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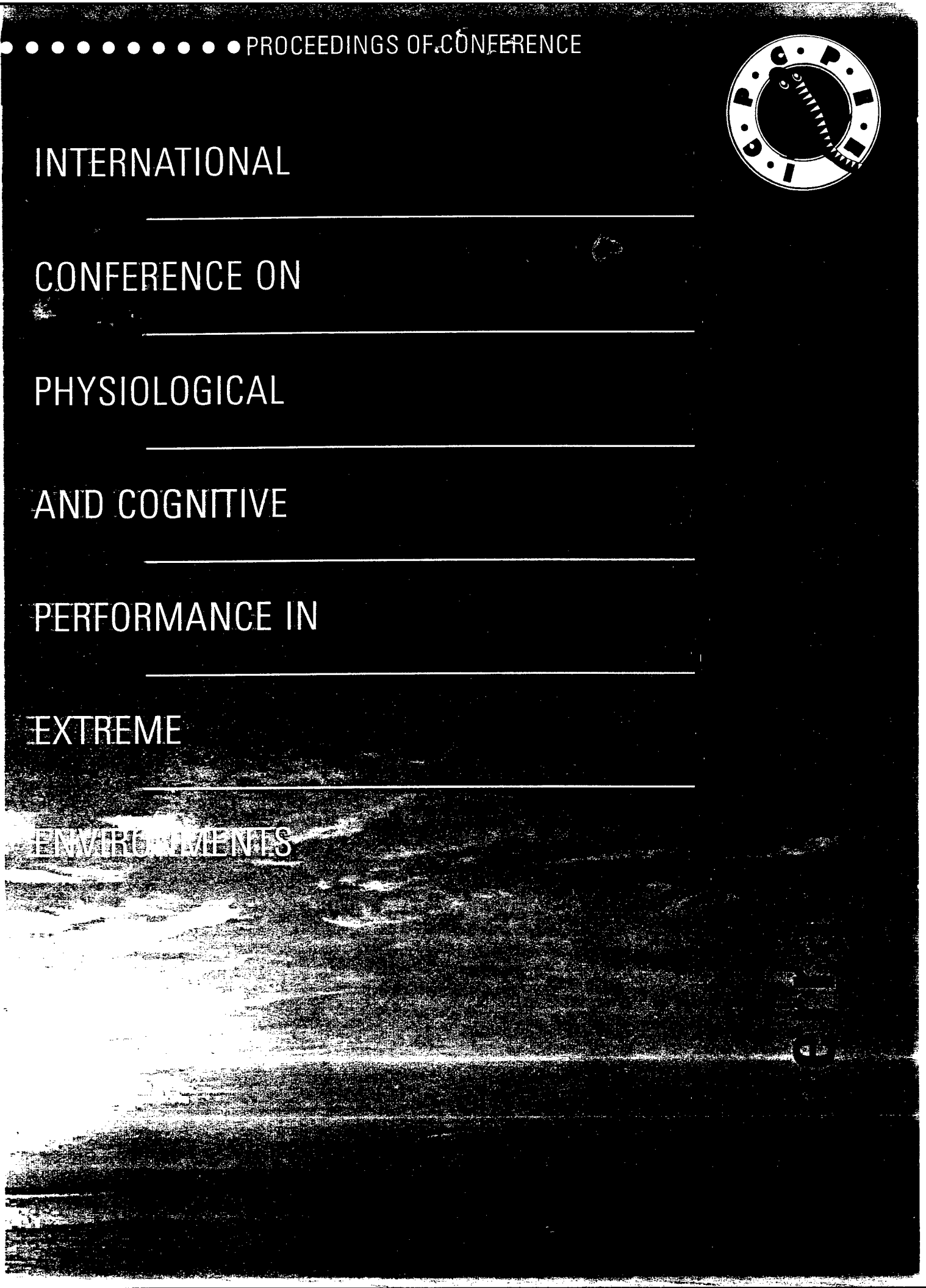

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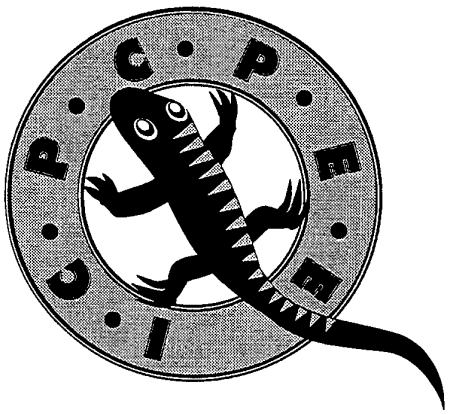

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INVITED LECTURE 1: EXERCISE, HEAT STRESS AND FATIGUE

M. Hargreaves

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An individual performing prolonged, strenuous exercise in a heat stressful environment experiences marked physiological and metabolic alterations due to the need to attain and maintain an adequate rate of heat loss. These include increased heart rate and body temperatures, elevated sweat rates and fluid losses, decreased stroke volume, cardiac output, central venous pressure and muscle blood flow, an accelerated rate of muscle and liver glycogenolysis, increased muscle and blood lactate accumulation, and elevated plasma catecholamines [1,2,3,4], to name only a few. Exercise performance, measured either by time to exhaustion [5,6,7,8] or average power output during exercise [9], is reduced with hyperthermia. For example, increasing environmental temperature from 20°C to 40°C reduced exercise time to fatigue at 70% VO_2 peak from 67 $\pm$ 1 min [mean $\pm$ SEM] to 30 $\pm$ 3 min [8]. Even the thermal load associated with exercise at 20°C may have performance implications, since at 3°C exercise time increased to 89 $\pm$ 10 min [8]. Notwithstanding the above-mentioned physiological and metabolic alterations during exercise in the heat, it has been suggested that the underlying cause of the premature fatigue observed under these conditions is the attainment of a critically high body core temperature that, in turn, impairs central nervous system function and motor drive [6,10]. Direct evidence of such a mechanism, however, is lacking.

Fatigue during exercise under mild environmental conditions is often associated with muscle glycogen depletion and hypoglycemia. Given that the reliance on muscle and liver glycogen is greater during exercise in the heat [1,2], it is possible that fatigue during exercise in the heat is a consequence of more rapid carbohydrate depletion. However, at the point of fatigue muscle glycogen and blood glucose levels can remain relatively high [8]. Furthermore, carbohydrate ingestion does not always enhance exercise performance in the heat [11], unlike the well described ergogenic benefits of carbohydrate ingestion during prolonged exercise at lower ambient temperatures [12]. This does not exclude the possibility of other metabolic alterations within contracting skeletal muscle with elevated temperature, but these have not been well studied. Interestingly, a recent study has observed enhanced exercise performance in the heat following a dietary carbohydrate loading protocol [13], despite indications that carbohydrate availability was not limiting in the low carbohydrate condition in the heat. This result raises the possibility that an increase in dietary carbohydrate may exert an effect via mechanisms other than increased skeletal muscle glycogen availability [eg. central fatigue?]. Ingestion of branched chain amino acids, suggested to alter central nervous system neurotransmitter balance, was also associated with improved exercise performance in the heat [14]. Further studies are required to investigate the role of central mechanisms in the etiology of fatigue during exercise in the heat.

Since hyperthermia appears to be the major determinant of exercise performance in the heat, strategies that delay the attainment of a critical body core temperature are likely to contribute to enhanced exercise performance. These include acclimation to heat, pre-exercise cooling and adequate rehydration during exercise. Repeated exposure to exercise and heat stress [acclimation] results in a lower heart rate and body core temperature during subsequent exercise in the heat and enhanced exercise tolerance [10]. Of interest in this study was the observation that the improved exercise performance appeared to be more related to a lower initial core temperature than to an enhanced rate of heat dissipation. Thus, pre-exercise cooling may also be an effective strategy for enhancing exercise in the

heat and this has been proven to be the case in a number of studies [6,15,16,17]. Results from one of these studies [6] are summarised in Figure 1 which demonstrates that pre-exercise cooling resulted in an increased exercise time to fatigue, while pre-exercise heating impaired exercise tolerance, relative to a control trial. Of note, exercise appeared to be terminated at a similar esophageal temperature in all three conditions. Finally, the rise in body core temperature during exercise in the heat is directly related to the degree of dehydration [18], suggesting that athletes should attempt to replace as much of the sweat fluid losses as is practicable.

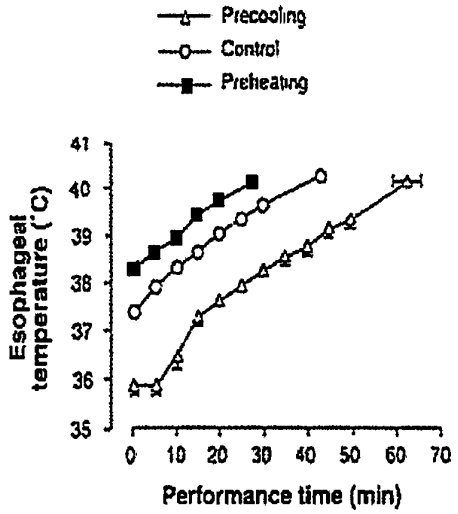

FIGURE 1: Esophageal temperatures during exercise to fatigue at 60% VO_2 peak and 40°C following either pre-cooling, pre-heating or no treatment. Values are means ± SEM for 7 subjects. Data from González-Alonso et al. [6] and Figure from Coyle [19].

In summary, exercise in the heat results in major alterations in circulatory, thermoregulatory and metabolic function, and the degree of hyperthermia appears to be the major determinant of exercise performance in the heat. Strategies that delay the attainment of a critically high core temperature are likely to contribute to enhanced performance. These include acclimation, pre-exercise cooling and fluid ingestion during exercise.

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PAPER 1: THE INFLUENCE OF WHOLE BODY VS TORSO PRE-COOLING ON PHYSIOLOGICAL STRAIN AND PERFORMANCE OF HIGH INTENSITY EXERCISE IN THE HEAT.

G.G. Sleivert[1], J.D. Cotter[2], W.S. Roberts[2] and M.A. Febbraio[3]

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INTRODUCTION

Pre-cooling before prolonged exercise in the heat has been studied extensively over the last 15 years, eg. [1-3], but little work has been published reporting on the effects of pre-cooling prior to high-intensity exercise in the heat. The literature suggests that pre-cooling is detrimental to this type of performance, largely because of decreased muscle temperature [4-6]. Conversely, a recent study that utilised water immersion to cool the torso, but not the legs reported a small enhancement of 70-s sprint cycling performance [7]. The cooling strategy in the latter study differed from others in that the leg muscles probably remained relatively warm. It is possible then that by maintaining the temperature of the working muscles but cooling the remainder of the body, high intensity exercise in the heat can be improved. Since muscle temperature and warm-up exercise may both influence high-intensity exercise performance [8-10], the interaction of pre-cooling the working muscles, and/or core and skin temperature with an exercise warm-up is not known. The purpose of the present study was therefore to determine whether pre-cooling with and without surface pre-cooling of the working muscles influenced high intensity (45 s) exercise performance, and to determine whether the effects of pre-cooling were influenced by a short exercise warm-up. Specifically, three conditions were examined in a balanced, cross-over fashion: 1. Warm thighs, cold remainder; 2. Cold thighs, cold remainder; 3. Warm thighs, warm remainder (control). It was hypothesised that pre-cooling the torso but not the legs would optimise high-intensity exercise, and that a short exercise warm-up would remove any detrimental effects of cooling the working muscles.

METHODS & MATERIALS

Subjects: After providing informed consent nine healthy males (age = 32.4 ±3.6 yr, weight = 80.8 ±9.9 kg) participated in these trials. All procedures followed the ethical approval of the Australian Defence Medical Ethics Committee. The experiments were conducted in two environmentally-controlled laboratories during the period of February – August, 1999. Subjects' pre-trial physical activity and hydration were standardised.

Protocol: Subjects completed at least two familiarisation trials, in which $\dot{V}O_{2peak}$ was determined during a maximal effort test, and opportunity was given to practice the high-intensity cycling work trial. Subjects completed three trials, presented in balanced order, at least one week apart. In two trials the subjects were cooled for 45 min by ice vest (Coollnz™, Dunedin, NZ) and cold (3°C) air, with the thighs warmed (T) or cooled (W) using water-perfused cuffs. In the other trial they sat quietly in the warm (31°C) chamber for 45 min (C). Subjects then performed a 6 min warm up at 45-50% $\dot{V}O_{2peak}$, followed by a 6-min rest, then a 45-s, 'all out' power test on an electrically-braked cycle ergometer (Lode, Netherlands) (air temperature = 33°C, relative humidity = 60%). The cycle ergometer was used in constant-load mode. Work load was set as a linear factor of 0.04 + 0.0001 * body mass (kg), determined in pilot work to optimise 45-s power output. The effect of no warm-up on high-intensity power output was then assessed by repeating these trials, minus the warm up, in five of these subjects, and in one additional subject (n=6 habitually-active males).

Measurements: External power was recorded at 50 Hz, from which peak and 45-s mean powers were calculated. Core temperature ($\bar{T}_c$) was taken as the unweighted mean of rectal (T_{re}) and oesophageal (T_{es}) temperatures. T_{re} was measured 10 cm beyond the anus, whilst oesophageal depth was estimated to be at right atrial level, based on the sitting-height formula of Mekjavic and Rempel [11]. Mean skin temperature ($\bar{T}_{sk}$) was calculated from area-weighting of nine local skin temperatures, measured using thermistors; forehead, chest, abdomen, back, forearm, hand, front thigh, posterior leg and dorsal foot. Muscle temperature was recorded using a thermister inserted into the *vastus lateralis* to a depth of 4 cm. Heart rate was measured at 5-s intervals by telemetry of the detection of ventricular depolarisation (R-R interval). Skin blood flow was estimated from forearm blood flow, as measured by venous occlusion plethysmography, in triplicate, from a mercury-in-silastic strain gauge.

Statistics: Dependent variables were analysed using 2-way repeated ANOVAs, with significant ($P<0.05$) differences isolated using the Neuman-Keuls post hoc test. Data are reported as means ± standard deviation of the mean.

RESULTS

Both peak and mean power were decreased after torso+thigh but not after torso-only cooling, and this finding was more pronounced in the absence of a short warm up (Figure 1). Core and skin temperatures were different between all conditions, with torso+thigh cooling eliciting the lowest skin and core temperatures, followed by torso cooling. There were no significant differences in muscle temperature between conditions after warm up, despite a trend for lower muscle temperature in torso+thigh cooling ($P= 0.16$). However, when no warm up was performed, torso & thigh cooling significantly decreased muscle temperature (Figure 2). Heart rate was higher in control than pre-cool conditions immediately prior to the 45-s test and mean heart rate over the 45-s test was lower after torso+thigh cooling than after no pre-cooling (Figure 3). Similarly, immediately prior to the 45-s test forearm blood flow was lower after torso+thigh cooling than after no pre-cooling (Figure 4).

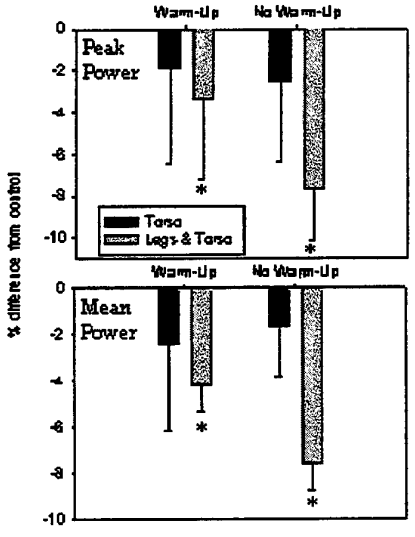

*Figure 1: Mean (SD) change in peak & mean 45 s power after pre-cooling the torso or torso and legs, relative to the control condition. * differs from control, P<0.05.*

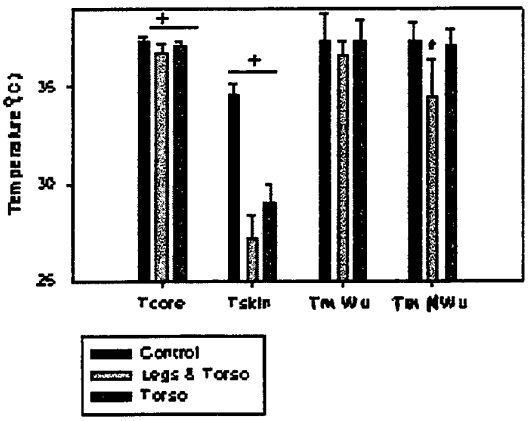

Figure 2: Mean (SD) mean core, mean skin and muscle temperatures (T_m) with or without warmup (WU) immediately prior to 45s power test in control condition and after pre-cooling the torso or the torso and legs. + difference between all conditions, # different from other conditions P<0.05.

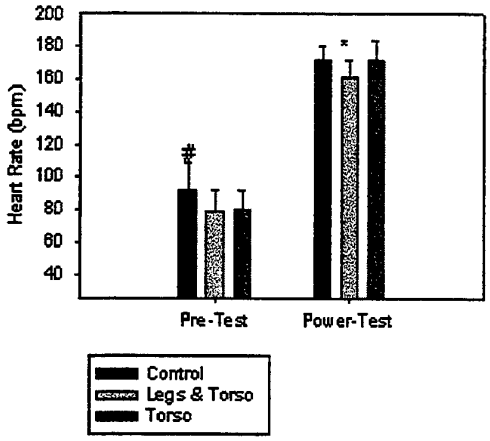

*Figure 3: Mean (SD) heart rate immediately prior to and during the 45-s power test in the control condition and after pre-cooling the torso or the torso and legs. # differs from other conditions, * differs from control, P<0.05.*

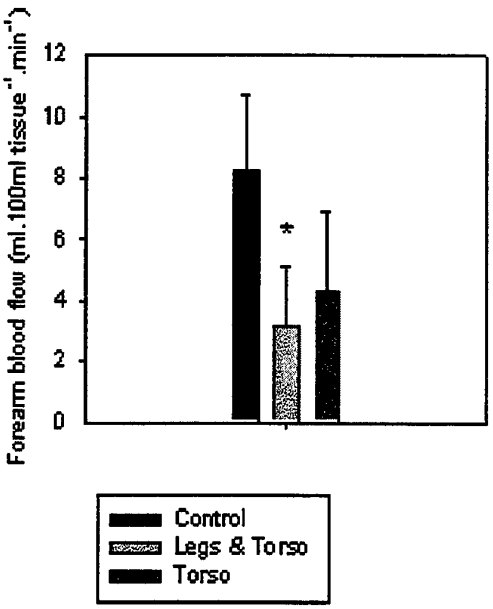

*Figure 4: Mean (SD) forearm blood flow immediately prior to 45-s power test in the control condition and after pre-cooling the torso or the torso and legs. * differs from control, P<0.05.*

DISCUSSION & CONCLUSIONS

The results indicate that pre-cooling of the torso+thighs impairs short-term, high-intensity exercise performance, but this impairment is minimised if cooling is not applied to the thighs. These effects were most strongly evident in the absence of an active warm up. The present findings contrast with those of a previous study demonstrating enhanced 70-s mean power after water immersion pre-cooling of the torso [7]. A longer warm-up (10 min) was used in that former study, which may have increased muscle temperature and activated aerobic metabolism to a greater extent than in the present protocol. Additionally, the longer performance test used in the study of Marsh & Sleivert [7], possibly involved greater reliance on aerobic re-phosphorylation of ATP than during the 45-s test used in this study. Together, these factors might explain the discrepancy observed in performance outcomes. Even so, it can be concluded that cooling applied over the muscles to be used should be avoided before high-intensity exercise performance since it impairs performance, but this may apply only if there is little or no opportunity for active warm up. Further, torso cooling does not impair high-intensity exercise performance.

