4 ± 0.3% body mass loss) or maintained hydration (<0.5% loss) with water (WAT) or a carbohydrate-electrolyte solution (CES). Mild DEH, compared to WAT and CES, increased on-ice mean and peak core temperature (NF: 39.1 ± 0.1°C, WAT: 38.6 ± 0.1°C, CES: 38.5 ± 0.1°C), heart rate and perceived fatigue and lowered scrimmage save percentage and reaction time. CES was superior to WAT for increasing peak lateral movement power and reducing on-ice perceived exertion. Central and peripheral fatigue, shootout performance and rebound control were similar between conditions. Overall, mild DEH impaired thermoregulation, performance and perceived fatigue during an on-ice scrimmage, shootout and drills compared to ingestion of either fluid.
DEDICATION

This thesis is dedicated to my grandparents for their altruistic, endless support.
ACKNOWLEDGEMENTS

First, I would like to thank my advisor Dr. Lawrence Spriet for the many incredible opportunities he has given me during my time in his lab. It truly has been an unforgettable experience and I will forever be grateful that you gambled on me as a fourth year undergraduate student with average grades because you thought I was keen. Dr. Spriet’s passion for science and ability to play as much hockey has he wants have inspired me to continue in academia research. Next, I would like to thank Dr. Jamie Burr for his contribution to my project and professional development.

To my lab mates and colleagues, both in ANNU 205 and not, thank you. I was incredibly fortunate to be surrounded by intelligent people who are so enthused about science. I would especially like to acknowledge all of the people who helped me collect and analyze my data, this process required many people to execute and it would have not been possible without their help. On that note, thank you to the goaltenders and skaters for all their hard work while participating in my study.

Last but not least, thank you to my family and friends who have been there along the way. To my parents and grandparents for being large supporters of my education; this thesis would not be possible without all time you dedicated to me. Finally, to Samantha, my vocabulary does not contain the words necessary to properly express my gratitude.
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<td>Save percentage</td>
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<td>AVI</td>
<td>Anticipatory visual information</td>
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<td>msec</td>
<td>Millisecond</td>
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CHAPTER I: REVIEW OF THE LITERATURE

The purpose of this literature review is to examine ice-hockey goaltenders by describing the unique requirements of this position and by demonstrating the stresses they are exposed to, which potentially impair athletic performance. An assessment of the current sweat loss and hydration data for all ice-hockey athletes is provided, as well as a comparison between positions. The review then analyzes how dehydration, as opposed to staying hydrated, affects physiological and central nervous system functions and how these influence fatigue, cognition and performance in sport. In the context of this review, mild dehydration is 1.5-2.0% BM loss and this magnitude has shown to decrease performance in stop-and-go sport (Nuccio et al. 2017). Also, this level of dehydration is mild and not clinically dangerous. Finally, the effects of carbohydrate ingestion during exercise will be reviewed.

1.1 The game of ice hockey and the positions

Ice-hockey is a sport where two teams skate on an ice surface with the purpose of winning games by scoring more goals than the other team within an established time frame. Typically, one team consists of 20 athletes, 12 of which are forwards, 6 are defensemen and 2 are goaltenders. During normal situations, each team plays 5 skaters and a goaltender at once. A typical game consists of three, 20 min periods that are separated by ~15 min intermissions. Play can extend beyond 60 min to determine a winner if the score is tied at the end of the game. Depending on the league and situation this can be sudden victory overtime played with a full ice roster until one team scores (again in 20 min periods) or a time restricted overtime (e.g. 5 min) with 3-4 skaters aside followed by a shootout if no goals are scored.
**Skaters.** Skaters are considered forwards and defensemen and these athletes play short duration, high intensity shifts. In a shift, 3 forwards and 2 defensemen from both teams skate around the entire ice surface trying to score and prevent goals. In a collegiate level ice hockey game shifts were 30-85 sec long and were separated by 2-5 min of rest and skaters played anywhere between 20-25 min per game depending on their position (Green et al. 1976). Due to the “shift nature” of ice hockey it is categorized as a stop-and-go or intermittent sport.

The physiological demand of ice hockey skaters is most commonly estimated by heart rate (HR) but a few studies have measured skeletal muscle (SM) glycogen use. Typical 5-on-5 play in a collegiate ice-hockey game elicited a mean HR during shifts of 174 beats per minute (bpm) or 87-92% HR max (Green et al. 1976). A more recent study on a similar population found comparable mean shift HR (forwards = 174 ± 6 bpm, defense = 168 ± 3 bpm) and that peak shift HR was near max (Jackson et al. 2017). Furthermore, mean shift HR was ~92% HR max in both 4-on-4 and 3-on-3 games (Lachaume and Lemoyne 2017). Also indicative of physiological and metabolic demand is SM glycogen utilization (van Loon et al. 2001). In one game, collegiate and professional ice hockey skaters used ~60% and ~55% of their SM glycogen stores respectively (Green et al. 1978). Thus, ice hockey is a physically demanding sport inducing significant cardiovascular and metabolic demand in skaters.

**Goaltenders.** A goaltender’s primary objective is to prevent pucks from entering the goal net, which they accomplish by blocking pucks shot at the net with their body or stick. Thus, the equipment a goaltender wears is larger, bulkier and has more protective padding compared to skaters. Performance is commonly measured as save percentage (SVP), the percentage of shots saved over shot attempts, and professional goaltenders save >90% of shots. Despite a seemingly high SVP, making saves is challenging when considering that pucks are shot at velocities up to
160 kilometers per hour (Ryan 2005). Making saves becomes more difficult than simply stopping a fast moving object because of the following: (1) there are 10 active skaters on the ice to mentally process at once; (2) pucks can be shot with or without a preceding deke move; (3) the puck can be shot immediately after the opposition player receives a pass, reducing the time the goaltender has to react and move; (4) shots directed at the goal can be deflected by a stick causing a late change in the initial puck projection; and (5) shots can come when vision is partially or fully occluded (Panchuk and Vickers 2009, Panchuk et al. 2017). All these factors can occur alone or in combination and the more factors involved the more difficult the save.

As a technique to improve SVP, ice hockey goaltenders used anticipatory visual information (AVI) (Bard and Fleury 1981, Panchuk and Vickers 2006). Goaltenders took 45-60 and ~20 milliseconds (msec) to react when pucks were shot from 10 and 5 meters (m) away (Panchuk and Vickers 2006, 2009). In comparison, it took ≥100 msec to visually process and react accordingly in tasks without AVI (Carlton 1992). Therefore, they initiated a save movement faster than the time required to visually process information. Furthermore, SVP was similar on shot attempts when the lower half of the shooter’s stick and the puck were visually occluded to when the entire shooter, stick and puck were visually occluded and both SVPs were significantly lower than all other occlusion combinations (Panchuk and Vickers 2006). Thus, goaltenders used AVI from the lower half of the shooter’s stick and the puck.

SVP is also strongly affected by quiet eye (QE), defined as the amount of time spent visually fixated on the puck prior to a shot. A longer QE duration was observed on shots saved vs. scored in both direct (Panchuk & Vickers 2006) and deflected attempts (Panchuk et al. 2017). Also, QE duration was longer in elite vs. non-elite ice hockey goaltenders when pucks were shot at them (Bard and Fleury 1981). QE was also effectively used to improve performance by soccer
goaltenders in penalty shootouts (Piras and Vickers 2011), basketball players for free throws (Harle and Vickers 2001), billiards players (Williams et al. 2002) and by golfers for putting (Vine et al. 2011). Thus, a longer QE duration prior to a challenging motor task improved performance on the task and making saves for ice hockey goaltenders is no exception.

Another important factor in making saves is positioning. Goaltenders navigate a 1.2 x 1.8 m portion of the ice called a goal crease and move in reaction to where the puck is going even before the puck is shot toward the net. Goaltenders primarily stay in their goal crease but will sometimes skate behind the net to stop and play the puck (Figure 1). In addition, sometimes goaltenders will play outside the goal crease to increase the amount of net they are blocking. When in the goal crease, an ideal movement ends with the goaltender positioned on a line between the center of the net and the puck. This maximizes the amount of net they are blocking and therefore the chance the puck will hit them. Goaltenders are on-ice for the entire game but are not always involved in play (e.g. when puck is in the other end). This means their activity patterns are intermittent like skaters; however, the movements they perform are very different.

Figure 1. Image of an ice hockey rink. Goaltenders are active in the goal crease (blue area) and behind the goal net (yellow area).

There has been very little research to determine the physiological demands in ice hockey goaltenders. In an early study, one goaltender was assessed throughout a game of ice hockey and
it was reported that blood [lactate] was ~15-30% that of the skater positions (Green et al. 1976). Unfortunately, blood [lactate] is challenging to contextualize without a quantified external workload and knowing when work was terminated relative to the blood draw. Similarly, one hydration study reported that goaltenders sweat less than skaters over three junior hockey games, but the goaltender’s team easily won all the games (Logan-Sprenger et al. 2011), suggesting a small external workload on the goaltenders in those games. The challenges associated with quantifying and experimentally controlling for goaltender workload in games limits the current research but may be overcome by studying goaltenders in practices where workload can be experimentally manipulated and more easily quantified.

Overall, ice hockey skater and goaltender positions are not similar. The many differences explain why research focuses on one group or the other. Some of the differences include: portion of ice they move on, movement type, equipment, primary objectives and potentially the physiological demand. The research on how ice hockey goaltenders made saves is thorough, but more research is needed to accurately define the physiological demand of this position.

1.2 Sweat loss, hydration and thermoregulation in ice hockey athletes

There have been many published field studies quantifying sweat loss and dehydration (DEH) in ice-hockey athletes but only three of these separated goaltender and skater results (Godek et al. 2006, Palmer and Spriet 2008, Batchelder et al. 2010, Palmer et al. 2010, Logan-Sprenger et al. 2011, Ozolina et al. 2014). In the current review, a larger sample size was created by combining unpublished practice results from 7 junior, 7 professional and 3 varsity ice hockey teams. These data, in combination with the published studies, were used to quantify whether athletes arrived at the rink hydrated or hypohydrated (HYPO), the volume of sweat lost during exercise and the
incidence of on-ice DEH. The primary purpose of this section is to document sweat losses, DEH and thermoregulatory responses in ice hockey skaters and goaltenders during practice and games. A secondary purpose is to compare aspects of hydration between goaltenders and skaters. In this review DEH will be considered the process of losing body water during acute exercise or heat stress, which is estimated by difference in body mass (BM) pre and post and HYPO is the state of being in a body water deficit that did not occur from acute exercise or heat stress.

It should also be noted that genetics and training status are strong factors in sweat rate and DEH risk, however these will not be further mentioned because genetics cannot be controlled for and professional athletes must be highly trained. In ice hockey, the risk of DEH is reduced because opportunities to drink are plentiful. Frequent stoppages in play and the short shifts played by skaters allow for fluid consumption. Goaltenders can keep fluid on the goal net for easy drink accessibility during stoppages in play. The ample opportunities to drink give athletes the option to replace their high sweat rates with fluid, therefore ice hockey was considered to be medium risk for DEH when considering the major sports (Nuccio et al. 2017).

**Sweat loss and hydration.** Sweat and hydration data in the literature are more abundant in skaters than goaltenders. This can be attributed to teams only carrying 2-4 goalies and ~20 skaters. The main points in this subsection are that both goaltenders and skaters in practices and games arrived at the arena HYPO, lost large amounts of sweat, and became mildly DEH.

Pre-practice hydration status was assessed using urine specific gravity (USG) when participants arrived at the arena. The literature contained USG data for six junior goaltenders obtained ~30 min pre-skate and all were determined HYPO (Palmer and Spriet 2008, Palmer et al. 2010). Data from 7 junior hockey teams showed that on average goaltenders (n = 27) arrived at the arena before practices HYPO and more than half produced a USG that indicated HYPO (Table
1). Similar trends were observed in professional goaltenders (n = 16), but varsity level goaltenders (n = 10) were, on average, hydrated upon arrival and only 33% were HYPO. Contrastingly, when USG was obtained 2-2.5 hours pre-game, junior goalies were adequately hydrated (USG = 1.015 ± 0.002) (Logan-Sprenger et al. 2011). Overall, the majority of goaltenders arrived at practice HYPO, but more research is required to determine a solid trend before a game.
Table 1. Sweat and hydration data from male ice-hockey skaters (S) and goaltenders (G) during intense practices at various levels.

<table>
<thead>
<tr>
<th>Duration (min)</th>
<th>Pre-practice</th>
<th>BM loss (%)</th>
<th>Sweat loss</th>
<th>Sweat Na⁺ Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>USG</td>
<td>USG≥1.020</td>
<td>BM (kg)</td>
<td>(L)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Junior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G (N=27)</td>
<td>64 ± 17</td>
<td>1.021 ± 0.008*</td>
<td>63%</td>
<td>1.22 ± 1.4*</td>
</tr>
<tr>
<td>S (N=261)</td>
<td>65 ± 7</td>
<td>1.017 ± 0.008</td>
<td>46%</td>
<td>0.90 ± 0.66</td>
</tr>
<tr>
<td>Professional</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G (N=16)</td>
<td>70 ± 15</td>
<td>1.019 ± 0.007</td>
<td>55%</td>
<td>2.22 ± 0.37*</td>
</tr>
<tr>
<td>S (N=168)</td>
<td>68 ± 15</td>
<td>1.020 ± 0.007</td>
<td>58%</td>
<td>1.44 ± 0.81</td>
</tr>
<tr>
<td>Varsity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G (N=10)</td>
<td>68 ± 15</td>
<td>1.015 ± 0.008</td>
<td>33%</td>
<td>0.82 ± 0.75</td>
</tr>
<tr>
<td>S (N=69)</td>
<td>74 ± 13</td>
<td>1.020 ± 0.007</td>
<td>66%</td>
<td>0.47 ± 0.69</td>
</tr>
</tbody>
</table>

Notes: Values are mean ± SD. WB sweat Na⁺ loss estimated as per Baker et al. (2009). FH, forehead; WB, whole body; BM, body mass; USG, urine specific gravity. *, significantly different between G and S within a level.
The USG measurement is most effective when obtained immediately pre-exercise. However, due to ice hockey skaters having to dress in protective equipment, USG was not obtained at this time. This gave athletes a chance to consume fluid between the USG collection and when the skate began. Thus, if one arrived HYPO there is an opportunity to rehydrate before the skate. When 600 mL of fluid was consumed in 15 min, USG went from HYPO (USG=1.022 ± 0.004) to adequately hydrated 30 (USG=1.013 ± 0.003) and 45 min (USG=1.010 ± 0.002) after beginning to drink (Logan-Sprenger and Spriet 2013). Therefore, hydration state upon arrival would not matter if the athlete consumed 600 mL 30-45 min before a game or practice.

The occurrence of DEH in ice-hockey games and practices is common for skaters and goaltenders because of large sweat losses. In a 95 min practice (arena temperature (T_a) = 11.4 ± 0.8°C, relative humidity (RH) = 52 ± 3%) skaters sweat ~1.5 L/hr and goaltenders ~1.9 L/hr (Palmer et al. 2010). During an intense 1 hr practice in elite junior hockey players (T_a~14°C, RH~67%), mean sweat loss was 1.8 ± 0.1 L/hr in skaters and significantly greater at 2.9 ± 0.2 L/hr in goaltenders (Palmer and Spriet 2008). The unpublished data also agrees with the literature, showing that in practices junior and professional goaltenders sweat at a greater rate vs. skaters. Interestingly, this trend was not found in varsity level athletes but could be because the varsity team used three goaltenders in their practices. Therefore, each goaltender had a smaller workload and more drinking opportunities. Altogether, the data demonstrated that both goaltenders and skaters produced significant amounts of sweat during practices, but goaltenders lost more.

