The Influence of Ethanol Consumption on Physiological and Perceptual Responses and Postural Control During Acute Heat Exposure in Older Adults

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Chapter 1: Introduction

In 2023, the earth's average surface temperature was the warmest on record since recordkeeping began in 1880; this unprecedented temperature rise can pose significant challenges to the human thermoregulatory system (*NASA Global Climate Change*, n.d.). The human thermoregulatory system protects us from potentially fatal changes in core temperature through physiological and behavioural mechanisms (Chithramol & Shine, 2023; Flouris, 2011; Mota-Rojas et al., 2021). Physiological responses like increasing heart rate and sweating facilitate heat dissipation, while thermal perception informs behavioural responses (Chithramol & Shine, 2023; Flouris, 2011; Mota-Rojas et al., 2021). The heightened demand can strain the body's ability to cope with heat, leading to more significant risks of heat-related illnesses. As extreme heat waves become more frequent, more intense and longer in duration (Bobb et al., 2014; Patz et al., 2014), the prevalence of heat-related illnesses are predicted to become more frequent (McMichael et al., 2006).

Public health agencies create heat-health action plans to mitigate the adverse health effects of extreme heat. Heat health action plans consistently suggest individuals avoid extreme heat, seek cooling centres, and remain adequately hydrated (Apfel et al., 2008). These plans emphasize these strategies for vulnerable populations like the elderly, children, and those with chronic illnesses who've been reported to be more sensitive to heat and to ensure they receive adequate protection and support (Apfel et al., 2008; *Heat Alert and Response Systems (HARS)*, 2023). Older adults are a particularly vulnerable demographic during extreme heat events (Apfel et al., 2008) because their physiological and behavioural thermoregulatory mechanisms gradually decline with age (Grosiak et al., 2020).

Additionally, heat health action plans advise the public to avoid alcohol consumption. In Canada, more than 75% of adults consume alcohol (Canada, 2021) and during broadcasted heat alerts, ~20% of adults report alcohol as a 'rehydration strategy' (Ravanelli et al., 2023). During the summer months, alcohol sales are the highest when compared to the rest of the year (Myran et al., 2021) despite being a proposed comorbidity for heat stroke (Dematte et al., 1998; Sohal et al., 2022). While heat health action plans advise against ethanol consumption owing to its diuretic effect which may increase heat-related injury risk, secondary to alterations in behavioural responses (*NCCEH - CCSNE*, 2010), the evidence supporting this guidance is extremely limited. Gaining empirical evidence on the effects of alcohol on physiological and behavioural responses is essential to inform health advisories.

A recent scoping review concluded that ethanol does not negatively affect young healthy males' thermoregulatory responses, hydration, or hormone markers in fluid balance in the heat (Morris et al., 2024). Although older adults are identified as a vulnerable population during extreme heat, owing to a higher risk of heat-related illnesses, no study has explored the age-related impacts of alcohol consumption in the heat. Therefore, this thesis aims to generate the evidence required to directly inform public health policies in their current mandate and support a better understanding of why alcohol may be harmful during extreme heat events.

Chapter 2: Literature Review

Responses to Heat Stress

Thermoregulation

Thermoregulation is the process by which the body attempts to maintain an internal temperature of approximately 37°C. A deviation of core temperature by ±3°C from the resting temperature can result in physiological impairments or worse, death (Moran & Mendal, 2002). For a healthy individual, a fluctuation in body temperature will occur during heat or cold stress as the body responds to the environment which will initiate necessary physiological or behavioural responses to maintain temperature equilibrium (Schlader & Vargas, 2019).

During heat exposure, autonomic temperature regulation is initiated by thermoreceptors distributed throughout the body, which are then sent to the hypothalamus; these signals indicate that the body's thermal homeostasis has been disturbed (Cramer et al., 2022). The autonomic nervous system initiates sympathetic efferent signals to the heat loss thermoeffector: cutaneous arterioles and eccrine sweat glands (Cramer et al., 2022). Activating eccrine sweat glands and vasodilation of the cutaneous arterioles (skin) promote heat dissipation from the body to regulate core temperature (Cramer et al., 2022; Ebi et al., 2021).

Maintaining the body's core temperature within safe limits is crucial for survival (Fuller-Jackson et al., 2017; T. A. Wang et al., 2019). A disturbance in core temperature is an important indicator of thermal strain in the body (Lim et al., 2008). Major fluctuations can significantly affect homeostasis, as core temperature reflects the amount of heat in the organs and circulating cells (Farnell et al., 2005; Folk, 1998). The hypothalamus detects the increases in core temperature via afferent signals received by the sympathetic nervous system (Cramer et al., 2022); the efferent signals will attempt to increase heat dissipation via cutaneous vasodilation

and sweating, thereby supporting thermal homeostasis (Werner, 1980). The thermoregulatory adjustments concerning the increase in core temperature are typically sufficient to satisfy the demand, ensuring the oxygen supply to vital organs is not jeopardized due to the increased heat (Johnson, 1996). However, under the conditions that core temperature cannot be restored, individuals will likely suffer uncompensable thermal stress (Ravanelli et al., 2019). Hyperthermia (core temperature >40°C) can impair the central nervous system and cause systemic inflammation, tissue necrosis and multiple organ failure (Bouchama & Knochel, 2002; Shapiro & Seidman, 1990).

Cardiovascular

Cutaneous vasodilation is a physiological response to regulate core temperature. Elevations in core temperature cause an increase in cutaneous vasodilation, which promotes the redistribution of heat from the core to the periphery to facilitate heat loss to the external environment. Heat dissipation responses are accompanied by cardiovascular adjustments (Crandall & González-Alonso, 2010). Increased cardiac output is required to support the increased blood flow and redistribution of blood from central organs to cutaneous vascular beds, which is mediated through increased heart rate and contractility (Crandall & González-Alonso, 2010). Heat stress may impair baroreflex function, which could compromise blood pressure control (Crandall & González-Alonso, 2010). Therefore, to prevent a substantial fall in arterial blood pressure due to increased total vascular conductance associated with cutaneous vasodilation, cardiac output must increase, and vascular conductance of non-cutaneous beds must decrease (Crandall & González-Alonso, 2010).

Orthostatic Tolerance

Maintaining an upright position, or orthostatic tolerance, is dependent upon circulatory adjustments (Goswami et al., 2017; Rodrigues et al., 2020). These adjustments necessitate coordinated postural muscular activity as well as integrated control of cerebral blood flow and blood pressure (Goswami et al., 2017; Rodrigues et al., 2020). The orthostatic challenge can lead to blood pooling in the limbs and splenic vascular bed (Mosqueda-Garcia et al., 2000) and likely to further decline of central venous pressure (Furlan et al., 2000) and subsequently a transient reduction in blood pressure in standing (Mukai & Lipsitz, 2002).

Orthostatic hypotension (OH), when combined with heat exposure, places a significant strain on the cardiovascular system, making it challenging for the body to maintain blood pressure and postural stability (Fleg et al., 2016; Veronese et al., 2015). To maintain blood pressure during orthostasis and sustain cardiac output, a variety of cardiovascular adaptations, mostly mediated by the baroreflexes and sympathetic nervous system must take place (Convertino, 2014; Esler, 2010; Fu & Levine, 2014; Mano & Iwase, 2003). However, if cardiovascular control mechanisms are impaired, adequate compensation can fail, and OH can result (Shaw & Claydon, 2014). Heat stress can also reduce orthostatic tolerance, resulting in an increased risk of syncope, and potential hospitalization due to falling (Galli et al., 2011).

Sweating & Dehydration

Sweating serves as one of the most important avenues for heat dissipation through evaporative heat loss. As core and skin temperature rise, sweat production increases to facilitate evaporative cooling. The thermoregulatory system relies on total body water; during heat exposure, water loss increases due to sweat production, which supports the body's thermoregulatory needs (Sawka et al., 2001). With an increased reliance on evaporative cooling

in hotter climates, sweating can cause significant water loss from the body; if this fluid loss is not replaced, the individual may suffer from dehydration (Sawka et al., 2001). An individual in a dehydrated state may present with lower local sweating rates (Bittel & Henane, 1975) and lower skin blood flow (Kenney et al., 1990), thus decreasing metabolic heat loss and potentially increasing the risk of heat-related illnesses (Sawka et al., 2001).

Behaviour

Behaviour, also known as voluntary choices, is another key mechanism of the thermoregulatory system (Allison & Reger, 1992; Desruelle et al., 1996; Gibiński et al., 1979). Attia (1984), suggests that behaviour, rather than the autonomic responses of the thermoeffectors, is the primary mechanism for thermal homeostasis. Unlike the involuntary responses of the thermoeffectors, thermoregulatory behaviours are motivated, adaptable, and driven by the expectation of reward (Carlton & Marks, 1958; Epstein & Milestone, 1968; Weiss & Laties, 1961), such as trying to stay cool by finding shade or drinking a cold beverage during warming temperatures. Engaging in proper cooling interventions can mitigate heat stress factors and potentially lower the physiological strain incurred during heat stress due to activation of the thermoregulatory system (Jacklitsch et al., 2016; Morris et al., 2020).

Impact of Age on Response to Heat

Thermoregulation

As a part of the natural aging process, the efficacy of the thermoregulatory system begins to decline gradually (Grosiak et al., 2020), thus older adults are considered a heat-vulnerable population (Apfel et al., 2008). Owing to the higher sensitivity to extreme heat, older adults are more prone to heat-related illnesses and are at a higher risk for hospitalizations during extreme

heat exposure (Bobb et al., 2014). In 2021, older adults (between the ages of 56-75 years) made up nearly a quarter of the Canadian population (24.9%) (S. C. Government of Canada, 2022).

Older adults have a compromised capacity to dissipate heat through dry mechanism relative to younger adults therefore increasing thermal and physiological strain (Millyard et al., 2020). Montagna & Carlisle (1979), suggested that lower cutaneous perfusion that occurs with age may be associated with a flattened underside of the epidermis, this transformation may be associated with the loss of capillary plexus functional units. Therefore, structural alterations may diminish the maximal skin blood flow capacities of older adults (Kenney & Munce, 2003).

As discussed previously, sweating is one of the primary mechanisms by which the body dissipates heat to maintain thermal homeostasis (Notley et al., 2023). A progressive age-related reduction in sweating capacity has been observed to begin by 40 years of age (Dufour & Candas, 2007; Larose et al., 2013). The core temperature threshold for the onset of sweating has been suggested to be higher in older adults when compared to younger adults (Hellon & Lind, 1956; Sagawa et al., 1988). The literature suggests that the loss of sweating capacity that older adults experience is likely due to a reduction in sweat gland output, causing a lower sweat rate (Kenney & Munce, 2003). The delayed onset of sweating coupled with a reduced ability to increase and maintain a high sweat rate may inhibit the effect of sweat-induced cooling, resulting in a higher core temperature and greater heat strain in older adults (Millyard et al., 2020).

Cardiovascular

Healthy older adults are at higher risk of cardiovascular events during heat exposure and are at even higher risk if cardiovascular co-morbidities are present (Anderson & Bell, 2009; Kenny et al., 2010). Healthy older adults exhibit an altered hemodynamic response to heat (Lucas et al., 2008; Minson et al., 1998) , which can negatively impact aspects of the autonomic

thermoeffector activation (Kenney & Munce, 2003), furthermore, the presence of cardiovascular co-morbidities can exacerbate these altered responses (Holowatz & Kenney, 2011; Kenney et al., 1984; Kenny et al., 2016).

In a heated environment, older adults have reduced skin blood flow compared to younger adults, which can be attributed to structural alterations in the skin and decreased sympathetic nerve activity (Kenney & Munce, 2003; Stanhewicz et al., 2016). In older adults, cardiac output is reduced by ~50% and can be attributed to a decrease in chronotropic response (Gagnon et al., 2016, 2017; Greaney et al., 2015; Minson et al., 1998). Owing to the blunted increase in cardiac output and the inability to maintain stroke volume, Minson et al. (1998) suggested that the aging heart is unable to adapt to the circulatory demands of heat stress. Moreover, aging is associated with impaired baroreflex function (Monahan, 2007) which can undermine the systemic cardiovascular response and the maintenance of blood pressure regulation during heat stress (Engelland et al., 2020; Schlader et al., 2016).

Orthostatic Tolerance

Empirical evidence suggests that the prevalence of OH increases with age (Hiitola et al., 2009; Luukinen et al., 1996; Shibao et al., 2007). Hence, older adults exhibit slower corrections in blood pressure during orthostasis while heat-stressed (Lucas et al., 2008), which may be due to age-induced alterations in baroreflex function (Monahan, 2007). Shaw and Clayton (2014) have hypothesized that as orthostatic tolerance decreases, the risk for falls increases; OH can produce bouts of light-headedness and syncope, raising the risk of falls, which is a leading cause of morbidity and mortality among older adults (Monahan, 2007). This increased risk of falls can also lead to a higher risk of fractures, as approximately 6% to 24% of reported falls with fractures have been associated with ethanol in older adults (Resnick & Junlapeeya, 2004).

However, the etiology of falls is complex and multifactorial (Shaw & Claydon, 2014); intrinsic factors such as cardiovascular impairments, side effects from medication, alcohol consumption, or orthostatic hypotension can impact risk in various ways (Carey & Potter, 2001; Narkiewicz et al., 2000). Extrinsic factors such as warming temperatures, could also indirectly lead to an increased risk of falls owing to the declining efficacy of the thermoregulatory system (Fleg et al., 2016; Veronese et al., 2015).

Dehydration

Older adults have a higher risk of developing dehydration (Jéquier & Constant, 2010) and is becoming more prevalent among older adults. For example, in the United States, dehydration-related hospitalizations among older adults rose by 40% between 1990 and 2000 (Xiao et al., 2004). During heat exposure, older adults have been recognized to have a diminished thirst perception (Phillips et al., 1984) and decreased thermal sensation (Miescher & Fortney, 1989), therefore feeling less thirsty and less hot when compared to younger adults. Thus, older adults are at risk of having insufficient water intake, due to blunted thirst response or less efficient renal urinary concentrating mechanisms (Phillips et al., 1984). In older adults, mild dehydration of just 1 or 2% of body water can impair cognitive functions, alertness, and exercise capacity (Jéquier & Constant, 2010). Dehydration is also associated with the increased risk of falls among older adults (Robinson & Rosher, 2002).

Behaviour

Behavioural adaptations to heat stress are often driven by thermal discomfort (Gagge et al., 1969), however, research suggests that, in comparison to younger adults, older adults may perceive heat less intensely (Khare et al., 2015). Khare and team (2015), found that older adults, despite being a vulnerable population, were less likely to take personal heat protection (e.g.,

sunscreen, electric fans, air conditioning, etc.) and less likely to report heat-related symptoms (e.g., dehydration, headaches, dizziness, etc.) when compared to younger adults. Other international studies have similar findings in which older adults' awareness of heat events is widespread, and very few were changing their behaviour accordingly (Abrahamson et al., 2009; Sheridan, 2007). In support, Wadlock et al. (2018) explored the perceptual responses of older adults during exercise in ambient temperatures of 15°C compared to 25°C and 35°C, and despite the rise in skin and core temperature, there was no change in subjective thermal comfort. Thus, raises the concern that older adults may be reluctant to adapt their behaviours when their body is encountering thermal strain (Millyard et al., 2020).

Alcohol, Ageing and Heat Stress

In Canada, the trend of older adults who have reported heavy drinking has been on an incline since 2015 (*Dry February, You Say?*, 2023). Heavy drinking can pose negative physiological and perceptual consequences and the rising rate of ethanol consumption can increase the likelihood of requiring medical attention (Sugarman & Greenfield, 2021). Wiberg et al., (1970) suggested that older rats may be more susceptible to acute ethanol toxicity when compared to younger rats; with identical ethanol dosage, the older rats had significantly higher brain and blood ethanol levels and a slower metabolism rate. Older adults tend to have lower body water content, which may impede the distribution of ethanol (Vestal et al., 1977). Owing to the distribution of ethanol being primarily governed by the water content of the body fluid (Wiberg et al., 1971), older adults can attain a higher blood alcohol level and more significant toxicity than younger adults when given the same amount of ethanol (Vestal et al., 1977). Ethanol can exacerbate the body's fluid loss during heat exposure, further straining the thermoregulatory system (Ajjan & Page, 2023), which is particularly concerning for older adults

who already face challenges in maintaining hydration and efficient thermoregulation due to age-related physiological changes.

Thermoregulation

Within our current literature, there are inconsistent findings on how our thermoregulatory system is affected by ethanol. In 1852, Lichtendelds and Frochlich concluded that ethanol ingestion could lower human body temperature at neutral ambient temperature. When comparing a neutral (22°C) versus hot (36°C) environment, Myers (1981) suggested that core temperature in rats was primarily dependent on ambient temperature, and that physiological mechanisms to dissipate heat seem to be unaffected by ethanol consumption. Other studies have observed a lower resting core temperature following ethanol consumption (Desruelle et al., 1996; Mekjavic et al., 1987; Yoda et al., 2005) proceeded by a sharper rise during heat exposure (Desruelle et al., 1996). In relation to our thermoeffectors, evidence is mixed; demonstrating no impact on sweating and cutaneous vasodilation (Allison & Reger, 1992; Gibiński et al., 1979) or increases in sweating and cutaneous vasodilation (Johnston et al., 1996; Proano & Perbeck, 1994; Yoda et al., 2005) following ethanol consumption in the heat.

A limitation of the current literature on the relationship between heat exposure and ethanol is they have small sample sizes and consist of young healthy male subjects which could lead to poor generalizability to the public (Morris et al., 2024). Further, some research has suggested women and younger adults have higher ethanol metabolic rates than men and older adults (Cole-Harding & Wilson, 1987; Mishra et al., 1989; G. D. Smith et al., 1993; Sutker et al., 1987); however, other research has shown no significant gender or age difference in ethanol metabolism (Arthur et al., 1984; Goist & Sutker, 1985; Sklar et al., 2012; Sutker et al., 1987;

Vestal et al., 1977). While alcohol consumption has various impacts on health, our understanding of how it increases the risk of heat-related illnesses remains largely unclear.