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PAPER 2: THE INFLUENCE OF TORSO AND WHOLE-BODY PRE-COOLING ON STRAIN AND PERFORMANCE DURING ENDURANCE WORK IN THE HEAT.

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INTRODUCTION

Exertional heat stress, whether it be exercise in a heat-stressful environment or work in protective clothing, causes heat to be stored in the body, thereby raising body core temperature (T_c). The ensuing demand for skin blood flow is met by progressive increases in heart rate and redistribution of the cardiac output. Substantial heat stress and dehydration compromises cutaneous perfusion [1,2] and perhaps even cardiac output and muscle perfusion [2], facilitating higher T_c, muscle temperature (T_m) and rate of muscle glycogen depletion [2]. The elevation in T_c and/or T_m *per se*, appears to limit exercise tolerance in the heat [1]. It is therefore not surprising that lowering of resting body temperature, as occurs in heat acclimation, confers greater heat tolerance [3]. Lowering body temperature before exercise by surface cooling, ie. pre-cooling, has also been shown to reduce thermal, cardiovascular, and metabolic strain during exercise in the heat [1,4,5,6], and usually to improve endurance performance in temperate [6] or hot [5] conditions. Water immersion, by virtue of its surface coverage and heat transfer capacity, provides effective pre-cooling [5]. However, perhaps the most relevant method of pre-cooling in many military settings is by cooling jacket (eg. ice vest) and cold air (eg. compartment interior). However, there appear to be few data available on the impact of this cooling procedure on strain and work performance effects. Therefore, since we were examining the effects of pre-cooling by ice vest and cold air - with and without cooling of the lower limbs - on high intensity power output, we further sought to determine the effectiveness of such pre-cooling in reducing heat-related strain and improving endurance performance in the heat. It was hypothesised that there would be no net adverse effect on endurance performance due to application of surface cooling over the muscles to be worked.

MATERIALS AND METHODS

Protocol: Nine male volunteers (mean ±SD age =32.4 ±3.6 y, weight =80.8 ±9.9 kg) completed two familiarisation sessions, during which $\dot{V}O_{2peak}$ was determined from a maximal effort test. Three trials were then performed, in balanced order, at least one week apart. In one trial subjects rested in 31°C air (C). In the other trials they were cooled for 45 min by ice vest (Cool1©, NZ) and cold (3°C) air, with the thighs warmed (T) or cooled (W) using cryogenic cuffs. Subjects then performed a 6-min warm up and a bout of high-intensity exercise in 33°C air (see previous paper, by Sleivert et al.), a short recovery, another 45 min period of C (34°C air) or pre-cooling (T, W), before 35 min cycling in the heat (35°C, 60% rh, v_a<0.5 m·s^{-1}). Cycling involved 20 min at a work rate of ~65% $\dot{V}O_{2peak}$, then a 15 min self-paced performance trial.

Measurements: Body weight and urine volume were recorded immediately before and after trials. Core temperature (T_c) was measured at 1-min intervals using thermistors in the rectum (T_{re}) and oesophagus (T_{es}). Mean core temperature ($\bar{T}_c$) was taken as the unweighted average of T_{re} and T_{es}. Skin temperature was measured at 1-min intervals using thermistors positioned on the forehead, chest, abdomen, back, forearm, hand, front thigh,

posterior leg and dorsal foot. Area weightings were used to calculate mean skin temperature ($\bar{T}_{sk}$). Muscle temperature (T_m) was measured periodically in *vastus lateralis*, 4 cm beneath the skin. Heart rate was measured at 5-s intervals from the R-R interval of ventricular depolarisation. Forearm blood flow (FBF) was measured by venous occlusion plethysmography, using triplicate samples from a mercury-in-silastic strain gauge. Cardiac output was determined by CO_2 rebreathing, using the Fick Principle, where F_{aCO2} was determined in duplicate samples by the exponential method, and initial F_{ICO2} was 0.039. Sensations of body temperature and thermal discomfort were recorded using 13 point and 5 point scales, respectively, adapted from Gagge et al. (1967). Perceived exertion was recorded for the legs and the whole-body using the 13 point Borg scale (Borg, 1962).

Statistics: The experimental design is a fully repeated, two-factor design, with three levels of factor one (treatment: C, T, W) and variable levels of factor two (time). Dependent variables were analysed using 2-way repeated ANOVAs. Significant ($P<0.05$) differences were isolated using the Neuman-Keuls post hoc test. Data are reported as mean ± standard error of the mean.

RESULTS

Constant-load work: At exercise onset the $\bar{T}_c$, $\bar{T}_{sk}$, FBF and HR were reduced in both W and T, relative to C. The $\bar{T}_c$ was different between all three conditions by 5 min and 20-min of exercise at ~65% $\dot{V}O_{2peak}$ (Figure 1, $P<0.05$). Whereas, $\bar{T}_{sk}$, FBF and HR were reduced equivalently between pre-cool conditions by 5 min, and remained equivalently lower than C at 20 min: $\bar{T}_{sk}$ (C: 35.7 ±0.2, T: 34.3 ±0.2, W: 34.1 ±0.3°C), HR (177 ±3, 163 ±3, 167 ±3 bpm) and FBF (15.5 ±1.6, 13.6 ±1.0, 11.4 ±1.0 mL·100mL tissue^{-1}·min^{-1}). The only exception was that FBF was equivalent between C and T at 20 min ($P=0.10$). In contrast, the lower muscle temperature for W before exercise was no longer present by 5 min ($P=0.07$, $N=7$). Perceptions of body temperature, thermal discomfort and work effort were lower ($P<0.05$) for both pre-cool conditions relative to no pre-cooling, at both 5 min and 20 min. Perceived effort of the legs was also lower for W relative to C at 20 min.

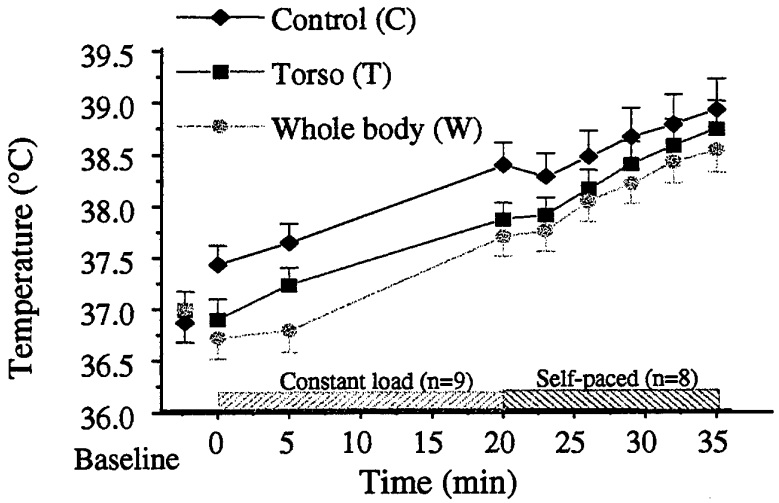

Figure 1. Mean (±SE) core temperature during exercise after no intervention (C) or after pre-cooling with (W) and without (T) cooling on the thighs. Core temperature is the mean of rectal and oesophageal temperatures. All conditions differed from each other after exercise onset ($P<0.05$).

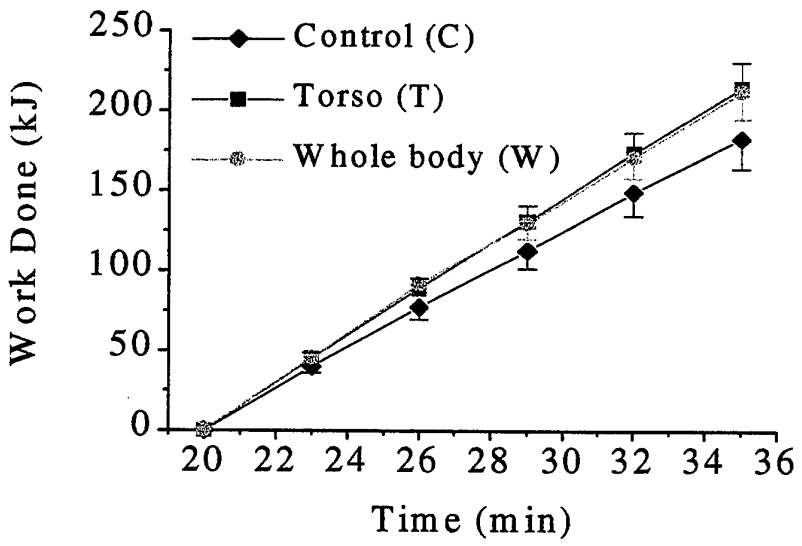

Figure 2. Mean (±SE) work done during the 15-min performance trial. See caption of Figure 1 for notation details. More work was done in both pre-cool trials than in the control trial. N=8. One subject is omitted due to very poor work tolerance in his control trial.

Self-paced work: Figure 2 shows that more work was done in the performance trial in both T (215 ±16 kJ) and W (212 ±16) than in CON (183 ±19, P=0.00, N=8), and that the additional work was performed throughout the 15-min period. Even so, $\bar{T}_c$ and FBF at completion remained slightly lower in T (38.7 ±0.3°C) and W (38.5 ±0.2) than in C (38.9 ±0.3, P<0.05, N=8). In contrast, HR and ratings of work effort, body temperature and thermal discomfort had become equivalent between all conditions by 3 min of this period, and remained so at completion.

DISCUSSION

Relative to pre-cooling by water immersion [4,7] or cold air alone [6], pre-cooling by ice vest and cold air was effective in reducing thermal, cardiovascular and psychophysical strain during subsequent exercise in the heat. In particular, thermal (T_c), cardiovascular (HR and FBF) and psychophysical indices of strain were generally still attenuated after 20 min of moderately-stressful exercise, which allowed subjects to perform more work throughout a subsequent 15 min period of self-paced exercise. Moreover, warming or cooling of the thighs during pre-cooling had little influence on physiological or psychophysical strain indices during exercise, other than further attenuation in T_c, and had no effect on the endurance performance. Coincident with the greater work output for both pre-cool trials was an equivalence of heart rate and psychophysical strain across all three trials, and a difference in T_c across all trials. It might therefore be speculated that the absolute level of T_c was not, by itself, limiting work performance under these conditions - ie. as distinct from the possible limiting factor(s) at the point of exhaustion.

CONCLUSIONS

Pre-cooling by ice-vest and cold air was effective in reducing physiological and psychophysical strain and improving endurance performance in the heat (35°C, 60% rh). The reduction in strain was evident to varying extents in body temperatures, cardiovascular strain, and in perceptions of thermal discomfort, work effort and temperature. The benefits

were mostly independent of whether the thighs were warmed or cooled during the pre-cool period. Therefore it is suggested that cooling garments should not be employed on the limbs to be worked.

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Trials were conducted in accordance with Australian Defence Medical Ethics Committee approval.

INVITED LECTURE 2: HEAT STRESS AND EXERCISE METABOLISM

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INTRODUCTION

Much of the research that has examined the interaction between metabolism and exercise has been conducted in comfortable ambient conditions. It is clear, however, that ambient conditions, particularly of heat and humidity, are major considerations for metabolism and exercise performance. When exercise is conducted in heat stressful environments, additional thermoregulatory activity is required to achieve adequate heat loss. This can ultimately impact upon hormonal and metabolic responses to exercise, which alters substrate utilisation and therefore also nutritional requirements. Hence, heat stress is an important factor to consider when examining exercise metabolism and performance.

SUBSTRATE UTILISATION

It is now generally accepted that exercise in a hot environment shifts substrate use. Intramuscular carbohydrate (CHO) utilisation has been demonstrated to be augmented when comparing exercise in the heat with that in a cooler environment [6, 7, 9, 11].

In addition, muscle glycogenolytic rate is reduced when the rise in body temperature is attenuated by heat acclimation [6, 14, 15], external cooling [16], a cooler environment [8, 17] or prevention of dehydration [10, 12]. The shift in respiratory exchange ratio [6, 11] and reduced intramuscular triglyceride use [9] demonstrate a substrate shift towards increased CHO utilisation and reduced fat utilisation when heat stress is exacerbated.

It appears that two mechanisms are responsible for these metabolic alterations. Exercise in the heat results in an approximate two-fold increase in the concentration of circulating epinephrine. Although some studies [1, 19] observed no effect of epinephrine on muscle glycogen use in untrained men, we have demonstrated that a two-fold increase in circulating epinephrine increases muscle glycogenolysis and muscle lactate accumulation in endurance trained individuals (Fig. 1) [5].

In addition to the influence of elevated epinephrine, a direct effect of temperature on substrate metabolism has also been observed. Manipulation of muscle temperature during intense, isometric [2] or dynamic [4] exercise results in elevated muscle lactate accumulation, increased ATP and PCr degradation and augmented muscle glycogen use. We have also recently conducted a study in which we heated one leg and cooled the other for 40 min prior to, and 20 min during, exercise at 70% VO_{2peak}. The difference in muscle temperature at exercise onset was reduced during exercise, but nonetheless remained significant at the completion of exercise. The warmed leg had an augmented rate of muscle glycogen use, but no differences in high-energy phosphagen metabolism when compared with the cooled leg [18]. Therefore, the data suggest that temperature *per se* plays a regulatory role in intramuscular CHO utilisation and appears to be responsible, in part, for the frequently observed increase in glycogen utilisation during exercise and heat stress.

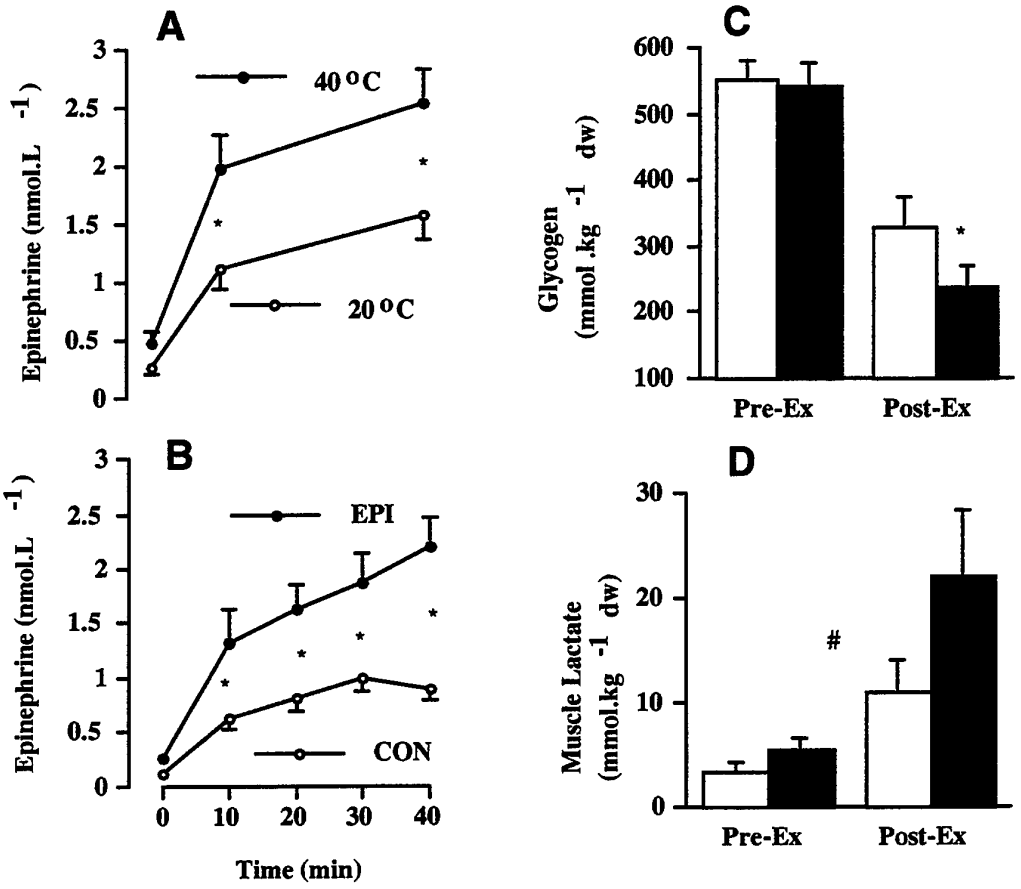

*Fig.1. Plasma epinephrine concentration during 40 min of exercise at 70% VO_{2peak} at 40°C and 20°C (Data from Febbraio et al. 1994a & Hargreaves et al. 1996a). Plasma epinephrine concentration (B), muscle glycogen use (C) and muscle lactate accumulation (D) during 40 min of exercise at 70% VO_{2peak} with (EPI, filled bars) and without (CON, unfilled bars) epinephrine infusion (Data from Febbraio et al. 1998). Values are means ± SE (n=20 for A; n=6 for B,C,D). * denotes difference (P<0.05) when comparing values at the common time point; # denotes main treatment effect (P<0.05).*

FATIGUE AND EXERCISE PERFORMANCE IN THE HEAT

Fatigue during prolonged exercise often coincides with depleted intramuscular glycogen concentrations, and glycogen use is exacerbated during exercise in the heat. It is somewhat paradoxical that fatigue during exercise in severe heat appears unrelated to substrate availability. When subjects exercise to exhaustion in hot conditions, they fatigue earlier, with higher intramuscular glycogen content and with an elevated body core temperature, compared with exercise in a cooler environment [17]. It has been suggested that the hyperthermia associated with exercise in the heat may be a major factor limiting exercise performance. It may be that either the attainment of a critical core temperature or the rate of rise in body heat storage may have negative effects on motor control centres. However, despite the fact that substrate availability is not limited during prolonged exercise and severe heat stress, one cannot rule out the possibility that temperature-induced metabolic perturbations take place within contracting muscle, causing mitochondrial dysfunction. It must be noted, however, that exercise performance may be limited by substrate availability if the conditions are mild to warm and the exercise is prolonged. This is likely because even small rises in body core temperature will increase the use of finite glycogen stores (for review see [3]).

GLUCOSE AVAILABILITY AND REQUIREMENT DURING EXERCISE AND HEAT STRESS

Consistent with the increase in muscle glycogenolysis during exercise and heat stress, hepatic glucose production is also augmented in these circumstances. This increase in liver glucose output is not matched by a concomitant increase in glucose disposal, resulting in a relative hyperglycaemia when comparing exercise in the heat with that in a cooler environment (Fig.2) [11].

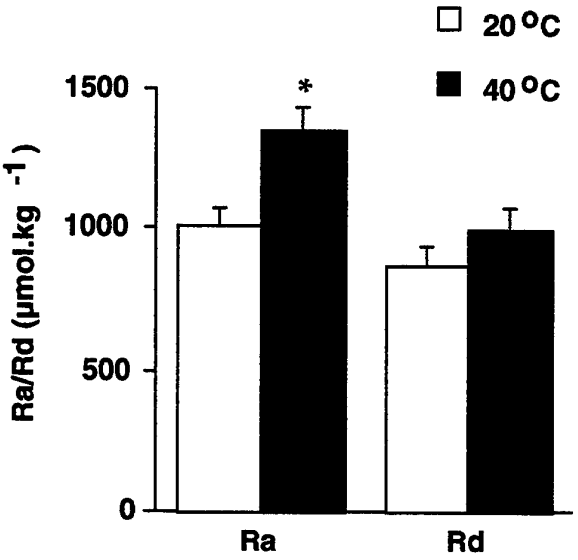

*Fig. 2. Total hepatic glucose production (HGP = R_a) and glucose uptake (R_d) during 40 min of exercise at 70% VO_{2peak} in different environmental conditions. Values are means ± SE (n=6). * denotes difference (P<0.05) from 20°C. Data from Hargreaves et al. (1996a)*

It is possible that this increase in glucose output is also mediated by the enhanced epinephrine which is observed during exercise and heat stress, since epinephrine plays a role in hepatic glucose production in exercising humans (Howlett et al., 1999). If exercise is performed in severe conditions, where it is likely that fatigue will be related to hyperthermia, carbohydrate ingestion does not appear to be warranted as blood glucose is not limiting. If, however, prolonged exercise is performed in warm to mild conditions, it would be wise to ingest carbohydrate since liver glycogenolysis will be augmented early during exercise.