Unfortunately sweat and hydration data during ice hockey games is scarce. High sweat losses occurred in a professional ice hockey game (3.7 ± 0.9 L) but goaltenders were not recorded (Godek et al. 2006). During three junior games goaltenders produced ~2 L of sweat, which was significantly lower than the 3.2 ± 0.2 L produced by skaters (Logan-Sprenger et al. 2011).
However, these goaltenders were not very active because their team easily won all the games. Therefore, the physical demand for goaltenders must be considered when reporting game sweat rates. It is evident that goaltenders produced more sweat than skaters when they were consistently involved – i.e. practice situations, but there is not enough evidence to make this claim for games.

Ice hockey athletes consistently allowed themselves to mildly DEH during practices and games despite many opportunities to drink. During a 90-min practice, professional ice hockey skaters lost $1.6 \pm 0.8\%$ BM and amateur $0.9 \pm 0.5\%$ BM (Ozolina et al. 2014). In two different practices, junior hockey skaters only lost $\sim 0.8\%$ BM (Palmer and Spriet 2008, Palmer et al. 2010) and collegiate skaters lost $\sim 1.1\%$ BM (Batchelder et al. 2010). The previously mentioned unpublished results also showed that during practice junior and professional goaltenders lost $\sim 1.8$ and $\sim 2.2\%$ BM respectively, which was significantly more than skaters (Table 1). Therefore, ice hockey goaltenders and skaters produced a considerable amount of sweat that was not entirely replaced during practices. Also, goaltenders lost more sweat and BM than skaters during practices.

In a pre-season game, professional skaters lost $1.5 \pm 0.7\%$ BM (Godek et al. 2006). In another game, 66% of junior goaltenders and skaters lost $\geq 1.8\%$ BM (Logan-Sprenger et al. 2011). The small data set from games suggested that skaters dehydrated notably more in games than practices, but more goaltender data is required to make this assessment. If hydration habits were similar in practices to games and goaltenders do indeed produce sweat like in practice, then they would be at risk for mild DEH. Furthermore, if the junior goaltenders did not drink any fluid during the game, they would have lost $\sim 2.2\%$ BM (Logan-Sprenger et al. 2011) and therefore, goaltenders are also at risk for DEH during games.

Altogether, these results showed that, 1) most goaltenders and skaters arrived at practice HYPO, 2) both skaters and goaltenders lost large amounts of sweat during games and practices, 3)
skaters sweat more and lost more BM during games than practices, but this was inconclusive in goaltenders and, 4) goaltenders sweat more and lost more BM than skaters during practice.

**Thermoregulation.** In the three studies reporting core body temperature (T_c) in ice hockey none have included goaltender results. During a 110 min collegiate level practice (T_a = 6.0 ± 1.7°C), core temperature (T_c) increased ~1°C from baseline to plateau at ~38.4°C 40 min into the practice (Batchelder et al. 2010). In a scrimmage (T_a = 3.0 ± 0.1°C, RH = 44 ± 2%), participants consumed either no fluid to induce DEH (1.9 ± 0.1% BM loss) or a sport drink in a volume to match sweat loss (Linseman et al. 2014). T_c peaked at 38.6 ± 0.1°C during the sport drink condition and 38.9 ± 0.1°C when DEH occurred (Figure 2). In a similar experimental design using female subjects, peak T_c was reduced ~0.3°C in the final 25 min of a 90 min scrimmage when hydration was maintained with either a sport drink (T_c = 38.5 ± 0.1°C) or water (T_c = 38.6 ± 0.1°C) compared to losing 1.2 ± 0.1% BM (T_c = 38.8 ± 0.1°C loss) (Boville 2015). Thus, DEH impaired thermoregulation in ice hockey skaters.

![Figure 2](image.png)

**Figure 2.** Core temperature in an ice hockey scrimmage when skaters drank no fluid (NF, 1.9 ± 0.1% body mass (BM) loss) a carbohydrate-electrolyte solution (CES) to maintain BM (Linseman et al. 2014). *, significant difference between conditions. Values are mean ± SE (n = 14).
In addition to DEH, thermoregulation is also affected by the $T_a$ and the micro-environment temperature under ice hockey protective equipment (skin temperature ($T_{sk}$)) must be considered. During a simulated ice hockey game, participants wore full equipment or standard gym shorts and a t-shirt, which resulted in ~2.6 and ~1.2% BM loss respectively (Noonan et al. 2007). In the equipment compared to no equipment condition, BM loss was 2-fold greater, a smaller percent of sweat evaporated, $T_{sk}$ was ~5°C higher ($34.1 \pm 0.2°C$ vs. $28.9 \pm 0.3°C$), and $T_c$ was increased ~0.26°C in the second half of the simulated game. Thus, the equipment worn by skaters impaired the ability to evaporate sweat and therefore dissipate heat, resulting in greater $T_c$. This study was conducted using skater equipment (Noonan et al. 2007), which is typically smaller and lighter than goaltender equipment. Although untested, the larger equipment used by goaltenders could result in greater hyperthermia compared to skaters exercising at the same intensity. However, it is difficult to accurately compare exercise intensity and duration between goaltenders and skaters. These challenges imply that $T_c$ and $T_{sk}$ should be recorded in both goaltenders and skaters.

A recurring theme of this section is that there are more data from skaters than goaltenders. The take home points are that ice hockey goaltenders and skaters lost large amounts of sweat during games and practices, which they failed to completely replace resulting in mild DEH. The magnitude of DEH achieved was shown to affect in-game thermoregulation in skaters, but this has not been tested in goaltenders.

### 1.3 Dehydration and hyperthermia: peripheral and central effects

**Fluid loss.** This subsection will review the mechanisms of sweating, the different methods of heat transfer, fluid lost in each compartment, and what influences sweat loss, rate, onset threshold and sensitivity. Then it will review how DEH affects sweat rate, onset threshold and
sensitivity, as well as heat storage. Sweat loss is most commonly measured as the difference in BM pre and post-exercise (accounting for fluid ingestion and urine output) but can also be measured as changes in blood, plasma and red cell volume as well as plasma osmolality (Dill and Costill 1974, Costill et al. 1976). Estimating sweat loss by changes in BM assumes that all BM lost is fluid and that respiratory and metabolic water losses/gains are negligible, which is correct during exercises lasting $\leq$ 1 hour (Maughan et al. 2007). It should also be considered that 2% BM loss equates to ~3% body fluid loss.

The purpose of sweating is to remove heat from the body. During cycling exercise, heat produced in leg SM increased muscle temperature ($T_m$) and was transferred to the femoral venous blood and the core and then was sent to the periphery via skin blood flow (Gonzalez-Alonso et al. 1999a). Correspondingly, forearm venous blood temperature was also increased during exercise but was not as high as leg venous blood (Gonzalez-Alonso et al. 1999a). It was concluded that blood-to-skin heat transfer was the most important factor for removing heat during exercise. However, this process requires fluid to be lost as well as heat. Therefore, heat generated in the body is moved to the skin via the blood and for heat to leave the body fluid must also be lost.

When the blood delivers heat to the skin there are four main methods of heat transfer: conduction, convection, radiation and evaporation (Jay and Morris 2018). Conduction requires contact and is minimal in sport but does occur within the body. Radiation is typically heat applied to the body from the sun, so it is greater in outdoor vs. indoor sports. Convection occurs when skin temperature $T_{sk}$ does not equal environment temperature. Lastly, evaporative heat transfer depends on the humidity, where a lower RH increases evaporative sweat loss and vice versa. Interestingly, increased air flow augmented convective and evaporative heat transfer (Jay et al. 2015).
The human body is comprised mostly of fluid, which is divided into two main compartments: extracellular (interstitial and plasma) and intracellular. Assuming a 73 kg human body is 60% water, it was estimated that 3.4 L of fluid was blood plasma, 11.0 L was interstitial, and 28.9 L was intracellular (Costill et al. 1976). During exercise when 1.4, 3.0 and 4.1 L of sweat was lost (2.2, 4.1 and 5.8% BM respectively), fluid volume decreased in all compartments and, more specifically, blood plasma volume (PV) decreased 4% for each 2% BM lost (Costill et al. 1976). Since blood was redirected to the skin during thermoregulatory sweating the fluid lost in sweat would have come directly from PV. Furthermore, because PV loss did not equal sweat loss (Costill et al. 1976), the interstitial and intracellular fluid compartment fluid must have redistributed to partially replenish PV. Thus, sweat loss reflected a decreased fluid volume in all compartments.

Considering that thermoregulatory sweating is advantageous during exercise (González-Alonso et al. 1999b) it is logical that greater exercise training status and heat acclimation increased sweat rate (Poirier et al. 2016). Professional ice hockey athletes are highly trained individuals, which partially explains their high sweat rates (Table 1). Sweat rate is also affected by genetics, exercise intensity, and environmental temperature and humidity; however, this review will not cover genetics. Exercise intensity and environmental temperature both independently and dependently increased sweat rate (Montain et al. 1995). Most important to certain sports (i.e. ice hockey, American football) is that protective equipment increased sweat rate (Noonan et al. 2007). This is because the temperature under the equipment was warmer than the ambient temperature, and sweat rate was proportional to the former. Therefore, when exercise intensity and environmental temperature increased so did sweat rate.
Exercise intensity also affects sweating sensitivity and threshold. Sweat onset threshold is the $T_c$ at which sweating commences and sweat sensitivity is a measure of how much sweat rate increases per increase in $T_c$. When exercise intensity was increased sweat onset threshold was unaffected but sensitivity decreased (Montain et al. 1995). These were also affected by body water loss as HYPO (3 and 5% BM loss) delayed the sweat onset threshold and reduced sweat sensitivity (Montain et al. 1995). Delaying sweat onset threshold and decreasing sensitivity can reduce sweat loss and therefore contribute to the impaired thermoregulation observed during exercise when DEH. However, it must be considered that these changes occurred at $\geq 3\%$ BM loss and this magnitude of DEH takes time to occur. Thus, these effects may not affect stop-and-go athletes.

Thus, during exercise or heat stress, body fluid is lost from all compartments and subsequently redistributed between compartments, which occurs to mitigate the rise in $T_c$. Sweat loss is greater as exercise intensity and environment temperature increases but decreases at higher magnitudes of HYPO or DEH ($\geq 3\%$ BM loss).

**Sweat electrolytes.** Sweat is composed primarily of fluid, but also electrolytes such as potassium, sodium ($Na^+$), magnesium, calcium and chloride ($Cl^-$) (Costill et al. 1976). The electrolyte concentration of blood plasma is an important factor in determining how much of the specified electrolyte is lost in sweat, in addition to the ability of the eccrine sweat gland to resorb the given electrolyte. In the blood plasma, there is 140 mM $Na^+$, 110 mM of $Cl^-$ and 5 mM potassium and the concentration of these ions in sweat is proportional to their plasma concentration (Costill et al. 1976). Magnesium, potassium and calcium loss can be ignored as they are lost in physiologically insignificant amounts (Costill et al. 1976). Normative sweat $Na^+$ from males ($n = 306$) indicates an estimated mean $\pm$ standard deviation whole-body (WB) sweat $[Na^+]$ of $39 \pm 10$ mM or $\sim 900$ mg $Na^+/L$ (Baker et al. 2016). Also, of note was the large inter-individual variability,
demonstrating that a large genetic component exists. Thus, Na\(^+\) and Cl\(^-\) were lost in the greatest amounts in sweat and these losses were variable between individuals.

An individualized sweat [Na\(^+\)] measurement is necessary for accurate sport nutrition guidelines to account for large individual differences. It must also be considered that sweat [Na\(^+\)] varied between sites within an individual, therefore taking a sweat sample from a single site may over or underestimate whole-body sweat [Na\(^+\)] and therefore WB Na\(^+\) losses (Patterson et al. 2000). Fortunately there are strong linear relationships between single site sweat [Na\(^+\)] and WB sweat [Na\(^+\)] (Baker et al. 2009) and WB sweat [Na\(^+\)] can easily be estimated by a single sweat collection patch.

Although not ergogenic during exercise, Na\(^+\) supplementation has been demonstrated to be useful pre and post-exercise. When Na\(^+\) was added to a solution consumed pre-exercise, PV was increased relative to placebo as was endurance capacity and 15 min TT performance (Coles and Luetkemeier 2005, Sims et al. 2007a). Additionally, when a Na\(^+\) solution was ingested pre-exercise it improved thermoregulation and reduced cardiovascular stress during exercise to exhaustion trials relative to water (Sims et al. 2007a, b). Thus, consuming Na\(^+\) with water pre-exercise reduced cardiovascular strain and improved thermoregulation and exercise performance.

Overall Na\(^+\) is lost in large amounts in sweat and the concentration lost is highly variable between individuals (Baker et al. 2016, Table 1). Replacing Na\(^+\) during exercise has not proven ergogenic, but Na\(^+\) consumed with water pre-exercise reduces thermoregulatory and cardiovascular strain and could be ergogenic. Even though Na\(^+\) consumption may not benefit the athlete during their event, it still must be replaced during recovery and therefore consuming Na\(^+\) before/during exercise is logical to reduce the need in recovery.
Circulatory and skeletal muscle responses. DEH increases $T_c$ during exercise, which affects both the circulatory system and SM metabolism. Therefore, both body water losses and $T_c$ will be reviewed. This section will begin with how DEH affects the heart, then the rest of the circulatory system and finally the SM.

The cardiovascular system will be reviewed as HR, stroke volume (SV) and cardiac output (Q). HR is the frequency the heart beats per min, SV the volume of blood pumped per beat, and Q is the volume of blood the heart pumps per min ($Q = HR \times SV$). During exercise Q increased to match the $O_2$ and metabolic demand of the contracting SM (González-Alonso et al. 1998, 1999a). When fluid was restricted during exercise Q was significantly reduced 90 min into exercise (~2.5% BM loss) but not at 60 min (~1.7% BM loss) compared to maintained BM (González-Alonso et al. 1998). Furthermore, during a 2-hour moderate intensity cycle, Q was unchanged when BM loss was 1% but was reduced at 2, 3 and 4% BM loss (Montain and Coyle 1992a). Interestingly, Q was unaffected independently by 4% BM loss or hyperthermia ($T_c = 39.3 \pm 0.1^\circ C$), but the combination significantly reduced Q (González-Alonso et al. 1997). Before Q was reduced during exercise, HR and SV were affected (González-Alonso et al. 1998). Four percent BM loss and hyperthermia both independently increased HR and decreased SV and together potentiated these changes (González-Alonso et al. 1997). More importantly, at more modest magnitudes of DEH (1.5-2% BM loss) HR and SV were still increased and decreased respectively (Montain and Coyle 1992a, Logan-Sprenger et al. 2013). Thus, DEH and hyperthermia both independently and collectively increased HR and decreased SV during exercise and these changes occurred around 1.5-2% BM. However only the combination of DEH and hyperthermia reduced Q.

Since blood flow (BF) is directed to the skin and contracting SM during exercise it is important to understand what happens to BF when Q is decreased. When subjects were
progressively DEH (~4% BM loss) during a 2-hr cycle, BF was first reduced to the forearm and non-contracting leg muscles and lastly the contracting leg muscles (Montain and Coyle 1992b, González-Alonso et al. 1998). The reduced peripheral BF was attributed to decreased skin BF, which could explain the impaired ability to dissipate heat while DEH (González-Alonso et al. 1998). Furthermore, vascular conductance was unchanged with DEH (≥4% BM loss) in the contracting SM but was reduced in the forearm and non-contracting SM (González-Alonso et al. 1998). In two studies, $T_c$ was increased at <2% BM loss compared to maintained BM despite similar sweat losses (Logan-Sprenger et al. 2012, 2013). It was speculated that less BF reached the skin and less heat was removed when DEH but a similar amount of sweat was excreted through sweat glands. Thus BF to and vasoconstriction/dilation around the contracting SM is preserved over inactive SM and the forearm/skin, which may explain why mild DEH impairs thermoregulation.

