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## Physiological strain of stock car drivers during competitive racing

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## ABSTRACT

Heat strain experienced by motorsport athletes competing in National Association for Stock Car Automobile Racing (NASCAR) may be significant enough to impair performance or even result in a life-threatening accident. There is a need to carefully quantify heat strain during actual NASCAR race competitions in order to faithfully represent the magnitude of the problem and conceptualize future mitigation practices. The purpose of this investigation was to quantify the thermoregulatory and physiological strain associated with competitive stock car driving. Eight male stock car drivers ( $29.0 \pm 10.0$  yr;  $176.2 \pm 3.3$  cm,  $80.6 \pm 15.7$  kg) participated in sanctioned stock car races. Physiological measurements included intestinal core ( $T_c$ ) and skin ( $T_{sk}$ ) temperatures, heart rate (HR), blood pressure, and body mass before and after completion of the race. Pre-race  $T_c$  was  $38.1 \pm 0.1$  °C which increased to  $38.6 \pm 0.2$  °C post-race ( $p=0.001$ ).  $T_{sk}$  increased from  $36.1 \pm 0.2$  °C pre-race to  $37.3 \pm 0.3$  °C post-race ( $p=0.001$ ) whereas the core-to-skin temperature gradient decreased from a pre-race value of  $2.0 \pm 0.3$  °C to  $1.3 \pm 0.3$  °C post-race ( $p=0.005$ ). HRs post-race were  $80 \pm 0.1\%$  of the drivers' age-predicted maximum HR. Physiological Strain Index (PSI) post-race was 4.9, which indicates moderate strain. Drivers' thermal sensation based on the ASHRAE Scale increased from  $1.3 \pm 0.5$  to  $2.8 \pm 0.4$ , and their perception of exertion (RPE) responses also increased from  $8.4 \pm 1.6$  to  $13.9 \pm 1.8$  after competition. Heat strain associated with competitive stock car racing is significant. These findings suggest the need for heat mitigation practices and provide evidence that motorsport should consider strategies to become heat acclimated to better meet the thermoregulatory and cardiovascular challenges of motorsport competition.

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## 1. Introduction

During National Association for Stock Car Automobile Racing (NASCAR) competition, motorsports athletes may be exposed to severe heat strain due to the summer months and southern climate of the competitive season. Motorsports athletes can drive at speeds of 322 km/h for 3–4 h of competition in a cockpit that can reach temperatures of  $\sim 50$  °C (Falkner, 1972; Walker et al., 2001a). The high temperature is due in part to the stock car design. The thin construction materials used to keep weight down and the aerodynamic design, force heat from the drive train (engine and transmission) into the car cockpit in order to reduce drag. Furthermore, motorsport athletes are required to wear fire protective clothing that adds insulation and impedes heat loss. It has

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been suggested that motorsport competition may increase heat stress, challenge the cardiovascular system, adding to driver fatigue, and possibly leading to catastrophic injury (Brearley and Finn, 2007; Carlson, 2013). In a recent review, Potkanowicz and Mendel (2013) stated that the goals of sports science research are to improve the fitness levels in all competitive athletes in order to minimize stress on the body during competition, but unfortunately, less is known regarding the stress motorsports drivers are faced with during competitive racing. To date, the thermoregulatory and cardiovascular stress and strain of actual stock car race competitions has not been carefully quantified.

Extreme heat has the potential to cause pronounced challenges to the cardiovascular system due to the increased demand for greater skin blood flow in order to dissipate heat. The increased heart rate (HR) and cardiac output associated with exercising in the heat is necessary to maintain blood pressure (Crandall, 2008) while perfusing active muscle, including the myocardium (Gonzalez-Alonso et al., 2008) and peripheral vessels. Jacobs et al. (2002) found that open-wheel (wheels are outside car's body in contrast to stock cars which have wheels under the