Cardiovascular

The long-term use of ethanol can have detrimental effects on the cardiovascular system, cellular immunity, hemostasis, and various physiological skin functions (K. E. Smith & Fenske, 2000); however, acute use of ethanol on the cutaneous arterioles is poorly understood within the literature (Eby & Majetschak, 2019; Lachenmeier, 2008). During heat exposure in resting humans, the initial autonomic response is to withdraw the normal constrictor tone acting on the cutaneous arterioles, which then promotes vasodilation however, since ethanol is suggested to depress vasoconstrictor response and promote higher skin blood flow, this may disrupt orthostatic stress and impair baroreflex (Maufrais et al., 2017). To our knowledge, only Yoda et al. (2005) have seen an increase in skin blood flow during passive heat exposure. However, Hughes et al. (1984) concluded that the influence of ambient temperature on skin blood flow appears to dominate over the influence of moderate amounts of ethanol.

In young healthy adults, ambient temperature, whether thermoneutral or heated, can result in an increased heart rate after ethanol consumption (Aranha Rosito, 1999; Kupari, 1983; Maufrais et al., 2017; Narkiewicz et al., 2000; T. A. Wang et al., 2019) and can remain elevated following ~12 hours of consumption in thermoneutral conditions (Aranha Rosito, 1999). In heated environments and underwater cycling, an increased skin blood flow has been observed with ethanol consumption (Hughes et al., 1984; Johnston et al., 1996; Yoda et al., 2005).

The effect of ethanol on blood pressure remains inconsistent, as the literature has shown a decrease (Narkiewicz et al., 2000), no change (Allison & Reger, 1992), and an increase after consumption (M. Q. Wang et al., 1995). In healthy subjects, the acute effects of ethanol on the

cardiovascular system have been shown to increase cardiac output due to reductions in total peripheral resistance (Kupari, 1983). Whether ethanol consumption in the heat modulates the blood pressure response in older adults remains unexplored.

Orthostasis Tolerance & Postural Control

It is suggested that ethanol may interfere with the autonomic nervous system as it disrupts the vasoconstrictor response, resulting in orthostatic stress and impairing the efficacy of the baroreflex (A.-R. A. Abdel-Rahman et al., 1987; Carter et al., 2011; Narkiewicz et al., 2000). Therefore, it can be hypothesized that alcohol consumption induces orthostatic hypotension (Maufrais et al., 2017). Narkiewicz et al., (2000) conducted a crossover study on young, healthy adults (18 males, 1 female), and when ingesting alcohol versus a placebo, the ethanol trial exhibited a larger decrease in blood pressure with orthostatic stress; although the increase in heart rate was similar between both trials, suggesting that ethanol inhibits the central response to orthostasis. The increased risk of OH when combined with ethanol can increase the risk of injury due to impaired balance, reflexes, and central nervous system depression. The combined effect of ethanol and OH can make it more challenging to maintain a stable blood pressure increasing the risk of dizziness, syncope, and a higher likelihood of falls and injuries.

There are few studies examining the acute effects of ethanol on the vestibular function with postural sway (Kubo et al., 1989; Lathers & Smith, 1976; Lukas et al., 1989; Nieschalk et al., 1999). Research concerning the effects of ethanol on the vestibular system have demonstrated a depression in the activity of neuron impairment of static balance control, resulting in an increased postural sway and the inability to coordinate postural activity (Woollacott, 1983). Early studies have demonstrated a decrease in tendon reflex latency with large doses of alcohol, which can be attributed to ethanol's depression in the central nervous

system pathways, and may delay spinal reflexes, resulting in larger sway response (Woollacott, 1983). However, other research has suggested that ethanol may increase spinal reflex latencies after consumption (Joachim & Weyer, 1975).

Dehydration

Ethanol inhibits the release of vasopressin, an antidiuretic hormone, promoting increased urine output and decreased water retention (Flores-Salamanca & Aragon-Vargas, 2014). The body then becomes more susceptible to dehydration when it cannot retain adequate fluid, resulting in potentially higher thermal strain (Flores-Salamanca & Aragon-Vargas, 2014). It is generally acknowledged that ethanol causes diuresis by blocking the release of vasopressin; as demonstrated by Murray (1932) the administration of a posterior pituitary extract prevented the diuresis caused by ethanol. Other research by Taivaninen and team (1995) suggests that there may be other mechanisms, such as decreased renal sensitivity to vasopressin (De Marchi et al., 1993; Linkola et al., 1978) or alterations to renal hemodynamics may also contribute to alcohol-induced diuresis. Hobson & Maughan (2010) hypothesized that the amount of ethanol ingested, and the state of hydration are assumed to have an impact on the degree of diuresis. Moreover, Eggleton (1942) and Stookey (1999) suggested that alcohol-induced diuresis was less pronounced when slightly dehydrated compared to people who were euhydrated. Eggleton (1942) also suggested that the ingestion of drinks with small amounts of ethanol when dehydrated did not significantly reduce the efficacy of rehydration. Further, although vasopressin plays a major role in the regulation of water secretion (Hobson & Maughan, 2010), research suggests that it does not directly influence sweating; vasopressin's main role in retaining fluid balance is crucial for overall thermoregulation, as adequate hydration is necessary for efficient sweat production and cooling (Sawka et al., 2001). Nevertheless, no evidence to date has

examined whether alcohol consumption in the heat amplifies the net reduction in total body water in older adults, thereby exacerbating dehydration.

Behaviour

Boisvert et al. (1994) demonstrated that after alcohol consumption, ambient temperatures may be perceived as significantly warmer when compared to a placebo trial. However, the warm sensation that ethanol often produces is suggested to be due to warming skin temperature caused by dilated peripheral vessels (Fleming et al., 2001).

In the United Kingdom, Khare et al., (2015), surveyed heat protection behaviours after a heatwave found that although younger adults agreed that avoiding alcohol during the heat is an effective intervention, only a small percentage of younger adults undertook this protective measure. Meanwhile, the younger adults responded with a higher percentage of heat-related health symptoms (e.g. dehydration, headaches, tiredness, etc.); therefore, Khare et al. (2015) hypothesized that the higher rates of heat-related symptoms may be related to engaging in risky behaviour such as increased alcohol consumption during the extreme heat. In support, Starkey & Charlton, (2014) have also proposed that drinking alcohol may increase risky behaviour; as blood alcohol concentration (BAC) increased, there was a corresponding rise in the frequency of crashes during a simulated driving test. Increased risk-taking behaviour can be disadvantageous for behavioural feedback and such impaired judgment may result in the inability to adopt cooling behaviours (*NCCEH - CCSNE*, 2010; Van Dyke & Fillmore, 2017). From our understanding, whether older adults perceive their thermal environment differently following alcohol consumption remains unknown.

Chapter 3: Summary and Research Purpose

Although public health authorities recommend avoiding alcohol consumption during heat exposure due to the increased risk of heat-related illnesses, the scientific rationale supporting their statements is inconsistent. The literature provides little definitive explanation of the physiological or perceptual effects of alcohol consumption during heat stress, nor does it fully explain the combined effects of alcohol and heat on postural control. Further, the current literature examining alcohol consumption and thermoregulatory responses only consists of young, healthy male subjects, thus leaving no analysis of older adults (Morris et al., 2024). Therefore, this study aims to investigate the effects of alcohol consumption during heat stress on older adults' perception, thermoregulatory response, and postural control. Unpublished data from our thermal lab indicates that during heat exposure, young healthy adults do experience adverse physiological and postural control effects after alcohol consumption, but no change in perception. Although thermoregulatory responses (e.g., perception, body temperature, sweating) remain relatively unchanged, during heat stress, ethanol increases cardiovascular strain, implying an increased risk of adverse cardiovascular events and increases urine output, indicating a higher risk of dehydration.

Based on previous evidence and existing literature within younger adults, it is hypothesized that alcohol consumption during acute heat exposure will affect older adults' postural control and perception but have no impact on thermoregulatory responses.

Chapter 4: Method

This study used a single-blind randomized placebo-controlled crossover method to evaluate the effects of heat and alcohol exposure on perception, thermoregulatory responses, and postural control in healthy older adult participants. Ethical approval for this study was received from Lakehead University Research Ethics Board (#1469888).

Preliminary Session

To orient participants to the study, a preliminary session was conducted via a Zoom® call to introduce the study details and procedures. Participants provided verbal consent during the call, and they signed written consent during their first visit. For the exclusion criteria, participants were asked to complete the Alcohol Use Disorders Identification Test (AUDIT); any scores above 8 were excluded from the study (n = 2). Exclusion criteria also included being pregnant, trying to conceive or breastfeeding, or being hospitalized with COVID-19. Participants had no history of cardiovascular disease, diabetes, chronic obstructive pulmonary disease, cystic fibrosis, cancer, or a history of alcohol addiction/dependence. They were screened with a demographics survey before the data collection began. No participants were excluded due to any pre-existing health conditions.

Experimental Protocol

Two experimental trials were scheduled at the participant's convenience, and both followed an identical experimental protocol. The experimental visits took place within one month of the preliminary session. All experimental trials were conducted at the Physiology Laboratory located at Lakehead University.

For each experimental trial, the participants were asked to arrive at the laboratory at the same time of day to avoid any influence of circadian rhythm. Upon arrival, they were asked to

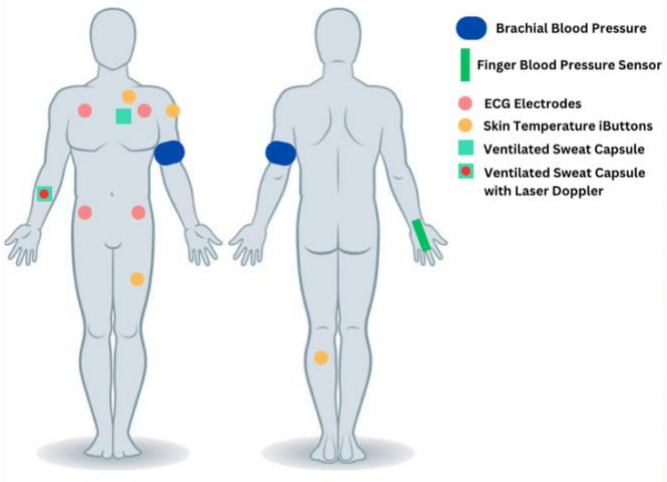
provide a urine sample to assess hydration status. Using a pocket refractometer (PAL-10S BLT/A+W, Atago), hydration status must be confirmed to be less than 1.025 (Kenefick & Cheuvront, 2012). If not, the participant was asked to drink 500ml of water and then provide another urine sample; however, all participants were hydrated upon arrival, therefore, this step was not taken. Once the urine sample was completed, participants were instructed to not drink anything until the trial was over unless provided by the researcher. Then, a nude body mass was done in complete privacy. The participants then reported their weight to the researcher after being re-dressed in shorts and tops. Participants were asked to self-insert a flexible pediatric grade thermistor (~2 mm diameter) through the anus into the rectum (~15 cm) in private (a step-by-step handout was provided) that remained inserted throughout the experimental trial and was used to measure core temperature. Those who felt uncomfortable obtaining their rectal temperature had the option of using an oral thermometer as an alternative to measure their core temperature (n=3).

Ambient conditions were measured using a temperature and humidity probe (HMT120, Vaisala). In a thermoneutral room, the participants were then instrumented with four (4) wireless skin temperature sensors (iButtons, Analog Devices) that were be taped to the skin surface with hypoallergenic tape skin (the sites located on the chest, shoulder, quadricep and calf), a ventilated sweat capsule on the upper chest and forearm (surface area coverage per capsule = 2.89cm^2) supplied with influent dry gas governed at 1.7 L/min using a glass flowmeter (FL3905G, Omega Engineering). Effluent air from the ventilated capsule was measured using a temperature and humidity probe (HMT330, Vaisala), laser doppler (PeriFlux System 5000), 4-lead electrocardiogram electrodes (MLA0115 ECG Lead Switch Box) (sensors were placed on the left and right upper chest and lower abdomen), an automated

brachial blood pressure cuff (Tango M2, SunTech), and a finometer (FMS, Finapres Medical System) (see Figure 1).

Figure 1

A schematic illustration of participant instrumentation



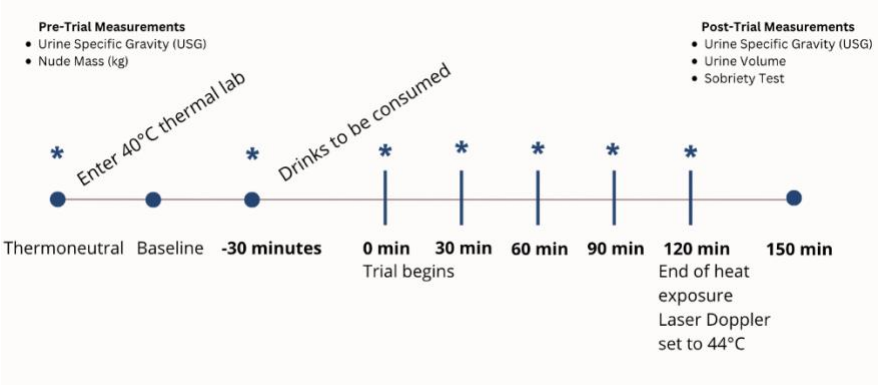
For the experimental trial, participants first completed a 10-minute baseline after instrumentation in 25°C, and then entered a thermal chamber set to 40°C. After an approximate 30-minute baseline period to equilibrate with the new environment, in a single-blind randomized counterbalance fashion, the participant was asked to consume either i) a placebo (i.e., non-alcoholic drink) or ii) gin-based alcoholic beverages. The researcher prepared one drink, partitioned into four equal parts, through standardized calculations described by Starkey and Charlton (2014). Women received doses of 0.75 ± 0.1 grams per kilogram of body mass, and men received doses of 1.0 ± 0.1 grams per kilogram of body mass. Alcohol (gin, 40% alcohol) was mixed with orange juice at a ratio of 25% gin to 75% orange juice. Previous data collected in younger adults confirms that this alcohol dose is expected to achieve a blood alcohol concentration of ~0.08% within 30 minutes following consumption (Lefebvre et al., 2025). The placebo drink was a ratio of 50% tonic water to 50% orange juice during the second experimental trial. Tonic water was chosen to mimic the bitter taste of gin; participants were

blinded to the order of trials to the best of our capacity. The target blood alcohol concentration (BAC) was selected at 0.08mg/100ml since studies have indicated that a moderate amount of alcohol consumption (BAC 0.05mg/100ml) may not consistently result in signs of intoxication until BACs of 0.08 to 0.10 are attained (Liu & and Fu, 2007; Moskowitz & Florentino, 2000). Our participants reached a BAC of ~0.08% within the 120 minutes following alcohol consumption (Figure 3).

The participants began drinking the beverages provided by the researcher. They were instructed to consume each of the four drinks in 6-8 minutes, totalling 30 minutes. After finishing the four drinks, the participants performed each test in our Physiological, Perceptual, and Postural Test Battery. This testing battery was repeated every 30 minutes during heat exposure (e.g., 30, 60, 90, and 120 minutes) (Figure 2). During the trial, if a participant had to urinate, a large urine specimen container was provided, and in private, the participant was asked to excrete all urine into the container. Once their bladder felt drained and redressed, the researcher would re-enter the room, document the time, weigh the urine on the scale and measure hydration using a pocket refractometer. Participants were also asked to void bladder at the end of each trial in which the same measurements were taken.

Figure 2

A schematic figure of the experimental design



Note. The asterisk (*) indicates when the battery of tests was taken.

Physiological, Perceptual and Postural Test Battery

First, participants used a visual analog scale to measure subjective thermal sensation, thermal comfort, and subjective intoxication level (Appendix A). Using a dry-erase marker, participants were asked to record their feelings on the plastic, laminated scale. The researcher then used a ruler to measure the distance from the left end of the scale.

Next, a seated blood pressure reading was taken with a brachial blood pressure cuff placed over the left arm. The participants were then asked to stand and complete the Romberg test. To evaluate postural control, a Romberg test, a 60-second trial (30 seconds with eyes open, 30 seconds with eyes closed), arms crossed over the chest and feet together on a postural sway board (Wii Fit Balance Board, Nintendo, Japan). Once the Romberg test was completed, the participants were asked to step on a digital scale to provide a body mass measurement. While on the scale, a standing blood pressure reading was taken. Then, the participants were asked to sit down until the next battery of tests.

To evaluate blood alcohol concentration, the participants were required to blow into a breathalyzer device (BACTRACK S80) every 30 minutes, irrespective of condition, to maintain blinding to the intervention.

After the 120-minute heating period, a laser doppler probe was heated to 44°C to acquire a measurement of maximum skin blood flow (~30 minutes). Participants were then de-instrumented by the researchers, with the exception of the rectal probe which the participants themselves removed. Prior to leaving the lab, the participants completed standardized field sobriety tests (see Appendix B) to confirm sobriety upon leaving. To maintain blindness between the trials, participants completed the sobriety tests after each trial.

Participants were reminded that they can terminate participation in the current study,

without prejudice, at any point, including during an experimental trial. The experiment would have also been terminated on the researcher's discretion if core temperature ratings exceeded 39.5°C or if the participant appeared to be in distress. However, no participant volitionally terminated their heat stress exposure, and no participant exceeded a core temperature of 39.5°C.

Data analysis

All continuous data acquisition was sampled at 1000 Hz using a Powerlab 16/35 (AD Instruments, New Zealand) and LabChart 8 software. Skin temperature was measured in 5-second averages extracted from OneWireViewer and then calculated using the Ramanathan (1964) equation to determine mean skin temperature. Whole-body sweat loss is calculated by subtracting the participant's weight from 0 minutes to 120 minutes, assuming the weight loss is due to sweat, while accounting for urine volume excretion.

Postural stability was assessed using a software developed by the University of Colorado, specifically CU BrainBLox (Cooper et al., 2014). These data were imported into Matlab (MathWorks, Inc., United States of America) to calculate total displacement (cm), mean displacement velocity (cm/second), maximum displacement (cm/second), mean medial-lateral (M/L) centre of pressure (COP) (cm), mean anterior-posterior (A/P) COP (cm), sway area (cm^2).

All statistical analyses were conducted using GraphPad Prism 9.5, with the exception of postural sway analysis and orthostatic tolerance which were performed using SPSS. We chose SPSS for the postural sway analysis because it handles missing data by excluding it from the analysis, rather than treating it as a zero value, which can help avoid skewing the results when data from specific time points are unavailable in a trial (*Missing Data | SPSS Learning Modules*, n.d.). Orthostatic tolerance was measured using systolic and diastolic responses extracted from

the Finometer. Systolic and diastolic measures were recorded at nadir and subtracted from baseline values, as described by Ferreira et al., (2024).