The cardiovascular system delivers substrate, including $O_2$, to SM and this must be considered when DEH. Despite reductions in contracting muscle BF when DEH, higher arterial $O_2$ concentration and SM extraction preserved leg $O_2$ uptake ($VO_2$) (González-Alonso et al. 1998). Contrastingly, contracting SM glucose and free fatty acid uptake were unaffected by DEH (González-Alonso et al. 1999a). Therefore, metabolic substrate and $O_2$ uptake to SM were unaffected by DEH. Indirect calorimetry at the WB and SM level suggested that >1.5% BM loss increased carbohydrate oxidation and was due to elevated SM glycogenolysis rather than exogenous glucose oxidation (Hargreaves et al. 1996, González-Alonso et al. 1998, Logan-Sprenger et al. 2013, 2015). Although DEH increased glycogenolysis, this response was due to elevated body temperature, specifically $T_m$, when DEH rather than body water loss (Febbraio et al. 1994, 1996a, b, Fernández-Elías et al. 2015).
Altogether, DEH and hyperthermia both in isolation and together increased aspects of cardiovascular strain. Furthermore, BF was preferentially redirected away from non-contacting SM and skin to the contracting muscle when DEH, which can account for decreased heat dissipation and increased heat storage. DEH and hyperthermia did not affect VO₂ or metabolic substrate uptake by SM, but SM glycogenolysis was increased by hyperthermia when in a DEH state.

Central nervous system and neuromuscular fatigue. This subsection will review the neuromuscular effects present with DEH and hyperthermia. The purpose is to review what happens to the muscle at fatigue onset outside of a metabolic and cardiovascular scope and then compare these effects to when athletes are DEH or hyperthermic, but not fatigued. Fatigue is defined as the inability to produce force or a loss of force production. Fatigue can result from central and/or peripheral factors. Peripheral fatigue (PF) is the reduction of force producing capacity of contracted muscle and is commonly measured as force produced during a brief maximal voluntary contraction (MVC). Central fatigue (CF) arises upstream of the neuromuscular junction and can occur without PF being present. CF is measured as voluntary activation (VA), or the difference in force produced during a maximal voluntary contraction (MVC) and the additional force produced when a maximal twitch stimulation is applied to the contracting muscle.

Demanding and fatiguing exercises cause both PF and CF. Immediately after fatiguing exercise, significantly less MVC force was produced with the leg flexor and extensor muscles (Sahlin and Segen 1995). Moreover, 5 hr of submaximal cycling decreased both MVC and CF in the leg extensors immediately post-exercise and throughout a 5 hr recovery (Lepers et al. 2002). Similar trends were observed in the same muscle groups after 30 min cycling at 80% peak power
output (Lepers et al. 2001) and from half time until the end of a simulated soccer match (Goodall et al. 2017). Therefore, CF and PF were present after challenging exercises and at fatigue onset.

Interestingly, $T_c$ and $T_m$ also affect CF. Using gradual heating and cooling, VA changed reciprocally with $T_c$, that is as $T_c$ increased VA decreased (Morrison et al. 2004, Thomas et al. 2006). Although the changes in VA were primarily attributed to $T_c$, it was later shown that $T_m$ contributed ~15% to this effect (Lloyd et al. 2015). Therefore, CF was caused by both an elevated $T_m$ and $T_c$ at rest but more so by the latter.

$T_c$ and $T_m$ are also increased by exercise and therefore exercise may interact with hyperthermia to potentiate CF. When $T_c$ was increased to ~38.5°C via passive heating or cycling in the heat, CF in the leg extensor muscles was unaffected in both conditions vs. control ($T_c$ ~37.0°C) (Périard et al. 2014). Likewise, using the same passive and active heating approach, CF in the leg extensors was similarly reduced compared to control ($T_c$ ~37.0°C) when $T_c$ was ~39.5°C after passive heating or exercise (Périard et al. 2011). Therefore, exercise did not potentiate the effects of hyperthermia on CF.

Interestingly, CF can explain worse exercise performance, aspects of fatigue, and endurance capacity during exercise in the heat when cardiovascular or metabolic effects could not (Nielsen et al. 1993, Nybo and Nielsen 2001, Drust et al. 2005). When cycling to exhaustion in a 40°C environment compared to 18°C, VA was reduced in the leg extensor muscles at fatigue in the heat trial ($T_c = 40.0 \pm 0.1°C$) vs. control ($T_c = 38.0 \pm 0.1°C$) despite cycling for a shorter duration (Nybo and Nielsen 2001). Similarly, less power was produced during repeated sprints when hyperthermic ($T_c = 39.5 \pm 0.2°C$) relative to control ($T_c = 38.2 \pm 0.2°C$) and this coincided with a reduced peak handgrip force (Drust et al. 2005). Since the forearm flexors are not regularly contracted during cycling, any decrease in force produced by the forearms would be from CF not
PF. Therefore, PF would not be a factor and decreased force production could be attributed to CF. Overall when hyperthermic, CF coincided with reduced performance in situations where other factors could not explain it.

When exercising in the heat sweat loss is increased and if the fluid is not replaced, DEH will occur. When exercised in the heat, DEH participants exhausted when $T_c$ was $39.7 \pm 0.2^\circ C$ (González-Alonso et al. 1999a). Comparatively, when hydration status was maintained during exercise in the heat, $T_c$ at exhaustion was always $>40^\circ C$ (González-Alonso et al. 1999b), suggesting that DEH potentiated the effects of hyperthermia (Nybo and González-Alonso 2015). Two studies have examined CF and PF under HYPO (~2.5, 4 and 5% BM loss) but neither found that any magnitude of body mass loss had an effect on CF in the knee extensor muscles (Judelson et al. 2007, Stewart et al. 2014). A meta-analysis found that DEH reduced lower and upper body strength and power, but CF was not measured and based on other studies that showed a lack of CF present with DEH or HYPO (Judelson et al. 2007, Stewart et al. 2014), it is likely that these changes would be from PF (Savoie et al. 2015). Thus, body mass losses $\leq 5\%$ did not exclusively cause CF but mild DEH induced PF.

It should be considered that impaired thermoregulation is a consequence of DEH. $T_c$ increased $\sim 1.5^\circ C$ when DEH vs. hydrated during cycling in the heat (Gonzalez-Alonso et al. 1999a) but this change was more modest ($\sim 0.3^\circ C$) in ice hockey skaters (Boville 2015, Linseman et al. 2014). Although the absolute $T_c$ difference was small for ice hockey skaters when DEH, CF occurred gradually during passive heating and cooling (Morrison et al. 2004). Therefore, if $T_c$ increased enough when DEH vs. hydrated, CF may arise but this theory has not been tested.

Overall, both fatiguing exercise and hyperthermia independently caused CF. Thus, when hyperthermic, CF caused a non-fatigued muscle to act more like a fatigued one and this effect
explained reduced exercise performance. Despite HYPO not affecting CF, if DEH elevates Tc by a large enough magnitude, CF may occur.

**Performance.** Becoming mildly DEH during exercise has many physiological consequences that may decrease performance and therefore, replacing fluid losses may be beneficial. In DEH performance studies, Tc must also be considered because of its similar effects (Nuccio et al. 2017). Compared to becoming mildly DEH (≥2% BM loss), the majority of research indicates that replacing sweat losses with water during steady-state cycling decreased Tc and improved exercise performance (Below et al. 1995, McGregor et al. 1999, Logan-Sprenger et al. 2015, Nuccio et al. 2017). However, not all steady-state or intermittent exercise performance studies showed this (Goulet 2011, 2013, Cheung et al. 2015, Palmer et al. 2017b). One reason for this discrepancy may be that steady-state and intermittent cycling exercises in the lab do not require the athlete to process as much external information compared to stop-and-go team sports; and therefore, these protocols do not completely simulate team sports.

DEH impairs cognitive function and movement power and strength. Specifically, mild DEH (≥2% BM loss) increased decision making duration, reaction time and perceptions of fatigue as well as worsened object tracking performance, short-term memory and mood (Cian et al. 2000, 2001, Grandjean and Grandjean 2007). Moreover, a recent meta-analysis showed that DEH worsened executive function, motor coordination and attention, and that this occurred progressively with BM loss (Wittbrodt and Millard-Stafford 2018). Also notable, is that a recent meta-analysis showed reduced strength and power occur when mildly DEH (Savoie et al. 2015). Cognitive function and movement power are important factors in sport and especially ice hockey goaltenders. Goaltenders, like other stop-and-go athletes, process a large amount of information during sport (i.e. reacting to where a pass is going, AVI used for making saves and motor function
for making saves). Therefore, DEH may decrease performance in goaltenders by reducing their ability to properly and/or quickly process the information cognitively. Furthermore, less powerful and slower movements when DEH (Savoie et al. 2015) could reduce performance in ice hockey goaltending, especially when considering that DEH reduced the available processing time to make a decision (i.e. when moving to the skater shooting the puck in reaction to a pass). Thus, DEH progressively impaired cognitive function and movement strength and power, all of which could contribute to decreased performance in sports that have a large cognitive component.

In soccer and basketball athletes, DEH impaired physical and sport-specific performance compared to maintained hydration with water (McGregor et al. 1999, Baker et al. 2007, Edwards et al. 2007). However, compared to maintaining BM with water, mild DEH (1.2 and 1.9% BM loss) did not effect on-ice performance in post-scrimmage skating, shooting and passing drills containing decision making components (Boville 2015, Eskedjian 2015). Thus, mild DEH compared to proper hydration with water sometimes impaired sport performance in sport-specific drills with and without a large cognitive component.

**Summary.** During exercise the increase in $T_c$ is regulated through sweating and this response increases with exercise intensity and/or heat stress. Sweat also contains a significant amount of $\text{Na}^+$ that is highly variable between individuals and necessary to replace during recovery. Sweat volume is also variable between individuals and increases with exercise intensity and/or heat stress. DEH during exercise strains the cardiovascular system, impairs the sweating response, shifts SM substrate preference to glycogen and increases $T_m$ and $T_c$. CF is largely affected by hyperthermia and not body water loss, however, if DEH increases $T_m$ and $T_c$ enough then CF could occur. Becoming mildly DEH during exercise sometimes impaired performance
during modalities with a low cognitive demand and when cognitive demand was increased – i.e. sport specific situations – so are the effects of DEH.

1.4 Carbohydrate effects during exercise

This section will review the literature on carbohydrate (CHO) intake and mouth sensing during exercise and its effect on SM metabolism and performance. It will also touch on CHO absorption in the gut and its interaction with hydration. It is acknowledged that there is a place for athletes to consume CHO pre- and post-exercise to optimize performance and recovery respectively. It is beyond the scope of this review to analyze the literature on these topics, but it should be noted that most elite athletes will be following a high CHO diet, and therefore consuming CHO before and after exercise.

Carbohydrate metabolism. Energy or adenosine triphosphate (ATP) is stored in very small amounts within the SM and would deplete within 5 sec of exercise, if not regenerated. Therefore, ATP is created through aerobic and anaerobic metabolic pathways during exercise. Anaerobic metabolism or substrate phosphorylation does not require O$_2$ to create ATP and does so through phosphocreatine (PCr) breakdown and anaerobic glycolysis. PCr generates ATP at the onset of exercise but is only dominant for the first 6 sec during all out sprints (Gaitanos et al. 1993, Parolin et al. 1999). The anaerobic capacity of the glycolytic pathway is about 3 times as large as PCr and can contribute energy for up to 1-2 min (Parolin et al. 1999). Glycolysis can make ATP both aerobically and anaerobically but more ATP is generated through aerobic glycolysis. Aerobic energy production requires glucose or FFA and O$_2$ to enter the mitochondria to generate ATP via oxidative phosphorylation. Aerobic metabolism creates ATP with both glucose and FFA at a given exercise intensity, but the proportion of ATP derived from the two substrates changes with
intensity. FFA oxidation plateaus around 65% VO$_2$ max while glucose oxidation increases with exercise intensity and at 85% VO$_2$ max most ATP was created by CHO oxidation (van Loon et al. 2001). Furthermore of the CHO oxidized at high intensity, the majority comes from glucose stored within SM (glycogen) not blood glucose (van Loon et al. 2001). Therefore, ATP is made both aerobically and anaerobically and at high aerobic intensities and anaerobic-dominated exercise (i.e. sprinting) relies heavily on SM glycogen as a substrate.

Movement patterns during stop-and-go sport are brief sprints or high intensity efforts separated by rest or low intensity efforts (Williams and Rollo 2014). During a 6 sec sprint, anaerobic metabolism created almost half of the ATP, about 50% was PCr breakdown and the other half glycolysis (Parolin et al. 1999). The aerobic system is also important because recovery of the anaerobic systems during the rest or low intensity portions requires O$_2$ and is positively related to aerobic fitness (Bogdanis et al. 1996). So, the higher the aerobic fitness of an individual, the faster the anaerobic systems recover. In addition, the more aerobically fit a stop-and-go athlete is, the less they rely on the anaerobic system at the onset of activity and in repeated sprints (Gaitanos et al. 1993, Parolin et al. 1999). This is beneficial because the anaerobic system provides energy for short durations and is not sustainable for long durations. Therefore, being more aerobically fit would better allow an athlete to maintain sprint power during a set of repeated sprints. Due to the high exercise intensity and the stop-and-go nature of team sports, SM glycogen would be the primary substrate used. Unfortunately, SM glycogen is limited and depletion results in fatigue (Coyle et al. 1986). Therefore, starting exercise with more glycogen and reducing SM glycogen use while maintaining the same exercise intensity is beneficial and can be achieved by consuming CHO during steady-state and intermittent exercises (Foskett et al. 2008).
**Gastric emptying and intestinal absorption.** Before CHO can have an effect in the body it must first be consumed and then moved into the blood. This subsection will review the effect CHO has on fluid delivery to and fluid absorption from the gut.

The process of contents leaving the stomach through the pyloric sphincter into the small intestine is called gastric emptying (GE). It has been shown that GE was reduced by increasing [CHO] from four to 18% in a CHO-electrolyte solution (CES) when consumed as a large bolus (Vist and Maughan 1992). Another study found that 15 min after drinking 400 mL 4% CES, GE was reduced ~28% compared to water (Coyle et al. 1978). When consuming CES in a repeated or serial manner – more applicable to athlete drinking patterns – it appears CHO has a smaller effect. When 3.0 mL/kg (~227 mL) of water or a 4, 6, or 8% CES were consumed every 15 min during low intensity exercise, only the 8% CES decreased GE vs. water (Murray et al. 1999). However, when even smaller boluses were ingested (2.3 mL/kg, ~180 mL) every 10 min during exercise the 8% CES emptied the stomach as fast as water (Ryan et al. 1998). Similarly, an 8.6% CES showed comparable GE to water during the second half of 90 min moderate intensity exercise (Jeukendrup and Moseley 2010). Thus, GE with a CES vs. water was less affected by [CHO] when consumed more frequently in smaller volumes.

It is important to note that the previously cited articles all used multiple CHO sources containing glucose and fructose (Coyle et al. 1978, Vist and Maughan 1992, Ryan et al. 1998, Murray et al. 1999, Jeukendrup and Moseley 2010, Osterberg et al. 2010). One study (Jeukendrup and Moseley 2010) found that an 8.6% CES (2:1 glucose:fructose) had a quicker GE time compared to an 8.6% glucose only solution. Glucose and fructose are absorbed with different transporters in the gastrointestinal (GI) tract so it possible that increased GI absorption may allow for faster GE.
Net fluid flux across the GI tract can be referred to as fluid availability (FA), which is important because this is the step where fluid enters the blood plasma. FA is affected by [CHO] in a CES. When fluids were consumed serially during moderate intensity exercise, net water absorption across the GI tract was decreased with 8 and 9% CES vs. both a 6% CES and water (Ryan et al. 1998). Thus when [CHO] >6% in a CES, FA decreased.

The delivery rate of CHO monomers from a CES may also affect FA. The absorption of glucose into the bloodstream from the intestine via the sodium-glucose transporter-1 (SGLT1) co-transporters a water molecule, whereas glucose transporter-5 (GLUT5) which transports fructose does not. Glucose transport via SGLT1 saturates at ~1.0 g glucose/min and since SGLT1 co-transporters a water molecule, theoretically water flux across the GI tract can be increased by ingesting glucose ≤1.0 g/min. When an 8.6% CES was ingested during exercise (1.0 g glucose/min) and SGLT1 was saturated, GE and FA were comparable to water (Jeukendrup and Moseley 2010). In contrast, when a smaller volume of an 8% CES (~0.6 g glucose/min) was consumed during exercise and SGLT1 was not saturated, GE was similar but intestinal fluid flux was reduced vs. isovolumetric water consumption (Ryan et al. 1998). Therefore, saturating the SGLT1 and maximizing the co-transportation of water may increase net fluid flux and FA. However, more research is required to prove this concept.