fenders) drivers during two non-competition, paced driving sessions on a road course, were reported to elicit oxygen uptake ( $\text{VO}_2$ ) and HR responses of approximately 79% and 82% respectively, of their maximal responses from treadmill testing. Additionally, drivers' rating of perceived exertion (RPE) responses ranged from 15 to 17 (hard to very hard) after the road course testing session (Jacobs et al., 2002). Jacobs et al. (2002) concluded that the physiological responses assessed during the driving sessions required similar energy expenditures and heart rates as observed in other competitive sports activities such as basketball and soccer. Brearley and Finn (2007) likewise reported that V8 Supercar drivers averaged HRs of 167 and 169 beats/min during both short and long road course races. Furthermore, amateur kart drivers exhibited heart rates of 150 beats/min during kart racing and significantly decreased blood pressure post-racing (Yamakoshi et al., 2010). Despite some cardiovascular monitoring during different types of motor sports, there is clear indication that this has not been documented in stock car racing, especially during actual competition. The constant pedal work required during a stock car race, in addition to the isometric muscular activation (Falkner, 1972) of the neck, trunk, abdomen, and legs to counter against the acute exposure to gravitational ( $G$ ) forces may not only contribute to an increase in metabolic heat (Brearley and Finn, 2007), but cardiovascular strain (Convertino, 2001). Allan and Crossley (1972) reported that elevated cockpit temperatures in military aircraft, which induced modest pilot core and skin temperatures, reduced grayout threshold (Grayout threshold is defined as the level of  $+G_z$  acceleration when maintained for 15 s, caused the subject to lose peripheral vision for at least 5 s without losing central vision) by  $\sim 1G$ . Stock cars, depending on the track, can reach between 3 and 4Gs for several hours. The cardiovascular strain associated with driving in hot conditions (Walker et al., 2001a) combined with exposure to  $G$  accelerations for several hours, may adversely affect driver ability. A consequence of this combination may possibly result in a lower blackout tolerance (Allan and Crossley, 1972) and greater likelihood of making a mistake on the track resulting in a poor performance, or even a life-threatening accident.

Thermoregulatory stress for motorsport athletes is likely increased due to the fact that: (1) drivers are subjected to a hot environment within the vehicle; (2) the moderate to high intensity work ( $\sim 45$ – $80\%$  of  $\text{VO}_{2\text{max}}$  during open-wheel racing on road and oval courses (Jacobs et al., 2002)) generates metabolic heat and; and (3) evaporative cooling is inhibited by heavy, protective clothing composed of a Nomex<sup>®</sup> fire-retardant suit, gloves, underwear, socks, boots, and a full helmet that drivers are required to wear. The increased metabolic expenditures of drivers coupled with prolonged heat exposure will ultimately result in elevated body temperatures and substantial sweat losses. In fact, Jareno et al. (1987) reported that during the *Grand Premio de España* at the Jerez Speedway in Spain where temperatures in the pitboxes averaged  $31^\circ\text{C}$  and the relative humidity was 53%, two drivers were clinically diagnosed with heat stroke, despite electric cooling devices in their helmets (Jareno et al., 1987). In addition, V8 Supercar drivers competing in hot conditions experienced elevations in core temperatures to approximately  $39^\circ\text{C}$ , despite the utilization of torso cooling shirts (Brearley and Finn, 2007), and kart racing drivers (Yamakoshi et al., 2010) have also experienced increases in core temperatures of  $0.5^\circ\text{C}$  at the end of a driving session. Thermoregulatory responses to these different modes of motorsports driving in different scenarios have been documented, yet none have quantified the core and skin temperatures of drivers in stock cars during a sanctioned competition on an oval track, and without the use of any cooling devices, which are not typically used during this type of competition. Given all of these factors, it is reasonable to assume that motorsport drivers undergo significant

thermoregulatory stress; however, this has not been quantified in competitive stock car racing. Moreover, the heat strain during summer months adversely affects performance. Anecdotally it has been noted that during June to September with the accompanying elevated ambient temperatures, there are increased wrecks at the end of the race (Fielden, 2007). Furthermore personal communications with infield car center staff state that during the summer months, there is increased number of drivers requesting intravenous fluids. Thus it stands to reason that during stock car racing there is increased thermal load that leads to performance impairment and possible increased risk of accidents. Accordingly, the purpose of this investigation was to quantify the thermoregulatory and physiological strain associated with stock car automobile driving during competition. There is a need to carefully quantify heat strain during actual NASCAR race competitions in order to accurately quantify the problem and thereby aid in the development of countermeasures.