SUMMARY

If exercise is performed in extremely hot environments, carbohydrate metabolism will be increased but exercise performance will be limited by factors associated with hyperthermia. In contrast, if the environmental temperature is slightly increased and/or the exercise intensity is moderate (ie. ultra endurance events), substrate availability may limit exercise performance.

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PAPER 3: EFFECT OF ENVIRONMENTAL TEMPERATURE ON STEADY-STATE AND MAXIMAL CYCLING

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INTRODUCTION

During exercise in warm and hot environments, dehydration and increased blood flow to the skin has the potential for reducing blood flow to the active muscles. Heart rate, skin temperature and cutaneous perfusion are increased during prolonged exercise in the heat, and there may be some effect of the hot environment on oxygen consumption ($\dot{V}O_2$), core temperature and blood lactate during prolonged exercise. The extent to which these occur in short duration exercises is equivocal. Steady-state $\dot{V}O_2$ has been reported as being increased [1, 2], decreased [3] or unchanged [4] in elevated ambient temperatures. Differing accounts of the influence of these temperatures on peak $\dot{V}O_2$ cite unchanged [5,6] or lowered [7,8] values. The above physiological changes during exercise in the heat may alter training thresholds. The influence of warm and hot environments on physiological indicators of the anaerobic threshold (AT) has not been extensively investigated. This investigation determined how $\dot{V}O_2$, heart rate, blood lactate and body temperature were affected during short duration steady-state and maximal cycling, and the implications for the AT measurement and application was determined.

MATERIALS AND METHODS

Six heat acclimatised subjects (25 ± 7 yr; 71.8 ± 4.4 kg; $\dot{V}O_{2peak}$ 56.8 ± 6.4 mL.kg^{-1}.min^{-1}) were recruited from Darwin triathlon and cycling clubs. A series of four incremental cycling tests were completed. Steady-state $\dot{V}O_2$ was measured during six consecutive five-minute bouts of cycling, beginning at 75 W (female) and 100 W (males) and increasing by 25 W each period. Peak $\dot{V}O_2$ was measured on separate occasions using a continuous incremental protocol to exhaustion (female 75 + 25 W.min^{-1}, males 60 + 30 W.min^{-1}). During the peak $\dot{V}O_2$ test AT was determined from the pulmonary ventilation curves ($\dot{V}_E$ and $\dot{V}_E/\dot{V}CO_2$). All tests were conducted in temperate (21.8 ± 0.5 °C; 52 ± 5 % humidity) and warm (29.6 ± 0.5 °C; 51 ± 9 % humidity) conditions representative of the environment under which the subjects train and compete.

Heart rate was recorded every five seconds with a heart rate monitor (Polar Vantage NV). Capillary blood samples were taken from a hyperaemised ear lobe pre and post exercise and a lactate analyser (YSI 2300 STAT PLUS) was used to determine the changes in concentrations of lactic acid. An electronic scale (AND) was used to measure changes in bodyweight. Core (T_c) and skin temperatures were monitored using a rectal probe and skin thermistors (YSI 400 Series), respectively. Mean skin temperature (T_{sk}) was calculated from a weighted mean of calf, forearm and chest skin temperatures.

Comparisons between the experimental treatments and between various points in time were made with repeated measures ANOVA. Tukey post-hoc comparisons were used in the event of statistically significant differences. Student's paired two-sample for means t-tests were used where appropriate for comparisons between experimental treatments.

RESULTS

The results for the physiological parameters measured for each condition are shown in Table 1. T_{sk} was higher in the warm conditions at rest and during all exercise tests. Resting core temperatures were similar for all tests, and the increases in T_{sk} and T_c during exercise was by approximately the same amount for each condition and in both the steady-state and maximal exercise tests. The mean differences between thermoneutral and hot conditions for sweat losses were 140 and 90 g for steady-state and maximal incremental exercise, respectively. During the hot condition, subjects drank on average an additional 100 and 20 g of water compared with the thermoneutral condition, for steady-state and maximal incremental exercise, respectively.

Steady-state and peak $\dot{V}O_2$ was not significantly different between temperate and warm conditions. There was also a significant difference between temperate and warm conditions for heart rates or blood lactate concentrations during steady-state or maximal cycling. The heart rate at AT threshold was the same for both temperate and warm conditions (169 ± 6 $b.min^{-1}$). This AT heart rate represented the same percentage of maximal heart rates recorded for the same condition (91 ± 2 %) and averaged the same power output (female 225 W, males 330 W). Despite the similarity of AT measures, there was a high degree of individual variability observed. The individual AT heart rates ranged from 6 beats higher in the warm condition to 9 beats lower.

		Steady state				$\dot{V}O_2$ peak	
		Rest	2nd	4th	6th	rest	maximum
Skin temp.#	temperate	32.2 ± 0.5	32.7 ± 1.0	33.6 ± 1.0	33.6 ± 1.2	32.4 ± 0.4	32.8 ± 1.2
(°C)	warm	34.1 ± 0.3	34.8 ± 0.5	34.7 ± 0.9	34.9 ± 0.9	33.9 ± 0.4	34.7 ± 0.6
Core temp.	temperate	37.4 ± 0.3	37.5 ± 0.3	37.8 ± 0.2	38.0 ± 0.2	37.4 ± 0.1	37.9 ± 0.2
(°C)	warm	37.3 ± 0.3	37.5 ± 0.3	37.7 ± 0.3	37.9 ± 0.3	37.4 ± 0.2	37.8 ± 0.3
$\dot{V}O_2$	temperate		1.73 ± 0.15	2.27 ± 0.19	2.86 ± 0.21		4.00 ± 0.61
($L.min^{-1}$)	warm		1.90 ± 0.13	2.43 ± 0.17	2.96 ± 0.17		3.94 ± 0.59
Heart rate	temperate	68 ± 5	115 ± 10	129 ± 12	146 ± 14	71 ± 12	183 ± 5
($b.min^{-1}$)	warm	69 ± 10	119 ± 13	139 ± 14	157 ± 14	64 ± 1	186 ± 5
La^-	temperate	0.7 ± 0.2	0.8 ± 0.2	1.1 ± 0.5	2.2 ± 1.3	0.8 ± 0.4	13.2 ± 4.2
($mmol.L^{-1}$)	warm	0.9 ± 0.4	0.8 ± 0.2	1.2 ± 0.4	2.4 ± 1.4	1.1 ± 0.5	14.5 ± 2.6

#Skin temperature = $(0.5 \times T_{manubrium}) + (0.14 \times T_{mid\ anterior\ forearm}) + (0.36 \times T_{mid\ posterior\ calf})$

Table 1. Physiological variables at rest and during steady-state (workloads 2, 4 and 6) and maximal exercise (mean ± s) for temperate (21.8 ± 0.5 °C; 52 ± 5 % humidity) and warm (29.6 ± 0.5 °C; 51 ± 9 % humidity) conditions.

DISCUSSION

Steady-state $\dot{V}O_2$ was not affected by increased environmental temperature in this study. This is consistent with an earlier report that found a significantly higher metabolic rate for men working at 37.7 compared with 29.4 and 21.2 °C but no difference in metabolic rate between the latter two temperatures [1]. These investigators concluded that there is a

temperature threshold at some point above 29.4°C at which an increase in metabolic rate that occurs. Other investigators who had reported differences for steady-state $\dot{V}O_2$ [2, 3] were above this threshold level. However, support for this temperature threshold effect on steady state $\dot{V}O_2$ is not universal, as Febbraio et al. [4] found no difference between 20 and 40 °C.

There is also support for a temperature threshold effect on $\dot{V}O_2$ peak test results. The observation that $\dot{V}O_2$ peak was not affected by increased environmental temperature is in agreement with that Rowell et al. [5] and Claremont [6]. Both Saltin et al. [7] and Sawka et al. [8] who tested above the aforementioned temperature threshold reported a 7% reduction in VO_2 peak in the heat when they compared 21 with 49 °C and 12 with 40 °C, respectively. Also, there was no decrease in AT in the heat in the present study, unlike the study of Dawson & Pyke [9] whose 35 °C hot condition could have been above this threshold.

Even though no difference was found for most of the physiological parameters measured during moderate heat stress, there was a high variation between individuals. Increased blood flow to the skin is a common response to exercise in the heat. However, the extent to which the heart rate increases to maintain blood supply to the active muscle varies between individuals. This is evident even in exercise of relatively short duration. Three of the six subjects in this study had differences of 5, 6 and 9 $b.min^{-1}$ in heart rate at AT. As maximum heart rates were similarly variable, the heart rates measured at the anaerobic threshold were at the same percentage of peak heart rates that were measured in the same test. Due to the high variability, and as absolute rather than relative heart rates are used as a marker of the AT for training in the heat, it is advisable that training thresholds be derived from tests in the heat. This may apply even if the athletes are heat acclimatised and the environmental temperature does not exceed 30 °C.

CONCLUSIONS

The results of this investigation indicated that $\dot{V}O_2$, heart rate and blood lactates were not significantly affected in heat acclimatised athletes during steady-state and maximal cycling in 30 °C. Although there was no significant difference between temperate and warm conditions for heart rate at the AT, individual variability was observed such that heart rates used as a marker of the AT for training in the heat, should be derived from tests in the heat.

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This study was approved by the Northern Territory University Human Ethics Committee.

INVITED LECTURE 3: HYDRATION EFFECTS ON THERMOREGULATION AND PERFORMANCE IN THE HEAT

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INTRODUCTION

Depending on the climatic conditions, the relative contributions of evaporative and dry (radiative and conductive) heat exchange to the total heat loss will vary [7]. The hotter the ambient temperature, the greater dependence on evaporative heat loss, and thus on sweating. Therefore, a substantial volume of body water may be lost via sweating to enable evaporative cooling to occur in the heat. Generally, individuals dehydrate during exercise because of fluid unavailability or a mismatch between thirst and body water requirements [5]. In these circumstances, the individual starts the exercise task in a euhydrated condition (normal total body water) but undergoes dehydration over a prolonged period of time.

FLUID & ELECTROLYTE NEEDS

A person's sweating rate is dependent upon the environmental conditions, clothing worn, exercise intensity and heat acclimatization state. Persons performing occupational activities in the heat often have sweating rates of 0.3 to 1.2 L/hour. Persons performing light intensity exercise while wearing protective clothing often have sweating rates of 1 to 2 L/hour. Likewise, athletes performing high intensity exercise in the heat commonly have sweating rates of 1.0 to 2.5 L/hour. Fluid requirements will vary in relation to ambient temperature, clothing worn, acclimatization state and physical activity levels. Daily fluid requirements might range (for sedentary to very active persons) from 2-4 L/day in temperate environments and from 4-12 L/day in hot environments.

Electrolytes, primarily sodium chloride and to a lesser extent potassium, are lost in sweat. Sweat sodium concentration averages ~ 40 mEq/L (range 10 - 100 mEq/L) and varies depending upon diet, sweating rate, hydration status and heat acclimatization state. Heat acclimatized persons have relatively low sodium concentrations (>50% reduction) in sweat. Sweat potassium concentration averages 5 mEq/L (range 3 - 15 mEq/L) , calcium averages 1 mEq/L (range 0.3 - 2 mEq/L) and magnesium averages 0.8 mEq/L (range 0.2 - 1.5 mEq/L. Electrolyte supplementation is not necessary, except for their first several days of heat exposure, as normal dietary sodium intake will cover the sweat losses.

During exercise-heat stress, a principal problem is to avoid dehydration by matching fluid consumption to sweat loss [3]. This is a difficult problem because thirst does not always provide a good indication of body water requirements. Thirst probably may not be perceived until an individual has incurred a water deficit of ~2% of body weight. Ad libitum water intake often results in incomplete water replacement or "voluntary" dehydration during exercise and/or heat exposure. Heat acclimatization status may also influence the "voluntary" dehydration incurred during exercise in the heat. Although heat acclimatization improves the relationship between thirst and body water needs, "voluntary" dehydration still occurs. Since thirst provides a poor indication of body water needs, many persons will dehydrate by 2% to 6% of their body weight during situations of prolonged sweating.

EXERCISE PERFORMANCE & THERMOREGULATION

In temperate climates, a body water deficit of less than 3% body water loss (BWL) does not alter maximal aerobic power [5]: maximal aerobic power decreased when

hypohydration (decreased total body water) equaled or exceeded 3% BWL. Therefore, a critical water deficit (3% BWL) might exist before hypohydration reduces maximal aerobic power in temperate climates. In hot climates, small (2% BWL) to moderate (4% BWL) water deficits result in large reductions of maximal aerobic power. Physical work capacity is decreased by marginal (1% - 2% BWL) water deficits; the reduction in work capacity is larger with increasing water deficit. Clearly, hypohydration results in larger decrements of physical work capacity in hot as compared to temperate climates.

Hypohydration will increase core temperature during exercise in temperate and hot ambient conditions [4]. As the magnitude of water deficit increases, there is a concomitant elevation of core temperature during exercise heat stress. The magnitude of core temperature elevation ranges from 0.10 to 0.23°C for every percent body weight lost, and this elevation is greater during exercise in hot than in temperate ambient conditions. Hypohydration not only elevates core temperature responses, but it negates the core temperature advantages conferred by high aerobic fitness and heat acclimatization.

Hypohydration impairs both dry and evaporative heat loss by delaying sweating onset and skin vasodilatation as well as sweating sensitivity [4]. Hypohydration is also associated with either reduced or unchanged sweating rates at a given metabolic rate in the heat. The physiological mechanisms mediating the reduced dry and evaporative heat loss from hypohydration include both the separate and combined effects of plasma hyperosmolality and reduced blood volume [4].

During hypohydration, a decreased blood volume reduces central venous pressure and cardiac filling, which reduces stroke volume and requires a compensatory increase of heart rate. The combination of exercise with heat strain results in competition between the central and peripheral circulation for a limited blood volume. As body temperature increases during exercise, cutaneous vasodilatation occurs, and the superficial veins become more compliant, thus decreasing venous resistance and pressure. As a result of decreased blood volume and blood displacement to cutaneous vascular beds, central venous pressure, venous return and thus cardiac output will decrease below euhydration values.

Hyperhydration, or greater than normal body water, has been suggested to improve, above euhydration levels, thermoregulation and exercise-heat performance [6]. The concept that hyperhydration might be beneficial for exercise performance arose from the adverse consequences of hypohydration. It was theorized that an increase in body water might reduce cardiovascular and thermal strain of exercise by expanding blood volume and reducing blood tonicity, thereby improving exercise performance. Studies have evaluated hyperhydration effects on thermoregulation in the heat. Some studies observe smaller core temperature increases during exercise with hyperhydration, however, most of those studies suffer from confounding experimental procedures [6]. Recent research from our laboratory has controlled for these confounding factors, and we have observed no thermal advantage with either water hyperhydration or glycerol hyperhydration during exercise-heat stress [1,2].

CONCLUSIONS

During exercise in the heat, sweating rate increases to defend body temperature. It is important that persons rapidly replace their sweat losses while performing exercise in the heat. If sweat losses exceed fluid replacement then a body water deficit, or hypohydration, will occur. Hypohydration reduces exercise performance and increases heat strain. The magnitude of adverse effects increase with the level of fluid deficit. Hypohydration negates the thermoregulatory benefits conferred by high aerobic fitness and heat acclimatization. The over-consumption of fluids to increase total body water, or

hyperhydration, provides no thermoregulatory benefits beyond those obtained from euhydration.

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PAPER 4: THE EFFECT OF GLYCEROL HYPERHYDRATION ON OLYMPIC DISTANCE TRIATHLON PERFORMANCE IN HIGH THERMAL STRESS.

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INTRODUCTION

Prolonged, high intensity exercise in a heat-stressful environment poses considerable threat to the health and physical performance of endurance athletes. Exercise in these conditions is associated with increased body temperature, increased sweat rates, reduced total body water content and decreased performance [1, 2]. Recently, glycerol has been suggested as an effective hyperhydrating agent producing minimal side effects [3, 4]. However little research to date has examined the effects of glycerol loading on long duration (> 1 hour), high intensity endurance performance events such as the ODT in heat-stressful environmental conditions [5, 6]. The purpose of this study was to examine the effect of prior glycerol loading on Olympic distance triathlon performance (ODT) in heat-stressful conditions.

METHODS

Subjects

Ten well-trained and acclimatised triathletes (7 male, 3 female) volunteered to be subjects in the study. The subject's mean age was 33.3 ± 6.7 yrs; body mass 70.9 ± 9.7 kg; $\dot{V}O_{2max}$ 58.4 ± 7.7 mL•kg^{-1}•min^{-1}; sum of nine skinfolds 84.3 ± 31.7 mm; and best ODT time 2:11:30 ± 0:13:20 (hr:min:sec).

Experimental Design

Subjects were required to complete two ODT's in uncontrolled environmental conditions on two separate occasions. The study was randomly assigned (placebo / glycerol) and double blind conducted two weeks apart. Participants were advised to abstain from strenuous physical activity and standardise their diets for 48 hours prior to each ODT.

Glycerol and Placebo Concentrations

The glycerol solution consisted of 1.2g of glycerol per kilogram (kg) of body mass (BM), combined with 25 mL of half-strength Gatorade® (4% carbohydrate) per kg of BM (Hitchins et al., 1996). The placebo solution consisted of identical volumes of half-strength Gatorade® and water with the addition of saccharine (Equal*, The NutraSweet Company, Australia).

Performance Measures

Time (hr:min:sec) for completion of the ODT and each ODT leg (swim, bicycle and run) were the primary measures of performance.

Blood Parameters

Standing venous blood samples (5 mL) were taken from the antecubital vein by trained phlebotomists immediately before and after the loading period and at completion of each ODT and analysed for haematocrit (Hct) and haemoglobin (Hb) in order to calculate blood plasma volume changes.

Statistical Analysis

Paired t-tests were used to examine the effect of day (hot / warm) on performance and the measured variables. Subsequent statistical analysis involved independent t-tests between

the effects of treatment (ie. glycerol/placebo) on each testing day (ie. hot/warm) for each variable measured. Furthermore, the amount of change for each variable measured in each condition (ie. placebo/glycerol) between days (hot/warm) was analysed using independent t-tests according to the method of Shultz (1989). Statistical significance for each analysis was accepted at an alpha level of 0.05.

RESULTS

Table 1 shows environmental conditions were different on testing days.

	WBGT Out	Globe	Dry	Wet
Day One	30.5 ± 1.3*	45.7 ± 4.2*	45.6 ± 1.2*	25.9 ± 0.8*
Day Two	25.4 ± 0.8	33.1 ± 2.0	30.1 ± 0.3	1

* Significant difference between days ($p < 0.05$).

Table 1. Environmental conditions on testing days (°C) (mean ± SD)

Table 2 shows results from both glycerol (G) and placebo (P) conditions on both the hot and warm testing days.