Analysis

Separate two-way repeated measures (rm) analysis of variance (ANOVA), with the repeated factors of beverage (two levels: ethanol or placebo) and time (six levels: pre-drink, 0, 30, 60, 90, 120 minutes) was used to analyze the dependent variables of core temperature, mean skin temperature, heart rate, local sweat rate, skin blood flow, systolic blood pressure, diastolic blood pressure, mean arterial pressure, thermal sensation and thermal comfort. Similar to the two-way ANOVAs, a mixed-effects analysis of variance was conducted to measure maximum skin blood flow and orthostatic tolerance.

A three-way repeated ANOVA with repeated factors of beverage (two levels: placebo and alcohol), eyes (open and closed) and time (seven levels: thermoneutral, pre-drink, 0, 30, 60, 90 and 120 minutes) was also used to analyze postural sway variables. Post-hoc multiple comparisons were conducted using the Bonferroni correction to control for Type I error.

An additional two-way rmANOVA was conducted to analyze the change in blood pressure during the orthostatic tolerance test. Moreover, total body water loss was measured combining whole-body sweat loss and urine output. The treatment effect will be calculated using the mean difference between ethanol and placebo with 95% confidence intervals.

For the rmANOVAs, interaction effects were evaluated first, followed by main effects. If an interaction effect was present, post-hoc tests were conducted using the Bonferroni correction to control for Type I error in multiple t-test comparisons. Significant differences between groups were set at $p<0.05$. Effect sizes are reported as partial eta squared (η^2). When interpreting partial eta squared, $\eta^2 = 0.01$ indicates a small effect, $\eta^2 = 0.06$ indicates a medium effect and $\eta^2 = 0.14$

indicates a large effect (Daines, 2025). Given the repeated measures design over time, the Greenhouse-Geisser correction was used to account for violations of sphericity.

Chapter 6: Results

Participants

All participants (n = 11) were over 45 years of age and completed two experimental trials in a randomized counterbalance order (one placebo, one alcohol). Participants were euhydrated (USG < 1.025) before heat exposure. Participant demographics (Table 1) and thermoneutral measures (Table 2) are presented below.

Table 1

Participant characteristics

Variable	**Participants** (n=11)	**Females** (n=6)	**Males** (n=5)
Age (yrs)	59 ± 4.82 [49-67]	56 ± 4.37 [49-61]	62 ± 3.27 [59-67]
Height (m)	1.71 ± 7.79 [1.57-1.82]	1.66 ± 6.15 [1.57-1.74]	1.77 ± 5.19 [1.70-1.82]
Weight (kg)	85.18 ± 18.95 [62-125]	82.3 ± 22.22 [62-125]	88.6 ± 25.4 [79-114]
BMI (kg/m^2)	28.95 ± 5.61 [23.34-43.25]	29.61 ± 6.98 [23.34-43.25]	28.17 ± 4.03 [24.91-35.19]
BSA (m^2)	1.96 ± 0.22 [1.67-2.32]	1.89 ± 0.24 [1.67-2.32]	2.06 ± 0.19 [1.83-2.32]
AUDIT	3.63 ± 1.86 [1-7]	3.50 ± 2.26 [1-7]	3.80 ± 1.48 [2-6]

Note. Body Mass Index (BMI), Body Surface Area (BSA), Alcohol-Use Disorder Identification Test (AUDIT). Data is presented as mean ± SD [range].

Table 2

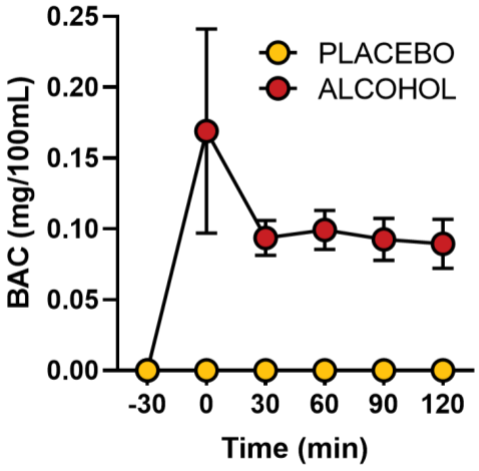
Thermoneutral baseline data between trials prior to drink ingestion

Variable	**Placebo** (n=11)	**Alcohol** (n=11)
Heart rate (BPM)	71 ± 11	72 ± 10
Systolic pressure (mmHg)	127 ± 10	126 ± 10
Diastolic pressure (mmHg)	86 ± 6	85 ± 7
Mean arterial pressure (mmHg)	100 ± 7	98 ± 8
Skin Blood Flow (%max)	4 ± 2	4 ± 2
Core temperature (°C)	36.9 ± 0.5	37.0 ± 0.5
Thermal comfort (cm)	2.3 ± 6.1	10.9 ± 18.7
Thermal sensation (cm)	65.1 ±24.1	75.5 ± 19.2

Note. Mean values ± standard deviations were derived from a thermoneutral environment and were taken prior to heat exposure. Blood pressure was taken in a seated position. A thermal comfort of 0 cm indicates 'not uncomfortable', and a thermal sensation of 80 cm indicates 'neutral' (see Appendix A).

Figure 3

Blood alcohol concentration over time (minutes)



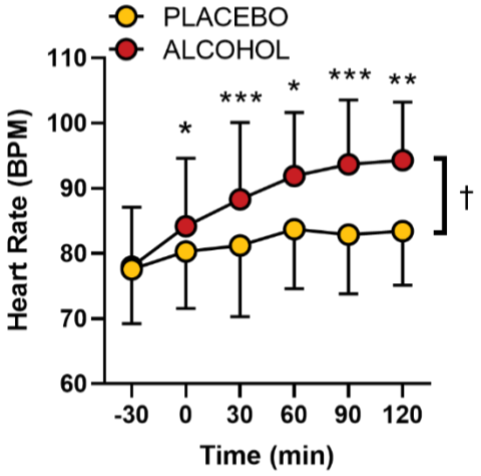
Note: The 0-minute Blood Alcohol Concentration (BAC) readings are overestimate due to the mouth alcohol phenomena (Spector, 1971). -30 refers to the 30 minutes preceding the start of drinking, while 0 minutes refers to the end of the drinking period and the beginning of the passive heating procedure. Data is presented as the mean ± SD.

Blood alcohol concentration (BAC) (mg/100 mL) levels over the 120 minutes of heat exposure. Each participant was measured with a BAC of 0.00 before heat exposure (Figure 3).

Cardiovascular Response

Figure 4

Heart rate responses as a function of time (minutes)

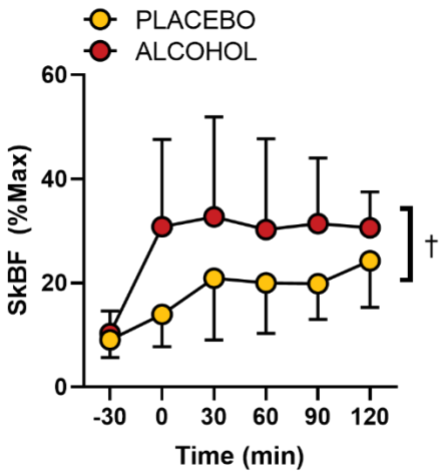


Note. Heart rate measured in beats per minute (BPM) responses over time during placebo and alcohol conditions. Data is presented in mean ± SD. Asterisks indicate significant differences between at specific time points. * $p < 0.05$, ** $p < 0.001$, *** $p < 0.001$. The dagger (†) indicates a main effect between drinks.

Heart rate exhibited a significant main effect of time ($F_{(1.681, 16.81)} = 16.240$, $p<.001$, $\eta_p^2 = .774$) and, a main effect of drink ($F_{(1,10)} = 49.440$, $p<.001$, $\eta_p^2 = .719$), with heart rate being higher with alcohol; an interaction was also present ($F_{(2.162,21.62)} = 7.66$, $p<.001$, $\eta_p^2 = .434$). Post-hoc comparisons revealed that heart rate was significantly elevated at 0- ($p<.03$), 30- ($p<.001$), 60- ($p<.020$), 90- ($p<.001$) and 120 minutes ($p<.005$) (Figure 4).

Figure 5

Skin blood flow responses as a function of time (minutes)

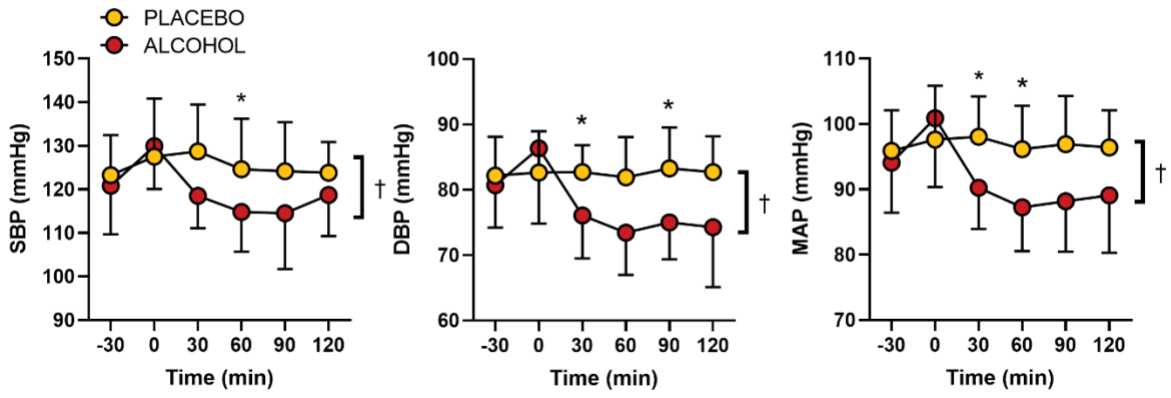


Note. Skin blood flow (SkBF, %Max; n = 7). The asterisk (*) indicates a significant effect of time. The dagger (†) indicates a main effect between drinks.

SkBF revealed significant main effects of time ($F_{(2.262, 13.57)}$ = 6.487, p<.008) and drink ($F_{(1.000, 6.000)}$ = 8.413, p<0.02) with no interaction ($F_{(2.190, 11.39)}$ = 1.282, p=0.44) (Figure 5).

Figure 6

Blood pressure responses as a function over time (minutes)



Note. Systolic blood pressure (SBP; left), diastolic blood pressure (DBP; middle), and mean arterial pressure (MAP; right) responses over time during placebo and alcohol conditions. Blood pressure was measured in millimetres of mercury (mmHg). Data is presented in mean ± SD.

Asterisks indicate significant differences between at specific time points. * $p < 0.05$. The dagger (†) indicates a significant main effect of drink.

Systolic blood pressure (SBP) showed a significant main effect of time ($F_{(3.038, 30.38)}$ = 5.100, $p<.001$, $\eta_p^2 = .388$) and drink ($F_{(1.000, 10.00)} = 5.262$, $p=0.045$, $\eta_p^2 = .350$), with SBP being lower with alcohol, as well as an interaction ($F_{(3.602, 36.02)} = 3.425$, $p=.021$, $\eta_p^2 = .255$); post-hoc analysis indicated a significant difference at 30 minutes during the alcohol trial ($p<0.028$) (Figure 6).

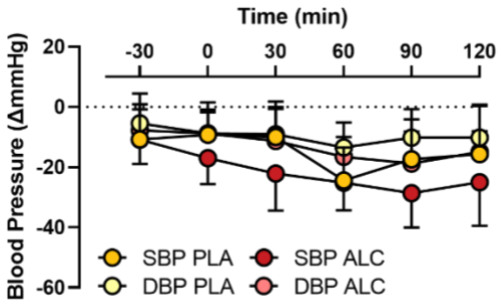
Diastolic blood pressure (DBP) showed a significant main effect of time ($F_{(2.664, 26.64)}$ = 5.021, $p<0.008$, $\eta_p^2 = .377$) and drink ($F_{(1.000, 10.00)} = 7.337$, $p=.022$, $\eta_p^2 = .415$), with DBP being lower with alcohol, as well as an interaction ($F_{(3.464, 34.64)} = 6.147$, $p<0.001$, $\eta_p^2 = .381$); post-hoc analysis indicated a significant difference at 30- ($p=.047$) and 90-minutes ($p=.040$) during the alcohol trial (Figure 6).

Mean arterial pressure (MAP) showed significant main effect of time ($F_{(3.045, 30.45)}$ = 6.003, $p<0.002$, $\eta_p^2 = .429$) and drink ($F_{(1.000, 10.00)} = 7.504$, $p=.021$, $\eta_p^2 = .453$), with MAP being lower with alcohol, as well as an interaction ($F_{(3.689, 36.89)} = 6.135$, $p<.001$, $\eta_p^2 = .381$); post-hoc analysis indicated a significant difference at 30- ($p=.021$) and 60-minutes ($p=.025$) during the alcohol trial (Figure 6).

Orthostatic Tolerance

Figure 7

Orthostatic tolerance response from sitting to standing position over time (minutes)



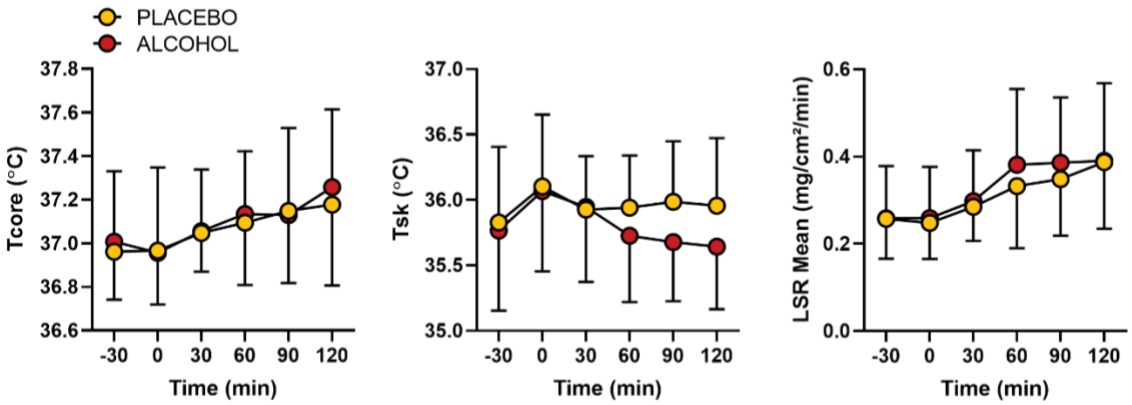
Note. Change (Δ) from baseline to nadir systolic (SBP) and diastolic blood pressure (DBP) during heat stress, comparing placebo and alcohol conditions (n = 7). A score of 0 indicates no change in blood pressure response.

Change in SBP exhibits a significant main effect of time, ($F_{(2.867,11.469)} = 5.370$, p=.003, $\eta_p^2 = .573$) and a significant effect of drink, ($F_{(1.00,4.00)} = 26.242$, p=.007, $\eta_p^2 = .868$), with ΔSBP being lower with alcohol; no interaction was found, ($F_{(2.754,11.015)} = 2.201$, p=.095, $\eta_p^2 = .355$) (Figure 7). Change in DBP exhibits no main effect of time, ($F_{(2.128,8.514)} = 2.145$, p=.175, $\eta_p^2 = .349$) no main effect of drink, ($F_{(1.00, 4.00)} = 0.605$, p=.480, $\eta_p^2 = .131$) and no interaction ($F_{(2.235, 8.939)} = 0.975$, p=.423, $\eta_p^2 = .196$) (Figure 7).

Thermoregulatory Response

Figure 8

Thermoregulatory responses over time (minutes)



Note. The effect of alcohol and placebo on the thermoregulatory system: core temperature (Tcore; left), skin temperature (Tsk; middle), and local sweat rate mean (LSR mean; right). Data is presented as a mean in degrees Celsius (°C) ± SD.

Tcore observed a significant main effect of time ($F_{(2.331, 23.31)} = 7.207$ $p<0.002$, $\eta_p^2 = .523$), with no significant main effect of drink ($F_{(1.000, 10.00)} = 0.086$, $p=.776$, $\eta_p^2 = .019$) and no interaction ($F_{(1.746, 17.46)} = 0.409$, $p=.643$, $\eta_p^2 = .039$) (Figure 8).

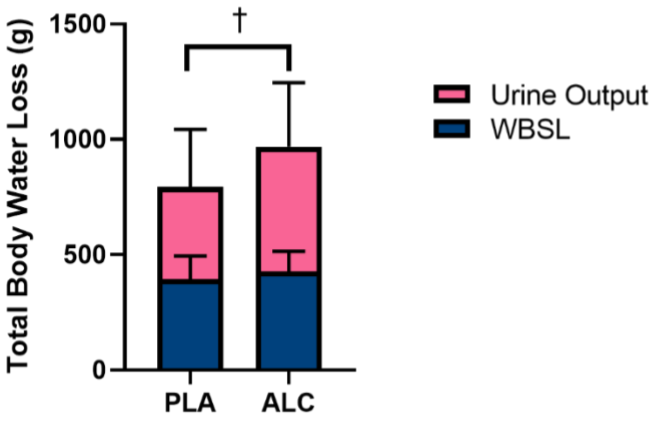
Tsk did not observe a significant main effect of time ($F_{(2.178, 21.78)} = 2.233$, $p=.128$, $\eta_p^2 = .300$) or drink ($F_{(1.000, 10.00)} = 1.360$, $p=.271$, $\eta_p^2 = .191$), and no interaction ($F_{(2.705, 27.05)} = 1.799$, $p=.175$, $\eta_p^2 = .152$) (Figure 8).

LSR mean observed a significant main effect of time ($F_{(1.591, 15.91)} = 6.619$, $p=.011$, $\eta_p^2 = .574$), with no significant main effect of drink ($F_{(1.000, 10.00)} = 1.290$, $p=.283$, $\eta_p^2 = .041$) and no interaction ($F_{(2.606, 26.06)} = 0.372$, $p=.746$, $\eta_p^2 = .036$) (Figure 8).