## 2. Materials and methods

### 2.1. Participants.

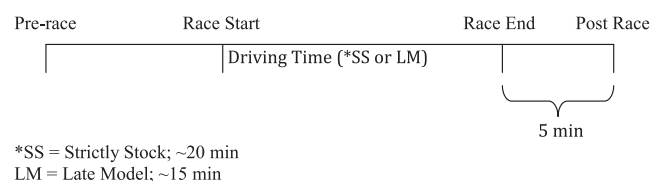
Eight male stock car drivers (mean age:  $20.0 \pm 10.0$  yr, body mass:  $80.6 \pm 15.7$  kg, height:  $176.2 \pm 3.3$  cm, and body fat:  $17.5 \pm 5.1\%$  (Jackson and Pollock, 1985)), and experience driving ( $8.8 \pm 3.5$  yr), identified by the Oxford Plains Speedway (Oxford, ME), volunteered for this study. All eligible participants gave their written informed consent to participate in this study, which was approved by the University of New England's Institutional Review Board for Human Subjects. Each driver was told that his physiological responses to competitive racing would be monitored.

### 2.2. Procedure

All measurements were taken during a sanctioned *Champion Series* Stock Car race (minor league series for NASCAR Sprint Cup) in July. Each driver underwent pre- and post-race assessments consisting of anthropometric and physiological measurements. Prior to the anticipated race start times, researchers recorded all initial pre-race measurements. After each race, the drivers returned to the pit areas and all post-race assessments were recorded within 5 min of completion of the race. Race and data collection timeline is depicted graphically in Fig. 1. Environmental temperature and humidity using a sling psychrometer (Reed, Montreal, Canada), and cockpit heat stress via a thermal laser (Craftsman, Sears Roebuck) were also assessed prior to and after competition races.

### 2.3. CORE and skin temperatures

To monitor core temperature ( $T_c$ ), drivers ingested a telemetry pill (Jonah core body temperature capsule, Mini Mitter, Bend, OR) at the racetrack 4.5 h before racing to allow adequate time for gastric emptying. Dermal temperature patches (Mini Mitter) were placed on the subject's right chest at the mid-clavicular line, approximately midway between the clavicle and the nipple, the



\*SS = Strictly Stock;  $\sim 20$  min  
LM = Late Model;  $\sim 15$  min

Fig. 1. Race and data collection timeline.

medial forearm, the anterior thigh, and the posterior calf. Core and skin temperatures readings were logged continuously and telemetrically on a monitor (Vitalsense monitor, Mini Mitter) that each volunteer wore around their waist in their fire suit. Mean skin temperatures ( $T_{sk}$ ) from the four dermal patch sites was calculated using a modified Ramanathan formula (Ramanathan, 1964):

$$T_{sk} = 0.3(T_{chest} + T_{arm}) + 0.2(T_{thigh} + T_{leg})$$

Since core and skin temperatures were logged continuously, temperatures were used from the start of the race (green flag) as each driver finished their race. A core-to-skin temperature gradient was calculated by subtracting  $T_{sk}$  from  $T_c$  at each 3 min interval.

#### 2.4. Cardiovascular and driver perception responses

Drivers were also fitted with a HR monitor (Polar Heartrate Monitor, Lake Success, NY), which continuously recorded HRs at 15-s intervals during their racing session. Due to interference from an electromagnetic field within each car, HR data storage was dismissed and only pre- and post-race HRs were utilized for the analysis. A Physiological Strain Index (PSI) was determined based on  $T_c$  and HR to evaluate heat stress using the following calculation (Moran et al., 1998):

$$PSI = 5(T_{re_t} - T_{re_0}) \times (39.5 - T_{re_0}) + 5(HR_t - HR_0) \times (180 - HR_0)^{-1}$$

Seated blood pressures via standard auscultation with a sphygmomanometer and cuff were also assessed prior to and immediately after their races. Pulse pressure (PP; mmHg) was calculated as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP). Mean arterial pressure (MAP; mmHg) was calculated as  $MAP = DBP + PP/3$ . Additionally, prior to and immediately after completion of the race, drivers were asked their rating of perceived exertion (RPE) utilizing a scale of 6–20 (Borg, 1998). Drivers were also asked before and after the racing competition to rate their thermal discomfort using a seven-point thermal scale (ASHRAE, 1966).