	Treatment	Day One (hot)	Day Two (warm)	Mean
ODT Time	P	2:28:09±0:11:29*	2:16:24±0:08:54	2:22:14±0:11:34
(hr:min:sec)	G	2:20:14±0:08:49*	2:18:24±0:10:34	2:19:22±0:09:11
	Mean	2:24:09±0:10:33*	2:17:26±0:09:14	
Run Time	P	0:52:00±0:06:52*	0:45:44±0:03:41	0:48:50±0:06:11
(hr:min:sec)	G	0:46:50±0:05:39*	0:45:18±0:06:08	0:46:00±0:05:53
	Mean	0:49:23±0:06:34*	0:45:27±0:05:04	
Loading Blood Plasma Volume Change (%)	P	2.49±2.35	2.97±4.26	2.74±3.26
	G	7.50±2.65§	5.75±3.61	6.62±3.13
	Mean	4.99±3.54	4.73±4.00	
ODT Plasma Volume Change (%)	P	-7.20±5.55	-5.61±3.64	-6.40±4.50
	G	-9.45±5.57	-7.07±6.44	-8.26±5.82
	Mean	-8.33±5.38	-6.33±4.99	
Fluid Retention (mL)	P	690±233	590±178	640±202
	G	920±76#	1080 ± 424#	1000±299#
	Mean	805±203	835±401	
Urine Output	P	986±385	1263±283	1124±350
(mL)	G	611±176#	859±80#	735±184#
	Mean	798±345	1061±289	

* Significant difference between days ($p<0.05$);
§ Significant difference to placebo group ($p<0.05$);
\# Significant difference between conditions

Table 2: Experimental results for glycerol and placebo conditions on both testing days (mean ± SD).

DISCUSSION

This study investigated the influence of glycerol loading on ODT performance in heat-stressful conditions. The present results demonstrated that in comparison to the placebo

condition, glycerol hyperhydration attenuated the decrease in performance in participants between the warm and hot days. Furthermore, the present data suggest that this ergogenic benefit appears to be related to an increase in blood plasma volume during the loading period.

ODT performance time was faster in the placebo (8.6%) and glycerol groups (1.3%) on the warm day when compared to the hot day. The decreased performance (4.6%) on the hot day (30.5 ± 1.3°C $WBGT_{out}$) was most likely due to increased sweat losses and greater thermal load.

Glycerol hyperhydration appeared to offer greater ergogenic benefits during day one, when environmental conditions were hotter than during day two (day one: 30.5 ± 1.3 $WBGT_{out}$; day two: 25.4 ± 0.8 $WBGT_{out}$). This was evidenced by a reduced decrement in performance time on the hot day in comparison to the warm day. The ODT performance times were 5.7% faster in the glycerol group during the hot day and 1.5% slower in the glycerol group on the warm day. This observation suggests that glycerol hyperhydration offers a physiological defence against the detrimental effects of dehydration in heat-stressful environmental conditions. This finding is in agreement with previous research investigating the ergogenic effects of glycerol hyperhydration on strenuous and prolonged physical activity in heat-stressful conditions [5, 6]. For example, Anderson and colleagues (1999) found that performance increased by 5% in six trained men during 90 mins of steady state cycling at 98±10% of lactate threshold in hot conditions (35°C).

The current investigation confirmed that glycerol hyperhydration enabled significantly greater fluid retention following the loading period compared to the placebo solution (1000 mL vs 640 mL) in both hot and warm conditions and is in agreement with previous investigations [5-7] that have observed a reduced urine output during the loading period. On average, the reduction in diuresis with glycerol ingestion contributed an additional 389 mL to total body water content in comparison to the placebo group. The current data is supported by previous research reporting that glycerol loading may promote fluid retention through reduced diuresis [3, 5, 6, 8, 9].

The present data revealed that acute blood plasma volume expansion was significantly greater in the glycerol group compared to the placebo group on the hot testing day. However, the results from the warm testing day demonstrated that despite a 21.2% increase in plasma volume in comparison to the placebo group, the pre-exercise glycerol ingestion did not increase plasma volume significantly compared to the placebo. This finding is in agreement with previous research [10] that observed a non-significant increase of blood plasma volume with glycerol hyperhydration in moderate to hot conditions (21 – 33°C; 25 –75 %RH).

The purpose of the current study was to investigate the effect of glycerol ingestion on ODT performance in heat-stressful environmental conditions. While it is acknowledged that both the method of plasma determination and the low subject numbers of the present study are limitations, results suggests that glycerol hyperhydration prior to ODT in such conditions may improve endurance performance. Importantly, the majority of the ODT performance improvement occurred during the 10-kilometre run leg on the hot testing day. The mechanisms responsible for the increased performance in the current investigation appear to be an improved hydration status secondary to a reduced diuresis and increased fluid retention with glycerol loading compared to the placebo solution. These findings suggest that glycerol-hyperhydration may offer ergogenic benefits when used prior to prolonged, strenuous exercise in heat-stressful conditions.

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PAPER 5: THE MAIN FUNCTION OF THERMOREGULATION AND THE SUBJECT OF TEMPERATURE CONTROL

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INTRODUCTION

It is common practice to study the mechanisms of thermoregulation during strong temperature actions on an organism, though the power of physiological thermoregulation is inadequate for maintaining the normal body temperature under such conditions. To protect themselves against strong, prolonged, or constant effects of the environmental temperature the animals use biological protective means: fur, feathers, fat, shelters, behavior. A man uses various techniques in addition to clothes and dwellings. Consequently, under adequate living conditions men and animals are always within the limits of the thermoneutral zone (TNZ). However the changes in the physiological state (for example, sleep - awake) or various kinds of physical activity are accompanied by the changes in the heat production. Thus, for example, when a man passes from a quiet "lying" position to a quiet "standing" position, his heat production increases almost twice. Taking food, changing the pose, various emotions - change the heat production to some extent or another. In this case quick and precise thermoregulation reactions are necessary to maintain the feeling of temperature comfort in a man or the optimal heat state in animals even within the limits of TNZ. This article is devoted to a short description of the temperature sensitivity and of the mechanisms of the activity of the physiological thermoregulation system in men and various kinds of animals, and also of the mechanisms of the initiation and regulation of powerful physiological reactions of thermoregulation under extremal temperature conditions. These studies have been carried out in the department of thermoregulation and bioenergetics of I.P. Pavlov Institute of Physiology for the last 15-20 years.

MATERIALS AND METHODS

The following methods were used in this work: the direct general and partial calorimetry, the measurements of the heat production by the oxygen consumption; the introduction of a dosed amount of heat into a man and animal body (the animals - via chronically implanted soft thermodes, the men - by drinking a certain volume of warm water), precise measurements of the temperature in various parts of the central nervous system, in various inner organs and tissues, in the blood of various vessels (without surgical intervention at the point of measurements), in various layers of the skin in man and animals (0.1 - 0.5 - 1.5 mm from the skin surface). Moreover the biopotentials were registered in animals from the neurons of various hypothalamus nuclei in acute and chronic experiments; the biopotentials of separate skin thermoreceptors were also registered. Some investigations were performed on specially designed thermophysical models of the rabbit and rat.

RESULTS AND DISCUSSION

It is generally agreed that occasional decreases or increases in the body temperature within the limits of TNZ can return to the initial value automatically, owing to a passive heat exchange of an organism with the environment. We checked this point of view with the help of quantitative investigations on a thermophysical model of a rabbit body, which we designed. The model represented a physical body, having approximately the same mass (≈3000 g), heat capacity (≈ 0.83 cal / g · °C) and heat conductivity, relative surface of the body, heat insulation (≈ 3 clo) of the body, power of heat production at a relative rest (7 W), intensity of heat and internal mass transfer (aided by moving liquid; circulation) as a real animal. When the temperature of the air in the room was 25-27°C (TNZ for a rabbit)

the temperature inside the model was set at the level of 38-39°C. Very rapid increases in temperature inside the model body (ie. 0.2-0.3°C within 1-1.5 min) were shown to return to the starting value during 6-7 hours by passive heat exchange. We showed that, when an animal is under the conditions of TNZ, the temperature sensitivity of its thermoregulation system increases abruptly. The smallest increases in the heat production (on changing the pose, the muscle tone, during an orienting reaction, masticatory muscle contraction) result in an increase in the hypothalamus temperature of a rabbit by 0.10-0.15°C. In answer to this small temperature change a reaction of ear vessel dilatation occurs immediately, so that the temperature of the hypothalamus (contrary to the thermophysical model) returns to the starting value in 10-20 min. An increased sensitivity of the thermoregulation system in animals under the TNZ conditions results in continuous fluctuations of the temperature of the blood in the arteries and hypothalamus with an amplitude of 0.1-0.2°C and a period of 10-20 min. These fluctuations are synchronous with the fluctuations of the ear vessel tone and of the ear temperature with an amplitude of several °C. In a man in TNZ at a complete rest the temperature of the tympanic membrane fluctuates constantly with the amplitude from 0.05 to 0.15°C and a period of 20-30 min. Continuous synchronous fluctuations of the tone of the hand skin vessels and of the skin temperature with an amplitude of 2-4°C occur respectively (hands are the most important heat exchange organ in a man in TNZ). These phenomena reflect a continuous operation of the thermoregulation system in TNZ, which ensures a precise and continuous compatibility between the heat production and the heat loss of a body. This ensures also a feeling of temperature comfort for a man and an optimal heat state for animals. Therein, as we think, lies the main function of the thermoregulation system, which resulted from biological evolution [1, 8].

The counteraction to abrupt temperature actions on an organism under extremal conditions is only an accessory function of the physiological thermoregulation system. When protecting against overcooling, for example, the thermoregulation system of a rat, rabbit, or man is really able to increase the heat production by a factor of 2-3 compared to the standard (basal) metabolism. According to precise measurements, which were carried out on our biophysical models, to preserve a normal body temperature in water with the temperature of 4-6°C a rat, for example, must increase its heat production by a factor of 40, and a rabbit, by 11-12. The physiological mechanisms of the protection against overheating cannot ensure the maintenance of a normal body temperature of a homoiothermal organism in a hot environment also for a long time, if the physiological thermoregulation is not supplemented with physiological protective methods (behavior).

The accuracy, sensitivity, and efficiency of the thermoregulation system depend on "the subject" of regulation. The search for this subject is an old and "painful" problem for physiological thermoregulation. Many researchers suggest that the subject of thermoregulation is the "mean" body temperature. Corresponding literature can be found in the recent reviews [2, 3, 4]. The simplest calculations show that the regulation of the temperature homeostasis of a living organism by its mean body temperature occurs much more quickly, accurately, and efficiently than by the temperature in any one site of the body (by the hypothalamus temperature, for example). However, nobody had given an explanation yet of how the thermoregulation system can measure the mean body temperature. Theoretically such a mechanism is very difficult to imagine, since measuring the mean body temperature requires a multitude of highly sensitive thermoreceptors distributed over the whole body mass. However, in a homoiothermal organism there are highly sensitive thermosensors only in peripheral tissues (skin) and in the central nervous system. Can the mean body temperature be determined with sufficient accuracy from only central and peripheral thermosensors? (the blood temperature in the right heart does not reflect with any accuracy the mean temperature of the whole body of animals and man!).

After years of studying this problem we were able to find the following:

1) only 60-65% of the cold skin thermoreceptors are located in the surface skin layers immediately under the epidermis, 35-40% of the thermoreceptors are located in the deep skin layers as far as the interface with the subcutaneous fat [5].

2) there is a nervous apparatus in the thermoregulation center which compares the intensity of the signals from the surface and deep skin thermoreceptors [6, 7], which (theoretically) allows the temperature gradient in the skin and the intensity of the heat flow ($W/cm^2 \cdot min$) through the skin to be determined [1]. The fluctuations of the intensity of the heat flow reflect the value of the fluctuation of the mean body temperature.

3) The reaction to a dosed introduction of heat into an animal or a man's body (from 40 to 300 cal/kg) the changes in the temperature gradients in the skin from the subcutaneous fat to the epidermis after the introduction of heat (actually from 0.05 to 1.0°C/mm) and in the heat flows through the skin support the suggestion that the thermoregulation in TNZ in men and animals is effected by the fluctuations of the mean body temperature (which corresponds to the fluctuation of the heat content of a body). Such a mechanism can increase the quickness and the accuracy of the thermoregulation within the limits of TNZ. Beyond the TNZ limits, when the external temperature actions exceed the possibilities of biological protection of an organism, the power of the thermoregulation reactions is proportional to the value of the temperature changes in the peripheral and central thermosensors. The intensity of the reactions decreases as the thermosensors lose their temperature sensitivity. Cold skin thermoreceptors decrease or lose their sensitivity at the skin temperature 5-10°C. The thermoregulation center ceases to function in a man at the brain temperature ≈31-32°C and in a rat at the brain temperature ≈19-20°C.

CONCLUSION

Literature [eg. 2, 3, 4, 1, 8] and our long-standing investigations suggest that the main function of physiological thermoregulation is to ensure thermal comfort during various kinds of physical activity within the limits of TNZ. The experiments suggest that the subject of thermoregulation in TNZ is the fluctuations of the mean body temperature, which ensures a high sensitivity, quickness, and efficiency of thermoregulation under these conditions. Under extremal conditions the power of thermoregulation reactions is proportional to the intensity of the signals from the thermosensors up to the moment of their decay or loss of temperature sensitivity. If these concepts are taken as the basis, the technical protection of a man from abrupt external temperature actions must be designed in such a manner that a precise compatibility between the heat production and the heat loss could, in the end, be set and regulated by the thermoregulation system, which ensures a constant temperature comfort and a high working capacity.

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All the experiments on animals and observations on men, carried out for the last 15 years, had the permission for publishing.

INVITED LECTURE 4: SWEATING IN EXTREME ENVIRONMENTS: HEAT LOSS, HEAT ADAPTATION, BODY-FLUID DISTRIBUTION AND THERMAL STRAIN.

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INTRODUCTION

Evaporation is an extremely powerful cooling process. When totally evaporated from the skin surface, sweat can remove body heat at a rate of 2.43 $kJ \cdot g^{-1}$. Humans therefore control sweat secretion to maintain thermal homeostasis. Since humans are capable of extended sweat rates approximating 30 $g \cdot min^{-1}$, it is possible to remove heat at rates ~73 $kJ \cdot min^{-1}$. Assuming a 20% efficiency, such heat loss will support a normothermic total energy use of 1520W. This equates with an external work rate of 304W, eliciting an oxygen consumption >3.5 $l \cdot min^{-1}$. However, while man has a great capacity to both work and dissipate metabolically-derived heat, exercise under various environmental extremes may impede heat dissipation. Under such conditions, the cumulative effects of metabolic and environmental thermal loads may represent an uncompensable heat stress, predisposing to hyperthermia.

THE PHYSIOLOGICAL SIGNIFICANCE OF SWEAT

Evaporative cooling in terrestrial beings is perpetual, occurring from the respiratory tract with every breath, and via water-permeable membranes. Resting normothermic man, within a cool-temperate environment, evaporates 30-33 $g \cdot h^{-1}$ from each surface, with the corresponding cooling effect accounting for the dissipation of about 25% of the resting metabolic heat production. This occurs without our awareness: insensible evaporation. When faced with an external heat load, thermosensitive cells within the skin, and eventually those within deeper tissues, communicate this altered thermal status to the hypothalamus for interpretation. Hypothalamic integration of thermal messages results in the generation of a `load error' message, to which a proportional sympathetic response is elicited: eccrine sweating.

Apart from man, a number of species possess the ability to sweat actively in response to thermal stress. In man, considerable inter-individual differences are apparent for sweat gland densities and secretion rates [1, 2]. Indeed, men generally sweat more than women [3], and even racial differences exist [4]. While euhydrated people can sustain insensible losses indefinitely, dehydration will occur during active sweating, if water replacement is not elevated proportionately. For instance, daily sweat rates can increase from 300-400 ml, to 10-15 litres during prolonged heat and exercise exposure. Extended-duration sweating of 1.5-1.8 $l \cdot h^{-1}$ (30 $g \cdot min^{-1}$) is commonly observed and, under severe heat stress, glands can secrete up to 3-4 $l \cdot h^{-1}$.

It has been estimated that we have between 1.6-4.0 million sweat glands [5], with considerable variability in gland density between regions. Each gland consists of a secretory coil, connected to the skin surface. As sweat moves through the duct, sodium, chloride and bicarbonate ions are reabsorbed [6]. Sweating typically starts by recruiting groups of glands innervated by the same sympathetic nerve. At rest, sweating generally starts at the extremities, moving towards the head as thermal strain increases [7]. However, we have shown that, with the exception of an earlier lower torso sweat onset, between-site sweat recruitment is generally uniform during upright exercise [8]. During sustained sweating, sweat is secreted across the body surface in a cyclic pattern, reflecting the rhythm of sympathetic activity. As thermal strain rises, the frequency of glandular stimulation is elevated. During the phase conversion from liquid to gas, the water

molecules do not themselves change temperature, but simply absorb thermal energy to drive evaporation. With the evaporation of 1 g of sweat, 2.43 kJ of heat is removed.

FACTORS WHICH AFFECT SWEAT EVAPORATION

The evaporation of sweat is influenced by the water vapour pressure of the surrounding air, but is mainly determined by that of the microclimate above the skin, with water passing down the vapour pressure gradient. Thus, any factor that affects this gradient will impact upon evaporative cooling. Six such factors will be covered within this presentation.

Immediately above the skin is a very thin layer of air, which behaves as though it was trapped in permanent contact with the skin: the boundary layer. For evaporation to occur, the water vapour pressure of the boundary layer must be less than that of the skin surface. Since both the size and composition of the boundary layer are an inverse function of air velocity (relative or absolute), both environmental water vapour pressure (reflected within relative humidity) and wind speed can modify evaporative cooling. The single greatest impact upon the boundary layer is brought about by the use of clothing. While some clothing ensembles allow air to pass through the fabric and apertures, less permeable garments trap air. In such ensembles, locomotion largely determines garment ventilation [9], and trapped air water vapour pressure.

The above factors represent physical changes. Evaporative cooling also depends upon physiological variations. Continuous sweating, at high flows, leads to sweat accumulation, and eventually sweat suppression (hidromeiosis: [10]). This is generally attributed to water-induced swelling of subcutaneous tissues, leading to pore blockage. Hydration status affects heat loss by reducing the core temperature threshold for sweating onset, the sensitivity of the sweat response to such changes, and local sweat rates [11]. On the other hand, adaptation to both endurance training and heat have been shown to increase sweat rates [12].

SWEATING AND BODY-FLUID COMPARTMENTS

The human body is about 60% water (500-600 $ml.kg^{-1}$: female-male), which, while stored within various compartments, is free to move between these sites. Water, the primary substrate for sweat, is drawn from this reservoir. The total volume of water stored is a function of hydration state [11], body composition [13], and endurance exercise adaptation [14]. For instance, a low adiposity is associated with the storage of a larger water volume relative to body mass, due to the higher water content of lean tissue relative to fat [13]. During exercise, we have shown that fluid losses are drawn almost equally from the intracellular and extracellular compartments [15]. The plasma volume forms a sub-division of the extracellular volume, and is generally defended in cool and temperate, but not within hot environments [15]. If not replaced, fluid losses result in progressive dehydration, impaired physiological function and dysthermia.

HEAT ADAPTATION

It was believed that heat adaptation (acclimation) might influence the regional distribution of sweating, favouring greater limb sweating. Past experiments from our laboratory have demonstrated that, while adaptation enhances sweating by elevating sweat rate and lowering its onset threshold [12, 16], it was not associated with a sweat redistribution [16]. For any site, the post-adaptation sweat responses appeared more closely related to differences in sweat gland density, than to altered control of the sweat glands.