Diuretic Response

Figure 9

Fluid loss pathways (whole-body sweat loss and total urine volume)



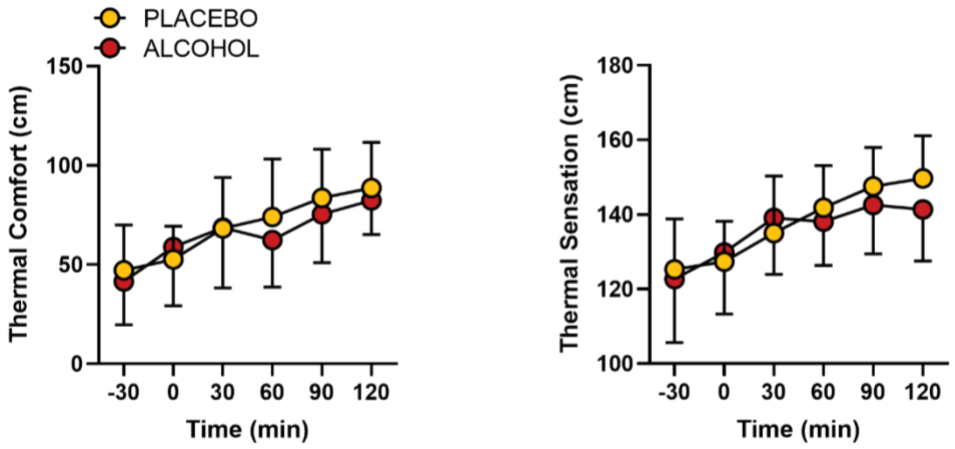
Note. Whole-body sweat loss (WBSL) and urine output were computed as total body water loss (grams) compared between the placebo (PLA) and alcohol (ALC) trials.

Total body water loss observed a significant main effect of drink ($F_{(1, 10)} = 9.286$, p=.012, $\eta_p^2 = .282$) being higher with alcohol, with no significant main effect of water loss ($F_{(1.000, 10.00)} = 1.225$, p=.294, $\eta_p^2 = .156$) and no interaction ($F_{(1,10)} = 1.536$, p=.244, $\eta_p^2 = .133$) (Figure 9).

Perceptual Response

Figure 10

Mean difference in thermal comfort (TC) and thermal sensation (TS) during the 2-hour heat exposure



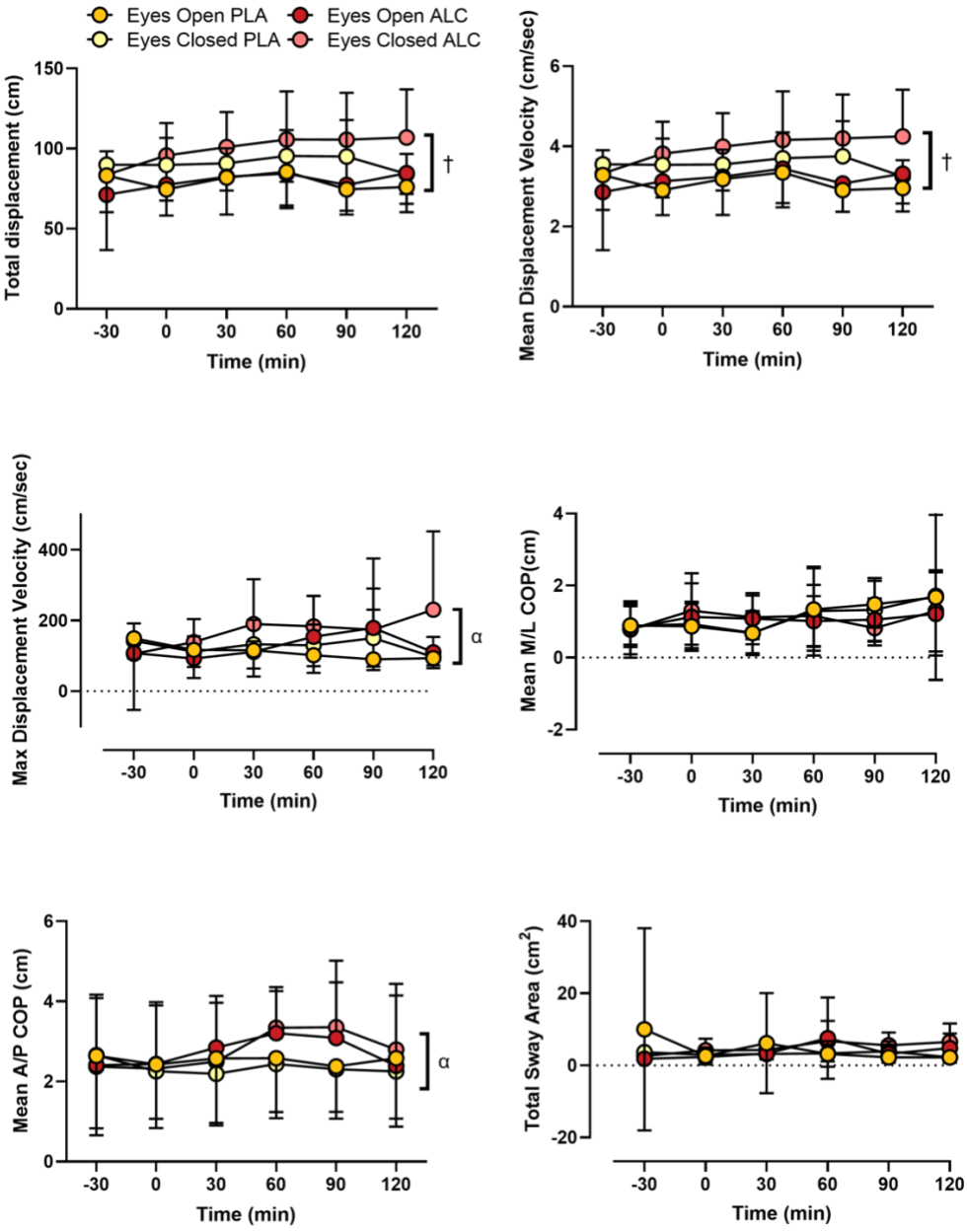
Note. Scores of subjective thermal comfort (left) and thermal sensation (right) during alcohol and control sessions. Mean difference was calculated between sessions; a score of 0 in thermal comfort indicates 'not uncomfortable' and 80 indicates 'neutral' in thermal sensation (see Appendix A).

Thermal comfort showed a significance main effect in time ($F_{(3.171, 31.71)} = 61.050$, $p<0.0001$, $\eta_p^2 = .887$) with no main effect of drink ($F_{(1, 10)} = 0.140$, $p=.716$, $\eta_p^2 = .018$) and no interaction ($F_{(3.33, 33.3)} = 1.589$, $p=.207$, $\eta_p^2 = .137$). Thermal sensation revealed a significant main effect of time ($F_{(1.575, 15.75)} = 73.280$, $p<0.0001$, $\eta_p^2 = .952$), no main effect of drink ($F_{(1,10)} = 0.022$, $p=.884$, $\eta_p^2 = .001$) and no interaction ($F_{(2.528, 25.28)} = 2.911$, $p=.062$, $\eta_p^2 = .225$) (Figure 10).

Postural Sway Response

Figure 11

Postural sway responses during passive heat stress using the Romberg test



Note. Postural sway metrics including total displacement (centimetres; top left), mean displacement velocity (centimetres/second; top right), maximum displacement velocity (centimeters/second; middle-left), mean medial-lateral centre of pressure (M/L COP; centimetres; middle right), mean anterior-posterior centre of pressure (A/P COP; centimeters; bottom left) and total sway area (centimeters; bottom right) during Romberg test conditions (eyes

open and eyes closed) under placebo (PLA) and alcohol (ALC) during heat stress. Data is presented in mean ± SD. The dagger (†) indicates a significant difference between drinks, and alpha (α) indicates a significance between eyes open and closed.

Total displacement revealed a significant main effect of time, ($F_{(3.045, 27.402)} = 5.202$, $p=.006$, $\eta_p^2 = .366$), a significant main effect of drink, ($F_{(1, 9)} = 17.537$, $p=.002$, $\eta_p^2 = .661$), with total displacement being higher with alcohol and no significant main effect of eyes, ($F_{(1, 9)} = 4.634$, $p=.060$, $\eta_p^2 = .34$). There was no significant interaction between time x drink, ($F_{(2.1, 18.897)} = 1.358$, $p=.282$, $\eta_p^2 = .131$), as well as no significant time × eyes interaction, ($F_{(2.091, 18.819)} = 1.848$, $p=.184$, $\eta_p^2 = .170$), and no drink × eyes interaction, ($F_{(1, 9)} = 1.795$, $p=.213$, $\eta_p^2 = .166$). Post-hoc tests for the main effect of time revealed no significant difference between time ($p>.05$) (Figure 11).

Mean displacement velocity revealed a significant main effect of time, ($F_{(3.046, 27.413)} = 3.125$, $p=.041$, $\eta_p^2 = .258$), a significant main effect of drink, ($F_{(1, 9)} = 6.437$, $p=.032$, $\eta_p^2 = .417$), with mean displacement velocity being higher with alcohol, and no significant main effect of eyes, ($F_{(1, 9)} = 1.055$, $p=.331$, $\eta_p^2 = .105$). There was also no significant interaction between time x drink, ($F_{(2.896, 26.061)} = 1.491$, $p=.241$, $\eta_p^2 = .142$), as well as no significant time × eyes interaction, ($F_{(1.452, 13.069)} = .837$, $p=.420$, $\eta_p^2 = .085$), and no drink × eyes interaction, ($F_{(1, 9)} = 1.055$, $p=.331$, $\eta_p^2 = .105$). Post-hoc tests for the main effect of time indicated a significant difference between time points -30 and 30 minutes ($p=.032$), but not between any other time points ($p>.05$) (Figure 11).

Maximum displacement velocity revealed no significant main effect of time, ($F_{(3.313, 29.818)} = .970$, $p=.426$, $\eta_p^2 = .097$), no significant main effect of drink, ($F_{(1, 9)} = 3.980$, $p=.077$, $\eta_p^2 = .307$), and a significant main effect of eyes, ($F_{(1, 9)} = 7.820$, $p=.021$, $\eta_p^2 = .465$). There was also no significant interaction between time x drink, ($F_{(2.804, 25.238)} = .583$, $p=.620$, $\eta_p^2 = .061$), as well

as no significant time × eyes interaction, ($F_{(4.177, 37.589)} = 1.839$, p=.139, $\eta_p^2 = .170$), and no drink × eyes interaction, ($F_{(1, 9)} = .617$, p=.452, $\eta_p^2 = .064$) (Figure 11).

Mean medio-lateral (M/L) centre of pressure (COP) revealed no significant main effect of time, ($F_{(2.227, 20.043)} = .941$, p=.416, $\eta_p^2 = .095$), no significant main effect of drink, ($F_{(1, 9)} = .002$, p=.965, $\eta_p^2 = .000$), and no significant main effect of eyes, ($F_{(1, 9)} = .796$, p=.395, $\eta_p^2 = .081$). There was also no significant interaction between time x drink, ($F_{(3.593, 32.340)} = 1.607$, p=.201, $\eta_p^2 = .151$), as well as no significant time × eyes interaction, ($F_{(2.365, 21.281)} = 1.070$, p=.370, $\eta_p^2 = .106$), and no drink × eyes interaction, ($F_{(1, 9)} = .028$, p=.871, $\eta_p^2 = .003$) (Figure 11).

Mean anterio-posterior (A/P) COP revealed no significant main effect of time ($F_{(4.205, 37.841)} = 1.148$, p=.350, $\eta_p^2 = .113$), no significant main effect of drink, ($F_{(1, 9)} = 1.263$, p=.290, $\eta_p^2 = .123$), and a significant main effect of eyes, ($F_{(1, 9)} = 5.105$, p=.050, $\eta_p^2 = .362$). There was also a significant interaction between time x drink, ($F_{(3.475, 31.271)} = 2.853$, p=.046, $\eta_p^2 = .241$), as well as no significant time × eyes interaction, ($F_{(3.923, 35.311)} = 1.279$, p=.297, $\eta_p^2 = .124$), and no drink × eyes interaction, ($F_{(1, 9)} = 1.590$, p=.239, $\eta_p^2 = .150$) (Figure 11).

Total sway area revealed no significant main effect of time ($F_{(1.241, 11.167)} = 1.466$, p=.259, $\eta_p^2 = .046$), no significant main effect of drink, ($F_{(1, 9)} = .006$, p = .940, $\eta_p^2 = .001$), and no significant main effect of eyes, ($F_{(1, 9)} = .429$, p=.529, $\eta_p^2 = .046$). There was also no significant interaction between time x drink, ($F_{(1.099, 9.894)} = .987$, p=.353, $\eta_p^2 = .099$), as well as no significant time × eyes interaction, ($F_{(1.122, 10.096)} = 2.007$, p=.188, $\eta_p^2 = .182$), and no drink × eyes interaction, ($F_{(1, 9)} = 1.453$, p=.259, $\eta_p^2 = .139$) (Figure 11).

Chapter 7: Discussion

The present thesis sought to evaluate how ethanol affects physiological, perceptual, and postural changes in older adults during heat stress. The primary outcome of this thesis demonstrated that a blood alcohol concentration of ~0.08 (mg/100mL), the threshold limit for operating a motor vehicle in Canada (D. of J. Government of Canada, 2018) can provoke increased cardiovascular strain, diuresis, orthostatic intolerance and postural instability in a hot environment (40°C). However, no significant differences were found in thermoregulatory and perceptual responses. These findings support the original hypothesis that alcohol consumption during acute heat exposure impairs postural control and does not significantly impact thermoregulation in older adults; however, contrary to the original hypothesis, perception remained unaffected.

Cardiovascular Responses

Heart Rate

Heart rate was higher and progressively rose during heat stress and was further exacerbated with alcohol consumption in older adults, which parallels our recent work in young adults (Brough et al., 2024; Lefebvre et al., 2025) (Figure 4). As described in young males by Rowell *et al.* (1969), heat stress increases cutaneous vasodilation, which reduces total peripheral resistance, resulting in a rise in heart rate to increase cardiac output to maintain systemic blood pressure. Ageing reduces the redistribution of blood from visceral circulation to peripheral cutaneous vascular beds, despite similar rises in heart rate during passive heat stress in comparison to young adults (Minson et al., 1998). Only three studies have measured heart rate in healthy younger adults during heat exposure and after alcohol consumption (Morris et al., 2024). One study reported no difference in heart rate when compared to a control trial (Allison & Reger, 1992), whereas

significant and non-significant elevations in heart rate have also been documented (Mekjavic et al., 1987; Yoda et al., 2005). It is speculated that either the independent effects of alcohol on i) peripheral resistance, and/or ii) stroke volume likely explain the nearly 2-fold rise in the heart rate response during heat stress relative to the thermoneutral environment (placebo: +12 BPM vs alcohol: +22 BPM). Previous evidence in men and women aged 24-47 reported a significant increase in heart rate after consuming a second glass of alcohol (blood alcohol to 72 ± 4 mg/dl for red wine or 80 ± 2 mg/dl for ethanol) in a thermoneutral environment (Spaak et al., 2008, 2010). Additional findings from Spaak *et al.* (2008 & 2010) suggest a dose-related impact of alcohol on heart rate, as increases in heart rate were only apparent when participants obtained higher blood alcohol concentrations. Further, cardiac output, muscle sympathetic nerve activity, and brachial artery diameter all increased after the second glass of alcohol (Spaak et al., 2008, 2010). Physiologically, alcohol inhibits vagal tone, which increases heart rate due to reduced parasympathetic restraint on the sinoatrial node (Newlin et al., 1990). Alcohol reduces vagal tone by inhibiting central vagal control, impairing baroreflex function and shifting autonomic balance towards the sympathetic nervous system (Newlin et al., 1990). Furthermore, alcohol causes peripheral vasodilation (Gillespie, 1967), which can lead to a drop in total peripheral resistance and thus blood pressure (as observed in Figure 6). In response, the carotid sinus and aortic arch detects this drop and triggers an increase in heart rate to maintain cardiac output and blood pressure (Goswami et al., 2017). Additionally, the maintenance of cardiac output is influenced by stroke volume. Acute alcohol consumption has been shown to incur a negative inotropic effect on the heart due to the depression of myocardial contractility (Danziger et al., 1991; Guarnieri & Lakatta, 1990). Impaired myocyte contractility results in reduced ventricular stroke volume and cardiac output (Starling, 1918). Thus, for a given cardiac output, a higher heart rate

is required to compensate for the reduced stroke volume. Older individuals demonstrate a reduced cardiac output during heat stress, influenced by the lower stroke volume as the heart rate response during passive heat stress is similar to young adults (Minson et al., 1998). Further work is needed to confirm whether stroke volume is further reduced in the heat in older adults following alcohol consumption.

Blood Pressure

During acute passive heat stress, expected cardiovascular responses maintain blood pressure well in young and older adults (Minson et al., 1998). However, ethanol alone produces hypotensive effects due to the diminished baroreceptor sensitivity (Abdel-Rahman et al., 1985; Rupp et al., 1996). There remains uncertainty regarding the combined effects of alcohol consumption and heat exposure on blood pressure; where in young adults, ethanol either produces no change (Allison & Reger, 1992) or a reduction in mean arterial pressure (Mekjavic et al., 1987). However, no study has evaluated the effect of ethanol on older adults during heat exposure (Morris et al., 2024). The present data (Figure 6) suggests that ethanol further decreases systolic and diastolic blood pressure in older adults when compared to the placebo condition. Older adults have impaired hemodynamic stability which is thought to contribute to their responses during heat stress (Kenney & Munce, 2003). Further, an older adults' reduced kidney function and impaired fluid regulation can also amplify alcohol's hypotensive effects (van de Borne et al., 1997). Thus, as older adults are already predisposed to impaired hemodynamic stability, it is likely that the combination of heat and alcohol may further exacerbate their symptoms. Our findings suggest that the increase in heart rate may have been insufficient to achieve steady blood pressure under the combined stresses of ethanol, heat, and age-related impacts on cardiovascular function.

Skin Blood Flow

Although older adults may exhibit attenuated responses during heat exposure, we observed a significant increase in skin blood flow after alcohol consumption compared to the placebo drink (Figure 5). Thus, ethanol may have an additive effect on cutaneous skin blood flow in the heat; however, we cannot confirm whether this increase in skin blood flow is due to enhanced vasodilation or differences in cardiac output due to rises in heart rate, warranting further investigation. The effect of alcohol consumption on young, healthy males has been noted to increase skin blood flow further when compared to a control (Yoda et al., 2005). The increased skin blood flow after alcohol consumption can be attributed to a decrease in vascular resistance due to alcohol-induced vasodilation (Gillespie, 1967; Kupari, 1983), which results from both a reduction in sympathetic vasoconstrictor tone and direct relaxation of vascular smooth muscles (Altura et al., 1978).