Altogether, these results demonstrated that consuming a CES with ≤6% CHO does not impair GE nor FA. However, the monosaccharide composition, low frequency of consumption, and large bolus volume decreases both GE and FA when the CES exceed 6% CHO.

**Exogenous carbohydrate oxidation.** In addition to affecting GE and FA, monosaccharaides transported into the blood from the gut using different transporters affect SM CHO oxidation. When glucose or glucose polymers are ingested at a rate <1.1 g/min during
exercise, peak exogenous CHO oxidation did not exceed ~1.0-1.1 g/min (Jentjens et al. 2004a, b, c, Jentjens and Jeukendrup 2005). Since infusing glucose directly into the blood elicited CHO oxidation rates >1.2g/min (Hawley et al. 1994) it was determined that SM glucose uptake was not limiting. Therefore, the limiting factor is GE or intestinal absorption. Nonetheless, when ingesting glucose or other monosaccharides transported across the intestines via SLGT1, peak CHO oxidation is maximized at ~1.0-1.1 g/min.

When fructose or sucrose, monosaccharides that use GLUT5 to cross the intestinal wall, are ingested along with glucose, CHO oxidation can exceed 1.2 g/min. Consuming 1.2 and 0.6 g/hr glucose and fructose respectively increased peak exogenous CHO oxidation at the 2 hr mark during moderate intensity steady state exercise compared to ingesting 1.8 g/hr glucose (Jentjens et al. 2004b). Furthermore, lesser GI upset was reported with glucose and fructose co-ingestion compared glucose (Jentjens et al. 2004b). In a separate experiment, peak CHO oxidation was ~1.75 g/min when 1.2 g/min of both glucose and fructose were co-ingested (Jentjens and Jeukendrup 2005). Therefore, co-ingestion of fructose and glucose resulted in the greatest exogenous CHO oxidation rates and did not cause significant GI upset.

These results suggest that athletes should aim to consume ~1.0g/min glucose and ~0.5 g/min fructose to maximize exogenous CHO oxidation during steady state exercise lasting >2 hr. However, these recommendations must be adjusted to stop-and-go sport due to the relatively short duration. When exogenous CHO oxidation was measured each 15 min, ingesting any CHO solution increased exogenous SM CHO oxidation 15 min into exercise (Jentjens and Jeukendrup 2005). Similarly, a mixture of glucose and fructose yielded greater exogenous CHO oxidation compared to ingestion of only glucose and occurred between 15 and 30 min into exercise (Jentjens et al. 2004a, b). Therefore, during shorter duration steady-state exercise, ingestion of glucose
increased exogenous CHO oxidation and co-ingestion of glucose and fructose further increased this. However, stop-and-go sport has intermittent exercise patterns usually performed at high aerobic or sprint intensities and therefore, these steady-state results are not directly applicable.

The practical applications for ingesting CHO during exercise stem from shifting substrate utilization. When a 2 g/kg glucose dose was ingested pre-exercise followed by 0.4 g/kg glucose boluses ingested each 20 min during moderate cycling, fatigue was delayed ~1 hr compared to placebo (Coyle et al. 1986). Interestingly, glucose ingestion spared muscle glycogen in type II but not in type I fibres (Coyle et al. 1986, De Bock et al. 2006) even though mixed muscle glycogen was unaffected by glucose ingestion (Coyle et al. 1986). It should be noted that differences in mixed muscle glycogen concentration with glucose intake during intermittent and steady-state exercise were not always present (Hargreaves et al. 1984, Coyle et al. 1986, Tsintzas et al. 1996). Sparing SM glycogen is important because it is limited and depletion results in fatigue (Coyle et al. 1986). Thus, CHO consumption during exercise reduced SM glycogenolysis and delayed fatigue onset. In addition to potentially sparing SM glycogen, ingesting CHO during exercise can maintain high CHO oxidation rates and blood glucose concentrations (Coyle et al. 1986). CHO is the primary SM fuel when exercising at high intensities, with a high proportion coming from glycogen (van Loon et al. 2001). In situations where CHO oxidation cannot be maintained, exercise intensity cannot persist; and therefore, power output decreases and so would sport performance.

**Carbohydrate mouth rinse.** It is evident that CHO can improve performance during long duration and/or high-intensity exercise by sparing SM glycogen and by maintaining blood glucose levels and high CHO oxidation rates (Coyle et al. 1986). However, in a 1 hr time trial (TT) none of these factors were limiting or affected when 60 g of CHO were infused (Carter et al. 2004b).
When 60 g of CHO were ingested during a 40 km TT (~1 hr) performance improved 2.3% vs. a water placebo (Jeukendrup et al. 1997). Therefore, CHO appeared to have an effect independent of blood glucose, CHO oxidation or SM glycogen. To test this hypothesis fasted subjects completed a 40 km cycling TT (~1 hr) while rinsing their mouth with, but not ingesting, a 6.4% tasteless CHO (maltodextrin) solution or a placebo for 5 sec after each 5 km completed (Carter et al. 2004a). Rinsing with CHO improved TT performance by 2.9%, which was comparable to ingesting CHO (Carter et al. 2004a, Jeukendrup et al. 1997). Thus, CHO had an ergogenic effect in the mouth.

Follow-up research attempted to find the mechanism of action for CHO mouth rinsing. When a glucose (sweet CHO) or maltodextrin (non-sweet CHO) solution or water was rinsed for 5 sec throughout exercise, functional magnetic resonance imaging revealed that areas of the brain associated with reward and motor control were more active and cycling time trial performance was increased when CHO was rinsed vs. placebo (Chambers et al. 2009). Furthermore, CHO vs. placebo rinsing improved aspects of neuromuscular performance during exercise and reduced indices of neuromuscular fatigue immediately after a 15 km TT after repeated elbow flexion contractions to fatigue (Gant et al. 2010, Jeffers et al. 2015, Bazzucchi et al. 2017). Thus, CHO mouth rinsing caused central effects that can explain its ergogenic benefits.

CHO also activated areas of the brain associated with reward and pleasure (Chambers et al. 2009), which could affect ratings of perceived exertion (RPE) during exercise and therefore improve pacing strategy and performance. Rinsing with CHO did not affect RPE or performance during a 1 hr TT (Pottier et al. 2010), a 5 km run (Clarke et al. 2017) or a 1 hr run (Rollo et al. 2010). On the contrary, participants that performed more work and improved performance when CHO vs. placebo rinsing indicated similar RPE scores (Lane et al. 2013; Rollo et al. 2008). In
conclusion, there is some evidence CHO mouth rinsing decreased RPE but also some to suggest it does not.

Another factor potentially influencing the performance effect of CHO is fed/fasted state. Exercise performance decreased when fasted (Lane et al. 2013) so current guidelines suggest consuming ~2.5 g of CHO/kg BM in the 2-3 hrs pre-exercise. Therefore, it is important to understand the effects of CHO mouth rinsing in fed subjects. One study had trained cyclists perform four 60 min TTs in the following conditions: 2.5 g CHO/kg BM breakfast or 9-10 hours fasted and CHO or placebo mouth rinse during the TT (Lane et al. 2013). When the 10% CHO solution was rinsed for 10 sec work completed significantly increased 3.4% and 1.6% within the fasted and fed trials vs. placebo respectively. The effect of CHO mouth rinsing was greater when fasted than fed, but not all studies find ergogenic effects when postprandial (Beelen et al. 2009, Trommelen et al. 2015). Two notable differences to explain the contrasting results were the duration and concentration of CHO solution rinsed. CHO tasting was beneficial when the 10% solution was rinsed for 10 sec and was not beneficial when a 6% CHO solution was rinsed for 5 sec (Beelen et al. 2009; Trommelen et al. 2015). However, there is evidence to suggest that rinsing the CHO solution for 10 sec is more ergogenic than 5 sec (Sinclair et al. 2014). Increasing [CHO] >7% has not proven to further benefit exercise performance over lower CHO concentrations (Ispoglou et al. 2015, Clarke et al. 2017, James et al. 2017). Overall, these results suggested that CHO mouth rinsing could be a potential performance aid in fed athletes.

Stop-and-go sport athletes typically perform repeated sprints and could benefit from CHO mouth rinsing. When a 6% CHO or placebo solution was rinsed before a 30 sec resisted sprint, CHO mouth rinsing increased peak power output but reduced mean power in the second half (Phillips et al. 2014). Similarly, when rinsing with CHO during five, 6 sec sprints, power output
was increased during sprint 1 but decreased in sprint 5 (Beaven et al. 2013). Another study showed no benefit of CHO mouth rinsing during a 30 sec repeated sprint (Chong et al. 2011). Thus, CHO mouth rinsing had equivocal effects on sprinting.

In conclusion, CHO mouth rinsing improved exercise performance in some sprints and ~1 hr TTs via central mechanisms. Although CHO mouth rinsing is interesting from a mechanistic point of view, it seems illogical for an athlete to avoid consuming CHO, especially because a CES also contains electrolytes and fluid. Even if drinking a CES did not improve performance it would reduce the fluid, electrolyte and CHO requirements in recovery. Finally, it seems logical that an athlete should swish the CES in their mouth before consuming it to get the full benefits of CHO.

**Performance.** It is established that CHO consumption during exercise compared to no CHO consumption decreased muscles glycogen breakdown, maintained blood glucose levels in some scenarios and increased/maintained whole body CHO oxidation rates (Coyle et al. 1986). Furthermore, tasting CHO also improved performance (Carter et al. 2004a). Thus, it is logical that CHO improved performance in team sports (Williams and Rollo 2015). The majority of research on CHO and team sport performance has focused on simulated intermittent sprint performance and has found improvements with CES ingestion relative to water and CES ingestion relative to mild DEH (Linseman et al. 2014, Williams and Rollo 2015, Palmer et al. 2017a). In addition to improved physical performance, CES consumption during simulated basketball playing improved motor skills, cognitive ability and mood (Winnick et al. 2005). Similar findings on mood existed in ice hockey skaters (Linseman et al. 2014, Boville 2015, Eskedjian 2015). CES ingestion to maintain BM compared to mild DEH reduced perceived fatigue, improved hockey-specific performance during a scrimmage and post-scrimmage drill completion time (Linseman et al. 2014). Furthermore, post-scrimmage skating and shooting performance were improved compared
to mild DEH with CES intake but not water in ice hockey skaters (Eskedjian 2015). However, post-scrimmage reaction time with the puck, passing performance and a different skating performance measure were not improved with CHO compared to water (Boville 2015, Eskedjian 2015). Overall, CHO consumption improved many aspects of sport performance compared to mild DEH, but the comparison between CES and water in ice hockey athletes is less clear.

**Summary.** Consuming CHO during exercise via a CES is recommended for athletes. Gastric emptying and intestinal absorption of a \(\leq 6\%\) CES were similar to water but the CES increased CHO oxidation. CHO ingestion decreased reliance on the limited SM glycogen stores, which improved performance later in exercise (Palmer et al. 2017a). Overall, consuming CHO via a CES proved more beneficial than consuming plain water during some sport type exercise. These improvements were likely due to central effects given the short duration of team sports but consuming CHO during exercise is still recommended because it will reduce the need in recovery.

1.5 Conclusions

Ice hockey goaltender and skater positions are different and warrant separate research. Although the physiological demand of ice hockey goaltending has not been reported in the literature, the observations while making saves suggested large cognitive and decision making components exist. Despite the differences between ice hockey goaltenders and skaters, both sweat at high rates and became mildly DEH during practices (Table 1). The losses observed in ice hockey goaltenders were greater than skaters during practice, but there is not enough game research to make this claim in goaltenders during games. Based on data in non-goaltenders, the magnitude of DEH achieved by goaltenders is predicted to impair thermoregulation, induce cardiovascular strain, alter SM metabolism and potentially reduce CNS function. Altogether, these consequences
of DEH worsened performance during cycling and team sport exercises and the consequences appeared larger when a cognitive component was required. DEH can be countered by consuming fluid during exercise to approximate sweat loss and the fluids typically consumed are water or a CES. Performance research showed that relative to mild DEH, maintaining hydration with water or CES was beneficial and that consuming CES was sometimes superior to water. Unfortunately, there is little research conducted on DEH in ice-hockey athletes and performance, and no research done on goaltender physiological demands and performance in this setting despite their high sweat losses and significant DEH.
CHAPTER II: AIMS OF THE THESIS

Background overview. Ice hockey has two positions, goaltenders and skaters. The literature on skaters shows they play fast, intense shifts interspersed by rest periods. There is not enough information available about goaltenders to conclude the physiological demands. Goaltenders are required to accurately navigate a small portion of the ice surface in reaction to what occurs with the puck and all 10 moving skaters on the ice at once. Therefore, they must be highly focused during games and the position has a large cognitive component. There has been more research in ice hockey athletes about sweat rates and dehydration (DEH). Goaltenders and skaters produced a large amount of sweat during practices, leading to DEH. Interestingly, goaltenders sweat more and were more DEH than skaters after practices. Unfortunately, there was only one game study that reported goaltender results and more game data are required. Overall, goaltenders produce a substantial volume of sweat, which suggests they are working at a high exercise intensity.

Ice hockey skater performance has been studied under mild DEH in field and simulated lab studies (Boville 2015, Eskedjian 2015, Linseman et al. 2014, Palmer et al. 2017a, b). Compared to maintaining hydration, mild DEH impaired thermoregulation, reduced on-ice skating speed, sprint performance and some hockey specific skills, in addition to increasing perceptions of fatigue. There is sparse literature on ice hockey goaltenders and no previous work testing the effects of mild DEH on physiological parameters and performance.

DEH is the process of losing body water and occurs progressively during exercise. Becoming mildly DEH during exercise increased cardiovascular strain, core body temperature ($T_c$) and perceptions of fatigue, in addition to decreasing sprint, endurance, cognitive and sport specific performance. Of most concern is the increased $T_c$ because hyperthermia interacts with DEH to
potentiate the previously mentioned effects. Furthermore, hyperthermia also fatigued the central nervous system (CNS), which independently caused fatigue.

The consequences of DEH during exercise can be mitigated by consuming fluid to replace sweat losses. Of greater benefit was consuming a carbohydrate (CHO)-electrolyte solution (CES) instead of water, which many athletes choose to do. Drinking a CES during simulated stop-and-go sports has improved performance compared to water (Rollo and Williams 2015). Also, tasting, but not ingesting, CHO improved performance during a variety of exercises. It is recommended that stop-and-go sport athletes consume 30-60 g/hr CHO during sport to improve performance and reduce the need for CHO during recovery (Thomas et al. 2016). Overall, consuming water (WAT) in a volume to match sweat loss was beneficial compared to becoming mildly DEH and consuming a CES was sometimes further beneficial to WAT.

Ice hockey goaltenders are a necessary population to study in this setting because of their high sweat losses and mild DEH that occurred during practices. There is research on ice hockey skaters and other stop-and-go athletes in this context that demonstrate reduced performance when mildly DEH. However, due to the large differences between positions and sports these results cannot be accurately transferred. Therefore, studying the effects of ice hockey goaltenders warrants its own research.

**Purpose.** The purpose of this thesis was to examine the physiology, thermoregulation, fatigue and performance in ice hockey goaltenders during a scrimmage and subsequent drills when DEH (~2% BM loss) relative to maintaining hydration with WAT or CES.