Heat adaptation is known to affect body-fluid volumes. Typically, post-adaptation elevations in body fluid, including the plasma compartment, are observed [17]. However, until recently, it was accepted that the plasma lost during combined heat and exercise stress would be diminished following heat adaptation. We now know this to be somewhat

imprecise. Instead, the post-adaptation plasma volume increase is also associated with a greater post-adaptation plasma loss [17]. We have observed that plasma losses, during heat and exercise stress, increase from 16% prior, to 22% following, extended heat adaptation [17]. Thus, the plasma volume was not preferentially defended. Instead, the heightened sweat losses and elevated resting plasma volume, resulted in an elevated plasma contribution to fluid losses following heat adaptation.

CONCLUSION

We have seen that, while the evaporation of sweat serves a very powerful cooling function, it may be impeded by changes in the boundary layer air and hydration state. On the other hand, endurance training and heat adaptation enhance sweat secretion. However, such changes need not necessarily be associated with greater evaporative cooling, which remains dependent upon the water vapour pressure gradient between the skin and the boundary layer air.

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PAPER 6: TO WHAT EXTENT DOES THERMAL SENSATION REFLECT PHYSIOLOGICAL HEAT STRAIN

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INTRODUCTION

The purpose of this study was to compare the effects of different environmental temperatures on the physiological strain index (PSI, [Moran et al., 1998]) and on thermal sensation (TS) on a group of subjects. The PSI is an index based on rectal temperature (T_{re}) and heart rate (HR) and indicates heat strain (on a scale of 0-10) online. PSI is calculated as follows: $PSI = [5(T_{re}t - T_{re}0) \bullet (39.5 - T_{re}0)^{-1}] + [5(HRt - HR0) \bullet (180 - HR0)^{-1}]$, where T_{re} and HR are measured at any time (t) during the exposure. The TS scale [modified from Gagge et al., 1967] is also measured from 0-10, and subjects are asked to concentrate on their internal thermal sensation.

MATERIALS AND METHODS

Fifteen students (8 male and 7 female) with a mean (sd) age of 25.0 (3.6) y from the University of Wales, Bangor, who volunteered for this study, completed a health assessment questionnaire and gave informed consent. Any who had a recent history of illness or who were taking any form of medication, were excluded from the experiment. The volunteers were made aware that they could withdraw from the experiment at any time.

Subjects' peak heart rate was established during a preliminary laboratory visit. All subjects exercised on a treadmill, at a constant speed corresponding to 65% of their maximum heart rate reserve, on three occasions; each at a different temperature (20°C, 30°C, 40°C) and 55% relative humidity (RH) and zero air velocity, for 45 minutes maximum. Subjects, wearing sports kit (shorts, vest and training shoes) were encouraged to continue exercising for the full time period, but exercise was terminated if HR exceeded 180 beats·min^{-1} or T_{re} exceeded 39.5^{0}C. To avoid habituation or acclimation, the order of testing was randomized but the timing of testing was standardized with a seven-day interval between tests. Throughout exercise, T_{re}, HR, skin temperature and TS were recorded every three minutes. T_{re} was monitored for a further 15 minutes after exercise for safety reasons. All subjects completed 21 min of exercise with some completing the 45-min period. However, complete data are presented here for 21 min of exercise.

Statistical analysis comprised four, two-factor (time • temp) ANOVA's with repeated measures, for each variable (HR, T_{re}, TS, and PSI). Bland and Altman [1986] calculations (a procedure for assessing agreement between two methods of measurement) were performed on TS and PSI data.

RESULTS AND DISCUSSION

Significant main effects were found for HR, PSI and TS across time ($p<0.05$) and across temperature ($p<0.05$). There was a significant main effects across time ($p<0.05$) for T_{re}, but T_{re} was not significantly different between environmental temperatures ($p>0.05$). There was a significant time by temperature interaction for PSI ($p<0.01$). Data treated by the Bland and Altman procedure are displayed in Figure 1 and shown in Table 1.

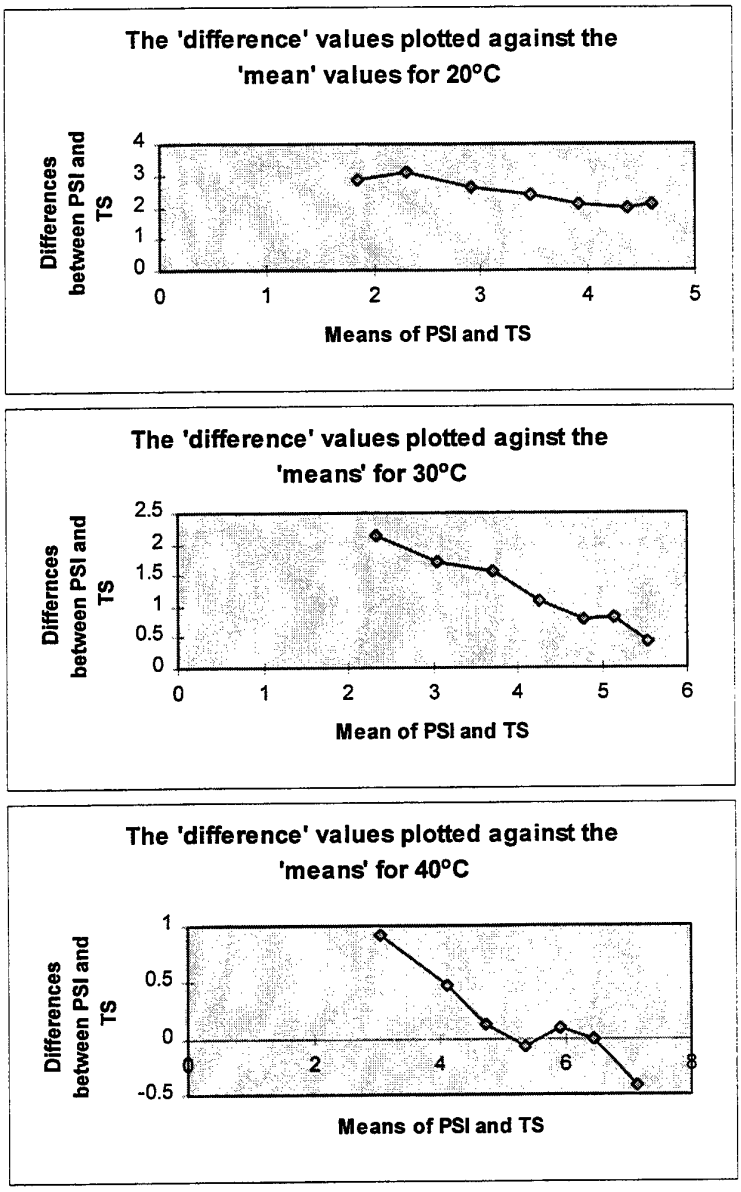

Figure 1. Mean and difference values between PSI and TS illustrated at 20 ^{0}C, 30^{0}C, and 40^{0}C . Data points are at 3 min intervals. Difference scores which approach zero indicates a closer agreement between variable measures.

The magnitude of mean values of TS and PSI increase with temperature (Figure 1); the last data point (21 min) for 20^{0}C was 4.75, for 30^{0}C was 5.65 and for 40^{0}C was 7.25. The magnitude of the difference between PSI and TS reduces progressively across time at each temperature. However, between temperature conditions the magnitude of the difference is greatest at 20^{0}C and reaches zero at 12 min during exercise at 40^{0}C. A zero value suggests that measurement on both variables (PSI and TS) is coincident.

There was a high correlation between the variables PSI and TS across the three temperatures (Table 1). The estimate of error between these variables was lowest (0.36) for the highest temperature (40^{0}C) suggesting that TS values more closely reflect those obtained from physiological heat strain at the higher temperature, as indicated from PSI.

Temperature	Relative Bias	Estimate of error	Correlation
20°C	2.45	0.41	0.94
30°C	1.22	0.56	0.99
40°C	0.16	0.36	0.93

Table 1: Results of the Bland and Altman Calculations. Relative bias represents the mean of the differences between TS and PSI and their standard deviations is the estimate of error. Lower values for relative bias and estimate of error with an increase in temperature suggest closer agreement between *the measures of TS and PSI.*

CONCLUSIONS

The study has shown, that when subjects exercise at either 20°C, 30°C or 40°C for a period of time, their level of heat strain continues to increase. Our results also suggest that subjects are better able to predict their heat strain, (on a TS scale 0-10), when exercising in an environment at 40°C (55% RH) more accurately than at either 30°C or 20°C. However, after 12 min of exercise at the higher temperature subjects tended to over-estimate TS progressively as heat strain increases. Further investigation is required to examine the robustness of temperature sensation as a measure of heat strain under a wider range of environmental conditions with subjects wearing heat protective ensembles.

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The Ethical Committee from the School of Sport Health and Exercise Sciences, University of Wales , Bangor, UK, approved this study

PAPER 7: THERMAL SWEATING FOLLOWING SPINAL CORD INJURY.

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INTRODUCTION

A complete spinal cord injury prevents neural connections between distal sites and higher neural structures. While it has previously been demonstrated that an isolated spinal cord can elicit non-thermal sweating independently of the hypothalamus [1-3], the ability of the spinal cord to control sweating in response to thermal stimuli, without hypothalamic influence, is less clear. The majority of early literature indicates that thermal sweating is absent below a complete spinal cord injury (SCI) [4-7], yet several studies suggest otherwise [8-11]. However, invasive measures have failed to observe altered sympathetic activity when thermally stimulating insensate regions [12], which is inconsistent with the observations of sweating below a SCI.

There are two main limitations within many of the above studies. Firstly, for the spinal cord to be deemed to have initiated sweating independently of the hypothalamus, it must be confirmed that neural connections are absent. Since most clinical verifications of SCI completeness do not evaluate autonomic completeness, and since sweat glands are sympathetically innervated, it is imperative to evaluate the integrity of autonomic function. In several cases, where sweating was observed below a SCI [9-11], autonomic completeness was not reported, and presumably not tested. Indeed, it has been demonstrated that complete somatosensory separation can exist while residual autonomic function remains [1, 13]. Hence, we are unable to determine whether sweating below a SCI is the result of surviving or regenerated neural connections, or whether it is spinally mediated. In those studies reporting sweating below a SCI, sweat rate is substantially reduced [8-10]. Such a pattern may occur in the presence of a massive, but incomplete disruption of the neural pathways. Thus, such sweating may have been initiated by the hypothalamus.

Secondly, if sweating is present below a confirmed complete SCI, it must be confirmed whether or not its origin is thermal, or resulting from other afferent feedback (*e.g.* pain or localised pressure). Autonomic dysreflexia and muscle spasms are the major cause of non-thermal sweating in spinal patients. Such sweating, both above and below the SCI, is a common clinical observation [1-3]. However, since sweat rate is directly affected by local temperature [14], non-thermal sweating may be similarly altered, despite being initiated by non-thermal factors. Therefore, the aim of the current project was to investigate the possible existence of thermally-induced sudomotor control in subjects with a complete SCI, using measures of sweat rate ($\dot{m}_{sw}$), and sweat expulsion frequency (f_{sw}).

MATERIALS AND METHODS

Eight subjects with clinically complete SCI (C5-L1) and 10 non-injured controls were studied. Clinical examination, performed by an experienced medical practitioner, verified somatosensory completeness. Since several studies have shown residual sensory and motor innervation despite clinically-verified complete SCI [13, 15-17], additional verification was achieved by clamping mean body temperature above the sweat threshold, while the leg blood flow was occluded and the leg was cooled with 7.9°C water. A concomitant decrease in forehead $\dot{m}_{sw}$ in SCI subjects was interpreted to have resulted from intact sensory

connections. Such subjects were classified as physiologically incomplete SCI, and are not reported herein.

Subjects rested supine in a climate-controlled chamber at 38.5°C (38.5% r.h.). Mean body temperature was clamped using a water-perfusion suit (T_w 38.8°C, SD 0.5). Core temperature was measured from the oesophagus, rectum and auditory canal, and skin temperatures from 14 sites. The $\dot{m}_{sw}$ was measured at six sites simultaneously, using ventilated sweat capsules (3.16 ±0.05 cm^2: Multi-Site Sweat Monitor, Clinical Engineering Solutions, Australia). Two capsules, attached to the forehead and foot, were modified to yield f_{sw} data. These data were used to determine whether sweating at the forehead was of the same rhythm as that below the SCI (if present). This was essential for determining whether or not sweat below the SCI was under hypothalamic control, and thereby having a synchronous f_{sw} pattern.

Since major causes of non-thermal sweating below a SCI are bladder and bowel distension, and local tissue ischaemia, subjects emptied their bladder and bowel prior to a trial. To limit pressure-induced ischaemia, all subjects rested on a dry floatation cushion (ROHO© Inc, Belleville, IL., U.S.A.). This is a specialised mattress commonly used with spinal patients to minimise local pressure. Great care was also taken to avoid any tubing or thermistor cables lying between the subject and the mattress, and subjects were moved regularly to reduce localised pressure. These precautions minimised both the incidence and severity of spastic responses in the SCI subjects.

RESULTS

All controls displayed sweat suppression with leg cooling. However, one SCI subject similarly experienced reduced forehead sweating, and was classified as an incomplete SCI. Seven clinically, and physiologically, complete SCI subjects remained. Sweating did not occur at the forehead in any quadriplegic subject (n=3). Since preganglionic sympathetic fibres originate from neurons in the thoracic and lumbar segments only, a complete SCI above the thoracic segments results in the loss of all sympathetic and, subsequently, sudomotor control. Therefore, the ability to verify SCI completeness using limb cooling was not applicable within these subjects.

Whole body $\dot{m}_{sw}$ (averaged across six sweat capsules) for the control group was greater than observed in the SCI subjects (1.03 and 0.3 $mg \cdot cm^{-2} \cdot min^{-1}$ respectively; P<0.05). This was primarily due to the absence of sweating at several sites in the majority of SCI subjects, regardless of large increases in mean body temperature (1.9°C SD 0.4). However, of the seven complete SCI subjects, two had sweating below the SCI, *albeit* of a substantially reduced magnitude: < 0.1 $mg \cdot cm^{-2} \cdot min^{-1}$.

In the control subjects, a delay of about 2 s occurred between a sweat expulsion at the forehead, and a corresponding expulsion at the foot. In the two SCI subjects with sweating below the SCI, the foot f_{sw} was only 53% of the forehead f_{sw}. However, when averaged over 50 min and across both subjects, 94% of the expulsions at the foot coincided with forehead sweat expulsions.

DISCUSSION AND CONCLUSION

The major finding of the present study was that the spinal cord, when isolated from the hypothalamus, could not independently induce sweating in response to thermal stimuli. These results also demonstrate that, despite the complete absence of spinal cord function below the site of injury, the autonomic nervous system can still be partially intact, and responsive to hypothalamic signals.

Consistently sequential sweat expulsions from both the sensate and insensate skin of two subjects with clinically-verified complete SCI, are consistent with hypothalamic control of thermally-induced sweating, and cannot be deemed to be a spinally-mediated sudomotor

response. Such control may, however, be due to the survival, or regeneration, of sympathetic ganglia, independently of a complete SCI. This finding may help explain why several previous studies have found sweating below a clinically, and sometimes anatomically, complete SCI.

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All experiments were approved by the University's Human Research Ethics Committee.

INVITED LECTURE 5: PARTICIPATION OF GASTROINTESTINAL ENDOTOXINS IN THE TOLERANCE OF HEAT AND EXERCISE

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INTRODUCTION

Normally, the gut contains massive quantities of gram-negative bacteria and endotoxin (lipopolysaccharide, LPS) formed from the bacteria, however, there is a highly effective gut wall barrier to movement of the LPS or bacteria into plasma. This presentation is based on two detailed discussions [1,2] of the proposals that heat and exercise tolerance can be reduced by LPS from the gut and that heat stroke and exercise exhaustion can involve fever being superimposed onto hyperthermia.

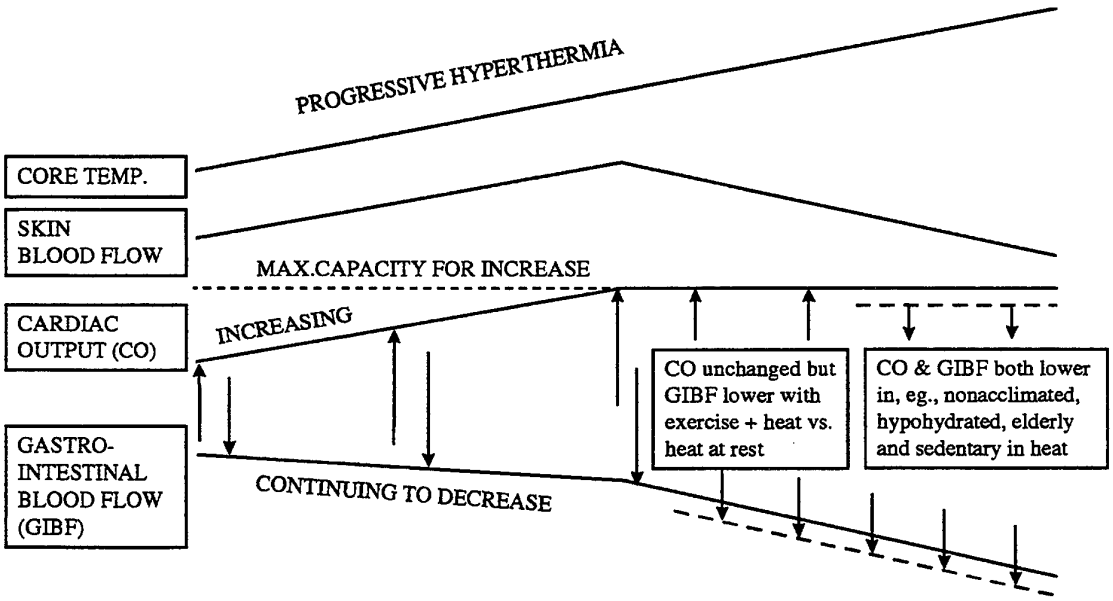

Figure 1. Schematic illustration of our basic rationale for gut ischaemia (and therefore endotoxin load) being determined by cardiovascular capacity. From [2] with permission.

It has been well established (in studies not involving thermoregulatory challenges or exercise) that gut ischaemia reduces the gut wall barrier to translocation of endotoxins. Fig.1 illustrates our basic concept: That is, gut wall ischaemia is an integral part of the normal changes in regional blood flows necessary to combat heat stress (or perform exercise). This brings about a reduction in the gut wall barrier to LPS by a magnitude and/or at a time of onset which depends upon cardiovascular capacity - which has been determined by, eg., heat acclimation, physical fitness and age. A greater capacity to increase cardiac output allows the circulatory changes necessary to combat heat stress, such as increased skin blood flow, to be achieved while gut blood flow is maintained at higher levels. Further, we know [3] that the survival of animals at specified levels of thermal stress is lower with combined exercise and heat than with heat alone; we propose that this is due to the additional blood flow requirements of the exercising muscles necessitating earlier and greater gut ischaemia, leading to earlier and greater entry of LPS. Likewise, for example,hypohydration will increase whereas physical fitness will decrease the endotoxin load and thereby heat tolerance will be decreased or increased, respectively.