#### 2.5. Fluid balance

Before and after the race, each subject undressed and nude body mass was recorded using a calibrated scale (Tanita, Arlington Heights, IL) to provide an estimate of fluid loss. Drivers were permitted to drink ad libitum prior to the race; however, drivers were not able to drink fluids during the competitive racing session, and were asked not to ingest fluids until after all measurements were taken post-race. The total protective clothing ensemble worn by the drivers consisted of a Nomex<sup>®</sup> fire-retardant suit, gloves, underwear, socks, and boots. Insulation (clo) and vapor resistance [defined as the ratio of intrinsic moisture permeability index ( $I_m$ ) to insulation, or  $I_m/clo$ ], of this ensemble were estimated from similar US military clothing ensembles (evaluated on thermal manikins) as 1.56 clo and 0.16  $I_m/clo$  (T. Karis, personal communication).

#### 2.6. Environmental conditions, the track, and stock cars

Ambient temperature and relative humidity were recorded pre- and post-race using a sling psychrometer. In addition, the climate of the cockpit of each stock car was also recorded via a thermal laser. A cockpit temperature pre- and post-race on the transmission plate inside all cars was recorded to represent the cars' heat storage. No cars or driver equipment provided any cooling system during the race.



Fig. 2. Late model stock car.

The Oxford Plains Speedway (OPS) track is a 0.6035 km paved asphalt surface with 6° of banking. Drivers competed in either the Strictly Stock (SS;  $n=3$ ) or the Late Model (LM;  $n=5$ ) race. SS race competition involved 30 laps whereas the LM race consisted of 40 laps. Vertical  $G$  force was calculated with the following equation (Knight, 2003):

$$G \text{ force} = (\text{Speed of the Racecar} / \text{radius of the race track turns}) / \text{the acceleration due to gravity}$$

All stock cars were American-Canadian Tour (ACT) registered and compete under ACT rules. Late Model Stock Car races are open to eligible models of American made passenger car production cars such as: Impala, Monte Carlo, Grand Prix, Fusion, Taurus, Charger, Intrepid and Camry. SS racecars are 1972 and newer models of American-made passenger cars like the LM cars; however, they have slight differences in performance specifications. Fig. 2 demonstrates the types of racecars used in this investigation.

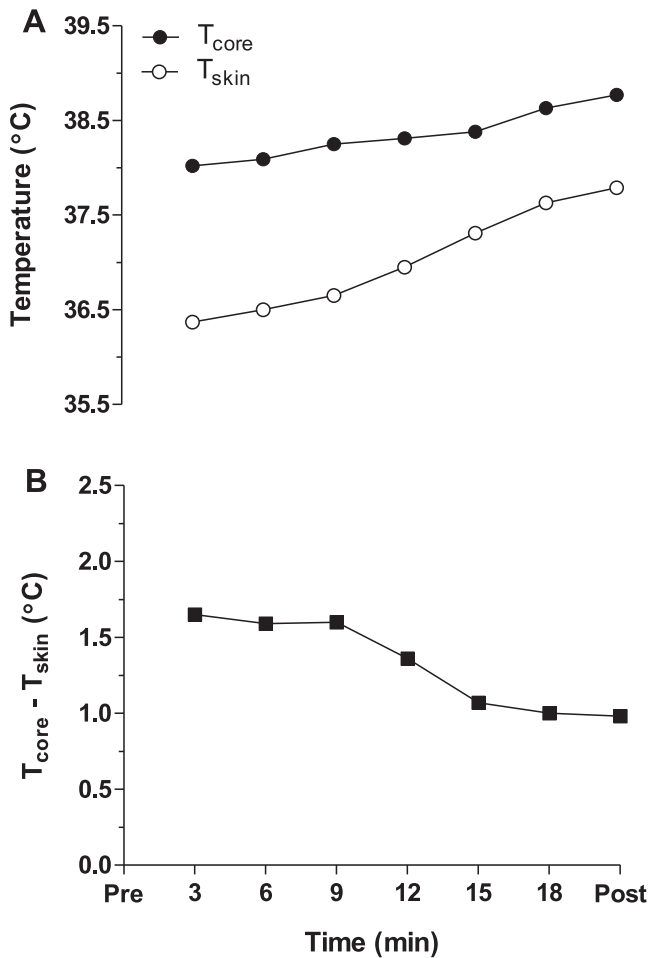
#### 2.7. Analysis

All physiological data for all drivers from both races were combined and are expressed as mean  $\pm$  SD. Statistical analyses were performed in JMP v. 7.0 (SAS, Cary, NC) by using a Student's  $t$  test to evaluate pre- and post-race measurements. An alpha level of 0.05 was set *a priori*. Data were checked for normality and a Grubb's test (Grubbs, 1950) was performed to identify any outliers. If an outlier was identified it was removed from analysis.