Orthostatic Tolerance

Our results demonstrate a main effect of both time and condition (Figure 7), indicating that during heat exposure, older adults are at higher risk for orthostatic intolerance with progressive heat exposure, which can be further exacerbated with alcohol consumption. In thermoneutral conditions, upon standing, aortic and carotid blood pressure decreases, thereby unloading the aortic arch and carotid sinus baroreceptors (Goswami et al., 2017). Hence, a rapid increase in heart rate, achieved through vagal withdrawal and sympathetic activation of peripheral vascular resistance, is attempted to maintain arterial blood pressure (Rowell, 1993). If blood pressure cannot be maintained, individuals will likely suffer from orthostatic hypotension and balance impairments.

Heat stress induces cutaneous vasodilation, leading to increased skin blood flow (Figure 5), which in turn reduces systemic vascular resistance (Rowell et al., 1969). Meanwhile, sweating can decrease circulating fluid volume (Mack & Nadel, 1996). Together, these responses compromise central blood volume and venous return, placing additional strain on the cardiovascular system (Crandall & González-Alonso, 2010; C. J. Smith & Johnson, 2016). When upright, this can impair the body's ability to maintain blood pressure, thus reducing orthostatic tolerance.

Heat, alcohol and aging are some of the predisposing factors to peripheral venous pooling, which can also increase the risk of orthostatic intolerance (Dani et al., 2021; Ferreira et al., 2024; Ricci et al., 2015). It has been hypothesized by Johnson et al. (1973) that as core temperature rises, the baroreflex mechanism is unable to overcome the heat-induced vasodilation, thereby lowering orthostatic tolerance (Mekjavic et al., 1987). Ferreira et al. (2024) suggest that the combined effects of heat-induced vasodilation and gravitational blood pooling provoked a transient central hypovolemia. This results in insufficient hemodynamic compensation, as stroke volume, cardiac output and blood pressure were not maintained in the hot condition compared to the thermoneutral control (Ferreira et al., 2024).

Furthermore, alcohol also depresses orthostatic tolerance by attenuating baroreflex sensitivity (Abdel-Rahman et al., 1987) and suppressing muscle sympathetic nerve activity (Carter et al., 2011), which reduces vasoconstrictor tone in peripheral circulation (Carter et al., 2011; Narkiewicz et al., 2000). Combined with alcohol-induced vasodilation and diuresis, this blunted sympathetic response can potentially impair maintenance of blood pressure during orthostatic stress. Alcohol, dehydration and warm environments are well-known behaviour-dependent triggers for orthostatic hypotension (Freeman et al., 2011). Within our results (Figure

7), we can hypothesize that these factors physiologically impair orthostatic tolerance, thereby compromising the body's ability to maintain blood pressure upon standing. Individuals with existing deficits to orthostatic stress, such as older adults, may exhibit more pronounced effects of orthostatic intolerance after drinking alcohol in a warm environment.

Thermoregulatory Responses

Core and Skin Temperature

As an extension of previous evidence, the present thesis confirms that alcohol does not impact core and skin temperature responses during heat stress in older adults (Figure 8), which is consistent with recent data collection in young adults (Brough et al., 2024; Lefebvre et al., 2025). A recent systematic scoping review concluded that there is no literature on the physiological thermoregulatory responses in older adults following ethanol consumption (Morris et al., 2024). In young male adults, evidence to date suggests that the consumption of alcohol does not negatively influence thermoregulation. In fact, some papers have reported a reduction in core temperature during heat stress following alcohol consumption (Desruelle et al., 1996; Yoda et al., 2005); however, this is inconsistent, whereas some research has shown no change (Allison & Reger, 1992). Consistently, however, mean skin temperature remains unaffected with alcohol consumption in younger adults (Allison & Reger, 1992; Desruelle et al., 1996; Livingstone et al., 1980; Yoda et al., 2005).

Sweat Rate

Local and whole-body sweat rates were not altered by alcohol consumption (Figure 8); however, a significant main effect of time was observed for local sweat rate, reflecting the expected progressive increase in sweat rate during prolonged heat exposure. However, alcohol

did not independently modulate this response. Data thus far show that in warm environments, neither younger nor older adults experience a difference in local sweat rates with alcohol consumption (Allison & Reger, 1992; Brough et al., 2024; Desruelle et al., 1996; Gibiński et al., 1979; Lefebvre et al., 2025).

Diuretic Effects

Urine Output

Although we observed a trend for increased urine output in our older adults (Figure 7), unpublished data from our younger adult cohort demonstrated a significant effect of alcohol on urine output. Thus, total body water loss (combination of urine output and sweating) was greater following alcohol consumption, which may reduce blood volume and further support the observed compensatory increase in heart rate to maintain cardiac output and blood pressure (Figure 4).

Exposure to heat increases the risk of dehydration, which is commonly identified by markers such as increased urine output and sweat loss. When consuming diuretics, like ethanol, in these conditions, the likelihood of dehydration is further amplified (Shirreffs & Maughan, 1997). The risk is especially concerning for older adults who are prone to presenting with reduced thirst sensation and less efficient renal urinary concentrating mechanisms (Phillips et al., 1984), making them more vulnerable to fluid imbalances and the dehydrating effects of both heat and ethanol. Emerging evidence suggests alcohol's diuretic effect may be acute and transient. In a recent study examining the diuretic action of alcohol in elderly men, results indicate that stronger alcoholic beverages (e.g., wine and spirits) provoked a short-term diuretic effect within two hours of consumption, but not after the 24-hour urine collection (Polhuis et al., 2017). Similar

dose-dependent and time effects are consistent throughout the literature (Eggleton, 1942; Hobson & Maughan, 2010; Saini et al., 1995; Shirreffs & Maughan, 1997; Strauss et al., 1950).

This may suggest that alcohol concentration, rather than net alcohol content, determines the acute effect of alcohol on cumulative urine output.

It must be acknowledged that older adults may exhibit a reduced diuretic response to alcohol due to age-related impairments in renal concentration ability and altered vasopressin regulation, which collectively can blunt fluid loss compared to younger adults (Beck, 1998). In older adults, glomerular filtration rate decreases, and tubular cells are less responsive to vasopressin (Beck, 1998). Alcohol inhibits arginine vasopressin (AVP), a hormone that promotes water reabsorption in the kidneys (Eisenhofer & Johnson, 1982; Taivainen et al., 1995); however, in older adults, AVP levels are often already elevated, and AVP regulation is usually less responsive (Cowen et al., 2013; A. G. Johnson et al., 1994). Therefore, alcohol may not reduce AVP as effectively, due to a higher AVP baseline when compared to younger adults (A. G. Johnson et al., 1994) and possibly expect less urine output from older adults, which likely explains the trend observed.

Perceptual Responses

The ability of older adults to perceive and respond to heat stress has been shown to be significantly impaired due to blunted thermal sensitivity, which can increase their risk of heat-related injuries. With the limited evidence that explores the effect of alcohol on thermal comfort within any demographic, inconsistent findings have been reported. In corroboration of our findings (Figure 10), Allison and Reger (1992) saw no change after alcohol consumption in young healthy men during hot water immersion. However, Yoda and colleagues (2005) did report young men feeling hotter after alcohol consumption; within the literature, these findings have not yet been reciprocated in older adults and were not significant in my thesis. It should be

noted that Yoda et al. (2005) participants identified as Asian, which could have contributed to the higher sensitivity of thermal comfort due to their pronounced physiological and perceptual reaction to alcohol when compared to Caucasians (Wolff, 1972; Yoda et al., 2005). While thermal sensation and comfort were not affected by alcohol in our older adults assessed, it still remains to be evaluated whether alcohol will impact the decision-making process to engage heat stress preventative measures.

Postural Sway

Our findings suggest that both ethanol and heat have a statistically significant influence on postural sway in older adults (Figure 11). Significant main effects of time and drink were observed for total displacement and mean displacement velocity. Additionally, the impact of eyes on maximum displacement velocity and mean A/P COP were observed, highlighting the importance of visual input in maintaining stability. The interaction between time and drink on mean A/P COP further suggests that the combined effects of heat and ethanol can exacerbate anterior-posterior sway.

In young, healthy adults, postural instability is present during heat exposure (Ferreira et al., 2024) and after alcohol consumption (Nieschalk et al., 1999). The overall impairment of static balance control caused by ethanol can be observed by a greater degree of postural sway and an inability to coordinate voluntary and postural movements (Begbie, 1966; Woollacott, 1983). It has been demonstrated that ethanol primarily affects the vestibulo-cerebellum (Ikeda et al., 1980; Kubo et al., 1989; Nieschalk et al., 1999) given that the vestibular nuclear neurons are more susceptible to ethanol than the medial geniculate and trigeminal nuclei (Ikeda et al., 1980). Moreover, older adults are susceptible to neuronal loss in the vestibular neural complex starting

at the age of 40 (Lopez et al., 1997); therefore, the combination of age and ethanol may be particularly detrimental to vestibular function, exacerbating postural sway impairments.

Furthermore, postural sway is often associated with a positive correlation with BAC (Kubo et al., 1989; Lukas et al., 1989; Nieschalk et al., 1999); yet, it has been hypothesized that postural sway does not significantly increase until BAC reaches 0.08 (Kubo et al., 1989; Nieschalk et al., 1999).

In thermoneutral conditions, it is hypothesized that an interaction exists among the cardiovascular, postural, and musculoskeletal systems, which is critical for maintaining postural stability and orthostatic tolerance (Verma et al., 2017). Subsequently, the role of the lower leg muscles may be more effective in regulating blood pressure compared to the baroreflex mechanism (Guyton et al., 1962; Verma et al., 2017). Therefore, slight increases in postural sway while standing have been shown to play a significant role in enhancing venous return and supporting orthostatic tolerance (Blaber et al., 2013; Inamura et al., 1996; Murata et al., 2012). However, in a hot environment, gastrocnemius activity was reduced in young adults compared to a thermoneutral environment (Ferreira et al., 2024), suggesting that the increased cardiovascular strain, such as increased skin blood flow during heat stress, may limit the capacity of skeletal muscles to support venous return and stabilize blood pressure (J. M. Johnson, 2010).

During heat stress, postural sway increases, which can be attributed to a decrease in the skeletal muscle pump (Ferreira et al., 2024). An alternative approach suggests that during heat exposure, rising body temperature impairs the nervous system's ability to process sensory information, resulting in a blunted somatosensory response (Dubois et al., 1981). Aging is associated with a decline in the functioning of the somatosensory and motor systems, which in turn can lead to poor standing balance (Hurley et al., 1998; Lord et al., 1991). Thus, when

compared to younger adults, older adults have also been shown to exhibit increased postural instability (Kollegger et al., 2008). Although this is the first study to be conducted on older adults in hot ambient temperatures and after alcohol consumption, our findings remain consistent with the literature that has evaluated each predisposing factor independently.

Current Perspective

Our findings suggest that alcohol consumption during heat exposure may pose a health risk for older adults. Specifically, this study is the first to examine the combined effects of ethanol and heat in an older population, revealing impairments in cardiovascular responses and postural stability. This new evidence provides critical insights for public health messaging and risk management during heatwaves, particularly as climate change intensifies. Our data complement existing assumptions, based primarily on younger male cohorts, that alcohol does not affect thermoregulation but does impair other physiological responses.

Limitations/Delimitations

This study is subject to certain limitations that should be considered when interpreting the findings. The first limitation is the small sample size. Although a sample size calculation was performed a priori to detect a statistically significant effect in heart rate [$\alpha = 0.05$, $\beta=0.8$], we were unable to reach the target number of participants due to recruitment challenges and our exclusion criteria. As a result, our final sample was smaller than anticipated ($n = 11$), which may have reduced the statistical power of our analyses. However, this study is designed to expose older adults to heat after consuming alcohol and, therefore, can act as a framework for future studies. Another limitation of this study is that the results cannot be generalized to all concentrations of alcohol, as previous research has shown that different alcohol concentrations and forms may produce varying physiological effects.

Furthermore, we chose to use a commercially available and commonly consumed spirit to enhance the ecological validity of our findings. Another limitation is that we studied healthy older adults in Canada; in Canada, approximately two-thirds of older adults take five or more prescription medications, and nearly one-quarter take at least ten, where medications may interact negatively with alcohol (Canadian Institute for Health Information, 2018). The results of this study can only be generalized to Caucasian populations, as all participants identified as Caucasian, limiting the applicability of the findings to other racial or ethnic groups. For example, some Japanese cannot drink alcohol as they lack mitochondrial aldehyde dehydrogenase activity, which is essential for alcohol metabolism (Harada et al., 1980; Shibuya et al., 1989). Another limitation of this study is that the postural sway board used was not research-grade equipment, which may affect the sensitivity and resolution of the balance measurements obtained. However, a comparative study was conducted using the Wii Balance Board (WBB) and the same software (CU BrainBLoX), and they concluded that the WBB can be used as a low-cost substitute for a medical-grade Posturography machine for quantification of balance in situations where precise measurement of balance is not required (Singh et al., 2021).

Conclusion

In conclusion, the findings highlight the intricate interaction between heat stress, ethanol, and older adults. The main finding of this study was that ethanol during heat exposure provoked increased cardiovascular strain, total body water loss and postural instability, and a trend for higher urine output. No significant differences were found in thermoregulatory and perceptual responses. Collectively, this suggest that older adults exhibit similar findings to younger adults, as supported by our unpublished data and existing literature. The results of this study represent the importance of understanding how environmental and behavioural factors interact with aging

physiology and how this information can be beneficial for guiding hydration strategies, assessing safety during heat exposure, and developing interventions to protect heat-vulnerable populations, such as older adults.

References

Abdel-Rahman, A. R., Dar, M. S., & Wooles, W. R. (1985). Effect of chronic ethanol administration on arterial baroreceptor function and pressor and depressor responsiveness in rats. *The Journal of Pharmacology and Experimental Therapeutics*, *232*(1), 194–201. https://doi.org/10.1016/S0022-3565(25)20099-4

Abdel-Rahman, A.-R. A., Merrill, R. H., & Wooles, W. R. (1987). Effect of acute ethanol administration on the baroreceptor reflex control of heart rate in normotensive human volunteers. *Clinical Science*, *72*(1), 113–122. https://doi.org/10.1042/cs0720113

Abrahamson, V., Wolf, J., Lorenzoni, I., Fenn, B., Kovats, S., Wilkinson, P., Adger, W. N., & Raine, R. (2009). Perceptions of heatwave risks to health: Interview-based study of older people in London and Norwich, UK. *Journal of Public Health*, *31*(1), 119–126. https://doi.org/10.1093/pubmed/fdn102

Ajjan, N., & Page, A. (2023, May 23). *Heat-related illnesses: Physiology, symptoms and management*. The Pharmaceutical Journal. https://pharmaceutical-journal.com/article/ld/heat-related-illnesses-physiology-symptoms-and-management

Allison, T. G., & Reger, W. E. (1992). Thermoregulatory, cardiovascular, and psychophysical response to alcohol in men in 40 degrees C water. *Journal of Applied Physiology*, *72*(6), 2099–2107. https://doi.org/10.1152/jappl.1992.72.6.2099

Altura, B. M., Carella, A., & Altura, B. T. (1978). Acetaldehyde on vascular smooth muscle: Possible role in vasodilator action of ethanol. *European Journal of Pharmacology*, *52*(1), 73–83. https://doi.org/10.1016/0014-2999(78)90023-7

Anderson, B. G., & Bell, M. L. (2009). Weather-Related Mortality: How Heat, Cold, and Heat Waves Affect Mortality in the United States. *Epidemiology*, *20*(2), 205. https://doi.org/10.1097/EDE.0b013e318190ee08

Apfel, F., Bickler, G., Biggelaar, M. van den, Bouchama, A., Cardeñosa Marín, N., Hales, S., Koppe, C., Kovats, S., Laaidi, K., Matthies, F., Menne, B., Morgan, A., Páldy, A., & Tavares, A. (2008). *Heat-health action plans: Guidance* (R. O. for E. World Health Organization, Ed.). World Health Organization. https://www.who.int/publications/i/item/9789289071918

Aranha Rosito, G. (1999). Dose-dependent biphasic effect of ethanol on 24-h blood pressure in normotensive subjects. *American Journal of Hypertension*, *12*(2), 236–240. https://doi.org/10.1016/S0895-7061(98)00237-4

[ARCHIVED] Heat advice: Alcohol and caffeine | National Collaborating Centre for Environmental Health | NCCEH - CCSNE. (2010, October 1). https://ncceh.ca/resources/evidence-briefs/archived-heat-advice-alcohol-and-caffeine

Arthur, M. J. P., Lee, A., & Wright, R. (1984). Sex Differences in the Metabolism of Ethanol and Acetaldehyde in Normal Subjects. *Clinical Science*, *67*(4), 397–401. https://doi.org/10.1042/cs0670397

Attia, M. (1984). Thermal pleasantness and temperature regulation in man. *Neuroscience and Biobehavioral Reviews*, *8*(3), 335–342. https://doi.org/10.1016/0149-7634(84)90056-3

Beck, L. H. (1998). Changes in Renal Function with Aging. *Clinics in Geriatric Medicine*, *14*(2), 199–210. https://doi.org/10.1016/S0749-0690(18)30113-7

Begbie, G. H. (1966). The Effects of Alcohol and of Varying Amounts of Visual Information on a Balancing Test. *Ergonomics*, *9*(4), 325–333. https://doi.org/10.1080/00140136608964388

Bittel, J., & Henane, R. (1975). Comparison of thermal exchanges in men and women under neutral and hot conditions. *The Journal of Physiology*, *250*(3), 475–489. https://doi.org/10.1113/jphysiol.1975.sp011066

Blaber, A. P., Zuj, K. A., & Goswami, N. (2013). Cerebrovascular autoregulation: Lessons learned from spaceflight research. *European Journal of Applied Physiology*, *113*(8), 1909–1917. https://doi.org/10.1007/s00421-012-2539-x

Bobb, J. F., Obermeyer, Z., Wang, Y., & Dominici, F. (2014). *Cause-Specific Risk of Hospital Admission Related to Extreme Heat in Older Adults*.