The principal evidence for endotoxin involvement in tolerance of heat and exercise is: (a) Heat stroke or exhaustive exercise has many symptoms in common with endotoxaemia. (b) Additional to gut ischaemia, normal responses to heat stress or exercise include acidosis and production of reactive oxygen species in gut tissue and elevated circulating catecholamine levels; any one or all of these changes will diminish the gut barrier to LPS. (c) The normal efficiency of endotoxin removal by the liver is reduced by severe heat stress or exhaustive exercise. (d) Plasma endotoxin levels increase during severe heat stress or exhaustive exercise and cytokyne levels are elevated in heat stroke patients. (e) Development of endotoxin tolerance improves heat tolerance and vice versa. (f) Removal of gut endotoxins by antibiotics or lavage improves heat tolerance. (g) Exogenous endotoxin given iv during early stages of heat exposure reduces heat tolerance. (h) Development of heat stroke has been delayed by pre-treatment with either corticosteroids or LPS-antibodies. We recently further demonstrated that indomethacin treatment (to block prostaglandins) elevates the heat tolerance of physically unfit [4] or old aged [5] animals to that of physically fit or young animals, respectively.

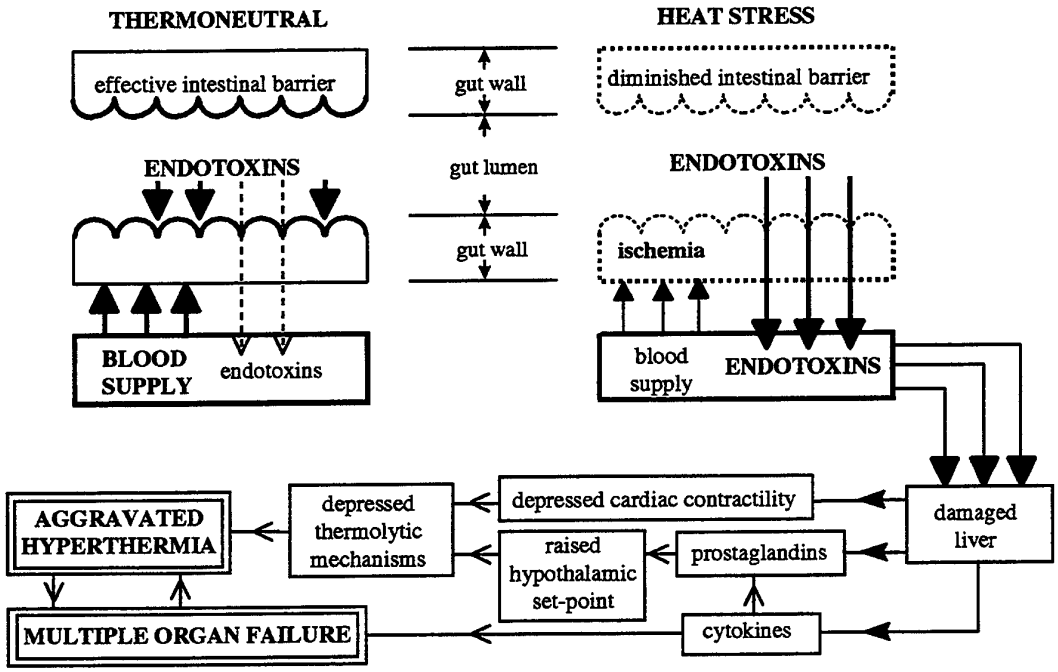

Fig.2 illustrates the proposed transfer of endotoxins from the gut into plasma due to gut ischaemia during heat stress, failure of the liver to clear those endotoxins and ultimately the aggravation of hyperthermia (and possible contribution to the development of heat stroke) by three means: (i) Depression of thermolytic mechanisms by raising hypothalamic set-point temperature, (ii) depression of thermolytic mechanisms by depressing cardiac contractility and (iii) contributing to the development of multiple organ failure. From [2] with permission.

Fig.2 illustrates the proposed transfer of endotoxins from the gut into plasma due to gut ischaemia during heat stress, failure of the liver to clear those endotoxins and ultimately the aggravation of hyperthermia by three means: (i) Depression of thermolytic mechanisms by raising hypothalamic set-point temperature (via prostaglandins directly or via cytokynes), (ii) depression of thermolytic mechanisms by depressing cardiac contractility (due to direct effects of endotoxin on cardiac muscle) and (iii) by contributing to the development of multiple organ failure (due to DIC and cellular effects of cytokynes).

CONCLUSIONS

There is increasing evidence that endotoxins may limit the tolerance of heat and exercise and that those endotoxins come from the gastrointestinal tract. This limitation is due to the hyperthermia of fever being superimposed onto the hyperthermia of exposure to a hot environment and/or performance of exercise. That is, gastrointestinal endotoxins can become a factor which causes a physiological state to become pathophysiological during exposure to hot environments and/or exercise.

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PAPER 8: PREVENTION AND TREATMENT OF AN EXPERIMENTAL HEAT STROKE MODEL

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INTRODUCTION

The clinical diagnosis of heat stroke is strongly suggested when hyperthermia is associated with neurological abnormalities including restlessness, delirium, coma or seizure, as well as multiple-organ damage after exposure to heat [1]. Whereas tissue damage was formerly attributed to hyperthermia itself, our previous studies [2, 3, 4] demonstrated that cerebral ischaemia rather than hyperthermia, is the main reason for the central nervous system dysfunction that occurs during heat stroke.

The treatment of heat stroke is cooling [1]. No pharmacological agent has been found to be beneficial in the treatment of heat stroke [1,5]. The aim of the present study was to test any possible therapeutic effects of pretreatment or treatment with monoamine depletors, NMDA receptor antagonists, dexamethasone, superior cervical ganglionectomy, hypervolaemic haemodilution, heat shock protein (HSP) 72 induction, chronic hypoxia, dl-tetrahydropalmatine or interleukin-1 (IL-1) β receptor antagonist in rat heat stroke.

MATERIALS AND METHODS

Adult male Sprague-Dawley rats weighing 250-300 g were purchased from National Yang-Ming University Animal Center (Taipei, Taiwan) and housed in a temperature- and light-controlled animal room (23±1.0°C; lights on from 0600 to 2000 h) with free access to rat chow and water.

The right femoral artery and vein of these rats, under urethane (1400 mg/kg, i.p.) anaesthesia, were cannulated with polyethylene tubing (PE50). Systemic arterial blood pressure was monitored continuously using a pressure transducer and chart recorder (Gould model 481). Animals were then fixed to a stereotaxic frame. Local cerebral blood flow was monitored with a Laserflo BPM2 laser Doppler flowmeter (Vasamedics, St. Paul MN, USA). A 24 gauge stainless steel needle probe (diameter, 0.58 mm; length, 40 mm) was inserted into the right corpus striatum, hypothalamus or cerebral cortex using the stereotaxic atlas and coordinates of Paxinos and Watson [6]. The colon temperature (T_{co}) was continuously monitored by thermocouples. The animals were exposed to an ambient temperature (T_a) of 42°C to induce heat stroke. The moment at which the mean arterial blood pressure or local cerebral blood flow (CBF) began to decrease from its peak level was taken as the onset of heat stroke [7].

In separate experiments, the autoradiography diffusible tracer technique was used for measuring local CBF [8]. At the end of the experiments, the rat brains were resected, fixed in 10% neutral buffered formalin and embedded in paraffin blocks. Serial (10μm) sections through the striatum were stained with haematoxylin and eosin for microscopic examination. The extent of striatal neural damage was scored on a scale of 0-3, modified from the grading system of Pulsinelli et al [9], in which 0 is normal, 1 means that a few neurons are damaged, 2 means that many neurons are damaged, and 3 means that all neurons are damaged.

RESULTS AND DISCUSSION

Table 1 summaries the mean and S.E.M. values for the normothermia control rats and rats with heat stress.

Physiological parameter	Time of heat stress (42°C)					
	0 min	40 min	60 min	70 min	80 min	
Rats kept at T_a 24°C:						
T_{co}(°C)	36.5±0.1	36.6±0.2	36.7±0.2	36.5±0.1	36.7±0.2	
MAP(mmHg)	92±4	90±3	93±2	95±3	91±4	
CBF(% baseline) in striatum	100±5	98±4	102±3	101±5	105±4	
Neural damage score(0-3)	0				0	
ST(min)						>300
Rats kept at Ta 42°C:						
T_{co}(°C)	36.5±0.2	39.5±0.2#	42.2±0.2#	42.7±0.3#	42.8±0.4#	
MAP(mmHg)	90±4	105±5#	154±8#	129±7#	35±4#	
CBF(% baseline) in striatum	100±4	151±5#	186±13#	155±6#	40±3#	
Neural damage score(0-3)	0				2.0±0.4#	
ST(min)						17±3#

# $P<0.05$, significantly different from the corresponding control value, Student *t*-test.

Table 1. Effects of heat stress (42 °C) on colon temperature (T_{co}), mean arterial pressure (MAP), local cerebral blood flow (CBF), neuronal damage score and survival time (ST) in rats.

The latency of onset of heat stroke was about 70 min and the survival time (interval between the onset of heatstroke and cardiac arrest) about 17 min for rats exposed to a T_a of 42°C. Ten min after the onset of heatstroke, animals displayed hyperthermia, arterial hypotension, cerebral ischaemia and neuronal damage. The heat stroke-induced arterial hypotension, cerebral ischaemia and neuronal damage were attenuated by pretreatment or treatment of animals with 6-hydroxydopamine [9], 5,7-dihydroxytryptamine [10], MK801 and ketamine [7], superior cervical ganglionectomy [11], heat shock protein induction [12], IL-1 receptor antagonist [13], dexamethasone [Lin et al., unpublished data], dl-tetrahydropalmatine [14] or 10% albumin [Lin et al., unpublished data].

As shown in our previous results [4,7,9,10,12], after the onset of heat stroke, animals displayed hyperthermia, arterial hypotension, intracranial hypertension, cerebral ischaemia, elevated DA, 5-HT or glutamate levels, and increased cerebral neuronal damage as compared with those of normothermia, control animals. However, when the brain DA, 5-HT, or glutamate levels were decreased, respectively by 6-hydroxydopamine, 5,7-dihydroxytryptamine, or MK801, the heat stroke –induced arterial hypotension, intracranial hypertension, increased release of DA, 5-HT, or glutamate in brain, and ischaemic damage to neurons in animals were greatly attenuated. In addition, either heat shock protein induction (induced by heat treatment or chronic hypoxia)[12] or pretreatment with IL-1 receptor antagonist [13], dexamethasone, dl-tetrahydropalmatine [14] or hypervolaemic haemodilution (produced by intravenous administration of 10% human albumin) decreased the enhanced release of brain DA, 5-HT or glutamate evoked by heat stroke and resulted in an increase of survival time in animals with heat stroke. These results indicate that excess release of DA, 5-HT, or glutamate is essential for pathogenesis of heat stroke in rats. Accordingly, the heat stroke syndromes could be prevented or treated by any measures or agents which are able to prevent excess release of DA, 5-HT or glutamate from cerebral neurons.

CONCLUSIONS

In the present results, the heat stroke-induced arterial hypotension, cerebral ischaemia and neuronal injury were attenuated by pretreatment with either DA or 5-HT nerve depletors (such as 6-hydroxydopamine or 5,7-dihydroxytryptamine), IL-1 receptor antagonist, NMDA receptor antagonists (such as MK801 and ketamine), dexamethasone (a synthetic glucocorticoid), dl-tetrahydropalmatine (a monoamine depletor), superior cervical ganglionectomy, hypervolaemic hemodilution (produced by intravenous infusion of 10% human albumin), HSP72 induction, or chronic hypoxia. In addition, the heat stroke-induced physiological alterations could be attenuated by treatment with IL-1 receptor antagonist or dexamethasone immediately after the onset of heat stroke. These results indicate that IL-1 receptor antagonist or dexamethasone is a good choice for prevention and treatment of CNS syndromes associated with heat stroke.

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PAPER 9: INTER-RELATIONSHIPS BETWEEN SWEATING, CORE AND INTRAMUSCULAR TEMPERATURES.

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INTRODUCTION

A number of studies have proposed the existence of thermoreceptors within skeletal muscle [1-3], but only one report has supported their existence [4]. Jessen *et al.* [4], in a study conducted on conscious goats, found evidence for the existence of thermosensitive elements in, or near, skeletal muscle. The authors observed that muscle cooling, at a constant body temperature, decreased respiratory evaporative heat loss. This response was considered to indicate that intramuscular afferents projected to the hypothalamus, resulting in modified evaporative heat loss.

While one would assume that thermoreceptors may best function if located close to sources of thermal energy, only circumstantial evidence for the existence of intramuscular thermoreceptors is available. Consequently, this project sought to obtain data consistent with their existence, by analysing the inter-relationships between thermal stimuli and sweating responses.

MATERIALS AND METHODS

Physically-active males (age: 28.8 y (SD 9.0), mass: 78.3 kg (SD 8.7), peak aerobic power: 4.53 $l \cdot min^{-1}$ (SD 0.57)) participated in exercise trials (cycling): a one-legged step (n=9; 39.3°C (SD 0.7), 44.5% r.h. (SD 0.7)); an incremental ramp (n=11; 25.1°C (SD 0.4), 37.6% r.h. (SD 3.3)); and a sinusoidal exercise function (n=10; 25.2°C (SD 0.4), 36.4% r.h. (SD 3.0)). The step protocol involved two stages: (i) an increment to 38.2 W (SD 1.5) held for 25 min (60 rev $\cdot min^{-1}$); and (ii) an increment to 76.3 W (SD 3.0) for 20 min (60 rev $\cdot min^{-1}$). For the ramp function, work rate increased from steady state cycling at 120 W (SD 11.9; 15 min; 60 rev $\cdot min^{-1}$) towards volitional fatigue at 20 W $\cdot min^{-1}$. The sinusoidal protocol followed steady state cycling (35 min, 119 W (SD 12.0), 60 rev $\cdot min^{-1}$), and involved three sinusoidal work rate variations between 30 watts and 60% of each subject's peak power (207.8 W (SD 22.6), 60 rev $\cdot min^{-1}$), with an 8-min period.

Core temperature was measured (0.2 Hz) from the oesophagus (T_{es}), while mean skin temperature ($\bar{T}_{sk}$; 0.2 Hz) was derived from eight sites. Intramuscular temperature was measured from the *vastus lateralis* (T_m; 1 Hz), using an indwelling thermocouple inserted 25-30 mm. Sweat rate () was assessed at five sites (ventilated capsules: 3.16 cm^2; 1 Hz): forehead, arm, chest, both thighs.

RESULTS

Simple linear regression analyses, across all three forcing functions, revealed strong correlations between $\dot{m}_{sw}$ (averaged across sites) and $\bar{T}_{sk}$ (r^2=0.57 (±0.23)), T_{es} (r^2=0.60 (±0.08)), and T_m (r^2=0.78 (±0.13)). These relationships were not significantly different from each other (P>0.05). In addition, thermal input and sudomotor output relationships, or control gains (mg $\cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$), were modelled linearly. Again, sudomotor gains did not differ between exercise forcing functions, or between thermal indices within each function (P>0.05).

The phase delay for each physiological response, in reaction to sinusoidal work rate changes, was determined at 90° increments (five points) between the second and third waveform peaks. At any point within the waveform, the phase delay for each physiological response was not significantly different from that of the average phase delay across the

waveform, indicating that the sinusoidal driving of deep tissue temperature was relayed through the hypothalamic processor, and appeared as thermoefferent output with the same sinusoidal waveform (period). Figure 1 illustrates typical thermal and sudomotor responses during this sinusoidal function. On average, across these five points within the waveforms, the T_{es} phase delay was not significantly different from forehead $\dot{m}_{sw}$ ($P>0.05$). However, T_m and $\bar{T}_{sk}$ phase delays both differed significantly from $\dot{m}_{sw}$ ($P<0.05$). The prolonged $\bar{T}_{sk}$ delay indicated that central rather than peripheral thermal input was driving sweating.

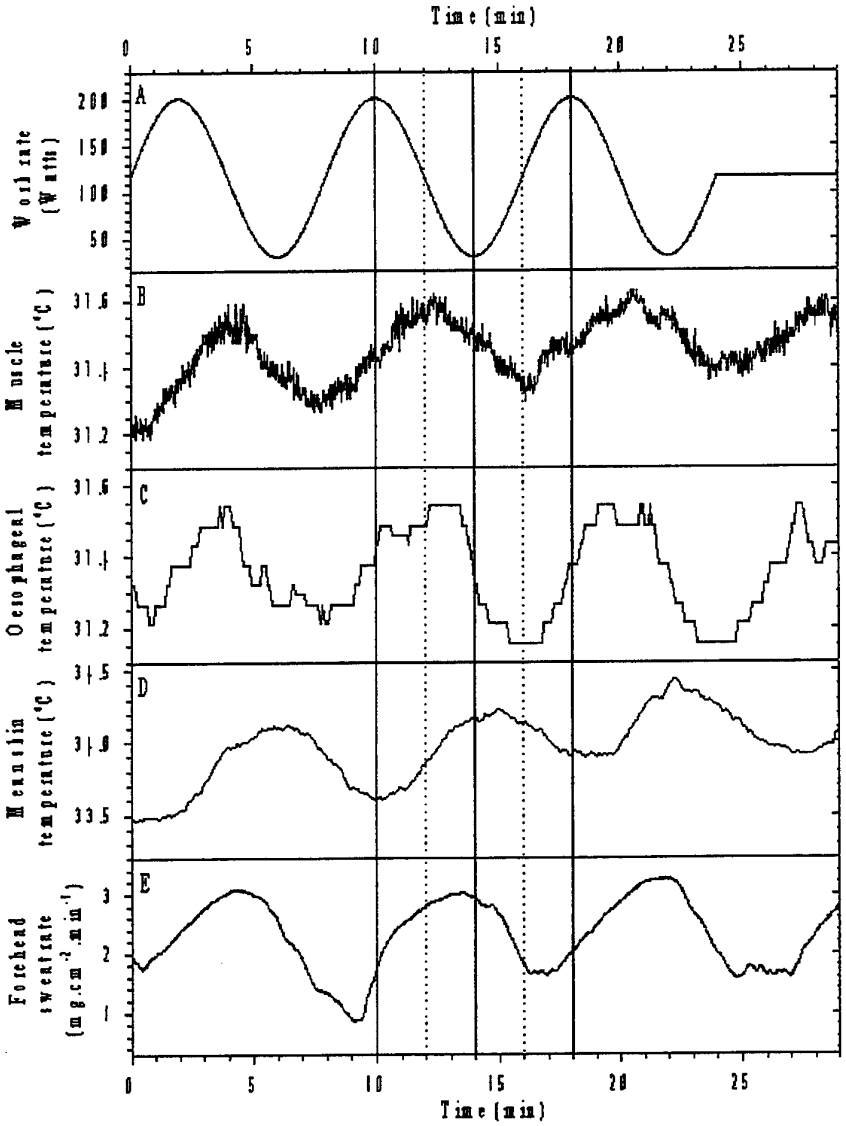

Figure 1: Sinusoidally-driven variations in work rate (A), muscle temperature (B), oesophageal temperature (C), skin temperature (D) and forehead sweat rate (E). Each graph contains raw data from the same subject. Vertical lines indicate 90° steps through the work rate sinusoidal function.

DISCUSSION

For some time, we have known that thermoreceptors are located within the skin [5] and the core tissues [3,6], and that these two sites exhibit a strong relationship with sweating [2]. The results of the linear regression analyses could be considered consistent with the provision of comparable thermoafferent input from core, skin and intramuscular tissues.

This is reinforced by the similarities of the sudomotor gains, relative to temperature changes at these sites. Indeed, convincing correlations between T_m and $\dot{m}_{sw}$ have been shown in previous studies [7,2], and such relationships would be expected from a system exhibiting proportional control. We similarly found a strong correlation, and since this relationship was not significantly different from that of the core with sweating, one may consider such evidence to be consistent with the possible existence of intramuscular thermoreceptors. However, such evidence is insufficient to ascribe a causal role to T_m changes.