**Hypotheses.** It was hypothesized that DEH would decrease performance, increase fatigue, and the thermoregulatory and physiological responses to exercise would be magnified as BM loss reached 1.5-2.0% compared to maintaining hydration with either fluid. A secondary hypothesis
was that although consuming either solution would be beneficial over DEH, maintaining hydration with a CES would be superior to WAT for performance and fatigue but not for thermoregulation and physiology.
CHAPTER III: METHODOLOGY

3.1 Subjects

Eleven skilled goaltenders were recruited for the study (age: 21 ± 1 years, height: 179 ± 2 cm, BM: 82.1 ± 4.6 kg, playing experience = 13 ± 2 years, skill level: AA minor hockey–college varsity). Forty-five skaters were also recruited (playing experience: 14 ± 5 years, skill level range was high school-NCAA D1) but data were only collected from goaltenders. Before obtaining written informed consent from all participants, they were informed in writing of the study risks and goaltenders were also informed orally. Ethical approval was obtained from the University of Guelph Research Ethics Board.

3.2 Study design

This randomized, crossover design study contained one familiarization and three experimental trials. For each trial, goaltenders arrived 60 min before skating for pre-skate measures, warmed-up on the ice for 10 min, competed in a 70 min scrimmage, and completed 24 min of drills and post skate measures (Figure 3). Trials were separated by >48 hours and were at the same time of day ± 4 hours. During each trial, two goaltenders were tested and the entire on-ice portion was filmed. On each occasion, goaltenders drank either a 6% CHO-electrolyte solution (CES) in a volume equal to sweat loss or the same amount of taste-matched water (WAT) or no fluid (NF). Commercially available CES powder was added to water to create a 6% CHO solution (Gatorade: 6% CHO, 18 mM Na⁺, 11 mM Cl⁻, 3-mM K⁺, 240 kcal/L), and the taste-matched placebo was prepared using low-calorie drink powder (2 g of Crystal Light per L water; 10 kcal/L). Before all trials, participants drank 250 mL of water in the dressing room between the time of arrival until the skate began. After putting on equipment, goaltenders performed a 120 sec
sustained maximum voluntary contraction (SMVC) with each hand 10 min before the skate. During the 104 min on-ice portion, goaltenders participated in a warm-up, a 3-on-3 scrimmage, a shootout, a shooting drill and a lateral movement drill (Figure 3). Immediately post-skate, goaltenders returned to the dressing room to repeat the SMVC protocol, then removed equipment, urinated if applicable, were weighed and completed a fatigue questionnaire. When this was completed subjects could drink *ad libitum*.

**Figure 3.** Schematic overview of the experimental trial timeline. Numbers are in min; HR, heart rate; T_c, core body temperature; RPE, rating of perceived exertion; * indicates if applicable; SMVC, sustained maximal voluntary contraction.

### 3.3 Familiarization trial

Goaltenders performed one familiarization trial where they were provided *ad libitum* water during breaks in play. The purposes of this trial were to calculate the sweat loss of each goaltender and to familiarize all participants with the procedures. The latter was necessary to remove any learning effect because goaltenders played the 3-on-3 scrimmage with no crease. The crease is important because it is a tool used by goaltenders to navigate their position relative to the goal net.

### 3.4 Experimental protocol

Goaltenders were instructed to consume the same foods and drinks on each trial day, and to finish their final meal 2-3 hours pre-skate. Caffeine intake was not restricted but was consumed
at the same time before each trial. Subjects were instructed not to change their diet or activity level during the study duration and abstain from alcohol and strenuous exercise the day before and the day of the trial. Three hours before the skate began, goaltenders ingested a core temperature (T<sub>c</sub>) thermistor with 250 mL of water (HQInc, Palmetto, FL). The subjects were then instructed to drink 700 mL of water at 60-90 min before the skate began (~45 min before arriving at rink). These were confirmed by a self-completed dietary recall on the day of all trials.

Subjects arrived at the University of Guelph arena (T<sub>a</sub>, 4.1 ± 0.5°C, RH: 63 ± 2%, Table 2) 45-60 min before the skate started. Upon arrival, goaltenders completely emptied their bladder and a ~100 mL urine sample was collected to determine hydration status (See section 3.5 for details). Participants were then weighed to obtain BM wearing only underwear, the heart rate (HR) equipment was applied (RS400, Polar Electro Canada, Lachine, QC) and T<sub>c</sub> was recorded (Section 3.5). A sweat patch (3M Tegaderm + Pad, London, ON) was applied to the goaltender’s forehead, and they completed a dietary recall for the day of the trial. While putting their equipment on in the dressing room they consumed 250 mL of water during the ~45 min prior to the skate.

**Table 2.** Arena temperature (T<sub>a</sub>) and relative humidity (RH) in the NF (no fluid), water (WAT) and carbohydrate-electrolyte solution (CES) conditions for all goaltenders (n = 11).

<table>
<thead>
<tr>
<th>Goaltender</th>
<th>NF T&lt;sub&gt;a&lt;/sub&gt; (°C)</th>
<th>NF RH (%)</th>
<th>WAT T&lt;sub&gt;a&lt;/sub&gt; (°C)</th>
<th>WAT RH (%)</th>
<th>CES T&lt;sub&gt;a&lt;/sub&gt; (°C)</th>
<th>CES RH (%)</th>
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</tr>
<tr>
<td>Mean</td>
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<td>64.5 ± 4.0</td>
<td>3.4 ± 0.3</td>
<td>57.5 ± 2.6</td>
<td>3.9 ± 0.5</td>
<td>64.1 ± 2.1</td>
</tr>
</tbody>
</table>
Goaltenders then performed the SMVC protocol 10 min before the skate. The 120 sec SMVC was performed once per hand, separated by 1 min of rest, starting with the stick holding hand and finishing with the glove holding hand. The maximal hand grip exercise was performed wearing full equipment except for gloves. Participants performed the SMVC seated with their shoulder in neutral position, 90° elbow flexion, and the wrist 90° pronated with no flexion. The LoggerPro software (Vernier Software & Technology, Beaverton, OR) was zeroed while the hand grip dynamometer (Vernier Software & Technology) was held without contraction. The subjects were asked to begin the contraction and were verbally encouraged throughout the task. Force data were recorded at 10 Hz throughout.

The on-ice component of the study consisted of a controlled 10-min warmup, a 70-min scrimmage, a shootout drill, a shooting drill and a lateral movement drill (Figure 3).

**Warmup.** The 10 min warm-up included two, 1 min movement drills that were separated by 1 min rest. These required consistent movement and navigation around the net in a similar fashion that occurs in a game. The remainder of the warm-up was spent stretching or casual skating that was not physically demanding.

**Scrimmage.** Everything on-ice from this point forward was filmed. Ten min after stepping onto the ice the 3-on-3 scrimmage began. Each team had a goaltender and three skaters playing at once. Skaters switched between playing and resting as they chose and there was an average of 5.2 ± 1.0 skaters per team (range: 3-7 skaters) for the trials. The scrimmage was played on a regular rink but across the ice from the end boards to the blue line with the nets placed in a middle position (Figure 4). If the puck passed the blue-line it was considered out of bounds and the team that did not touch the puck last received possession in their defensive end. Following a goal or if the goaltender covered the puck, the defensive team received possession and the offensive team
retreated to the half-way point of the rink. All penalties were penalty shots. Six, 10 min intervals were played each separated by 2 min rest. An investigator did the timing, put pucks into play and called penalties. A second investigator recorded shots on net and goals. During rest periods RPE and $T_c$ were recorded by the two investigators and then goaltenders consumed fluid if applicable. The forehead sweat patch was removed during the third or fourth rest period.

![Scrimmage Setup Diagram](image)

**Figure 4.** Overhead image of the scrimmage set up. The solid blue line represents a boundary.

**Shootout.** During the final scrimmage rest period, the goal nets were repositioned to their standard spot on the ice. The two goaltenders then each received three rounds of shot attempts, 12 attempts per round for a total of 36 attempts. They alternated between receiving shots and resting. During the rest periods RPE and $T_c$ were recorded followed by fluid consumption if applicable. A shot attempt consisted of the shooter skating uninterrupted from the center of the ice towards the goal net with the intention of scoring. The attempt ended after the puck was shot, regardless of the outcome, and the next shooter would take their attempt. This would repeat until the goaltender received 12 shots. Each shooter was instructed to take at least one shot and make one deke attempt of their three attempts per goaltender (goaltenders were unaware of this instruction). Goaltenders
were simply instructed to prevent the puck from entering the net, but not specifically to control rebounds as in a game shootout situation. Data were recorded on-ice by an investigator and confirmed by film. The entire shootout lasted ~12 min.

**Shooting drill.** The shooting drill required two passers located beside the net behind the goal line and two shooters positioned by the faceoff circle (Figure 5). Each goaltender faced the same shooters for all trials (hockey experience = Jr. A – NCAA D1). In cases where goaltenders and skaters could not be matched, the skater was replaced by another with similar shooting ability as assessed by the lead investigator. Prior to trials and the drill, goaltenders were instructed to control rebounds like they would in a game situation and they were informed of all the potential drill scenarios (Figure 6). To begin a sequence, the goaltender would start on the goal post closest to the passer. Passers were instructed to make at least two passes to each shooter in a random order per set (goaltenders were unaware of this instruction). The first passer would pass the puck to either of the two shooters and the goaltender would react to the pass by moving to a position to make a save. The shooter receiving the initial pass could either shoot the puck on goal or pass the puck to the other shooter, however if the latter occurred, the shooter receiving the pass would have to shoot the puck on goal. A sequence was terminated if: (1) the shot on goal resulted in a goal, (2) the goaltender covered the puck, or (3) if the puck rebounded off the goaltender out of the shooters reach. If a rebound was within reach of the shooter, the shooter was to shoot the puck on goal immediately. Following this shot a sequence was terminated regardless of outcome. Following a terminated sequence, the goaltender immediately retreated back to the post opposite to the one they started on. The next sequence began immediately with a pass from the other passer. This continued until 10 sequences were completed and then the other goaltender took over. During the rest phase, \( T_c \) and RPE were recorded followed by fluid consumption if applicable. Each set
contained 10 shooting sequences and a rest period. The shooting drill was then repeated so each
goaltender completed the drill twice, receiving two sets of 10 sequences for a total of 20 sequences.

There was no tabulation on-ice, data were obtained from the film.

**Figure 5.** Image outlining the set up for the shooting drill. The drill contained one goaltender
(G), two passers (P) and two shooters (S). The drill was filmed by a camera (C).

**Figure 6.** A flowchart outlining the potential situations during one sequence of shots in the
shooting drill. Following a “sequence terminated” the goaltender retreated to the opposite goal
post they started on and when this happened another sequence began. No rebound* indicates either
a puck that did not rebound off the goalie or rebounded out of the skaters’ reach.

**Lateral movement drill.** The goaltenders completed the lateral movement drill at the end
of the rink opposite the shooting drill. A string connected to the 1080 Sprint (1080 Motion,
Lidingo, Sweden) was tethered to a loop on the goaltender’s pants via a carabiner (Figure 7). If no loop was accessible on the goaltender’s pants, a belt was placed around their waist and the string was tethered to the belt. The 1080 Sprint was set to isotonic mode with the lowest resistance (resistance = 1-N, assistance = 1-N) and with maximal string recoil (velocity = 14 m/sec, acceleration = 14 m/sec²). These settings allowed goaltenders to move as if they were not tethered and to keep the tethered string taught. The 1080 Sprint was zeroed ≥3 m away from the goaltender’s starting position to maintain a taught string. Velocity, acceleration and force were recorded at 1000 Hz and uploaded to an online data base (1080 Motion). Goaltenders performed 10 lateral standing shuffle pushes (shuffles) in each direction per set for a total of 20 shuffles per set. The length of each shuffle was the width of the net, 1.83 m. Each shuffle had goaltenders start on one goal post with their chest facing perpendicular to their plane of movement, the goaltender then moved laterally to the other post after the researcher’s cue. When the goaltender reached a complete stop, the researcher would give a subsequent push cue. This set of 20 shuffles was repeated twice, where goaltenders alternated between pushing and resting. During rest Tc and RPE were recorded followed by fluid consumption if applicable.

Figure 7. Photograph of a goaltender in the lateral movement drill immediately before (left) and after (right) performing a lateral shuffle push. The 1080 Sprint (not in image) is tethered to the goaltender’s pant loop via carabiner and string (highlighted in orange).
Post-skate. Goaltenders returned to the dressing room after they finished the lateral movement drill. The SMVC protocol was immediately performed in an identical fashion as pre-skate. Goaltenders then removed their equipment and completely voided their bladder into a cup (if needed) and the volume was measured. They were then weighed wearing the same clothing as pre-skate. Before leaving, goaltenders completed the post-skate questionnaire (post-Q) and indicated the solution they consumed if they felt they could correctly identify it. Fluid was completely restricted until the post-Q was finished.

3.5 Measurements
Urine specific gravity (USG) was obtained by submerging a refractometer (Atago USA Inc., Bellevue, WA) into the urine sample. RPE was recorded using the 20-point Borg scale (Borg et al. 1982). $T_c$ was recorded via a telemetric thermistor that communicated with the data acquisition device when it was placed near the goaltender’s low back (CorTemp). The HR watch was calibrated to the arena clock, and HR data were collected throughout the trials. The forehead sweat patch was centrifuged post-scrimmage and the aliquot was run through a conductivity analyzer (ELITechGroup North America, Princeton, NJ) to obtain forehead sweat [Na$^+$]. For the SMVC measure, the area under the force x time curve was obtained for the entire 120 sec and selected time periods via Logger Pro software (Vernier Software & Technology). The post-Q contained 7 questions relating to their perceived on-ice fatigue (Table 7). Goaltenders circled an integer from 1-10 corresponding to their perceived fatigue. A score of 1 indicated very little fatigue, 5 was moderate fatigue, and 10 was extremely fatigued.
3.6 Calculations

Percent body mass loss was calculated as \((\text{pre BM} - \text{post BM})/\text{pre BM}\) x 100 and sweat rate = \((\text{pre BM} - \text{post BM}) + \text{fluid intake} - \text{urine output}\). Forehead \([\text{Na}^+]\) in mM was corrected to accurately estimate whole body sweat \([\text{Na}^+]\) loss \(([\text{Na}^+_{\text{forehead}}] \times 0.61 - 2.98, \text{Baker et al. 2009})\). Whole body \([\text{Na}^+]\) loss (g) equaled the volume of sweat loss x molecular mass of sodium x corrected forehead \([\text{Na}^+]\). Sodium consumed was molecular mass of sodium x \([\text{Na}^+]\) in CES (18 mM) x volume of CES consumed. CHO consumed was 6 g CHO/100 mL of CES drank. Save percentage (SVP) was calculated as saves divided by total shots (shots saved and goals) x 100. Instantaneous force and velocity obtained from the 1080 Sprint were multiplied to calculate power.

3.7 Analyses

**Video analysis.** The entire on-ice portion of every trial was video recorded (HDR-CX405BKIT, Sony, Toronto, ON). In the scrimmage shots were categorized by: game situation, i.e. breakaway, 3-on-2, 2-on-1, shot off a rebound attempt, etc.; shot or deke attempt; whether there was a preceding pass; if there was a preceding pass, whether the pass was stopped and held before shooting or not (one-timed); goaltender visual occlusion; initial shot deflected; and the location of the rebound. Rebounds were classified four ways: (1) puck did not rebound off goaltender, i.e. caught with glove; (2) puck rebounded off goaltender but was within reach to cover; (3) puck rebounded off goaltender out of their reach but was directly in front of them, and (4) puck rebounded off goaltender out of their reach and was off to either side. In the shootout, attempts were categorized as a shot or deke attempt. For the shooting drill, video was analyzed frame-by-frame to determine the following for each sequence: time the first pass was initiated to the time the goaltender began moving their leg (reaction time), from the first leg movement to the onset of
movement (movement generation), the time to push from the post to the final position to make save (push movement duration), and the time the pass was initiated to time when the goaltender finished their movement (full movement duration). These were further categorized by whether the pass crossed the goaltender’s body.

**1080 Sprint data analyses.** In the lateral movement drill, data were only usable from shuffle pushes away from the 1080 Sprint and therefore data represent 10 shuffles per set. Peak velocity, force, power and acceleration were determined for each shuffle. Individual shuffles were averaged into set one and set two as well as shuffles 1-3, 4-7, and 8-10 within set 1 and 2. All shuffle data were analyzed using a custom Matlab software (The Mathworks, Natick, MA).