### 3. Results

All drivers finished the competition races. Drivers in the SS race completed 30 laps with an average speed of 117.47 km/h, totaling  $19.46 \pm 0.05$  min in duration. LM drivers completed 40 laps with an average speed of 129.54 km/h which took  $15.12 \pm 0.06$  min in duration. The vertical  $G$  load on the SS class was 1.6Gs in the turns while the  $G$  load on the LM drivers was 1.8Gs in the turns. The environmental conditions for both the SS and LM races were similar as the ambient temperatures were 32.0 °C and 31.7 °C, respectively and the relative humidity was 75% and 73%, respectively. The cockpit surface temperature averaged  $36.9 \pm 2.1$  °C prior to the start of the race, and increased ( $p=0.001$ ) to  $66.3 \pm 8.2$  °C post-race.





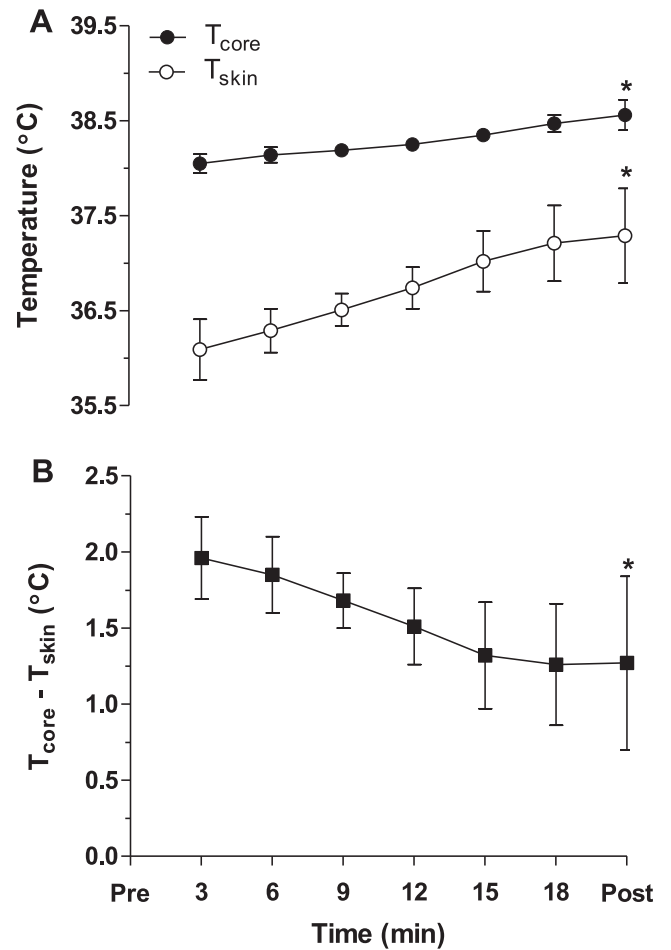
**Fig. 3.**  $T_c$  and  $T_{sk}$  responses (A) and  $T_c - T_{sk}$  gradient (B) in individual driver during competitive stock car race.

### 3.1. Thermoregulatory responses

Mean driver core temperatures increased from a pre-race value of  $38.1 \pm 0.1$  °C to a post-race value of  $38.6 \pm 0.2$  °C ( $p=0.005$ ; Fig. 3). All skin temperatures (chest, forearm, thigh and calf) also had a significant ( $p=0.001$ ) increase in temperatures during competition and are presented in Fig. 3.  $T_c$ ,  $T_{sk}$ , and  $T_c - T_{sk}$  gradient of an individual driver is presented in Fig. 3. Overall, mean skin temperatures increased ( $p=0.001$ ) as well from  $36.1 \pm 0.2$  °C pre-race to  $37.3 \pm 0.3$  °C post-race and the core-to-skin temperature gradient decreased ( $p=0.005$ ) from a pre-race value of  $2.0 \pm 0.3$  °C to  $1.3 \pm 0.3$  °C post-race (Fig. 4).