It was hypothesised that we may find possible causal evidence within our sinusoidal manipulation of tissue temperatures, with phase delays between changes at the heat source (T_m) and the effector response ($\dot{m}_{sw}$) being consistent with such a relationship. However, this was not the case, and the body core dominated the drive for sweating during sinusoidal exercise. Since sweating is sympathetically controlled [8], one would expect to find a delay of only a few seconds between thermoreceptor stimulation and the resultant effector response. We unequivocally found this was the case with the T_{es} and $\dot{m}_{sw}$ phase delays.

CONCLUSION

At present, we know thermoreceptors are located within the core and skin. Since the relationship between T_m and $\dot{m}_{sw}$ across three separate forcing functions, was similar to that of the core and skin, our evidence is indicative of thermoreceptors also being located within skeletal muscle. On the other hand, the phase delays for each thermal stimuli and effector response imply that primarily central, rather than peripheral, thermal input drive sweating during sinusoidal exercise. Nevertheless, we cannot discount a possible role of non-thermal (feed-forward) sudomotor drive.

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All experiments were approved by the University's Human Research Ethics Committee.

INVITED LECTURE 6: TIME COURSE OF HEAT ACCLIMATION AND ITS RETENTION

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INTRODUCTION

This report briefly reviews the key literature on the time course of heat acclimation and its retention. The perceptual, performance and physiological adaptations known to occur during heat acclimation are also characterized. The published literature is far more robust and plentiful on the time course of heat acclimation than its retention. More complete discussions of this topic can be found in other recent reviews [1, 2].

TIME COURSE AND ADAPTATIONS DURING HEAT ACCLIMATION

Repeated daily heat exposure reduces perceptual and physiological strain while improving exercise performance during subsequent heat exposures. Such changes are called *acclimatization* if found following exposure to a naturally occurring environment and *acclimation* if observed in a controlled environmental setting. Exercise in the heat is the most effective method for developing heat acclimation; however, resting in the heat can result in a lesser degree of acclimation [1, 2]. Exercise-heat acclimation does not need to involve daily 24-hour exposure for its full development. A continuous, daily 100 min exposure produces an "optimal" exercise-heat acclimation response for dry heat while longer daily exposures may be necessary to produce optimal acclimation for humid heat [2, 3].

Historically, many of the classic studies involving exercise-heat acclimation/acclimatization occurred in the 1940s [4, 5, 6]. From these classic studies, the following conclusions were drawn concerning the time course of heat acclimation. The time course for the acquisition of acclimation was similar for hot-dry and hot-humid environments [5, 6]. About 80% of the improvement in temperature regulation seen during dry-heat acclimation occurred during the first 7 days of the 23 day exposure period [6]. However, two recent reviews [1, 2] also suggested about two-thirds to 75% of the physiological adjustments and improvements in performance were seen in 4 to 6 days. Heat acclimation began on the first day of exposure, progressed rapidly during the next 2 to 4 days and was virtually complete in 7 to 10 days [5]. Relatively short periods of physical exercise (1 to 1½ hours/day) were required, but some very limited acclimation occurred while resting in the heat [5, 6]. Physically fit men acclimated more rapidly than less fit men in both dry and humid heat [4, 5, 6]. The cardiovascular adaptations during heat acclimation resulted in a rapid decline in syncopal events after the first day of exposure [5].

Table 1 reviews the major perceptual, performance and physiological adaptations known to occur during exercise-heat acclimation/acclimatization as noted in a recent publication [7]. During heat acclimation, body core temperature is reduced while cardiovascular strain is lessened through a lowered heart rate, increased stroke volume and a better defended blood pressure. Although somewhat debatable, metabolic rate has been shown by some to be lowered after acclimation. Sweating is generally thought to be improved through an earlier onset time, higher rate, and for humid environments the distribution is improved while resistance to hidromeiosis is increased. Skin blood flow is increased through an earlier onset time and higher flow. Improved fluid balance occurs through an increased thirst, reduced electrolyte loss, increased total body water, and an increased and better defended plasma volume. Overall, both thermal comfort and exercise performance are improved

during heat acclimation. No single cause can explain this adaptive process; exercise-heat acclimation probably results from the interplay of many mechanisms.

Thermal Comfort – *Improved*	Exercise Performance - *Improved*
· Core Temperature - *Reduced*	· Metabolic Rate - *Lowered*
· Sweating – *Improved*	· Cardiovascular Strain - *Reduced*
Onset – *Earlier*	Heart Rate - *Lowered*
Rate – *Higher*	Stroke Volume - *Increased*
Distribution – *Improved* (tropic)	Blood Pressure - *Better Defended*
Hidromeiosis – *Reduced* (tropic)	· Fluid Balance - *Improved*
· Skin Blood Flow – *Increased*	Thirst - *Improved*
Onset – *Earlier*	Electrolyte Loss - *Reduced*
Flow – *Higher*	Total Body Water - *Increased*
	Plasma Volume -*Increased & Better Defended*

Table 1. Adaptations during heat acclimation

Researchers generally agree that high aerobic fitness achieved through endurance training in a temperate environment reduces physiological strain and increases tolerance to exercise in the heat [2, 8]. In addition, endurance-trained individuals exhibit many of the characteristics of heat-acclimated individuals while exercising in the heat, and are thought to be partially but not fully heat acclimated [8]. Several authors suggest that high levels of maximal aerobic power are related to improved exercise-heat tolerance and/or a more rapid rate of heat acclimation [2, 8].

RETENTION OF HEAT ACCLIMATION

Heat acclimation is a transient process and gradually decays or is lost if not maintained by repeated exercise-heat exposure [1, 2, 8]. The heart rate improvement which takes place more rapidly during heat acclimation is also lost more rapidly than the other improvements in thermoregulatory responses [1, 2, 8]. However, there is considerable variability in the published literature concerning the rate of decay or loss of exercise-heat acclimation [1, 2, 8]. Some authors report that significant losses of heat acclimation occur in less than one week while others indicate that acclimation responses are fairly well maintained for nearly one month.

As shown in Table 2, eight studies were published between 1943 and 1963 on the decay or loss of heat acclimation of which four (upper half of Table 2) involved exposure to dry heat [3, 4, 6, 9] and four (lower half of Table 2) concerned humid-heat exposure [5, 10, 11, 12]. These early studies on the decay or loss of heat acclimation were flawed by either (a) the use of very small samples [4, 5, 6, 10], (b) incomplete heat acclimation [3, 9], or (c) inappropriate physiological measurements [11, 12]. Nevertheless, the observations from these early studies are pioneering from the standpoint that they generally showed that the retention of heat acclimation was better for hot-dry compared to hot-humid environments, and aerobically-fit individuals also displayed greater retention of the heat acclimation benefits.

Two somewhat more recent and better controlled studies [13, 14] generally support the major conclusions drawn from the earlier studies reported in Table 2. After humid-heat acclimation, Williams and colleagues [14] showed significant losses for both rectal temperature and heart rate after the first two weeks in cool conditions with 45% (rectal temperature) and 92% (heart rate) losses after the third week. In contrast, Pandolf and colleagues [13] evaluated the rate of loss/decay after dry-heat acclimation and reported non-significant losses for both rectal temperature and heart rate after 18 days in cool conditions. Thus, these two studies support the contention from the earlier studies (see Table 2) that the retention of heat acclimation is greater for hot-dry compared to hot-humid environments.

STUDY	CONDITIONS	WEAKNESS	CONCLUSIONS
Robinson et al. (1943) [6]	40°C DB/23% RH	n=1 for each decay period (13,15,24,26,28 days)	".. only slightly less efficient 2-3 weeks after acclimation.."
Bean & Eichna (1943) [4]	49°C DB/15-22% RH	n=1 for each decay period (1,2,3,5,6,13 weeks)	"..acclimation well retained for at least 1 week..after 1 month major portion lost.."
Henschel et al. (1943) [9]	43-49°C DB/20-25% RH	only 2 days of heat acclimation	"..heat acclimation persists during at least 3 weeks of cold weather."
Lind & Bass (1963) [3]	49°C DB/17% RH	groups not equally heat acclimated	"..for periods up to 8 days practically no loss..some loss after 17 days.."
Eichna et al. (1945) [5]	33°C DB/96% RH	n=2 after 3 weeks in cool conditions	"..lost most of their adaptation ..acclimation retained best by men who remained physically fit.."
Stein et al. (1949) [10]	42°C DB/50% RH	n=3 after 3 and 5 weeks in cold and cool conditions, respectively	"..most of the acclimation retained after 3 weeks..considerable loss after 5 weeks.."
Wyndham & Jacobs (1957) [11]	??°C DB/33°C WB	used mouth temperature	"..significant loss of acclimation after 6-day decay period.."
Adam et al. (1960) [12]	36°C DB/76% RH	few data to critically evaluate	"..substantial part of acclimation lost after 6 days..after 34 days the decay in acclimation was virtually complete.."

TABLE 2. EARLY FINDINGS ON RETENTION OF HEAT ACCLIMATION

CONCLUSIONS

Nearly complete exercise-heat acclimation for both hot-dry and hot-humid environments occurs after 7 to 10 days of exposure. However, about two-thirds to 75% of the physiological adjustments are seen in 4 to 6 days. Continuous, daily 100-min exercise bouts appear optimal to induce the heat-acclimation process. High levels of aerobic fitness can partially but not fully acclimate individuals to the heat. Retention of the benefits of heat acclimation appear to be longer for dry compared to humid heat. High levels of aerobic fitness seem to be associated with greater retention of heat acclimation. Further well-designed and definitive studies on the decay/loss of heat acclimation are necessary.

DISCLAIMER

The views, opinions and/or findings contained in this report are those of the author and should not be construed as an official Department of the Army position, policy or decision, unless so designated by other official documentation. Approved for public release; distribution is unlimited.

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PAPER 10: URINALYSES AND BODY MASS CHANGES DURING AN ULTRA-DISTANCE ENDURANCE EVENT: THE SIMPSON DESERT CYCLE CHALLENGE

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INTRODUCTION

Hypohydration during high intensity and/or prolonged exercise in thermally stressful environments may lead to decreases in physical performance, thermoregulatory ability and cardiovascular function (1). Athletes competing in thermally stressful environments are advised to observe sweat losses and measure both fluid intake and body mass in order to monitor fluid losses. However, these techniques may not be accurate unless all food intake, fluid intake, sweat loss, respiratory fluid loss, and faeces losses are accurately monitored (2). It has also recently been suggested that urinalysis is a valid and non-invasive screening tool to monitor hydration status in athletes (2). The purpose of the present study was to describe the body mass and urine changes that occurred over a five-day, 580 km multi-stage cycling race (Simpson Desert Cycle Challenge - SDCC) across Australia's Simpson Desert in ambient temperatures ranging from 16-47°C.

MATERIALS AND METHODS

Seventeen cyclists (15M, 2F) volunteered to participate in the study (Table 1).

Age (yr)	Height (cm)	Mass (kg)	Skinfold total (mm) *
34.4±8.9	180.8±7.9	75.7±11.3	75.4±24

* Sum of seven skinfolds (3)

Table 1: Physical characteristics (Mean ± SD) of the Simpson Desert Cycle Challenge volunteers (n=17).

For logistical reasons (exhaustion, race drop-outs, illness, adherance), only nine of the original seventeen volunteers completed all urinalyses and various numbers of athletes completed all pre - post race stage weigh-ins. Body mass was measured to 0±1 kg before and immediately after each race stage using previously calibrated weighing scales (Mercury, Australia). Fluid intake of a wide variety of beverages during each stage was *ad libitum* with race organisers ruling a minimum of 1.5 litres of fluid was to be carried and collected at every 15-20 km checkpoint. Urinalysis was conducted early morning prior to food or fluid ingestion. A clean-caught midstream sample was directly placed on a multiple reagent strip (Mutistix 10SG, Ames, Miles Australia). Ambient conditions (temperature, WBGT) were measured using a heat stress monitor (Questemp 10, Oconomowoc, USA). One-way analysis of variance for repeated measures and Scheffe's post-hoc analysis was used to determine significant changes over time for the urine parameters and Student's t-tests were used to determine body mass changes during each stage of the race. Significance was determined at the 0.05 level.

RESULTS

Table 2 shows the ambient conditions and changes in body mass during each of the nine stages.

Parameter	Temp (°C)	Range (°C)	WBGT (°C)	Pre-BM (kg)	Post-BM (kg)
Day 1 am	37.5±8.8	16.2-43.0	25.8±6.0	79.0±11.9	78.4±11.3 *
Day 1 pm	41.0±2.8	38.6-41.1	28.6±1.7	78.7±12.1	77.1±11.8 *
Day 2 am	38.5±9.9	18.8-47.0	26.1±6.3	79.1±11.5	79.0±11.3
Day 2 pm	44.3±2.9	41.8-47.5	29.5±2.8	79.1±12.4	74.7±8.3 *
Day 3 am	35.5±8.5	22.8-40.1	26.2±6.2	75.1±9.1	76.2±7.2
Day 3 pm	41.3±0.7	40.8-42.4	29.8±0.3	77.1±6.9	75.9±10.7 *
Day 4 am	33.9±9.9	22.6-40.7	25.0±7.7	76.9±11.6	76.3±7.4
Day 4 pm	40.7±0.3	40.5-41.1	28.9±0.7	76.9±11.7	75.8±11.4 *
Day 5 am	34.0±7.4	23.8-40.2	25.3±4.1	79.6±12.5	77.7±12.3 *

** denotes significant changes at the 0.05 level. Values are mean±SD.*
BM = Body mass. WBGT = Wet bulb globe temperature.

Table 2: Ambient conditions and body mass changes during each of the nine stages of the SDCC.

Significant changes in body mass were observed during each of the afternoon stages and the morning leg of days 1 and 5 (t = 2.04-4.99). Table 3 shows the results of the urinalysis parameters where observable changes occurred, together with body mass changes over the five days.

Day	1	2	3	4	5
Body mass (kg)	75.6±10.6	74.4±11.2	74.5±10.2.	74.8±10.9	74.2±11.1
Protein	2.1±0.8	2.8±0.7	2.7±0.7	3.2±0.7	2.9±1.2
Ketones	1.7±0.5	1.1±0.3	1.1±0.3	1.7±0.5	1.8±0.4[2]
Blood	2.9±0.6	2.8±0.7	2.2±1.0[4]	3.0±1.2	3.0±0.9
Bilirubin	1.4±0.5	1.3±0.5	1.2±0.4[4]	1.9±0.3	1.6±0.5
pH	6.2±0.3	5.8±0.4	6.0±0.8	5.4±0.5[1]	6.4±0.8
Specific gravity	1.02±0.0[4]	1.03±0.0	1.03±0.0	1.03±0.0	1.02±0.0[3]

Table 3: Results of body mass and urine changes (Mean±SD) using clinical reagent strips. Values for protein, blood, ketones, bilirubin are coded. The body mass, pH and specific gravity results are actual values. [1] different from day 5. [2] different from day 2 and 3. [3] different from day 2 and 4. [4] different from day 4.

ANOVA revealed main effects of time for urinary bilirubin (F[4,8]=3.89, p=0.01); urinary blood (F[4,8]=2.56, p=0.05); urine specific gravity (F[4,8]=6.14, p=0.009); urine pH (F[4,8]=4.73, p=0.004); and urinary ketones (F[4,8]=5.8, p=0.001).

DISCUSSION

The present results confirm previous studies that have observed urinary abnormalities following prolonged strenuous exercise in trained athletes (1, 4-7). Urinary specific gravity (SG) approaching 1.03 has recently been suggested to be a marker of hypohydration status in athletes (2). The observed increased SG from 1.02 pre-event to 1.03 most likely suggests dehydration, excessive sweating or inadequate fluid intake. It is interesting to note that even the pre-event SG (1.02) was at the higher end of the normal range of 1.015 to 1.024 for adults on a normal fluid intake (8). This may reflect the fact that most participants in the SDCC were from southern and thus cooler parts of Australia

and had travelled to race start for many days in air-conditioned vehicles in high ambient temperatures. Moreover, previous research suggests that ultraendurance athletes are chronically dehydrated prior to competition (9), probably as a result of inadequate hydration during training.

The observed hematuria, even prior to the race start, is commonly observed in athletes undertaking long duration or high intensity exercise, particularly athletes such as runners with high impact forces (4,6). Blood in the urine may be due to a number of factors including menstruation (two menstruating females in cohort), high impact forces (rough terrain and running up sandhills, through boggy sand), mechanical organ damage, or gastrointestinal bleeding through dehydration (observed in SDCC).

The current data suggest a significant increase in urinary protein. Such a finding is commonly observed in endurance athletes and is related to both the intensity and duration of exercise, and is most likely due to mechanical trauma, breakdown of the blood-urine barrier, or prolonged muscular exertion (4,6,7).

The present data also suggest a decreased urinary pH, at least up to the last day of the race. Such observations are commonly observed following metabolic acidosis or dietary carbohydrate insufficiency that may lead to ketoacidosis (8). Given the nature of the terrain, the duration of each stage of the race, and the multiple stage nature of the SDCC, it is possible that both metabolic acidosis and carbohydrate insufficiency may have occurred.

Ketones are the end-product of fatty acid breakdown. The observed significant changes in urinary ketones, particularly on day 5, may be due to the high degree of fatty acid metabolism that would be taking place during prolonged endurance exercise over many stages where an athlete may become carbohydrate depleted. The observed significant changes in urinary bilirubin are not normal in healthy individuals and may reflect use of vitamin C, antibiotics, oral contraceptives, or non-steroidal anti-inflammatory drugs such as Indocid (8).

Significant body mass changes were observed during the six of the 11 stages of the SDCC. Each of the four afternoon stages recorded significant body mass changes when high ambient temperatures (41.0-44.3 ^{o}C) were observed. For example, the greatest mean body mass loss (5.6% BM) was observed during the afternoon stage of day 2 where ambient temperatures ranged between 41.8 and 47.5 ^{o}C. Acute body mass changes are commonly observed during ultraendurance events (10,11) and stage cycling races similar to the SDCC (12) where fluid losses of 1-2 litres per hour are commonly observed in moderate ambient conditions. Given that the SDCC organisers only allow up to 2.0 litres to be carried and replaced every 15-20 km (1-2 hrs), it might be suggested that the athletes may become chronically dehydrated, particularly given the suggestion that ultraendurance athletes are chronically dehydrated prior to competition (9).

CONCLUSIONS

Competing in the SDCC leads to significant changes in a number of urine parameters suggesting hemolysis, dehydration and carbohydrate insufficiency.

- SDCC competitors need to ensure hyperhydration prior to each race stage.
- SDCC organisers need to allow increased water availability during each stage.
- Significant body mass and thus by definition fluid losses are observed during the SDCC suggesting the need for greater prior education of participants.

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INVITED LECTURE 7: HUMAN HEAT ACCLIMATION: WHAT IS THE BEST METHOD?

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PAPER 11: THE THERMOREGULATORY STRAIN PRODUCED BY PROTECTIVE PVC SUITS DURING SIMULATED CHEMICAL SPILL CLEAN-UP OPERATIONS IN A HOT ENVIRONMENT IS NOT REDUCED BY PASSIVE COOLING VESTS.

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INTRODUCTION

The North Queensland region of Australia endures high levels of heat and humidity with temperatures and humidities averaging 31°C and 71% respectively during the summer months [1]. Fire officers must perform under these conditions whilst also having the added heat load of wearing fully enclosed polyvinylchloride (PVC) protective suits. The combination of the high environmental heat stress, metabolic heat production and minimal heat dissipation from the suit may lead to an elevated risk of heat-related illness [2, 3].