**Statistical analysis.** Normality was tested with Q-Q plots and a Shapiro-Wilks test. Variables recorded at multiple time points were analyzed using a repeated measures (RM) two-way ANOVA (condition x time). Any variable obtained at a single time point was compared between conditions using a RM one-way ANOVA (condition). A significant F test was followed up with a Tukey’s HSD post-hoc analysis to compare means between conditions within a time point. A Sidak post-hoc test was used to examine differences in means within a condition between times. Statistical analyses were performed using Prism (v7.0, Graphpad, La Jolla, CA). All values are reported as mean ± standard error (SE) and significance was set as \( p \leq 0.05 \).
CHAPTER IV: RESULTS

**Dietary standardizations.** Pre-skate meal contents and fluid intake were the same for all trials. Two subjects consumed the same volume and brand of coffee 2.5 hours prior to the beginning of all trials.

**Blinding effectiveness.** Goaltenders could not be blinded to the NF condition. All goaltenders felt they could correctly identify the fluid they consumed on the ice after all fluid trials. After the first fluid trial, 1/5 correctly identified WAT and 5/6 correctly identified the CES (Total: 6/11). After the second fluid trial, 6/6 correctly identified the WAT and 3/5 the CES (Total: 8/11).

### 4.1 Hydration

Mean pre-skate USG, BM and sweat loss were not significantly different between trials (Table 3). On-ice fluid intake was similar between WAT and CES conditions but significantly lower in the NF condition. Therefore, BM loss was minimal and similar when fluid was ingested and significantly greater in the NF condition (Table 3). One goaltender was allowed to consume ~900 mL of water pre-skate in all trials and 425 mL on-ice during the NF condition to offset a very high sweat loss. The mean sweat [Na⁺] obtained from the forehead patches was 68 ± 6 mM and the estimated whole-body Na⁺ losses were not different between conditions. However, mean Na⁺ balance was significantly greater in CES condition compared to NF and WAT conditions (NF: -1749 ± 308 mg, WAT: -1689 ± 280 mg, CES: -1019 ± 245 mg). CHO was only consumed in the CES condition (99 ± 10 g).
Table 3. Hydration and sweat data for the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions.

<table>
<thead>
<tr>
<th></th>
<th>NF</th>
<th>WAT</th>
<th>CES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-skate USG</td>
<td>1.011 ± 0.003</td>
<td>1.013 ± 0.002</td>
<td>1.014 ± 0.003</td>
</tr>
<tr>
<td>Pre-skate BM (kg)</td>
<td>82.1 ± 4.6</td>
<td>82.4 ± 4.7</td>
<td>81.8 ± 4.9</td>
</tr>
<tr>
<td>Pre-skate fluid intake (L)</td>
<td>0.30 ± 0.70</td>
<td>0.28 ± 0.66</td>
<td>0.30 ± 0.61</td>
</tr>
<tr>
<td>On-ice fluid intake (L)</td>
<td>0.039 ± 0.39*</td>
<td>1.50 ± 0.14</td>
<td>1.65 ± 0.16</td>
</tr>
<tr>
<td>Sweat loss (L)</td>
<td>1.95 ± 0.28</td>
<td>1.96 ± 0.21</td>
<td>1.90 ± 0.27</td>
</tr>
<tr>
<td>BM Loss (%)</td>
<td>2.4 ± 0.3*</td>
<td>0.39 ± 0.2</td>
<td>0.39 ± 0.1</td>
</tr>
<tr>
<td>Whole body Na+ loss (mg)</td>
<td>-1749 ± 308</td>
<td>-1689 ± 280</td>
<td>-1749 ± 291</td>
</tr>
</tbody>
</table>

Notes: One goaltender consumed 425 mL of water during the NF trial, all others consumed 0 mL. Data are presented as mean ± SE (n = 11). *, different from WAT and CES. USG, urine specific gravity; BM, body mass.

4.2 Physiology

Core temperature. $T_c$ data were recorded in 9, 7 and 6 goaltenders during the NF, WAT and CES conditions, respectively. A full $T_c$ data set was obtained from n = 5, which showed significantly greater $T_c$ in the NF condition vs. WAT and CES from 44-min until the end of the skate (Figure 8). Interestingly, $T_c$ at 32 min trended towards significantly greater in the NF condition vs. CES (p = 0.053) but not WAT (p = 0.74). Peak $T_c$ was significantly lower when fluid was ingested vs. NF at any time on-ice (NF: 39.1 ± 0.1°C, WAT: 38.6 ± 0.1°C, CES: 38.5 ± 0.1°C), during the scrimmage (NF: 38.9 ± 0.2°C, WAT: 38.5 ± 0.1°C, CES: 38.4 ± 0.1°C) and in post-scrimmage drills (NF: 39.0 ± 0.1°C, WAT: 38.4 ± 0.1°C, CES: 38.4 ± 0.1°C). Similarly, mean $T_c$ was significantly lower when fluid was ingested vs. NF throughout the scrimmage (NF: 38.7 ± 0.1°C, WAT: 38.3 ± 0.1°C, CES: 38.2 ± 0.1°C) and in post-scrimmage drills (NF: 38.9 ± 0.2°C, WAT: 38.3 ± 0.1°C, CES: 38.2 ± 0.1°C). $T_c$ in the NF condition vs. WAT and CES separately had the same trends as when all 3 conditions were compared (Figure 9). Mean and peak $T_c$ in the NF condition compared to WAT and CES individually (n = 7 and n = 6 respectively) showed the same trends as when all 3 conditions were compared. A significant correlation was found between peak $T_c$ and % BM loss in the NF condition (Figure 10).
Figure 8. Core body temperature ($T_c$) pre-skate, in the scrimmage, shootout (SO), shooting drill (D1) and the lateral movement drill (D2) in the no fluid (NF), water (WAT), and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 5). *, NF different from WAT and CES.

Figure 9. Core body temperature ($T_c$) pre-skate, in the 3-on-3 scrimmage, shootout (SO), shooting drill (D1) and the lateral movement drill (D2) in the no fluid (NF) and water (WAT) conditions (top, n = 7) and the NF and carbohydrate-electrolyte solution (CES) conditions (bottom, n = 6). Values are mean ± SE. *, NF different from WAT or CES.
Heart rate. Mean HR during activity was significantly greater in the NF vs. WAT condition during the second half of the scrimmage and the shootout and vs. the CES condition in the final 20 scrimmage minutes and in the last two shootout rounds (Figure 11). Interestingly, mean HR was significantly greater in the CES condition in the first shootout round compared to the WAT condition. Mean HR was similar between conditions in the first half of the scrimmage (NF: 151 ± 6, WAT: 151 ± 7, CES: 153 ± 6 bpm) but was significantly greater in the NF condition vs. both fluid conditions in the second half of the scrimmage (NF: 157 ± 7, WAT: 148 ± 7, CES: 150 ± 6 bpm, Figure 12). When mean HR was analyzed post-scrimmage, all conditions were different during the shootout (NF: 177 ± 5, WAT: 166 ± 6, CES: 173 ± 5 bpm), were similar in the shooting drill (NF: 180 ± 4, WAT: 177 ± 5, CES: 178 ± 5 bpm) and NF was greater in the lateral movement drill vs. both fluid conditions (NF: 175 ± 4, WAT: 171 ± 5, CES: 172 ± 4 bpm, Figure 12). Peak and recovery HR were unaffected by condition.
Figure 11. Active mean heart rate (HR) during the scrimmage, shootout (SO), shooting drill (D1) and the lateral movement drill (D2) in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). *, NF different from WAT and CES; #, NF different from WAT; Ψ, WAT different from CES.

Figure 12. Active mean HR during the shootout (SO), shooting drill (D1) and lateral movement drill (D2) in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). *, NF different from WAT and CES; Ψ, WAT different from CES.

4.3 Fatigue

Perceived fatigue. RPE was significantly greater in the NF condition compared to WAT and CES conditions 68 min into the skate (Figure 13). Interestingly, post-scrimmage (88-min) until the end of the skate, only the CES condition had lower RPE scores vs. the NF condition. Mean
RPE was not different between conditions during the scrimmage (NF: 13.1 ± 0.6, WAT: 12.7 ± 0.6, CES: 12.8 ± 0.5) but was post-scrimmage such that CES<WAT<NF (NF: 16.6 ± 0.6, WAT: 16.0 ± 0.5, CES: 15.4 ± 0.8, Figure 14). When only analyzing post-scrimmage drills, mean RPE was significantly greater in the NF condition vs. the CES condition only. Peak RPE was similar between conditions during the scrimmage (NF: 15.2 ± 0.7, WAT = 14.5 ± 0.6, CES: 14.5 ± 0.6) and post-scrimmage (NF: 17.9 ± 0.5, WAT: 17.6 ± 0.6, CES: 16.7 ± 0.7).

Mean of all post-Q scores were significantly greater in the NF condition (6.2 ± 0.5) compared to fluid conditions (WAT: 4.8 ± 0.6, CES: 4.7 ± 0.5, Figure 15). Lightheaded scores were significantly reduced in the WAT and CES conditions compared to NF but no other individual question reached significance (Table 4). Individual question scores in the NF condition were compared to WAT and CES separately and showed that CES significantly reduced lightheaded, winded, overheated, whole-body fatigue and leg fatigue scores; and WAT significantly reduced winded and lightheaded scores.

Figure 13. Rating of perceived exertion (RPE) during the scrimmage, shootout (SO), shooting drill (D1) and the lateral movement drill (D2) in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). *, NF different from WAT and CES; #, NF different from CES.
Figure 14. Rating of perceived exertion (RPE) during the scrimmage and post-scrimmage drills in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). *, NF different from WAT and CES; $, different from post-scrimmage.

Figure 15. Post-skate questionnaire (Post-Q) results in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). Score of 1: very little fatigue, 5: moderate fatigue, 10: extreme fatigue. *, NF different from WAT and CES.

Table 4. Post-skate fatigue questionnaire (Post-Q) results in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions.

<table>
<thead>
<tr>
<th>Fatigue element (/10)</th>
<th>NF</th>
<th>WAT</th>
<th>CES</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lightheaded</td>
<td>5.1 ± 0.8*</td>
<td>3.1 ± 0.9</td>
<td>3.0 ± 0.8</td>
<td>0.024</td>
</tr>
<tr>
<td>Winded</td>
<td>5.6 ± 0.7</td>
<td>4.6 ± 0.8</td>
<td>4.0 ± 0.8</td>
<td>0.085</td>
</tr>
<tr>
<td>Overheated</td>
<td>5.9 ± 0.8</td>
<td>4.6 ± 0.9</td>
<td>4.1 ± 0.9</td>
<td>0.14</td>
</tr>
<tr>
<td>Muscles Cramping</td>
<td>5.4 ± 0.8</td>
<td>3.3 ± 0.5</td>
<td>3.8 ± 0.8</td>
<td>0.064</td>
</tr>
<tr>
<td>Whole-body Fatigue</td>
<td>7.6 ± 0.4</td>
<td>6.3 ± 0.6</td>
<td>6.5 ± 0.6</td>
<td>0.11</td>
</tr>
<tr>
<td>Leg Fatigue</td>
<td>7.6 ± 0.3</td>
<td>6.7 ± 0.5</td>
<td>6.3 ± 0.6</td>
<td>0.13</td>
</tr>
<tr>
<td>Arm fatigue</td>
<td>5.9 ± 0.5</td>
<td>5.2 ± 0.7</td>
<td>5.1 ± 0.3</td>
<td>0.34</td>
</tr>
</tbody>
</table>

Notes: Data are presented as mean ± SE (n = 11). Reported p-values are from repeated measures one-way ANOVA. *, post-hoc test NF different from WAT and CES.
**Sustained Maximal Voluntary Contraction.** The trends for area under the force x time curve at 60, 90 and 120 sec (AUC₁₂₀) yielded similar results; therefore, all area under the curve data are presented as AUC₁₂₀. The coefficient of variation for pre-skate AUC₁₂₀ was 8.0 ± 1.5% and 10.0 ± 1.2% in the glove and stick hands respectively. Force produced during the SMVC decreased over time but plateaued at 80 sec in the glove hand and 70 sec in the stick hand (Figure 16). Pre-skate AUC₁₂₀ was similar between conditions in the glove hand but was significantly greater in the NF compared to WAT and CES conditions in the stick hand (Table 5). In both hands, post-skate AUC₁₂₀ was significantly lower than pre-skate in all conditions. Post-skate AUC₁₂₀, both absolute and relative to pre-skate, were unaffected by condition in both hands (Figure 17, Table 5). Relative AUC₁₂₀ in the glove hand was 76.6 ± 4.1%, 82.7 ± 2.8 and 83.3 ± 3.8% in the NF, WAT and CES conditions, respectively. In the same order for the stick hand, relative AUC₁₂₀ was 73.3 ± 4.7%, 82.5 ± 4.4% and 78.7 ± 2.7%. No measure of CF was correlated to % BM loss, Tₑ or USG.

![Figure 16](image-url) Force produced during the pre-skate 120 sec sustained maximal voluntary contraction in the stick and glove hands in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). *, not different from 120 sec.
Table 5. Area under the entire force*time curve (AUC, N*sec) for the 120-sec sustained maximal handgrip contraction (SMVC) performed pre- and post-skate with the glove and stick hands in the no fluid (NF), water (H₂O) and carbohydrate-electrolyte solution (CES) conditions.

<table>
<thead>
<tr>
<th></th>
<th>Pre-skate</th>
<th>Post-skate⁵</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glove</td>
<td>18295 ± 1136ᵃ</td>
<td>14223 ± 1324</td>
</tr>
<tr>
<td>Stick</td>
<td>20623 ± 1088 *</td>
<td>15048 ± 1125</td>
</tr>
<tr>
<td>WAT</td>
<td>17936 ± 1030</td>
<td>14920 ± 1073</td>
</tr>
<tr>
<td></td>
<td>18438 ± 1028</td>
<td>15173 ± 948</td>
</tr>
<tr>
<td>Glove</td>
<td>17599 ± 1026ᵃ</td>
<td>14812 ± 1177</td>
</tr>
<tr>
<td>Stick</td>
<td>19037 ± 918</td>
<td>15055 ± 955</td>
</tr>
</tbody>
</table>

Notes: Values are mean ± SE (n = 11).ᵃ, glove different than stick hand within a condition; *, NF different from CES and WAT in the stick hand pre-skate; ⁵, post-skate different than pre-skate in both hands and all conditions.

Figure 17. Area under the force*time curve (AUC) every 10-sec during the sustained maximal voluntary contraction for the glove and stick hands post-skate relative to pre-skate in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). No significant findings existed.

Peak force was unaffected by condition pre- or post-skate in the stick hand (Table 6). In the glove hand, peak force was similar between conditions post-skate but was significantly lower pre-skate in the CES condition vs. NF and WAT. Peak force was significantly greater pre-skate vs. post-skate in all conditions and in both hands. Peak force post-skate relative to pre-skate was similar between conditions in the stick hand but was significantly greater in the glove hand in the CES condition vs. WAT and NF.
Table 6. Peak force produced during the 120-sec sustained maximal voluntary contraction pre- and post-skate as well as post-skate relative to pre-skate in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions.