Bouchama, A., & Knochel, J. P. (2002). Heat Stroke. *New England Journal of Medicine*, *346*(25), 1978–1988. https://doi.org/10.1056/NEJMra011089

Brough, A., Lefebvre, K., Mihalcin, E., & Ravanelli, N. (2024). The Physiological and Perceptual Responses During Acute Heat Stress Following Alcohol Consumption. *Physiology*, *39*(S1), 2560. https://doi.org/10.1152/physiol.2024.39.S1.2560

Canada, H. (2021, December 20). *Canadian Alcohol and Drugs Survey (CADS): Summary of results for 2019* [Surveys]. https://www.canada.ca/en/health-canada/services/canadian-alcohol-drugs-survey/2019-summary.html

Carey, B., & Potter, J. (2001). Cardiovascular causes of falls. *Age and Ageing*, *30 Suppl 4*, 19–24. https://doi.org/10.1093/ageing/30.suppl_4.19

Carlton, P. L., & Marks, R. A. (1958). Cold exposure and heat reinforced operant behavior. *Science (New York, N.Y.)*, *128*(3335), 1344. https://doi.org/10.1126/science.128.3335.1344

Carter, J. R., Stream, S. F., Durocher, J. J., & Larson, R. A. (2011). Influence of acute alcohol ingestion on sympathetic neural responses to orthostatic stress in humans. *American Journal of Physiology-Endocrinology and Metabolism*, *300*(5), E771–E778. https://doi.org/10.1152/ajpendo.00674.2010

Chithramol, M. K., & Shine, S. R. (2023). Review on modelling approaches of thermoregulation mechanisms. *Journal of Thermal Analysis and Calorimetry*, *148*(17), 9343–9360. https://doi.org/10.1007/s10973-023-12132-1

Cole-Harding, S., & Wilson, J. R. (1987). Ethanol metabolism in men and women. *Journal of Studies on Alcohol*, *48*(4), 380–387. https://doi.org/10.15288/jsa.1987.48.380

Convertino, V. A. (2014). Neurohumoral mechanisms associated with orthostasis: Reaffirmation of the significant contribution of the heart rate response. *Frontiers in Physiology*, *5*. https://doi.org/10.3389/fphys.2014.00236

Cowen, L. E., Hodak, S. P., & Verbalis, J. G. (2013). Age-Associated Abnormalities of Water Homeostasis. *Endocrinology and Metabolism Clinics of North America*, *42*(2), 349–370. https://doi.org/10.1016/j.ecl.2013.02.005

Cramer, M. N., Gagnon, D., Laitano, O., & Crandall, C. G. (2022). Human temperature regulation under heat stress in health, disease, and injury. *Physiological Reviews*, *102*(4), 1907–1989. https://doi.org/10.1152/physrev.00047.2021

Crandall, C. G., & González-Alonso, J. (2010). Cardiovascular function in the heat-stressed human. *Acta Physiologica*, *199*(4), 407–423. https://doi.org/10.1111/j.1748-1716.2010.02119.x

Daines, R. (2025, July 10). *Partial Eta Squared* [National University, Academic Success Center]. https://resources.nu.edu/statsresources/eta

Dani, M., Dirksen, A., Taraborrelli, P., Panagopolous, D., Torocastro, M., Sutton, R., & Lim, P. B. (2021). Orthostatic hypotension in older people: Considerations, diagnosis and management. *Clinical Medicine*, *21*(3), e275–e282. https://doi.org/10.7861/clinmed.2020-1044

Danziger, R. S., Sakai, M., Capogrossi, M. C., Spurgeon, H. A., Hansford, R. G., & Lakatta, E. G. (1991). Ethanol acutely and reversibly suppresses excitation-contraction coupling in cardiac myocytes. *Circulation Research*, *68*(6), 1660–1668. https://doi.org/10.1161/01.RES.68.6.1660

Darowski, A., Chambers, S.-A. C. F., & Chambers, D. J. (2009). Antidepressants and Falls in the Elderly. *Drugs & Aging*, *26*(5), 381–394. https://doi.org/10.2165/00002512-200926050-00002

De Marchi, S., Cecchin, E., Basile, A., Bertotti, A., Nardini, R., & Bartoli, E. (1993). Renal Tubular Dysfunction in Chronic Alcohol Abuse—Effects of Abstinence. *New England Journal of Medicine*, *329*(26), 1927–1934. https://doi.org/10.1056/NEJM199312233292605

Dematte, J. E., O'Mara, K., Buescher, J., Whitney, C. G., Forsythe, S., McNamee, T., Adiga, R. B., & Ndukwu, I. M. (1998). Near-Fatal Heat Stroke during the 1995 Heat Wave in

Chicago. *Annals of Internal Medicine*, *129*(3), 173–181. https://doi.org/10.7326/0003-4819-129-3-199808010-00001

Desruelle, A.-V., Boisvert, P., & Candas, V. (1996). Alcohol and its variable effect on human thermoregulatory response to exercise in a warm environment. *European Journal of Applied Physiology and Occupational Physiology*, *74*(6), 572–574. https://doi.org/10.1007/BF02376775

Dry February, you say? (2023, February 2). Statistics Canada. https://www.statcan.gc.ca/o1/en/plus/2877-dry-february-you-say

Dubois, M., Coppola, R., Buchsbaum, M. S., & Lees, D. E. (1981). Somatosensory evoked potentials during whole body hyperthermia in humans. *Electroencephalography and Clinical Neurophysiology*, *52*(2), 157–162. https://doi.org/10.1016/0013-4694(81)90163-2

Dufour, A., & Candas, V. (2007). Ageing and thermal responses during passive heat exposure: Sweating and sensory aspects. *European Journal of Applied Physiology*, *100*(1), 19–26. https://doi.org/10.1007/s00421-007-0396-9

Ebi, K. L., Capon, A., Berry, P., Broderick, C., Dear, R. de, Havenith, G., Honda, Y., Kovats, R. S., Ma, W., Malik, A., Morris, N. B., Nybo, L., Seneviratne, S. I., Vanos, J., & Jay, O. (2021). Hot weather and heat extremes: Health risks. *The Lancet*, *398*(10301), 698–708. https://doi.org/10.1016/S0140-6736(21)01208-3

Eby, J. M., & Majetschak, M. (2019). Effects of ethanol and ethanol metabolites on intrinsic function of mesenteric resistance arteries. *PLOS ONE*, *14*(3), e0214336. https://doi.org/10.1371/journal.pone.0214336

Eggleton, M. G. (1942). The diuretic action of alcohol in man. *The Journal of Physiology*, *101*(2), 172–191.

Eisenhofer, G., & Johnson, R. H. (1982). Effect of ethanol ingestion on plasma vasopressin and water balance in humans. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *242*(5), R522–R527. https://doi.org/10.1152/ajpregu.1982.242.5.R522

Engelland, R. E., Hemingway, H. W., Tomasco, O. G., Olivencia-Yurvati, A. H., & Romero, S. A. (2020). Neural control of blood pressure is altered following isolated leg heating in aged humans. *American Journal of Physiology-Heart and Circulatory Physiology*, *318*(4), H976–H984. https://doi.org/10.1152/ajpheart.00019.2020

Epstein, A. N., & Milestone, R. (1968). Showering as a coolant for rats exposed to heat. *Science (New York, N.Y.)*, *160*(3830), 895–896. https://doi.org/10.1126/science.160.3830.895

Esler, M. (2010). The 2009 Carl Ludwig Lecture: Pathophysiology of the human sympathetic nervous system in cardiovascular diseases: the transition from mechanisms to medical management. *Journal of Applied Physiology*, *108*(2), 227–237. https://doi.org/10.1152/japplphysiol.00832.2009

Farnell, S., Maxwell, L., Tan, S., Rhodes, A., & Philips, B. (2005). Temperature measurement: Comparison of non-invasive methods used in adult critical care. *Journal of Clinical Nursing*, *14*(5), 632–639. https://doi.org/10.1111/j.1365-2702.2004.00916.x

Ferreira, F. C., Padilha, M. C. S. V., Araujo-Leite, M. A., Da Silva Soares, P. P., & Rodrigues, G. D. (2024). Heated environment increases blood pressure drop and postural sway during initial orthostasis in healthy subjects. *European Journal of Applied Physiology*. https://doi.org/10.1007/s00421-024-05546-8

Fleg, J. L., Evans, G. W., Margolis, K. L., Barzilay, J., Basile, J. N., Bigger, J. T., Cutler, J. A., Grimm, R., Pedley, C., Peterson, K., Pop-Busui, R., Sperl-Hillen, J., & Cushman, W. C. (2016). Orthostatic Hypotension in the ACCORD (Action to Control Cardiovascular Risk in Diabetes) Blood Pressure Trial. *Hypertension*, *68*(4), 888–895. https://doi.org/10.1161/HYPERTENSIONAHA.116.07474

Flores-Salamanca, R., & Aragon-Vargas, L. F. (2014). Postexercise rehydration with beer impairs fluid retention, reaction time, and balance. *Applied Physiology, Nutrition, and Metabolism*, *39*(10), 1175–1182. https://doi.org/10.1139/apnm-2013-0576

Flouris, A. D. (2011). Functional architecture of behavioural thermoregulation. *European Journal of Applied Physiology*, *111*(1), 1–8. https://doi.org/10.1007/s00421-010-1602-8

Folk, G. E. (with Riedesel, M. L., & Thrift, D. L.). (1998). *Principles of integrative environmental physiology*. Austin & Winfield Publishers.

Freeman, R., Wieling, W., Axelrod, F. B., Benditt, D. G., Benarroch, E., Biaggioni, I., Cheshire, W. P., Chelimsky, T., Cortelli, P., Gibbons, C. H., Goldstein, D. S., Hainsworth, R., Hilz, M. J., Jacob, G., Kaufmann, H., Jordan, J., Lipsitz, L. A., Levine, B. D., Low, P. A., … Van Dijk, J. G. (2011). Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. *Autonomic Neuroscience*, *161*(1–2), 46–48. https://doi.org/10.1016/j.autneu.2011.02.004

Fu, Q., & Levine, B. D. (2014). Pathophysiology of neurally mediated syncope: Role of cardiac output and total peripheral resistance. *Autonomic Neuroscience*, *184*, 24–26. https://doi.org/10.1016/j.autneu.2014.07.004

Fuller-Jackson, J.-P., Clarke, I. J., & Henry, B. A. (2017). Animal Models for Manipulation of Thermogenesis. In P. M. Conn (Ed.), *Animal Models for the Study of Human Disease*

(Second Edition) (pp. 281–312). Academic Press. https://doi.org/10.1016/B978-0-12-809468-6.00012-7

Furlan, R., Porta, A., Costa, F., Tank, J., Baker, L., Schiavi, R., Robertson, D., Malliani, A., & Mosqueda-Garcia, R. (2000). Oscillatory Patterns in Sympathetic Neural Discharge and Cardiovascular Variables During Orthostatic Stimulus. *Circulation*, *101*(8), 886–892. https://doi.org/10.1161/01.CIR.101.8.886

Gagge, A. P., Stolwijk, J. A. J., & Saltin, B. (1969). Comfort and thermal sensations and associated physiological responses during exercise at various ambient temperatures. *Environmental Research*, *2*(3), 209–229. https://doi.org/10.1016/0013-9351(69)90037-1

Gagnon, D., Romero, S. A., Ngo, H., Sarma, S., Cornwell, W. K., Poh, P. Y. S., Stoller, D., Levine, B. D., & Crandall, C. G. (2016). Healthy aging does not compromise the augmentation of cardiac function during heat stress. *Journal of Applied Physiology*, *121*(4), 885–892. https://doi.org/10.1152/japplphysiol.00643.2016

Gagnon, D., Romero, S. A., Ngo, H., Sarma, S., Cornwell, W. K., Poh, P. Y. S., Stoller, D., Levine, B. D., & Crandall, C. G. (2017). Volume loading augments cutaneous vasodilatation and cardiac output of heat stressed older adults. *The Journal of Physiology*, *595*(20), 6489–6498. https://doi.org/10.1113/JP274742

Galli, A., Barbic, F., Borella, M., Costantino, G., Perego, F., Dipaola, F., Casella, F., Duca, P. G., Diedrich, A., Raj, S., Robertson, D., Porta, A., Furlan, R., & Investigators, on behalf of the St. (2011). Influence of Climate on Emergency Department Visits for Syncope: Role of Air Temperature Variability. *PLOS ONE*, *6*(7), e22719. https://doi.org/10.1371/journal.pone.0022719

Gibiński, K., Giec, L., Zmudzifiski, J., & Markiewicz, A. (1979). ADH and thermal sweating. *European Journal of Applied Physiology and Occupational Physiology*, *42*, 1–13. https://doi.org/10.1007/BF00421099

Gillespie, J. A. (1967). Vasodilator properties of alcohol. *British Medical Journal*, *2*(5547), 274–277. https://doi.org/10.1136/bmj.2.5547.274

Global Surface Temperature | NASA Global Climate Change. (n.d.). Climate Change: Vital Signs of the Planet. Retrieved July 24, 2024, from https://climate.nasa.gov/vital-signs/global-temperature?intent=121

Goist, K. C., & Sutker, P. B. (1985). Acute alcohol intoxication and body composition in women and men. *Pharmacology Biochemistry and Behavior*, *22*(5), 811–814. https://doi.org/10.1016/0091-3057(85)90532-5

Goswami, N., Blaber, A. P., Hinghofer-Szalkay, H., & Montani, J.-P. (2017). Orthostatic Intolerance in Older Persons: Etiology and Countermeasures. *Frontiers in Physiology*, *8*. https://doi.org/10.3389/fphys.2017.00803

Government of Canada, D. of J. (2018, June 22). *Impaired Driving Laws*. https://www.justice.gc.ca/eng/cj-jp/sidl-rlcfa/

Government of Canada, S. C. (2022, April 27). *A generational portrait of Canada's aging population from the 2021 Census*. https://www12.statcan.gc.ca/census-recensement/2021/as-sa/98-200-X/2021003/98-200-X2021003-eng.cfm

Greaney, J. L., Stanhewicz, A. E., Proctor, D. N., Alexander, L. M., & Kenney, W. L. (2015). Impairments in central cardiovascular function contribute to attenuated reflex vasodilation in aged skin. *Journal of Applied Physiology*, *119*(12), 1411–1420. https://doi.org/10.1152/japplphysiol.00729.2015

Grosiak, M., Koteja, P., Bauchinger, U., & Sadowska, E. T. (2020). Age-Related Changes in the Thermoregulatory Properties in Bank Voles From a Selection Experiment. *Frontiers in Physiology*, *11*. https://doi.org/10.3389/fphys.2020.576304

Guarnieri, T., & Lakatta, E. G. (1990). Mechanism of myocardial contractile depression by clinical concentrations of ethanol. A study in ferret papillary muscles. *The Journal of Clinical Investigation*, *85*(5), 1462–1467. https://doi.org/10.1172/JCI114592

Guergova, S., & Dufour, A. (2011). Thermal sensitivity in the elderly: A review. *Ageing Research Reviews*, *10*(1), 80–92. https://doi.org/10.1016/j.arr.2010.04.009

Guyton, A. C., Douglas, B. H., Langston, J. B., Richardson, T. Q., & Abernathy, B. (1962). Instantaneous Increase in Mean Circulatory Pressure and Cardiac Output at Onset of Muscular Activity. *Circulation Research*, *11*(3), 431–441. https://doi.org/10.1161/01.RES.11.3.431

Harada, S., Misawa, S., Agarwal, D. P., & Goedde, H. W. (1980). Liver alcohol dehydrogenase and aldehyde dehydrogenase in the Japanese: Isozyme variation and its possible role in alcohol intoxication. *American Journal of Human Genetics*, *32*(1), 8–15.

Heat Alert and Response Systems (HARS). (2023). Public Health Ontario.

Hellon, R. F., & Lind, A. R. (1956). Observations on the activity of sweat glands with special reference to the influence of ageing. *The Journal of Physiology*, *133*(1), 132–144. https://doi.org/10.1113/jphysiol.1956.sp005571

Hiitola, P., Enlund, H., Kettunen, R., Sulkava, R., & Hartikainen, S. (2009). Postural changes in blood pressure and the prevalence of orthostatic hypotension among home-dwelling elderly aged 75 years or older. *Journal of Human Hypertension*, *23*(1), 33–39. https://doi.org/10.1038/jhh.2008.81

Hobson, R. M., & Maughan, R. J. (2010). Hydration Status and the Diuretic Action of a Small Dose of Alcohol. *Alcohol and Alcoholism*, *45*(4), 366–373. https://doi.org/10.1093/alcalc/agq029

Holowatz, L. A., & Kenney, W. L. (2011). Acute localized administration of tetrahydrobiopterin and chronic systemic atorvastatin treatment restore cutaneous microvascular function in hypercholesterolaemic humans. *The Journal of Physiology*, *589*(19), 4787–4797. https://doi.org/10.1113/jphysiol.2011.212100

Hughes, J. H., Henry, R. E., & Daly, M. J. (1984). Influence of ethanol and ambient temperature on skin blood flow. *Annals of Emergency Medicine*, *13*(8), 597–600. https://doi.org/10.1016/S0196-0644(84)80282-6

Hurley, M. V., Rees, J., & Newham, D. J. (1998). Quadriceps function, proprioceptive acuity and functional performance in healthy young, middle-aged and elderly subjects. *Age and Ageing*, *27*(1), 55–62. https://doi.org/10.1093/ageing/27.1.55

Ikeda, Y., Sasa, M., & Takaori, S. (1980). Selective Effect of Ethanol on the Vestibular Nucleus Neurons in the Cat. *The Japanese Journal of Pharmacology*, *30*(5), 665–673. https://doi.org/10.1254/jjp.30.665

Inamura, K., Mano, T., Iwase, S., Amagishi, Y., & Inamura, S. (1996). One-minute wave in body fluid volume change enhanced by postural sway during upright standing. *Journal of Applied Physiology*. https://doi.org/10.1152/jappl.1996.81.1.459

Jacklitsch, B., Williams, J., Musolin, K., Coca, A., Kim, J.-H., & Turner, N. (2016). *NIOSH Criteria for a Recommended Standard: Occupational Exposure to Heat and Hot Environments*.