### 3.2. Cardiovascular and perception responses

Drivers' HRs significantly ( $p=0.001$ ) increased from pre-race value of  $105.6 \pm 9.1$  to  $152.0 \pm 10.1$  beats/min post-race. Overall, drivers' HRs post-race were  $80 \pm 0.1\%$  of the drivers' age-predicted maximum HR. Systolic ( $p=0.352$ ) blood pressure did not significantly decrease from a pre-race value of  $135.5 \pm 7.9$  mmHg to  $128.3 \pm 19.6$  mmHg post-race. Diastolic blood pressure did not have a significant ( $p=0.416$ ) change with a pre-race value of  $81.3 \pm 7.5$  mmHg and a post-race value of  $78.3 \pm 6.8$  mmHg. Prior to running the Student's  $t$  test all data were subject to a Grubb's test to identify outliers. Single values were removed if deemed an outlier based on the Grubb's test (Grubbs, 1950). Once the outlier was removed, the data was then analyzed by  $t$  test ( $n=7$ ). The results of the Grubb's test showed one subject was an outlier in



**Fig. 4.**  $T_c$  and  $T_{sk}$  responses (A) and  $T_c - T_{sk}$  gradient (B) throughout a competitive stock car race. Denotes significant difference ( $p < 0.05$ ) between pre- and post-race values, mean  $\pm$  SD.

the MAP and PP measurement. That subject was removed from analysis and Student's  $t$  test was run to determine significant changes between pre and post-race. There was not a significant ( $p=0.246$ ) change in PP with pre-race values of  $52.3 \pm 9.6$  mmHg and post-race values of  $45.2 \pm 13.5$  mmHg; however, there was a significant ( $p=0.011$ ) decrease in mean arterial pressure (MAP) from  $98.6 \pm 5.4$  mmHg pre-race to  $91.8 \pm 3.8$  mmHg post-race. PSI post-race was 4.9, which indicates moderate strain. At the completion of the races, the drivers' perception of thermal sensation based on the ASHRAE Scale increased ( $p=0.001$ ) from  $1.3 \pm 0.5$  to  $2.8 \pm 0.4$ , and the participants RPE responses also increased ( $p=0.001$ ) from  $8.4 \pm 1.6$  (extremely light) to  $13.9 \pm 1.8$  (somewhat hard).

### 3.3. Fluid balance

Despite our inability to account for fluid intake or output during the time period of the race, the average driver nude body mass pre-race was  $80.6 \pm 15.7$  kg and decreased to  $80.0 \pm 15.8$  kg post-race ( $p > 0.05$ ). The percent dehydration determined following the race competition was  $0.71 \pm 0.6\%$ . It was not possible to control all fluid intake and outtake pre- and post-racing so while post body mass values are reported, sweat rates could not be calculated.

#### 4. Discussion

The purpose of this study was to quantify the physiological strain of stock car drivers during an actual sanctioned stock car race competition in hot weather. No research to date provides complete information about the physiological effects of driving a stock car in which all the motorsports athletes are competing during a race (Potkanowicz and Mendel, 2013). The major findings of this study were that driving a stock car during a competitive race resulted in significant increases in skin and core temperatures along with increases in heart rate, a significant decline in mean arterial pressure, and increased physiological strain. Much of the existing exercise literature documents the concerted impact of environmental heat stress and elevated  $T_c$  and  $T_{sk}$ , and its implication on endurance performance (Cheuvront et al., 2010; Kenefick et al., 2007; Sawka et al., 2012); however, although the effects of heat stress on exercise physiology have been well studied, less is known about its impact during more specialized tasks like driving a stock car where all drivers are competing in a race under the same ambient conditions. The combination of the stock car drivers' required fire-retardant protective equipment, extremely hot race car cockpit temperatures, elevated ambient temperature and relative humidity, and increase in metabolic work resulted in considerable body heat storage in a short period of time, as evidenced by our drivers' increase in  $T_c$  and  $T_{sk}$ .