<table>
<thead>
<tr>
<th></th>
<th>Pre-skate (N)</th>
<th>Post-skate (N) $^*$</th>
<th>Relative Peak Force (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NF</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glove</td>
<td>334 ± 12</td>
<td>285 ± 18</td>
<td>84.6 ± 3.7</td>
</tr>
<tr>
<td>Stick</td>
<td>344 ± 9</td>
<td>309 ± 13</td>
<td>89.9 ± 3.0</td>
</tr>
<tr>
<td><strong>WAT</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glove</td>
<td>329 ± 15</td>
<td>277 ± 16</td>
<td>83.8 ± 2.4</td>
</tr>
<tr>
<td>Stick</td>
<td>320 ± 15</td>
<td>308 ± 13</td>
<td>96.9 ± 3.0</td>
</tr>
<tr>
<td><strong>CES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glove</td>
<td>309 ± 16$^*$</td>
<td>285 ± 16</td>
<td>91.9 ± 2.5$^*$</td>
</tr>
<tr>
<td>Stick</td>
<td>321 ± 14</td>
<td>293 ± 15</td>
<td>91.6 ± 3.7</td>
</tr>
</tbody>
</table>

*Notes: Data are presented as mean ± SE (n = 11). N = newtons. $^*$ post-skate different than pre-skate in both hands and all conditions; $^*$, CES different from WAT and CES in the glove hand.

4.4 Performance

**Scrimmage.** Shot attempts were similar between conditions throughout the scrimmage. There was no difference in SVP when all six scrimmage intervals were compared. However, to test the hypothesis that the effects of DEH would appear over time, SVP during the last 10 scrimmage min were compared the first 10 (Figures 18 and 19). Mean SVP was similar for all conditions in the first 10 min (NF: 81.5 ± 1.9%, WAT: 78.8 ± 1.9%, CES: 79.0 ± 1.9%) but was significantly lower in NF vs. WAT and CES conditions during the last 10 min (NF: 75.8 ± 1.9%, WAT: 81.7 ± 2.3%, CES: 81.3 ± 2.3%). Correspondingly, mean SVP was only significantly changed in the final vs. first 10 min of the 3-on-3 scrimmage in the NF condition. There were no significant effects for SVP when comparing the final 20 min of scrimmage to the first 10 or when scrimmage segments 0-10, 48-58 and 60-70 min were compared. Similarly, there was no effect of condition or time on scrimmage SVP when shots were categorized by: 1) the different game situations, 2) if a pass preceded the shot, 3) if the preceding pass went across the goaltender’s body, 4) shot type (one-timer, shot/deke attempt or deflected shot), or 5) when the goaltender was
visually occluded. Scrimmage SVP when categorized in any of the 5 ways above was not affected by condition or time under the following comparisons: all six scrimmage intervals; and scrimmage minutes 0-10 vs. 60-70, 0-10 vs. 48-70, and 0-10 vs. 48-58 vs. 60-70. Likewise, rebound control in the scrimmage was similar between conditions and times.

Figure 18. Save percentage in the first and last 10 min of a 70 min 3-on-3 scrimmage in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11). *, NF different from WAT and CES at 60-70 min; $, 0-10 min different from 60-70 min in the NF condition.

Figure 19. Individual save percentage in the final 10-min relative to the first 10-min of the scrimmage in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions (n = 11).
**Shootout.** On average there was significantly more deke attempts than shot attempts (21 ± 1 vs. 15 ± 1) but these proportions were similar between conditions. Shootout SVP was unaffected by condition or shooting round (Figure 20). In round one SVP was 79.5 ± 3.5%, 72.2 ± 2.8% and 70.2 ± 4.4% in the NF, WAT and CES conditions, respectively. In the same order SVP was 75.7 ± 4.5%, 68.5 ± 4.2% and 77.9 ± 4.8% in round two and 72.8 ± 4.9% 75.5 ± 3.8% and 70.3 ± 4.7% in round three. Similarly, SVP on shot (NF: 75.4 ± 3.3%, WAT: 72.9 ± 3.0%, CES: 67.8 ± 5.2%) and deke (NF: 75.1 ± 4.2%, WAT: 74.5 ± 4.4%, CES: 74.0 ± 4.2%) attempts was unaffected by condition (Figure 21).

**Figure 20.** Save percentage on all attempts during the shootout from each of the three rounds in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11).

**Figure 21.** Save percentage from all three shootout rounds when categorized into shot and deke attempts in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions. Values are mean ± SE (n = 11).
**Shooting Drill.** SVP and rebound location in the shooting drill were similar between rounds and unaffected by condition in all sequences, sequences resulting in shots on goal, sequences with or without a pass. Similarly, rebound location was unaffected by shooting round or condition. Compared to the NF condition, reaction time to the initial pass was shorter in both fluid trials and movement generation was shorter compared to CES only (Table 7). There was no effect of condition on full movement duration, but the push movement duration was shorter in WAT vs. both CES and NF conditions in both sequences. Table 7. Shooting drill movement durations (msec) during the first and second sequence in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions.

<table>
<thead>
<tr>
<th></th>
<th>NF</th>
<th>WAT</th>
<th>CES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sequence #1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reaction to pass</td>
<td>0.32 ± 0.03 *</td>
<td>0.26 ± 0.01</td>
<td>0.28 ± 0.04</td>
</tr>
<tr>
<td>Movement generation</td>
<td>0.62 ± 0.04 #</td>
<td>0.59 ± 0.04</td>
<td>0.53 ± 0.02</td>
</tr>
<tr>
<td>Push movement</td>
<td>0.52 ± 0.01 *</td>
<td>0.49 ± 0.02 b</td>
<td>0.52 ± 0.01 a</td>
</tr>
<tr>
<td>Full movement</td>
<td>1.10 ± 0.02</td>
<td>1.05 ± 0.02</td>
<td>1.08 ± 0.01</td>
</tr>
<tr>
<td><strong>Sequence #2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reaction to pass</td>
<td>0.30 ± 0.03*</td>
<td>0.23 ± 0.01</td>
<td>0.25 ± 0.02</td>
</tr>
<tr>
<td>Movement generation</td>
<td>0.61 ± 0.04#</td>
<td>0.57 ± 0.05</td>
<td>0.54 ± 0.02</td>
</tr>
<tr>
<td>Push movement</td>
<td>0.52 ± 0.01 a</td>
<td>0.49 ± 0.02 b</td>
<td>0.56 ± 0.02 c</td>
</tr>
<tr>
<td>Full movement</td>
<td>1.11 ± 0.02</td>
<td>1.06 ± 0.04</td>
<td>1.10 ± 0.02</td>
</tr>
</tbody>
</table>

Notes: Data are presented in milliseconds as mean ± SE (n = 11). *, significant main effect NF vs. WAT and CES; #, significant main effect: NF vs. CES; different letters indicate significant differences between conditions.

**Lateral movement drill.** Data was unsuccessfully obtained from one goaltender in the NF condition and therefore the lateral movement drill represents n = 10. All metrics of peak force, velocity and acceleration were unaffected by condition (Table 8), although the mean data showed a trend for peak velocity to be greater in CES vs. WAT and NF in sets 1 and 2. Peak power in set one was significantly greater in the CES condition compared to NF. Compared to NF, significantly more power was produced in the WAT condition during pushes 8-10 of set 1
and 1-3 of set 2 and in the CES condition during pushes 1-3, 4-7 and 8-10 of set 1 (Figure 22). Peak power was similar in pushes 1-3, 4-7 and 8-10 in all conditions within set 1 and 2.

Table 8. Mean peak power, force, velocity, and acceleration for all 10 shuffle pushes from sets one and two during the lateral movement drill in no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions.

<table>
<thead>
<tr>
<th></th>
<th>NF</th>
<th>WAT</th>
<th>CES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Set #1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Power (W)</td>
<td>46.4 ± 1.7</td>
<td>48.0 ± 2.7</td>
<td>50.4 ± 3.0*</td>
</tr>
<tr>
<td>Peak Force (N)</td>
<td>28.9 ± 0.4</td>
<td>27.2 ± 0.7</td>
<td>28.3 ± 1.3</td>
</tr>
<tr>
<td>Peak Velocity (m*sec⁻¹)</td>
<td>1.90 ± 0.05</td>
<td>1.97 ± 0.06</td>
<td>2.00 ± 0.07</td>
</tr>
<tr>
<td>Peak Acceleration (m*sec⁻²)</td>
<td>10.53 ± 0.57</td>
<td>10.25 ± 0.81</td>
<td>10.87 ± 1.10</td>
</tr>
<tr>
<td><strong>Set #2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Power</td>
<td>49.4 ± 2.9</td>
<td>50.5 ± 3.5</td>
<td>49.0 ± 3.4</td>
</tr>
<tr>
<td>Peak Force (N)</td>
<td>27.7 ± 0.9</td>
<td>28.2 ± 1.0</td>
<td>27.4 ± 0.7</td>
</tr>
<tr>
<td>Peak Velocity (m*sec⁻¹)</td>
<td>1.94 ± 0.07</td>
<td>2.03 ± 0.08</td>
<td>1.98 ± 0.08</td>
</tr>
<tr>
<td>Peak Acceleration (m*sec⁻²)</td>
<td>10.84 ± 0.86</td>
<td>11.02 ± 1.19</td>
<td>10.39 ± 0.72</td>
</tr>
</tbody>
</table>

*Notes: Values are mean ± SE (n = 10). *, different from NF.*

Figure 22. Mean peak power of lateral shuffle pushes 1-3, 4-7 and 8-10 during sets 1 and 2 from the lateral movement drill in the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) trials. Values are mean ± SE (n = 10). *, different from NF.
CHAPTER V: DISCUSSION

This study tested the effects of mild DEH on performance, thermoregulation and multiple aspects of fatigue during an on-ice scrimmage and drills in ice hockey goaltenders. Previous unpublished research showed that professional ice hockey goaltenders reached mild DEH (>2% BM loss) via sweating during practices (Table 1). This level of mild DEH was achieved in the current study by prohibiting fluid intake and thermoregulation, cardiovascular stress, perceived fatigue, scrimmage performance, reaction time and movement power were all worse compared to maintaining hydration with water or CES. Furthermore, consuming a CES was superior to water at reducing perceived fatigue and improving aspects of on-ice movement.

5.1 Hydration

Pre-exercise hydration. The goal of this research was to examine DEH during on-ice hockey playing when beginning the study in a hydrated state (USG < 1.020, Sawka et al. 2007). The average USG measured in all 3 conditions suggested that this was achieved (Table 3), although there was more variability than anticipated (see Appendix A). To better control for between-trial variability in pre-exercise hydration status, future research should have participants arrive at the arena 90 min pre-skate and immediately consume ~600 mL of fluid in the dressing room under researcher supervision (Logan-Sprenger and Spriet 2013).

The large unpublished data set showed that slightly more than half the goaltenders were HYPO when they arrived at the arena pre-practice (Table 1). Before a game, 2 junior goaltenders were euhydrated upon arrival (Logan-Sprenger et al. 2011) and one professional goaltender was HYPO upon arrival (USG: 1.029). Although about half the goaltenders arrived at practices and games HYPO, we chose to have goaltenders arrive euhydrated for better pre-exercise control.
Since thermoregulation, circulation and performance in a variety of exercises were impaired by pre-exercise HYPO (Armstrong et al. 1997, Savoie et al. 2015) the present findings may have larger effects for goaltenders that arrive at the arena HYPO and fail to rehydrate pre-skate.

**Sweat loss, fluid intake and dehydration.** This study was successful at mildly dehydrating goaltenders (2.4 ± 0.3% BM loss) during a scrimmage and drills. On average, goaltenders would have reached ~1% BM loss during the fourth scrimmage sequence (49 min) and 2% BM loss in the shootout (88 min), assuming a constant sweat rate and sweat onset occurring at the beginning of the scrimmage. The current level of DEH achieved was greater than that of one professional (1.9% BM loss) and one junior goaltender (0.3% BM gain) during games and mean junior and varsity goaltender data during practices (Table 1). However, the magnitude of DEH experienced in the current study was similar to professional goaltenders during practice (Table 1).

Sweat loss was highly variable between goaltenders, which rendered the individual hydration plans necessary. The observed sweat rate (~1.95 L in total or 1.24 L/hr, Table 3) was notably less than professional and junior ice hockey, but similar to varsity goaltenders in practices (Table 1). Absolute sweat loss was also similar to junior goaltenders during a game where they faced a low number of shots (Logan-Sprenger et al. 2011) and much lower than a professional goaltender during a game (4.3 L sweat loss). Comparing the current sweat loss to Table 1 is challenging because of uncontrolled exercise modalities. For example, the varsity goaltenders practiced in a cold environment (Tₐ ~3°C) and the junior and professional goaltenders played in much warmer arenas (Tₐ ~11-18°C) and sweat more (Godek et al. 2006). Since increasing environment temperature increased sweat loss (Pitsiladis and Maughan 1999), the cold arena temperature may explain the present lower sweat rate compared to junior and professional goaltenders. Additionally, training status can partially explain the differences because training in
a hot environment increased sweat rate (Nielsen et al. 1993) and it can be assumed that the skilled goaltenders studied were less trained than professional and junior ice hockey goaltenders. Most importantly, DEH could exceed 2.4% BM loss because of the high sweat losses of the trained professional goaltenders. If this occurred, one would expect greater effects of DEH than reported in the current study, especially in situations where BM loss was small such as the scrimmage.

Both fluid conditions successfully maintained euhydration during the skate (<0.5% BM loss) and the CES provided 758 ± 74 g of Na⁺ and 99 ± 10 g of CHO (~57 g/hr). Sport nutrition guidelines recommend that team sport athletes consume 30-60 g of CHO/hr (Thomas et al. 2016). Therefore, the dose of CHO consumed was on the high end of the recommended range. Although GI symptoms were not recorded, no goaltender complained about GI, which is not surprising given the CHO dose and the glucose, fructose mixture (Jeukendrup 2017). However, it should be noted that GI symptoms during exercise are highly individualized and future research should add questions to the post-Q to assess GI symptoms (De Oliveira et al. 2014).

Although exercise intensity was not measured, it can be assumed that CHO would have been the dominant substrate utilized by skeletal muscle for energy production because of the repetitive explosive movements and intermittent exercise pattern (Parolin et al. 1999). It is also very likely that exogenous CHO oxidation would have increased with CES ingestion, potentially sparing muscle glycogen use during exercise (Tsintzas et al. 1996, Jentjens et al. 2004b). Also, muscle glycogenolysis was increased during DEH by hyperthermia not body water loss (Fernández-Elias et al. 2015), which would suggest that intake of either fluid may have spared glycogen compared to mild DEH. Overall, glycogenolysis may have been different in all conditions such that NF>WAT>CES. However, since glycogen content was correlated to breakdown and self-selected work performed during intermittent exercise (Palmer et al. 2017a),
glycogen utilization may have been greater in the fluid conditions in the later stages of exercise compared to mild DEH because there was more available for use. This phenomenon may have contributed to improved performance when drinking WAT and CES. Additionally, tasting CHO in the CES condition may have also improved performance via central mechanisms (Carter et al. 2004a, Chambers et al. 2009).

5.2 Physiology and fatigue

Thermoregulation. Similar to hydration interventions in ice hockey skaters (Linseman et al. 2014, Boville 2015), mild DEH increased $T_c$ compared to maintaining euhydration in goaltenders when BM loss was $< \sim 1\%$ (Figures 8 and 9). Interestingly, the magnitude of thermoregulatory impairment appeared to be higher in goaltenders. When skaters were mildly DEH (1.9 ± 0.1 and 1.2 ± 0.1% BM loss) peak $T_c$ was increased ~0.3°C compared to maintained BM, while the 2.4 ± 0.3% BM loss experienced by goaltenders in the present study increased peak $T_c$ ~0.6°C. Although goaltenders experienced a ~2-fold greater increase in peak $T_c$ when DEH compared to skaters, they were also more DEH. A positive correlation between DEH level peak $T_c$ has been reported, albeit not in ice hockey skaters (Figure 10, Montain and Coyle 1992a). When peak $T_c$ and % BM loss in skaters and goaltenders was plotted on the same regression line, the data from both groups matched well (Figure 23). Also, when hydration was maintained, peak on-ice $T_c$ was ~38.6°C in both goaltenders and skaters (Linseman et al. 2014, Boville 2015). Thus, the higher $T_c$ observed in goaltenders compared to skaters was a function of DEH level. The data do suggest that the goaltender equipment does not reduce heat dissipation any more than skater equipment does because of the similar $T_c$ data when DEH was equal (Figure 23). However, the additional
sweat loss in goaltenders vs. skaters may be due to their bigger equipment, but it is challenging to compare sweat loss between the two positions because of the very different activity patterns.