Jéquier, E., & Constant, F. (2010). Water as an essential nutrient: The physiological basis of hydration. *European Journal of Clinical Nutrition*, *64*(2), 115–123. https://doi.org/10.1038/ejcn.2009.111

Joachim, H., & Weyer, H. (1975). UEBER DIE WIRKUNG DES ALKOHOLS UND DER ERMUEDUNG AUF DAS VERHALTEN EINIGER MENSCHLICHER EIGENREFLEXE. *UEBER DIE WIRKUNG DES ALKOHOLS UND DER ERMUEDUNG AUF DAS VERHALTEN EINIGER MENSCHLICHER EIGENREFLEXE.*, *76*, 187–210.

Johnson, A. G., Crawford, G. A., Kelly, D., Nguyen, T. V., & Gyory, A. Z. (1994). Arginine Vasopressin and Osmolality in the Elderly. *Journal of the American Geriatrics Society*, *42*(4), 399–404. https://doi.org/10.1111/j.1532-5415.1994.tb07488.x

Johnson, J. M. (2010). Exercise in a hot environment: The skin circulation. *Scandinavian Journal of Medicine & Science in Sports*, *20*(s3), 29–39. https://doi.org/10.1111/j.1600-0838.2010.01206.x

Johnston, C. E., Bristow, G. K., Elias, D. A., & Giesbrecht, G. G. (1996). Alcohol lowers the vasoconstriction threshold in humans without affecting core cooling rate during mild cold exposure. *European Journal of Applied Physiology and Occupational Physiology*, *74*(3), 293–295. https://doi.org/10.1007/BF00377453

Kenefick, R. W., & Cheuvront, S. N. (2012). Hydration for recreational sport and physical activity. *Nutrition Reviews*, *70 Suppl 2*, S137-142. https://doi.org/10.1111/j.1753-4887.2012.00523.x

Kenney, W. L., Kamon, E., & Buskirk, E. R. (1984). Effect of mild essential hypertension on control of forearm blood flow during exercise in the heat. *Journal of Applied Physiology*, *56*(4), 930–935. https://doi.org/10.1152/jappl.1984.56.4.930

Kenney, W. L., & Munce, T. A. (2003). Invited Review: Aging and human temperature regulation. *Journal of Applied Physiology*, *95*(6), 2598–2603. https://doi.org/10.1152/japplphysiol.00202.2003

Kenney, W. L., Tankersley, C. G., Newswanger, D. L., Hyde, D. E., Puhl, S. M., & Turner, N. L. (1990). Age and hypohydration independently influence the peripheral vascular response to heat stress. *Journal of Applied Physiology*, *68*(5), 1902–1908. https://doi.org/10.1152/jappl.1990.68.5.1902

Kenny, G. P., Sigal, R. J., & McGinn, R. (2016). Body temperature regulation in diabetes. *Temperature*, *3*(1), 119–145. https://doi.org/10.1080/23328940.2015.1131506

Kenny, G. P., Yardley, J., Brown, C., Sigal, R. J., & Jay, O. (2010). Heat stress in older individuals and patients with common chronic diseases. *CMAJ*, *182*(10), 1053–1060. https://doi.org/10.1503/cmaj.081050

Khare, S., Hajat, S., Kovats, S., Lefevre, C. E., De Bruin, W. B., Dessai, S., & Bone, A. (2015). Heat protection behaviour in the UK: Results of an online survey after the 2013 heatwave. *BMC Public Health*, *15*(1), 878. https://doi.org/10.1186/s12889-015-2181-8

Kollegger, H., Baumgartner, C., Wöber, C., Oder, W., & Deecke, L. (2008). Spontaneous Body Sway as a Function of Sex, Age, and Vision: Posturographic Study in 30 Healthy Adults. *European Neurology*, *32*(5), 253–259. https://doi.org/10.1159/000116836

Kubo, T., Sakata, Y., Matsunaga, T., Koshimune, A., Sakai, S., Ameno, K., & Ijiri, I. (1989). Analysis of Body Sway Pattern after Alcohol Ingestion in Human Subjects. *Acta Oto-Laryngologica*, *108*(sup468), 247–252. https://doi.org/10.3109/00016488909139056

Kupari, M. (1983). Acute cardiovascular effects of ethanol A controlled non-invasive study. *Heart*, *49*(2), 174–182. https://doi.org/10.1136/hrt.49.2.174

Lachenmeier, D. W. (2008). Safety evaluation of topical applications of ethanol on the skin and inside the oral cavity. *Journal of Occupational Medicine and Toxicology*, *3*(1), 26. https://doi.org/10.1186/1745-6673-3-26

Larose, J., Boulay, P., Sigal, R. J., Wright, H. E., & Kenny, G. P. (2013). Age-Related Decrements in Heat Dissipation during Physical Activity Occur as Early as the Age of 40. *PLOS ONE*, *8*(12), e83148. https://doi.org/10.1371/journal.pone.0083148

Lathers, C. M., & Smith, C. M. (1976). Ethanol effects on muscle spindle afferent activity and spinal reflexes. *Journal of Pharmacology and Experimental Therapeutics*, *197*(1), 126–134.

Lefebvre, K., Brough, A., Chaseling, G., Morris, N., & Ravanelli, N. (2025). The Sex-Dependent Effect of Alcohol Consumption on Physiological Responses and Perception to Acute Heat Stress. *Physiology*, *40*(S1), 1641. https://doi.org/10.1152/physiol.2025.40.S1.1641

Lim, C. L., Byrne, C., & Lee, J. K. (2008). Human Thermoregulation and Measurement of Body Temperature in Exercise and Clinical Settings. *Annals of the Academy of Medicine, Singapore*, *37*(4), 347–353. https://doi.org/10.47102/annals-acadmedsg.V37N4p347

Linkola, J., Ylikahri, R., Fyhrquist, F., & Wallenius, M. (1978). Plasma vasopressin in ethanol intoxication and hangover. *Acta Physiologica Scandinavica*, *104*(2), 180–187. https://doi.org/10.1111/j.1748-1716.1978.tb06265.x

Liu, Y.-C., & and Fu, S.-M. (2007). Changes in Driving Behavior and Cognitive Performance with Different Breath Alcohol Concentration Levels. *Traffic Injury Prevention*, *8*(2), 153–161. https://doi.org/10.1080/15389580601161623

Livingstone, S. D., Kuehn, L. A., Limmer, R. E., & Weatherson, B. (1980). The effect of alcohol on body heat loss. *Aviation, Space, and Environmental Medicine*, *51*(9 Pt 2), 961–964.

Lopez, I., Honrubia, V., & Baloh, R. W. (1997). Aging and the Human Vestibular Nucleus. *Journal of Vestibular Research*, *7*(1), 77–85. https://doi.org/10.3233/VES-1997-7107

Lord, S. R., Clark, R. D., & Webster, I. W. (1991). Postural stability and associated physiological factors in a population of aged persons. *Journal of Gerontology*, *46*(3), M69-76. https://doi.org/10.1093/geronj/46.3.m69

Lucas, R. a. I., Cotter, J. D., Morrison, S., & Ainslie, P. N. (2008). The effects of ageing and passive heating on cardiorespiratory and cerebrovascular responses to orthostatic stress in humans. *Experimental Physiology*, *93*(10), 1104–1117. https://doi.org/10.1113/expphysiol.2008.042580

Lukas, S. E., Lex, B. W., Slater, J. P., Greenwald, N. E., & Mendelson, J. H. (1989). A microanalysis of ethanol-induced disruption of body sway and psychomotor performance in women. *Psychopharmacology*, *98*(2), 169–175. https://doi.org/10.1007/BF00444687

Luukinen, H., Koski, K., Kivela, S.-L., & Laippala, P. (1996). Social status, life changes, housing conditions, health, functional abilitiesand life-style as risk factors for recurrent falls among the home-dwelling elderly. *Public Health*, *110*(2), 115–118. https://doi.org/10.1016/S0033-3506(96)80057-6

Mack, G. W., & Nadel, E. R. (1996). Body fluid balance during heat stress in humans. In *Fregly MJ, Blatteis CM, eds. Environmental physiology, vol. 1*. Published for the American Physiological Society by Oxford University Press.

Mano, T., & Iwase, S. (2003). Sympathetic nerve activity in hypotension and orthostatic intolerance. *Acta Physiologica Scandinavica*, *177*(3), 359–365. https://doi.org/10.1046/j.1365-201X.2003.01081.x

Maufrais, C., Charriere, N., & Montani, J.-P. (2017). Cardiovascular and Cutaneous Responses to the Combination of Alcohol and Soft Drinks: The Way to Orthostatic Intolerance? *Frontiers in Physiology*, *8*, 860. https://doi.org/10.3389/fphys.2017.00860

McMichael, A. J., Woodruff, R. E., & Hales, S. (2006). Climate change and human health: Present and future risks. *The Lancet*, *367*(9513), 859–869. https://doi.org/10.1016/S0140-6736(06)68079-3

Mekjavic, I. B., Gaul, C. A., White, M. D., & Mittleman, K. D. (1987). Cardiovascular responses during 70 degrees head-up tilt: The effect of elevated body temperature and high alcohol blood levels. *The Physiologist*, *30*(1 Suppl), S56-57.

Miescher, E., & Fortney, S. M. (1989). Responses to dehydration and rehydration during heat exposure in young and older men. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*. https://doi.org/10.1152/ajpregu.1989.257.5.R1050

Millyard, A., Layden, J. D., Pyne, D. B., Edwards, A. M., & Bloxham, S. R. (2020). Impairments to Thermoregulation in the Elderly During Heat Exposure Events. *Gerontology and Geriatric Medicine*, *6*, 233372142093243. https://doi.org/10.1177/2333721420932432

Minson, C. T., Wladkowski, S. L., Cardell, A. F., Pawelczyk, J. A., & Kenney, W. L. (1998). Age alters the cardiovascular response to direct passive heating. *Journal of Applied Physiology*, *84*(4), 1323–1332. https://doi.org/10.1152/jappl.1998.84.4.1323

Mishra, L., Sharma, S., Potter, J. J., & Mezey, E. (1989). More Rapid Elimination of Alcohol in Women as Compared to Their Male Siblings. *Alcoholism: Clinical and Experimental Research*, *13*(6), 752–754. https://doi.org/10.1111/j.1530-0277.1989.tb00415.x

Missing data | SPSS Learning Modules. (n.d.). Retrieved July 10, 2025, from https://stats.oarc.ucla.edu/spss/modules/missing-data/#:~:text=As%20a%20general%20rule%2C%20SPSS,not%20addressing%20commands%20like%20sort.)

Monahan, K. D. (2007). Effect of aging on baroreflex function in humans. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *293*(1), R3–R12. https://doi.org/10.1152/ajpregu.00031.2007

Montagna, W., & Carlisle, K. (1979). Structural Changes in Aging Human Skin. *Journal of Investigative Dermatology*, *73*(1), 47–53. https://doi.org/10.1111/1523-1747.ep12532761

Moran, D. S., & Mendal, L. (2002). Core Temperature Measurement. *Sports Medicine*, *32*(14), 879–885. https://doi.org/10.2165/00007256-200232140-00001

Morris, N. B., Jay, O., Flouris, A. D., Casanueva, A., Gao, C., Foster, J., Havenith, G., & Nybo, L. (2020). Sustainable solutions to mitigate occupational heat strain – an umbrella review of physiological effects and global health perspectives. *Environmental Health*, *19*(1), 95. https://doi.org/10.1186/s12940-020-00641-7

Morris, N. B., Ravanelli, N., & Chaseling, G. K. (2024). The effect of alcohol consumption on human physiological and perceptual responses to heat stress: A systematic scoping review. *Environmental Health*, *23*(1), 73. https://doi.org/10.1186/s12940-024-01113-y

Moskowitz, H., 1925-, & Florentino, D. (2000). *A Review of the Literature on the Effects of Low Doses of Alcohol on Driving-Related Skills* (DOT-HS-809-028). https://doi.org/10.21949/1525468

Mosqueda-Garcia, R., Furlan, R., MD, J. T., & Fernandez-Violante, R. (2000). The Elusive Pathophysiology of Neurally Mediated Syncope. *Circulation*, *102*(23), 2898–2906. https://doi.org/10.1161/01.CIR.102.23.2898

Mota-Rojas, D., Titto, C. G., Orihuela, A., Martínez-Burnes, J., Gómez-Prado, J., Torres-Bernal, F., Flores-Padilla, K., Carvajal-de La Fuente, V., & Wang, D. (2021). Physiological and Behavioral Mechanisms of Thermoregulation in Mammals. *Animals*, *11*(6), 1733. https://doi.org/10.3390/ani11061733

Mukai, S., & Lipsitz, L. A. (2002). Orthostatic hypotension. *Clinics in Geriatric Medicine*, *18*(2), 253–268. https://doi.org/10.1016/S0749-0690(02)00008-3

Murata, J., Murata, S., Horie, J., Ohtao, H., & Miyazaki, J. (2012). Relationship Between Orthostatic Blood Pressure Changes and Postural Sway When Standing up from a Chair in Older Adult Females. *International Journal of Gerontology*, *6*(3), 182–186. https://doi.org/10.1016/j.ijge.2012.01.011

Murray, M. M. (1932). The diuretic action of alcohol and its relation to pituitrin. *The Journal of Physiology*, *76*(3), 379–386.

Myers, R. D. (1981). Alcohol's effect on body temperature: Hypothermia, hyperthermia or poikilothermia? *Brain Research Bulletin*, *7*(2), 209–220. https://doi.org/10.1016/0361-9230(81)90085-X

Myran, D. T., Smith, B. T., Cantor, N., Li, L., Saha, S., Paradis, C., Jesseman, R., Tanuseputro, P., & Hobin, E. (2021). Changes in the dollar value of per capita alcohol, essential, and non-essential retail sales in Canada during COVID-19. *BMC Public Health*, *21*(1), 2162. https://doi.org/10.1186/s12889-021-12226-1

Narkiewicz, K., Cooley, R. L., & Somers, V. K. (2000). Alcohol Potentiates Orthostatic Hypotension: Implications for Alcohol-Related Syncope. *Circulation*, *101*(4), 398–402. https://doi.org/10.1161/01.CIR.101.4.398

Newlin, D. B., Byrne, E. A., & Porges, S. W. (1990). Vagal Mediation of the Effect of Alcohol on Heart Rate. *Alcoholism: Clinical and Experimental Research*, *14*(3), 421–424. https://doi.org/10.1111/j.1530-0277.1990.tb00498.x

Nieschalk, M., Ortmann, C., West, A., Schmäl, F., Stoll, W., & Fechner, G. (1999). Effects of alcohol on body-sway patterns in human subjects. *International Journal of Legal Medicine*, *112*(4), 253–260. https://doi.org/10.1007/s004140050245

Notley, S. R., Mitchell, D., & Taylor, N. A. S. (2023). A century of exercise physiology: Concepts that ignited the study of human thermoregulation. Part 1: Foundational principles and theories of regulation. *European Journal of Applied Physiology*, *123*(11), 2379–2459. https://doi.org/10.1007/s00421-023-05272-7

Phillips, P. A., Rolls, B. J., Ledingham, J. G. G., Forsling, M. L., Morton, J. J., Crowe, M. J., & Wollner, L. (1984). Reduced Thirst after Water Deprivation in Healthy Elderly Men. *New*

England Journal of Medicine, *311*(12), 753–759. https://doi.org/10.1056/NEJM198409203111202

Polhuis, K. C. M. M., Wijnen, A. H. C., Sierksma, A., Calame, W., & Tieland, M. (2017). The Diuretic Action of Weak and Strong Alcoholic Beverages in Elderly Men: A Randomized Diet-Controlled Crossover Trial. *Nutrients*, *9*(7), Article 7. https://doi.org/10.3390/nu9070660

Proano, E., & Perbeck, L. (1994). Effect of exposure to heat and intake of ethanol on the skin circulation and temperature in ischaemic limbs. *Clinical Physiology*, *14*(3), 305–310. https://doi.org/10.1111/j.1475-097X.1994.tb00387.x

Ramanathan, N. L. (1964). A new weighting system for mean surface temperature of the human body. *Journal of Applied Physiology*, *19*(3), 531–533. https://doi.org/10.1152/jappl.1964.19.3.531

Ravanelli, N., Bongers, C. C. W. G., & Jay, O. (2019). The Biophysics of Human Heat Exchange. In *Heat Stress in Sport and Exercise* (pp. 29–43). Springer, Cham. https://doi.org/10.1007/978-3-319-93515-7_2

Ravanelli, N., Morris, N., & Morrison, S. A. (2023). 24-h movement behaviour, thermal perception, thirst, and heat management strategies of children and adults during heat alerts: A pilot study. *Frontiers in Physiology*, *14*. https://doi.org/10.3389/fphys.2023.1179844

Resnick, B., & Junlapeeya, P. (2004). Falls in a community of older adults: Findings and implications for practice. *Applied Nursing Research*, *17*(2), 81–91. https://doi.org/10.1016/j.apnr.2004.03.004

Ricci, F., De Caterina, R., & Fedorowski, A. (2015). Orthostatic Hypotension. *Journal of the American College of Cardiology*, *66*(7), 848–860. https://doi.org/10.1016/j.jacc.2015.06.1084

Robinson, S. B., & Rosher, R. B. (2002). Can a beverage cart help improve hydration? *Geriatric Nursing*, *23*(4), 208–211. https://doi.org/10.1067/mgn.2002.126967

Rodrigues, G. D., Gurgel, J. L., dos Santos Galdino, I., da Nóbrega, A. C. L., & da Silva Soares, P. P. (2020). Respiratory pump contributions in cerebrovascular and postural control responses during orthostatic stress in older women. *Respiratory Physiology & Neurobiology*, *275*, 103384. https://doi.org/10.1016/j.resp.2020.103384

Rowell, L. B. (1993). *Human Cardiovascular Control*. Oxford University Press.

Rowell, L. B., Brengelmann, G. L., & Murray, J. A. (1969). Cardiovascular responses to sustained high skin temperature in resting man. *Journal of Applied Physiology*, *27*(5), 673–680. https://doi.org/10.1152/jappl.1969.27.5.673

Rupp, H., Brilla, C. G., & Maisch, B. (1996). Hypertension and alcohol: Central and peripheral mechanisms. *Herz*, *21*(4), 258–264.