The present findings from our study showed that the combined ambient environment, stock car cabin conditions, and required protective clothing while driving created a circumstance that was very stressful to the drivers' ability to maintain a thermal steady-state. Our investigation demonstrated that uniformed stock car drivers encounter thermal stress from short duration, competitive driving in hot conditions. Although an investigation like this has never been done in the motorsports world, our findings are consistent with some limited observations among an analogous cadre. Similar heat stress responses were also found among helicopter aircrew wearing immersion protection suits during flight operations (Ducharme, 2006). Whereas the cockpits of our racecars averaged  $\sim 66^\circ\text{C}$ , Ducharme (2006) reported that with military helicopter cabin temperatures as low as  $18^\circ\text{C}$  aircrew experienced thermal discomfort while wearing immersion suits of 2.2 clo (insulation) rating. Additionally, exposure for 140 min to  $25^\circ\text{C}$  increased rectal and skin temperatures up to  $38.4^\circ\text{C}$  and  $35.7^\circ\text{C}$  respectively, and heart rates up to 160 beats/min (Ducharme, 2006). Similarly, aviation pilots wearing protective clothing of 2.2 clo rating in  $40^\circ\text{C}$  conditions also had increased rectal and skin temperatures, and heart rates (Faerвик and Reinertsen, 2003). Likewise, interiors of armored vehicles have been reported to reach as high as  $54.5^\circ\text{C}$  (Kuennen et al., 2010) and with the protective gear soldiers must wear, increases the risks of heat illness which may impair muscle activation and mental alertness (Racinais et al., 2008). A common hallmark of the current study and the military personnel studies is that the microclimate observations in a vehicle with elevated cabin temperatures, and the addition of protective clothing, which hinders evaporative cooling, results in increased heat storage and creates a stressful microclimate.

Prior to the start of the races, our drivers'  $T_c$  and  $T_{sk}$  were starting to rise, which advocates for pre-cooling techniques to be employed prior to getting into the racecar. As a result of the stressful environment, our drivers reported an increase in their perceived thermal sensation based on the ASHRAE Scale from 1.3 to 2.8 by the completion of the races. We found that all our driver skin temperatures (chest, forearm, thigh and calf) had significant elevations in temperature. Interestingly, the significant thermal exposure our drivers' endured was only between 15 and 20 min in duration, and yet their mean core and skin temperatures rose to  $38.6 \pm 0.2^\circ\text{C}$  and  $37.3 \pm 0.3^\circ\text{C}$

correspondingly. Sawka et al. (2012) defined hot skin as  $35^\circ\text{C}$  and above, which our drivers clearly presented as  $\sim 37^\circ\text{C}$  post-race in full protective clothing which may have limited evaporative cooling. It is important to note that ASHRAE limits  $T_c$  to  $< 38.5^\circ\text{C}$  for continuous work (ASHRAE, 1986). The increase in core temperature appears small as we only observed a  $0.5^\circ\text{C}$  increase within  $\sim 20$  min. However, if we assume a constant rate of heat storage, a 1 h race could produce a  $\sim 1.5^\circ\text{C}$  elevation in  $T_c$ . In both laboratory and field studies, Sawka et al. (2001) reported that 50% of subjects experienced heat exhaustion with core temperatures of  $38.6^\circ$ , and skin temperatures of  $> 35^\circ\text{C}$ , indicating a very narrow core–skin temperature gradient. Meanwhile our average core–skin temperature gradient declined by 35%. An increase in skin temperature narrows the difference between the core and skin temperatures, which consequently reduces central blood volume and decreases the amount of the heat delivered to the skin (Crandall, 2008; Sawka et al., 2012). As a result, more blood flow is directed to the skin surface, which further stresses cardiovascular function (Rowell, 1986; Sawka et al., 2012) via reducing cardiac filling and increasing heart rate in order to sustain cardiac output (Sawka et al., 2012; Trinity et al., 2010). It is well established that elevated core and skin temperatures inflict significant cardiovascular strain through increases in heart rate and reductions in blood pressure caused by augmented cutaneous blood flow (Brearley and Finn, 2007; Cheuvront et al., 2010; Cheuvront and Haymes, 2001; Cheung et al., 2000; Gonzalez-Alonso et al., 2008; Sawka et al., 2012). Elevated skin temperatures were likely accompanied by greater cutaneous skin blood flow, further exacerbating cardiovascular strain (Brearley and Finn, 2007; Cheung et al., 2000; Jacobs et al., 2002).

While collecting data during an actual competitive event increases the 'real-world' application of the findings, it also introduces some limitations. For example, we did not have full access to the drivers other than during the pre- and post-race data collection time points and as a result, we were unable to account for all fluid intake and output throughout the course of data collection and thus were unable to calculate sweating rate. Despite this limitation, we did calculate a  $\sim 1\%$  change in body mass, which suggests that some degree of dehydration may occur during this type of event.