Figure 23. Regression line for % body mass (BM) loss in the no fluid condition (NF) and peak core temperature ($T_c$) generated from all goaltenders in the current study (Figure 10). Data points are means ± standard errors from all goaltenders in the current study (n = 9, diamond symbol), the goaltenders that lost <2% BM loss (n = 4, downward triangle) and >2% BM loss (n = 5, upright triangle), as well as the same data from male (n = 14, circle, Linseman et al. 2014) and female skaters (n = 19, square, Boville 2015) during an on-ice scrimmage.

Heart rate. HR was increased at the end of scrimmage and in post-scrimmage drills when goaltenders had lost <1% BM (Figures 11 and 12). This matched the results from steady-state cycling lab experiments, where the effect of DEH on HR appeared at ~1.1 and ~1.5% BM loss (Montain and Coyle 1992a, Logan-Sprenger et al. 2013). However, mild DEH does not always increase HR in field studies (Baker et al. 2007, Edwards et al. 2007), which is usually attributed to low study control. For example, 1.9% DEH in ice-hockey skaters did not affect HR (Linseman et al. 2014), but this may not have been because of unequal work completed in the two conditions. Palmer et al. (2017a, b) compared the effects of mild DEH (1.9% BM loss) to maintaining hydration with WAT or a 6% CES during a lab simulated ice-hockey game for skaters and found that neither hydration intervention affected sprint or recovery HR. Therefore, it may be that the
exercise patterns performed by skaters did not allow HR differences to occur when BM loss was
less than 2%.

Interestingly, DEH increased HR in the shootout and the lateral movement drill but not the
shooting drill. In the shooting drill, goaltenders received fast and difficult shots from a close
distance and the “stress” may have increased plasma epinephrine (Epi) and therefore HR, so much
so that it masked the effect of mild DEH. In line with this theory was that HR was higher in the
shooting drill compared to the shootout where the shot difficulty was lower and in the lateral
movement drill where there were no shots. However, it is challenging to compare the physiological
demand of the drills and it could have been that the shooting drill was the most physiologically
demanding, which would alternately explain the increased HR. Alternately, movement distance
was uncontrolled in the shooting drill, so it is possible that goaltenders covered less distance with
each push when mildly DEH, which would have resulted in a similar HR compared to both fluid
conditions because of less work completed.

**Perceived fatigue.** Goaltenders perceived they were working harder on-ice when mildly
DEH compared to fluid intake (Figures 13 - 15, Table 4), which aligned with results from cyclists
(Logan-Sprenger et al. 2012, 2013) and ice hockey skaters (Linseman et al. 2014, Boville 2015,
Eskedjian 2015). Interestingly, fluid ingestion reduced the RPE at 58 min into the scrimmage but
not at the end (Figure 13). Goaltenders saved more shots in the last 10 min of the scrimmage (60-
70 min) while consuming fluid (Figure 18), which could have been a result of increased work
performed when BM was maintained compared to mild DEH. Therefore, goaltenders may have
experienced similar RPE in the final 10 min of the scrimmage despite performing more work when
euhydrated vs. mildly DEH. To test the hypothesis, RPE was analyzed post-scrimmage because
DEH was greatest (≥1.8% BM loss). Post-scrimmage, goaltenders felt best while consuming the
CES, followed by WAT and felt worst when mildly DEH (Figure 14). This may have been due to the CHO in the CES because the presence of CHO in the mouth and/or ingesting CHO during exercise decreased perceptions of fatigue (Chambers et al. 2009). Overall, maintaining hydration was beneficial for reducing perceived fatigue in goaltenders and CHO had an additional benefit.

Central and peripheral fatigue. Contrary to the hypothesis, hydration did not affect CF measures post-skate (SMVC), but the lower AUC$_{120}$ post-skate compared to pre-skate suggested CF may have been present in all conditions (Figure 17, Table 5). CF can also be estimated by changes in maximum MVC force produced with non-contracted muscles since no PF would exist (Drust et al. 2005). In line with the AUC$_{120}$ results, peak post-skate handgrip force showed no effect of mild DEH on CF, but PF was observed in all conditions (Table 6). Therefore, the temporal changes that suggested CF existed were confounded by PF. Nonetheless, it seems likely that both PF and CF were present post-skate. In support of this, both CF and PF were found immediately after a simulated soccer match using direct measurements (Goodall et al. 2017). This suggests that both CF and PF would have contributed to impaired SMVC force in the current study. On the contrary, one study suggested that CF may have not been present in the current study as CF was similar when T$_c$ was between 38.0-39.0°C (Morrison et al. 2005) and a second study reported that CF was primarily caused by changes in T$_c$ (Lloyd et al. 2015). Thus, mild DEH may not have increased T$_c$ enough compared to euhydration to induce CF.

Future research should ensure that PF would not be present during the post-skate SMVC so all changes could be attributed to CF or directly measure CF. Furthermore, there was no measurement of effort during the SMVC and the variable pre-skate results suggested this was necessary (Table 5). However, the nature of the study made directly measuring effort during the SMVC impractical. To encourage consistent and maximal efforts, future studies could explore
using a sham effort measurement to hold the participants accountable when the interpolated twitch technique is not practical or available.

5.3 Performance

**Save percentage and rebounds.** To test the effects of DEH, the last 10 min of the scrimmage were compared to the first 10. In the final 10 scrimmage min, mild DEH (1.5-1.8% BM loss) reduced SVP on all shots compared to intake of either fluid, but contrary to the hypothesis CES did not improve performance compared to WAT (Figures 18 and 19). However, condition did not affect shootout and shooting drill SVP (Figures 20 and 21) or SVP when scrimmage shots were categorized by situation. Likewise, rebound analyses were not affected by condition. These results were partially due to a small sample size in each category. For example, in the shooting drill only 20 shots were taken in total and many shots missed the net. Even if all 20 shots hit the net, these were placed into 4 rebound categories. Therefore, there was not a large enough sample size of shots to make any rebound conclusions in both the shooting drill and the end of the scrimmage. Overall, designing realistic game like drills was difficult and future research must account for sample size when categorizing shots and rebounds. Alternately, it may be more meaningful to extend the scrimmage or game like portion of the testing by ~30 min to increase the level of DEH. Especially when considering that mild DEH decreased scrimmage SVP when BM loss was ~1.5-1.8%.

**Movement analyses.** Reaction time to a pass that had 2 recipients was longer when mildly DEH, which indicated that goaltenders required more time to make a decision and/or move (Table 7). Interestingly, only the CES was beneficial compared to mild DEH for reducing movement generation. Thus, drinking CES had goaltenders beginning their push movement earliest, followed
by WAT and lastly NF. Interestingly in the NF and WAT conditions, push duration was shorter compared to CES, which made total movement time similar in all conditions. Therefore, although the goaltenders started off faster in the CES condition, the increased push speed in NF and WAT conditions were able to compensate for this.

It must be considered that the increased push movement duration in the CES condition was not the result of a reduced ability to move because maintaining hydration with a CES increased mean peak power during the first 10 lateral shuffle pushes compared to mild DEH (Table 8). Therefore, more powerful movements while ingesting CES were possible in the shooting drill as well but this was not the case. One explanation may have to do with movement efficiency. Since RPE was lowest post-scrimmage in the CES condition, goaltenders may have performed more efficient movements in the shooting drill when consuming CES. When goaltenders reacted and initiated movement quicker, as done in the CES condition, they would have had more time to process what the shooter was doing with the puck. They may have then adjusted movement speed to minimize the work or effort they had to exert to accomplish the same result. For example, they may have noticed a shooter was stopping the puck before shooting it rather than one-timing it, and slowed their movement. Alternately, the goaltenders could have performed shorter pushes in while mildly DEH.

Compared to mild DEH, maintaining hydration with a CES increased mean peak power during the first set of lateral shuffle pushes, while consuming the same volume of water did not (Table 8). Although, WAT ingestion improved mean peak power in the final 3 shuffles of set 1 and first three shuffles of set 2 (Figure 22). Therefore, the CES was best for performance in set 1 of the lateral movement drill, followed by WAT and lastly NF. The present results are in agreement with a recent meta-analysis that found HYPO reduced performance in non-body weight dependent
movements (Savoie et al. 2015). Interestingly, only WAT had some benefits in set 2 compared to mild DEH. During repeated sprints while rinsing with a CES, performance with the CES compared to placebo was better during earlier sprints but became worse in later sprints (Beaven et al. 2013). The current findings support this as the improvements with CES were lost in set 2. This may be because goaltenders exerted themselves more in set 1 in the CES condition and simply were not able to maintain that intensity in set 2.

5.4 Limitations

Blinding and fluid restriction. Blinding was not completely effective between WAT and CES conditions. Goaltenders were asked to guess which fluid they consumed if they thought they knew after all fluid trials and all goaltenders thought they could differentiate the solutions after all trials. Anecdotally, some goaltenders found the WAT solution “too sweet”, which led to incorrect identification. So decreasing the amount of zero calorie sweetener in the WAT could be useful for blinding. In contrast, some goaltenders could not tell the difference. Further support for good blinding was that 10/11 goaltenders guessed that they received the CES during their first fluid intake trial. This suggested that they associated the sweet beverage with the CES despite knowing the WAT was artificially sweetened. Future research should ask goaltenders at the end of the second fluid trial whether they could distinguish the solutions and if so, then guess.

An obvious limitation was goaltenders were not blinded to the NF condition. Although blinding subjects to DEH is challenging during field research, perception of thirst could have been reduced by rinsing their mouths with but not swallowing water (Cheung et al. 2015). Goaltenders routinely consumed fluid during skates, and therefore rinsing their mouth’s with water may indeed be more applicable to the real world than complete fluid restriction. It should also be considered
that some research suggests a feed-forward mechanism for WAT exists that is similar to CHO mouth rinsing. Furthermore, DEH increases thirst sensation like fluid restriction does, so if rinsing with water completely removes thirstiness then it may also not be realistic. Overall, blinding to fluid restriction is very tough and future research may think about implementing a water rinse conditions to replace the fluid restriction condition but this comes with its own limitations.

**Core temperature.** Obtaining $T_c$ from goaltenders was not as successful as desired in this study. During the NF condition, $T_c$ was not recorded in 2/11 goaltenders, which was caused by either a malfunctioned thermistor or the goaltender not swallowing the thermistor. Recording $T_c$ during either fluid condition was much less successful than in NF trials. Goaltenders indicated they ingested the thermistor 3 hours pre-skate, which was not enough time to be unaffected by fluid temperature for some goaltenders. During the scrimmage there was 10 min between fluid ingestion and $T_c$ readings for fluid temperature to be heated by the core, but readings were still low. It was not uncommon for $T_c$ to read normal before fluid was ingested on-ice and then to decrease to $<35^\circ C$. Fluid temperature was not measured but would have been much less than $T_c$ since solutions were mixed using cold tap water and stored in a crate directly on the ice. Future research should have participants consume the thermistor at an earlier time relative to skate initiation, so the thermistor would have travelled further along the GI tract. But, not so early that it would be excreted through a bowel movement, which was challenging in morning trials for subjects that had normal morning bowel movements. Therefore, performing experiments later in the day would have been beneficial.

**Scrimmage.** The scrimmage played was not the same as a real game. It was 3-on-3, played on a smaller portion of the ice and goaltenders did not have a goal crease to aid in navigation. Furthermore, by design there were a very large amount of high percentage scoring chances.
compared to a real game. All these factors likely would have reduced SVP compared to a 5-on-5 game played on a full ice surface. Therefore, the effects of DEH may have been exaggerated because there were more high percentage or challenging shots and the modified playing surface would have increased cognitive demand vs. a real game. It may be the case that on low percentage or easy shots DEH may not impair performance, but there were not enough low percentage shots to make this conclusion with standard statistics.

The ability to test scrimmage performance was also limited by the level of DEH. Initially the scrimmage was designed to induce DEH and the drills were to test its effects. On average, goaltenders did not reach ~2% BM loss during the scrimmage, so it could be argued that there would not have been any effects to find, especially early on when BM loss was <1%. This is further support for extending the scrimmage length to test for in-game effects of DEH when it is more severe.

The workload of the scrimmage was much larger than what would be expected during a game. To elicit BM losses like professional ice hockey goaltenders, the shot demand was increased to offset the cooler Tₐ. The elevated workload would have increased muscular fatigue and a recent meta-analysis found that activity interacted with BM loss to impair performance (Savoie et al. 2015), therefore artificially increasing the workload could have exacerbated the effects of 2.4% BM loss. On the contrary, the same meta-analysis also demonstrated that trained athletes were more susceptible to impaired performance from DEH compared to untrained athletes. The current study population was deemed “skilled” and would have fallen somewhere between trained and untrained. Therefore, the current study population would have been affected to a lesser extent by DEH vs. elite goaltenders.
**Human shooter variability.** Human shooters were used to avoid having to familiarize goaltenders to a puck shooting machine. A limitation to this choice was additional variability. It can be assumed this variability was similar across all trials but for on-ice performance in the shootout and lateral movement drill, the variability was too large to detect any effects assuming there were effects to find. Future research should stick to assessments that do not quantify performance based on human shooters. For example, in the shooting drill performance measures were reaction time and movement time, which had nothing to do with the shooter executing their shot. Having the shooter present was necessary for applicability so goaltenders would perform the drill attempting to make saves.

**Lateral movements.** Due to limitations with the 1080 Sprint and drill design, lateral movements were only accurately recorded during shuffles away from the machine. This meant that, 10 of 11 goaltenders shuffled towards their stick hand and one goaltender toward their glove hand. Pushing from goal post to goal post is more challenging when going towards the stick hand compared to the glove hand because the goaltender must move the stick so it won’t knock the post. Theoretically, the additional stick movement required when pushing to the stick hand would have slowed down goaltenders compared to glove side pushes. The only situation where this occurs is during a post-to-post shuffle, therefore the results are only applicable to this situation. The post-to-post shuffle going to the glove side may be better representative of all goaltender movements but was only obtained in one goaltender. Furthermore, the lateral movement was performed in one plane and movements most commonly cover two. In multiaxial movements, goaltenders must first pivot and then make a lateral push; however, this was challenging to capture because of study temporality and the 1080 Sprint only records in one plane of motion. Having multiple 1080 Sprint machines placed in multiple axes may be able to obtain data from movements that are more game-
like. So while it is not a limitation per se, the 1080 Sprint was not used to its full potential for interest of time. For example, it can add resistance to movements and this feature could have been useful to study force x velocity profiles under the different study conditions.

5.5 Conclusions

In conclusion, ice hockey goaltenders produced sufficient sweat to mildly DEH when no fluid replacement occurred. Compared to replacing sweat losses with fluid consumption, mild DEH impaired thermoregulation, strained the cardiovascular system and increased perceptions of fatigue during an on-ice scrimmage and drills but did not affect CF or PF. This translated to decreased performance in the scrimmage, slower reaction times and less powerful movements. Furthermore, co-ingesting CHO and electrolytes with water was superior to water alone for improving perceived fatigue, and generating motion and lateral movement power in the goal net. The primary finding was that ice hockey goaltenders benefited from individualized hydration strategies and it is recommended that ice hockey goaltenders minimize in-game DEH. Secondly, adding CHO and electrolytes to in-game nutrition strategies was sometimes more beneficial for goaltenders compared to water and it is recommended in addition to replacing fluid losses. It should be noted that the responses to WAT and CES intakes were not completely uniform and inter-individual variability existed. Therefore, these strategies should be practiced before implemented in competition.
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**APPENDIX A**

Table 1 – Individual urine specific gravity (USG) and body mass (BM) measurements obtained prior to the no fluid (NF), water (WAT) and carbohydrate-electrolyte solution (CES) conditions, as well as the mean and max within-subject USG differences.

<table>
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<th>Goaltender</th>
<th>Pre-skate BM (kg)</th>
<th>USG</th>
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<tr>
<td></td>
<td>NF</td>
<td>WAT</td>
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