Sagawa, S., Shiraki, K., Yousef, M. K., & Miki, K. (1988). Sweating and Cardiovascular Responses of Aged Men to Heat Exposure. *Journal of Gerontology*, *43*(1), M1–M8. https://doi.org/10.1093/geronj/43.1.M1

Saini, J., Boisvert, P., Spiegel, K., Candas, V., & Brandenberger, G. (1995). Influence of alcohol on the hydromineral hormone responses to exercise in a warm environment. *European Journal of Applied Physiology and Occupational Physiology*, *72*(1), 32–36. https://doi.org/10.1007/BF00964111

Sawka, M. N., Montain, S. J., & Latzka, W. A. (2001). Hydration effects on thermoregulation and performance in the heat. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, *128*(4), 679–690. https://doi.org/10.1016/S1095-6433(01)00274-4

Schlader, Z. J., & Vargas, N. T. (2019). Regulation of Body Temperature by Autonomic and Behavioral Thermoeffectors. *Exercise and Sport Sciences Reviews*, *47*(2), 116–126. https://doi.org/10.1249/JES.0000000000000180

Schlader, Z. J., Wilson, T. E., & Crandall, C. G. (2016). Mechanisms of orthostatic intolerance during heat stress. *Autonomic Neuroscience*, *196*, 37–46. https://doi.org/10.1016/j.autneu.2015.12.005

Shapiro, Y., & Seidman, D. S. (1990). Field and clinical observations of exertional heat stroke patients. In *Medicine & Science in Sports & Exercise* (Vol. 22, pp. 6–14). The American College of Sports Medicine. https://oce-ovid-com.ezproxy.lakeheadu.ca/article/00005768-199002000-00003/HTML

Shaw, B. H., & Claydon, V. E. (2014). The relationship between orthostatic hypotension and falling in older adults. *Clinical Autonomic Research*, *24*(1), 3–13. https://doi.org/10.1007/s10286-013-0219-5

Sheridan, S. C. (2007). A survey of public perception and response to heat warnings across four North American cities: An evaluation of municipal effectiveness. *International Journal of Biometeorology*, *52*(1), 3–15. https://doi.org/10.1007/s00484-006-0052-9

Shibao, C., Grijalva, C. G., Raj, S. R., Biaggioni, I., & Griffin, M. R. (2007). Orthostatic Hypotension-Related Hospitalizations in the United States. *The American Journal of Medicine*, *120*(11), 975–980. https://doi.org/10.1016/j.amjmed.2007.05.009

Shibuya, A., Yasunami, M., & Yoshida, A. (1989). Genotypes of alcohol dehydrogenase and aldehyde dehydrogenase loci in Japanese alcohol flushers and nonflushers. *Human Genetics*, *82*(1), 14–16. https://doi.org/10.1007/BF00288263

Shirreffs, S. M., & Maughan, R. J. (1997). Restoration of fluid balance after exercise-induced dehydration: Effects of alcohol consumption. *Journal of Applied Physiology*, *83*(4), 1152–1158. https://doi.org/10.1152/jappl.1997.83.4.1152

Singh, A., Datta, R., & Singh, R. (2021). The Use of a Low-Cost Gaming Platform as a Substitute to Medical-Grade Force Plate in Objective Evaluation of Balance. *Indian Journal of Otolaryngology and Head & Neck Surgery*, *74*(2), 217. https://doi.org/10.1007/s12070-021-02963-5

Sklar, A. L., Gilbertson, R., Boissoneault, J., Prather, R., & Nixon, S. J. (2012). Differential Effects of Moderate Alcohol Consumption on Performance Among Older and Younger Adults. *Alcoholism: Clinical and Experimental Research*, *36*(12), 2150–2156. https://doi.org/10.1111/j.1530-0277.2012.01833.x

Smith, C. J., & Johnson, J. M. (2016). Responses to hyperthermia. Optimizing heat dissipation by convection and evaporation: Neural control of skin blood flow and sweating in humans. *Autonomic Neuroscience*, *196*, 25–36. https://doi.org/10.1016/j.autneu.2016.01.002

Smith, G. D., Shaw, L. J., Maini, P. K., WARD, R. J., Peters, T. J., & Murray, J. D. (1993). Mathermatical Modelling of Ethanol Metabolism in Normal Subjects and Chronic Alcohol Misusers. *Alcohol and Alcoholism*, *28*(1), 25–32. https://doi.org/10.1093/oxfordjournals.alcalc.a045345

Smith, K. E., & Fenske, N. A. (2000). Cutaneous manifestations of alcohol abuse. *Journal of the American Academy of Dermatology*, *43*(1, Part 1), 1–18. https://doi.org/10.1067/mjd.2000.104512

Sohal, A., Bains, K., Dhaliwal, A., Chaudhry, H., Sharma, R., Singla, P., Gupta, G., Dukovic, D., Sandhu, S., Roytman, M., & Tringali, S. (2022). Seasonal Variations of Hospital Admissions for Alcohol-Related Hepatitis in the United States. *Gastroenterology Research*, *15*(2), 75–81. https://doi.org/10.14740/gr1506

Spaak, J., Merlocco, A. C., Soleas, G. J., Tomlinson, G., Morris, B. L., Picton, P., Notarius, C. F., Chan, C. T., & Floras, J. S. (2008). Dose-related effects of red wine and alcohol on hemodynamics, sympathetic nerve activity, and arterial diameter. *American Journal of Physiology. Heart and Circulatory Physiology*, *294*(2), H605-612. https://doi.org/10.1152/ajpheart.01162.2007

Spaak, J., Tomlinson, G., McGowan, C. L., Soleas, G. J., Morris, B. L., Picton, P., Notarius, C. F., & Floras, J. S. (2010). Dose-related effects of red wine and alcohol on heart rate variability. *American Journal of Physiology-Heart and Circulatory Physiology*, *298*(6), H2226–H2231. https://doi.org/10.1152/ajpheart.00700.2009

Spector, N. H. (1971). Alcohol Breath Tests: Gross Errors in Current Methods of Measuring Alveolar Gas Concentrations. *Science*, *172*(3978), 57–59.

Stanhewicz, A. E., Greaney, J. L., Alexander, L. M., & Kenney, W. L. (2016). Blunted increases in skin sympathetic nerve activity are related to attenuated reflex vasodilation in aged human skin. *Journal of Applied Physiology*, *121*(6), 1354–1362. https://doi.org/10.1152/japplphysiol.00730.2016

Starkey, N. J., & Charlton, S. G. (2014). The effects of moderate alcohol concentrations on driving and cognitive performance during ascending and descending blood alcohol concentrations. *Human Psychopharmacology: Clinical and Experimental*, *29*(4), 370–383. https://doi.org/10.1002/hup.2415

Starling, E. H. (1918). *The Linacre Lecture on the Law of the Heart: Given at Cambridge, 1915. With Diagrams*. London.

Stookey, J. D. (1999). The Diuretic Effects of Alcohol and Caffeine and Total Water Intake Misclassification. *European Journal of Epidemiology*, *15*, 181–188.

Strauss, M. B., Rosenbaum, J. D., & Nelson, W. P. (1950). THE EFFECT OF ALCOHOL ON THE RENAL EXCRETION OF WATER AND ELECTROLYTE 1. *Journal of Clinical Investigation*, *29*(8), 1053–1058. https://doi.org/10.1172/JCI102336

Sugarman, D., & Greenfield, S. (2021, September 24). Rising alcohol use among older adults. *Harvard Health*. https://www.health.harvard.edu/blog/rising-alcohol-use-among-older-adult-202109242599

Sutker, P. B., Goist Jr, K. C., Allain, A. N., & Bugg, F. (1987). Acute Alcohol Intoxication: Sex Comparisons on Pharmacokinetic and Mood Measures. *Alcoholism: Clinical and Experimental Research*, *11*(6), 507–512. https://doi.org/10.1111/j.1530-0277.1987.tb00162.x

Taivainen, H., Laitinen, K., Tähtelä, R., Kiianmaa, K., & Välimäki, M. J. (1995). Role of Plasma Vasopressin in Changes of Water Balance Accompanying Acute Alcohol Intoxication. *Alcoholism: Clinical and Experimental Research*, *19*(3), 759–762. https://doi.org/10.1111/j.1530-0277.1995.tb01579.x

van de Borne, P., Montano, N., Pagani, M., Oren, R., & Somers, V. K. (1997). Absence of Low-Frequency Variability of Sympathetic Nerve Activity in Severe Heart Failure. *Circulation*, *95*(6), 1449–1454. https://doi.org/10.1161/01.CIR.95.6.1449

Van Dyke, N. A., & Fillmore, M. T. (2017). Laboratory analysis of risky driving at 0.05% and 0.08% blood alcohol concentration. *Drug and Alcohol Dependence*, *175*, 127–132. https://doi.org/10.1016/j.drugalcdep.2017.02.005

Verma, A. K., Garg, A., Xu, D., Bruner, M., Fazel-Rezai, R., Blaber, A. P., & Tavakolian, K. (2017). Skeletal Muscle Pump Drives Control of Cardiovascular and Postural Systems. *Scientific Reports*, *7*(1), 45301. https://doi.org/10.1038/srep45301

Veronese, N., De Rui, M., Bolzetta, F., Zambon, S., Corti, M. C., Baggio, G., Toffanello, E. D., Maggi, S., Crepaldi, G., Perissinotto, E., Manzato, E., & Sergi, G. (2015). Orthostatic Changes in Blood Pressure and Mortality in the Elderly: The Pro.V.A Study. *American Journal of Hypertension*, *28*(10), 1248–1256. https://doi.org/10.1093/ajh/hpv022

Vestal, R. E., McGuire, E. A., Tobin, J. D., Andres, R., Norris, A. H., & Mezey, E. (1977). Aging and ethanol metabolism. *Clinical Pharmacology & Therapeutics*, *21*(3), 343–354. https://doi.org/10.1002/cpt1977213343

Waldock, K. A. M., Hayes, M., Watt, P. W., & Maxwell, N. S. (2018). Physiological and perceptual responses in the elderly to simulated daily living activities in UK summer climatic conditions. *Public Health*, *161*, 163–170. https://doi.org/10.1016/j.puhe.2018.04.012

Wang, M. Q., Nicholson, M. E., Richardson, M. T., Fitzhugh, E. C., Reneau, P., & Westerfield, C. R. (1995). The acute effect of moderate alcohol consumption on cardiovascular

responses in women. *Journal of Studies on Alcohol*, *56*(1), 16–20. https://doi.org/10.15288/jsa.1995.56.16

Wang, T. A., Teo, C. F., Åkerblom, M., Chen, C., Fontaine, M. T.-L., Greiner, V. J., Diaz, A., McManus, M. T., Jan, Y. N., & Jan, L. Y. (2019). Thermoregulation via Temperature-Dependent PGD2 Production in Mouse Preoptic Area. *Neuron*, *103*(2), 309-322.e7. https://doi.org/10.1016/j.neuron.2019.04.035

Weiss, B., & Laties, V. G. (1961). Behavioral thermoregulation. *Science (New York, N.Y.)*, *133*(3461), 1338–1344. https://doi.org/10.1126/science.133.3461.1338

Werner, J. (1980). The concept of regulation for human body temperature. *Journal of Thermal Biology*, *5*(2), 75–82. https://doi.org/10.1016/0306-4565(80)90003-0

Wiberg, G. S., Samson, J. M., Maxwell, W. B., Coldwell, B. B., & Trenholm, H. L. (1971). Further studies on the acute toxicity of ethanol in young and old rats: Relative importance of pulmonary excretion and total body water. *Toxicology and Applied Pharmacology*, *20*(1), 22–29. https://doi.org/10.1016/0041-008X(71)90085-8

Wiberg, G. S., Trenholm, H. L., & Coldwell, B. B. (1970). Increased ethanol toxicity in old rats: Changes in LD50, *in vivo* and *in vitro* metabolism, and liver alcohol dehydrogenase activity. *Toxicology and Applied Pharmacology*, *16*(3), 718–727. https://doi.org/10.1016/0041-008X(70)90077-3

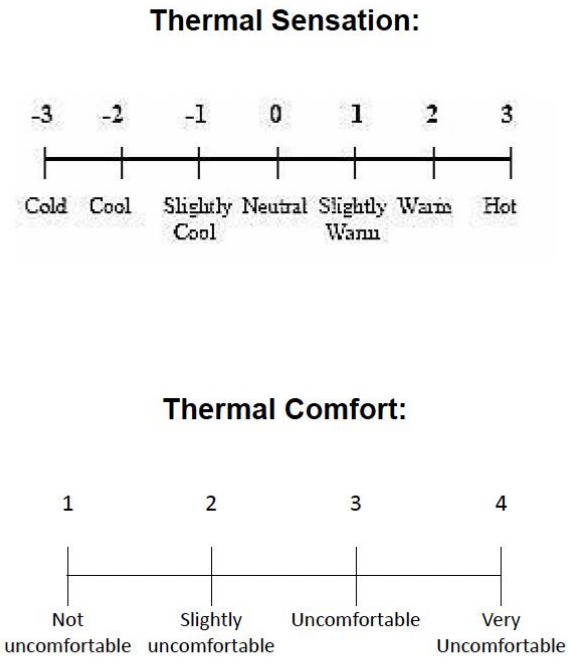
Wolff, P. H. (1972). Ethnic Differences in Alcohol Sensitivity. *Science*, *175*(4020), 449–450. https://doi.org/10.1126/science.175.4020.449

Woollacott, M. H. (1983). Effects of ethanol on postural adjustments in humans. *Experimental Neurology*, *80*(1), 55–68. https://doi.org/10.1016/0014-4886(83)90006-7

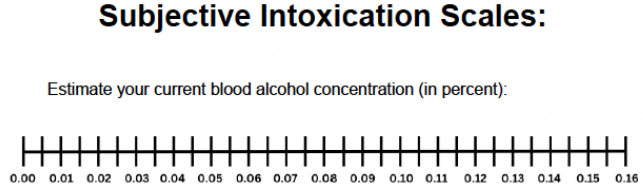
Xiao, H., Barber, J., & Campbell, E. S. (2004). Economic burden of dehydration among hospitalized elderly patients. *American Journal of Health-System Pharmacy*, *61*(23), 2534–2540. https://doi.org/10.1093/ajhp/61.23.2534

Yoda, T., Crawshaw, L. I., Nakamura, M., Saito, K., Konishi, A., Nagashima, K., Uchida, S., & Kanosue, K. (2005). Effects of alcohol on thermoregulation during mild heat exposure in humans. *Alcohol*, *36*(3), 195–200. https://doi.org/10.1016/j.alcohol.2005.09.002

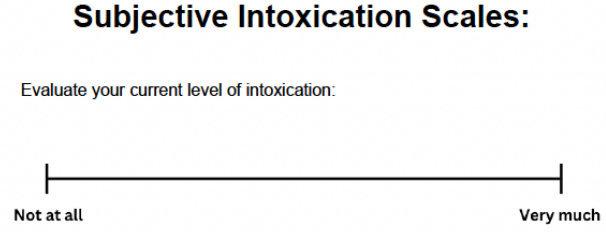
Appendix A



Note. Subjective thermal sensation and comfort scales.



Note. Subjective blood alcohol concentration scale.



Note. Subjective intoxication level scale.

Appendix B

THE NATIONAL HIGHWAY TRAFFIC SAFETY ADMINISTRATION
STANDARDIZED FIELD SOBRIETY
TESTING PROCEDURES

HORIZONTAL GAZE NYSTAGMUS INSTRUCTIONS

1. Please remove your glasses (if worn).
2. Put your feet together, hands at your side. Keep your head still and look at and follow this stimulus with your eyes only.
3. Keep looking at the stimulus until told the test is over.
4. Do not move your head.
5. Do you understand the directions?

WALK AND TURN INSTRUCTIONS

1. Put your left foot on the line, then place your right foot on the line ahead of your left, with the heel of your right foot against the toe of your left foot.
2. Do not start until I tell you to do so.
3. Do you understand? (must receive affirmative response)
4. When I tell you to begin, take 9 heel-to-toe steps on the line (demonstrate) and take 9 heel-to-toe steps back down the line.
5. When you turn on the ninth step, keep your front foot on the line and turn taking several small steps with the other foot (demonstrate) and take 9 heel-to-toe steps back down the line.
6. Ensure you look at your feet, count each step out loud, keep your arms at your side, ensure you touch heel-to-toe and do not stop until you have completed the test.
7. Do you understand the instructions?
8. You may begin.
9. If the suspect does not understand some part of the instructions, only the part in which the suspect does not understand should be repeated.

ONE-LEG-STAND INSTRUCTIONS

1. Stand with your feet together and your arms at your side (demonstrate)
2. Maintain position until told otherwise.
3. When I tell you to, I want you to raise one leg, either one, approximately 6 inches off the ground, foot pointed out, both legs straight and look at the elevated foot. Count out loud in the following manner: 1001, 1002, 1003, 1004 and so on until told to stop
4. Do you understand the instructions?
5. You may begin the test.

SUBJECT NAME _____

INCIDENT # _____

HORIZONTAL GAZE NYSTAMUS TEST

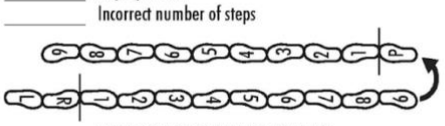
* Check for equal tracking, pupil size and resting nystagmus

_____ Lack of smooth pursuit: Left eye
_____ Lack of smooth pursuit: Right eye
_____ Distinct and sustained nystagmus at maximum deviation: Left eye
_____ Distinct and sustained nystagmus at maximum deviation: Right eye
_____ Onset of nystagmus prior to 45degrees: Left eye
_____ Onset of nystagmus prior to 45 degrees: Right eye
_____ Check for vertical gaze nystagmus

_____ **TOTAL CLUES OBSERVED (6 MAX)**

WALK AND TURN TEST

_____ Can't keep balance during instructions
_____ Starts too soon
_____ Stops walking
_____ Misses heel-to-toe
_____ Steps off line
_____ Uses arms for balance
_____ Improper turn
_____ Incorrect number of steps



_____ **TOTAL CLUES OBSERVED (8 MAX)**

ONE-LEG STAND TEST

_____ Sways while balancing
_____ Uses arms to balance
_____ Hopping
_____ Puts foot down

_____ Foot Raised

_____ **TOTAL CLUES OBSERVED (4 MAX)**

DATE: _____

TIME: _____

Note. Standardized field sobriety tests.