Driving a Stock Car imposes substantial physiological strain due to combined uncompensable heat stress and G-forces (Allan and Crossley, 1972). For example, our drivers' heart rates post-race competition increased to an average of  $152 \pm 10.1$  beats/min which was  $80 \pm 0.1\%$  of the drivers' age-predicted maximum HR. Oxygen consumption and HR responses to open-wheel road course driving have been reported to be similar to those found in more traditional sport settings such as running or swimming (Jacobs et al., 2002). During open-wheel roadway course driving, oxygen uptake and HR responses have been found to reach 79% and 82%, respectively, of values obtained in maximal graded treadmill exercise tests, (Jacobs et al., 2002). Heart rates were most often above 90% of maximum heart rate during motorcycle racing (D'Artibale et al., 2008) while HR responses during kart driving averaged 150 beats/min (Yamakoshi et al., 2010). Coinciding, our drivers' RPE during the race changed from extremely light pre-race to somewhat hard post-race, and the PSI increased to a moderate strain (4.92) during the racing competition. The elevated heart rates observed in our drivers were likely the result of the rise in core and skin temperatures, the demands of driving a stock car, and the increased sympathetic drive from the stimulating emotional response of racing (Brearley and Finn, 2007). Although driving a racecar increases arousal and sympathetic drive (Jacobs et al., 2002), it is not the sole mechanism to be considered elevating heart rate. With an increase in both core and skin temperatures, there will be an increase in skin blood flow and a reduction in central blood volume, thus a decrease in venous return and an increase in heart rate to maintain cardiac output (Cheuvront et al., 2010; Crandall, 2008; Jacobs et al., 2002).

In this study of stock car racing, we found that driver systolic blood pressures and MAPs both decreased  $\sim 7$  mmHg from pre-race to post-race. Although not statistically significant, PP also decreased  $\sim 4$  mmHg from pre-race to post-race. A PP reduction of  $\sim 5$  mmHg has been associated with a reduced stroke volume owing to central hypovolemia (Convertino et al., 2006). Furthermore, the reduction of our drivers' MAPs supports our findings regarding the  $T_c - T_{sk}$  observations and the narrowing of the core-skin temperature gradient. Taken together, these data suggest that elevated  $T_{sk}$  was likely accompanied by greater cutaneous skin blood flow, thus further magnifying cardiovascular strain by reducing central blood volume. The addition of  $G$  forces to the thermal racing situation also supports the potential for reduced grayout tolerance. As reported by Allan and Crossley (1972), elevated cockpit temperatures in military aircraft induced pilot core temperatures of  $38.5^\circ\text{C}$  which reduced grayout threshold by  $\sim 1G$ . What is both fascinating and concerning, is our drivers exhibited core temperatures of  $38.6^\circ\text{C}$  during short term racing coupled with 1.6 (SS) and 1.8 (LM)Gs. While these  $G$  loads are not the high  $G$ s as seen in Sprint Cup racing, the combination of an increased thermal stress, significant  $G$  forces, and heightened cardiovascular involvement all connected with a lower threshold for grayout tolerance is relevant to the performance and safety of drivers racing at high-speeds.

## 5. Conclusions

In summary, we are the first to quantify the thermoregulatory and cardiovascular strain of drivers during an ecologically valid stock car competition on an oval speedway. Our prospective findings suggest that drivers endure increased thermal stress (elevated skin and core body temperatures), increased cardiovascular strain, and potential fluid losses during competitive stock car racing in hot conditions. Our observations are in accord with the current view that other individuals who wear full protective uniforms in hot conditions may become susceptible to thermal strain; especially those not acclimated to the heat (Walker et al., 2001b). These results are of concern in the motorsports realm as significant thermal stress may affect driver safety. From a practical point of view, these findings are important because NASCAR motorsports athletes can compete in races for 3–4 h of competition under stressful thermal conditions with repetitive  $G$  force. Here, our drivers' core and skin temperatures are in the range in which grayout tolerance was found to be reduced according to findings by Allan and Crossley (1972). Taken together with the current literature regarding other motorsports drivers, the present study provides evidence that motorsports drivers and teams should consider strategies to improve cardiovascular fitness, heat tolerance (acclimatization), and monitor fluid replacement, to better meet the thermoregulatory and cardiovascular challenges of motorsport competition. The results of this investigation provide support for more research on techniques for mitigating heat stress for drivers and for preparing them for competition in the heat to reduce accidents. Stock car drivers are athletes and we should advocate for their safety when competing in their sport.

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