To counteract the high heat stress associated with wearing personal protective equipment, a number of researchers have studied the use of cooling vests (CV) under personal protective equipment in fire personnel [4-8]. Cooling vests worn under fire fighting ensembles have been shown to attenuate the rate of heat storage, prolong exposure tolerance time in hot/humid environments, limit and delay elevation in rectal temperature, skin temperature and minimise whole body sweating rates [4-9].

The purpose of this study was to determine the susceptibility of fire officers to heat stress whilst wearing protective suits during a simulated chemical spill clean-up in a hot/humid environment. A further aim was to investigate whether a "ETC Thermal Wear" body management cooling vest could prevent any incidence of heat stress under these conditions.

MATERIALS AND METHODS

The seven male subjects used in this experiment were full time fire fighters who were familiar with the protective suits used in the clean-up of hazardous substances. The physical characteristics of the subjects are shown in Table 1.

	Mean ± SD	Range
Years of Service	8.4 ± 7.1	1 – 17
Age (years)	34.7 ± 7.9	25 – 47
Weight (kg)	84.2 ± 10.0	69.3 – 96.6
Height (cm)	179.7 ± 6.7	172 – 188.2
$\dot{V}O_{2\ max}$ (mL/kg/min)	46.5 ± 9.2	33.7 – 62.7

Table 1. Physical characteristics of subjects.

Measures of urine specific gravity (U_{sg}), body weight (B_w), heart rate (HR), blood pressure (BP) and core body temperature approximated by temperatures taken at the tympanic membrane (T_{tym}) were recorded prior to the start of exercise. The protocol consisted of a random, repeated measure crossover design where each subject performed the simulated clean-up operation twice, once with the "ETC Thermal Wear" CV and once without the vest (NV). The CV was worn on the outside of their regulation fire clothing. A Level Three Respirex PVC outfit with breathing apparatus was worn by the fire officers. The duration of the test protocol was limited to 20 minutes when the subjects wore the PVC

suits. Temperature and humidity were measured inside the suit of two subjects in addition to recording the external environmental conditions. The exercise protocol was chosen to mimic a chemical spill clean-up and consisted of walking at a steady pace on a bitumen carpark over a distance of 22m whilst alternatively carrying a 20 kg drum or wheeling wheelbarrows with a weight of 20 kg. Heart rate was recorded every minute and rating of perceived exertion (RPE) and thermal comfort (TC) recorded every five minutes. Core temperature was measured at 5, 10 and 15 minutes or if the subject finished earlier than the 20-minute time limit. A finger prick blood sample was taken to measure blood lactate (Lac) levels five minutes after the completion of the exercise protocol. Body weight and U_{sg} were recorded immediately post exercise. The protocol was repeated after a one-hour rest period when T_{tym} returned to 37.1 ± 0.3°C. Subjects who had not previously worn the CV were now required to wear them inside their PVC suits and vice versa (cross-over). The same measurements were recorded for this second trial. The results for HR, T_{tym}, B_w and U_{sg} were compared using two-way analysis of variance with repeated measures. The Lac, RPE, TC and environmental conditions inside the suit were analysed using paired t-tests. Data are presented as mean ± SD.

RESULTS

The environmental conditions in North Queensland on the day of testing had lower than average relative humidity (46.1%), but high ambient (36.2°C) and radiant (45.1°C) temperatures. Under these conditions all subjects failed to complete the entire 20-minute exercise protocol over the two trials due to either high T_{tym} (>39°C), fatigue or depleted oxygen supply. The average exercise time for the seven subjects in the two trial protocols was 13.3 ± 3.2 minutes with the average total exercise time for the trials with the CV (12.1 ± 3.8 minutes) not significantly different (p=0.31) to the NV trials (14.4 ± 2.6 minutes).

Core temperature increased significantly from a resting average of 37.3 ± 0.1°C to 39.2 ± 0.3°C at the completion of the exercise protocol (p<0.001). However, there was no significant difference in the absolute and the rises of core temperature during the course of the exercise between the CV and NV trials (p=0.96) (see Figure 1).

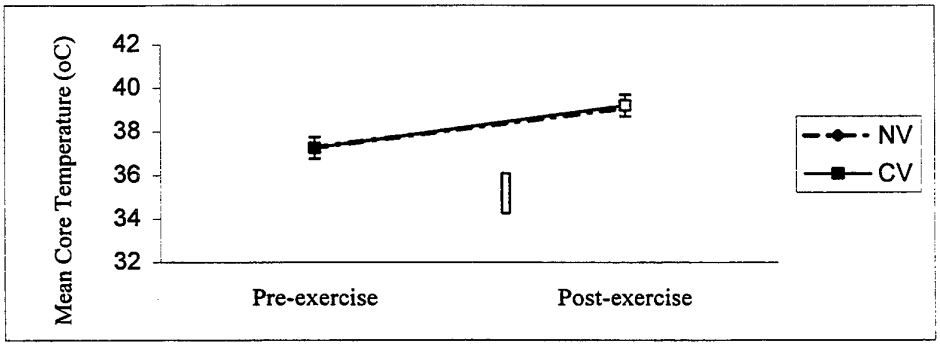

Figure 1: Average core temperature (°C) of the seven subjects before and after exercise with CV and NV.

With one subject withdrawing after seven minutes due to soreness of neck muscles, only the first seven minutes of the heart rate data was statistically analysed. There was a significant increase in HR over time (p<0.001), however, there was no significant difference in the absolute and the changes of HR between CV and NV trials (p>0.05). A *post hoc* Tukey test revealed that HR was significantly increased from resting during each minute of exercise up to seven minutes. The average HR expressed as a percentage of age predicted maximum measured at seven minutes was 84.9% for the trial with the CV and 86.1% without (NV). The final HR for all subjects just prior to conclusion of exercise

averaged 95.7% and 93.9% of age predicted maximum HR for the NV and CV trial respectively.

There was no significant difference in body weight before and after the exercise protocol (p=0.88). However, subjects lost an average of 0.56 ± 0.05 kg during the exercise protocol, equating to 560 mL of body fluid in less than 15 minutes. Furthermore, there was no significant difference in weight loss between the CV and NV trials (p = 0.98). Urine specific gravity, Lac, RPE, TC, temperature or relative humidity inside the suit did not differ between CV and NV trials (P>0.05).

The respective temperature and relative humidity inside the PVC suit was on average 4.2°C and 28% higher than the outside environmental temperature.

CONCLUSIONS

This study demonstrated that fire officers undertaking a chemical spill clean-up operation in the summer months in North Queensland may be exposed to increasing risk of thermal heat injury as a result of significant increases in core body temperature and near maximal HR after an average activity time of 13.3 minutes. Therefore, we recommend that a 10-15 minute time span be introduced to fire officers wearing the PVC suit during summer months rather than the national regulation time of 20 minutes. In contrast to previous studies, the wearing of a "ETC Thermal Wear" cooling vest had no significant benefit on core temperature, final heart rate, blood lactate, fluid loss, perceived rate of exertion or thermal comfort during the mock chemical spill clean-up operation.

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This study was approved by the James Cook University Ethics Committee, which complies with the provisions of the NHMRC statement on human experimentation.

PAPER 12: ENHANCEMENT OF PERFORMANCE THROUGH HEAT ACCLIMATION AND RACE SIMULATION AMONGST MOTORSPORT ATHLETES

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INTRODUCTION

Many elite motorsport athletes (MSA) undertake a physical training program to improve their performance. Although an improvement in physical fitness will enhance the MSA tolerance to the environmental and physical stressors, it is questionable whether an improvement in psychomotor performance will occur. This research examined the practicality of MSA using simulation conducted under replicated race conditions to heighten the psychomotor skills required in controlling a racecar (1).

MATERIALS AND METHODS

Eight male Australian Rally Championship level MSA (mean ± SD age, weight and height, respectively, = 24 ± 4 y, 77 ± 12 kg and 180 ± 6 cm) undertook four exposures to a simulated rallycar micro-environment, with 24 hours between each exposure. The rallycar micro-environment was replicated through the construction of an enclosed simulator inside an environmental chamber. During each simulation the temperature was maintained at 50°C and subjects were required to wear FIA safety equipment (3-layer nomex suit, gloves, boots and helmet). The simulation protocol consisted of two parts: first, a physical fatigue protocol consisting of riding a Monark cycle ergometer for 15min @ 125W, and second, three simulated rally stages, each of 12 minutes duration. During the simulated rally stages the co-pilot was seated next to the subject and called the pace notes for the rally stages. Between each rally stage there was a 2-minute rest period. Psychomotor fatigue was accelerated by having the subject undertake a 30 second cognitive task that tested his ability to interpret a verbal pace note into a motor action, prior to the commencement of each rally stage. During each simulation, Δbody mass, Δ heart rate and Δ body temperatures (rectum, sternum, forearm and calf) were recorded. Psychomotor performance was monitored through the recording of stage times, perceived levels of thermal strain and a validated psychomotor test that was conducted at the end of the first and fourth simulation to determine the level of mental fatigue.

RESULTS

The results are outlined in Table 1.

	1st simulation	4th simulation	Δ	Significance
Combined Stage Times (s)	2,258 ± 120	2,170 ± 78	88 ↓	$p < 0.003$
Psychomotor test (s)	140 ± 20	122 ± 21	18 ↓	$p < 0.001$
Δ Body mass (g)	1,350 ± 320	1,330 ± 140	20 ↓	NS
Sweat sensitivity ($g \cdot h^{-1} \cdot °C^{-1}$)	1.20 ± 0.42	1.53 ± 0.30	0.33 ↑	$p < 0.031$
Average heart rate (bpm)	134 ± 20	119 ± 14	15 ↓	$p < 0.003$
Δ Core temperature (°C)	1.18 ± 0.23	0.90 ± 0.19	0.28 ↓	$p < 0.001$
Δ Mean skin temperature (°C)	3.89 ± 0.31	3.03 ± 0.36	0.86 ↓	$p < 0.005$
Stored body heat ($W \cdot m^{-2}$)	68.5 ± 6.8	53.4 ± 4.7	15.1 ↓	$p < 0.001$
Physiological index of strain	1.71 ± 0.30	1.33 ± 0.21	0.38 ↓	$p < 0.003$
Perceived thermal strain	12.3 ± 2.4	8.6 ± 2.4	3.7 ↓	$p < 0.012$

Table 1:

DISCUSSION

All subjects recorded a significant improvement in the time taken to complete the simulated rally course and the psychomotor test. However, it is impossible to determine the relative contributions to the improvement in performance made by enhanced thermoregulation, familiarisation to the hot rallycar micro-environment, and task learning. The use of a control group (i.e. cool) may have removed the effect of acclimation. However, previous research by the authors has indicated that a cool simulation probably produced a reduction in the MSA arousal level of sufficient magnitude to affect the psychomotor results (1). Although the improvement in performance occurred under simulation, it is proposed that the MSA will use similar cognitive processes in controlling an actual rallycar. By practising these psychomotor tasks under replicated physical and environmental stressors, one may limit the deterioration in performance during an actual rally.

A significant decrease ($p<0.003$) in Physiological Index of Strain occurred over the duration of the simulation protocol. Previous literature has indicated that up to 75% of heat acclimation benefits can be achieved within the first 4-5 days of exposure (3). It is proposed that acclimation occurred at a faster rate than reported in previous literature due to the low energy expenditure ($\dot{V}O_2$ 0.7-0.9 $l \cdot min^{-1}$) required in performing the driving task. Accompanying the decrease in Physiological Index of Strain was a significant decrease ($p<0.012$) in the perceived level of thermal strain from 'very hot' to 'very warm'. Statistically there was no increase in sweat rate. However, a significant increase ($p<0.05$) in sweat sensitivity occurred by the end of the fourth simulation Although a racecar micro-environment has a high radiant heat value, the use of an impermeable racesuit by the MSA results in a 100% humidity micro-environment developing at the skin level. Saturation of the skin surface (as sweat is unable to evaporate) may result in hidromeisosis - a physical mechanism that blocks sweat secretion from the sweat glands (6).

The first simulation produced an average heart rate of 134 bpm, which was significantly reduced ($p<0.003$) to 119 bpm by the completion of the fourth simulation. Heart rate measurements recorded under racing conditions and published data indicate that the MSA has an elevated heart rate above 150-160 bpm (4,5). This difference in heart rate may be caused by a lower secretion of adrenaline, due to the absence of sympathetic nervous activity (1). It is proposed that the decrease in heart rate that occurred over the duration of the simulation protocol was due to: i.) familiarisation to the psychomotor stressors present within the simulator, and ii.) an increase in blood plasma volume due to heat acclimation, limiting cardiovascular drift. Hypothetically, if the in-car measurements were repeated after an environmental simulation protocol, then a reduction in heart rate of between 10-20 bpm may have been observed (in comparison to a subject who did not complete the protocol).

An examination of the physiological results may suggest that the improvements in performance may have occurred as the subjects adapted to the physical fatigue protocol. However, it is unlikely that a 15 $min \cdot d^{-1}$ exercise bout at a low energy expenditure would be a sufficient stimulus to improve the mechanical efficiency of the MSA (7). Instead, it is proposed that the initial bout of exercise enhanced the rate of acclimation by raising the core and skin temperatures for the duration of the simulation. A greater level of acclimation may have occurred by increasing the stress during the physical fatigue protocol (i.e. increasing time or intensity). However, psychometric analysis of the MSA has indicated that mental confidence plays a large part in a successful outcome of a race (8). It is proposed that as MSA develop acclimation-like adaptations, there is an improvement in their ability to efficiently complete the psychomotor tasks. Successful completion of the psychomotor tasks would promote mental confidence going into a race. It is important that

the MSA finishes the protocol believing that he is competent at performing the vigilance, tracking and reactive tasks required to successfully finish a race.

CONCLUSION

In summary, it has been shown that the environmental simulation protocol is a useful tool in preparing the MSA for the harsh micro-environment that exists within a rallycar. Subjective feedback by subjects, after performing the protocol prior to a rally, indicated that it improved their ability to withstand the high thermal load, while improving their confidence in controlling the rallycar. Undertaking similar psychomotor tasks at a higher stress level than normally occurs during a rally event may heighten the MSA mental performance, when required to perform under actual rallying conditions.

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INVITED LECTURE 8: THE IMPORTANCE OF AEROBIC FITNESS IN DETERMINING TOLERANCE TO UNCOMPENSABLE HEAT STRESS

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A high level of cardiorespiratory fitness has been associated with an improved exercise-heat tolerance since the initial theoretical connection was made by Robinson et al. [1] and Bean and Eichna [2]. These suggestions were based largely on anecdotal evidence, but have been supported by subsequent studies [3-5]. For example, Piwonka and Robinson [3] reported that trained distance runners exhibited a decreased physiological strain compared to untrained individuals during exercise-heat stress. Gisolfi and Robinson [6] were among the first to report a reduction in physiological strain and an improvement in exercise tolerance in compensable heat stress environments following a relatively long-term (6 weeks) interval training program. Four weeks of interval training also produced significant improvements in exercise-heat tolerance, with the improvement reaching a plateau after 8 or more weeks of training of approximately 50% of the adaptive responses brought about by heat acclimation [7].

It has been most common for researchers to select environmental conditions that favour evaporative heat loss when work performance in the heat is examined before and after an endurance training program [8] or when comparisons are made among groups with different aerobic fitness levels [3]. Whether the documented benefits of an increased aerobc fitness on work in the heat would be evident during exposure to more humid environmental conditons that do not favour evaporative heat loss is not as clear. Because nuclear, biological and chemical (NBC) protective clothing restricts evaporative heat loss, it is possible that the increased sweat rate that accompanies an increase in aerobic fitness would have little impact on the rate of heat storage when the clothing is worn [9]. It could be argued that individuals with an increased aerobic fitness would dehydrate faster and actually perform worse while wearing the protective clothing if fluid was not provided during the exercise.

We have recently performed a cross-sectional study comparing the responses of active endurance-trained ($\dot{V}O_{2max}$ of 60 $mL \cdot kg^{-1} \cdot min^{-1}$) versus inactive untrained individuals ($\dot{V}O_{2max}$ of 43 $mL \cdot kg^{-1} \cdot min^{-1}$) during light exercise at 40°C while wearing the NBC protective clothing [10]. Exercise-heat tolerance was greater in the fit compared to unfit individuals, with tolerance times averaging 110 and 88 min, respectively. All subjects walked at the same speed of 3.5 $km \cdot h^{-1}$ so the more fit individuals were exercising at a lower % $\dot{V}O_{2max}$. Nevertheless, individual differences in tolerance time are more closely related to an absolute rather than to a relative expression of metabolic rate because of the limits to evaporative heat loss created by the clothing layers. Interestingly, distinct differences between fitness groups were observed in the reasons for ending the experiment and also in both the initial T_{re} and the final T_{re} at which subjects terminated the experiment. The highly fit subjects generally terminated the trials due to their T_{re} reaching our ethical limit of 39.3°C and felt they could have continued. In contrast, the moderately fit subjects predominantly ended the trial due to exhaustion, with an average final T_{re} of 38.6°C. Overall, our observations during uncompensable heat stress support the general consensus observed during compensable heat stress that an association exists between the level of cardiorespiratory fitness and improvements in physiological responses to exercise in a hot environment [5].

Few studies have compared the heat tolerance response of unfit subjects during uncompensable heat stress before and after a controlled endurance training program designed to improve aerobic fitness. We have examined this response with the use of 2 different training models. Our first attempt used the more classical endurance training model that involved typical progressions in intensity, frequency and duration over an 8 week period [11]. This training program increased $\dot{V}O_{2max}$ approximately 15% from 40 to 46 $mL \cdot kg^{-1} \cdot min^{-1}$ and produced significant decreases in heart rate and T_{re} responses during 2 hours of compensable heat stress exposure at 40°C. In addition, control subjects showed no change in $\dot{V}O_{2max}$ over the 8-week period and no change in their thermoregulatory or cardiovascular response during the 2 hours of compensable heat stress. However, the endurance training program offered no benefit to the subjects during the uncompensable heat stress which involved wearing the NBC protective clothing ensemble. Tolerance times remained unchanged during the heavy exercise at 50 minutes and T_{re} and heart rate responses also were unaffected by the training program. Interestingly, sweat rates were elevated after the training but evaporative heat loss was unchanged when the protective clothing was worn indicating that the characteristics of the clothing determine the amount of evaporative heat loss to the environment.

Our next approach [12] was to use a short-term aerobic training model which had been reported to induce rapid cardiovascular and thermoregulatory changes during submaximal exercise [13] following 3 to 14 daily aerobic training sessions at 60 to 80% $\dot{V}O_{2max}$ for 1 to 3 hours. We rationalised that in a military environment there may be little time available prior to deployment to increase aerobic fitness and also that many military personnel may be unwilling to commit to the long-term training required to achieve aerobic fitness levels equivalent to a $\dot{V}O_{2max}$ of 60 $mL \cdot kg^{-1} \cdot min^{-1}$. As a result, unfit subjects with a $\dot{V}O_{2max}$ below 45 $mL \cdot kg^{-1} \cdot min^{-1}$ were evaluated before and after 12 days of treadmill walking at 65% $\dot{V}O_{2max}$ for 1 hour each day [12]. The 12 days of training led to significant decreases in the rise in T_{re} and heart rate at the end of the 1 hour of walking in a thermoneutral environment. During the heat-stress test which involved light exercise walking at 3.5 $km \cdot hr^{-1}$, sweat rate was increased but evaporative heat loss from the clothing ensemble was unchanged after the 12 days of training. In addition, there were no changes in the heart rate or T_{re} responses and tolerance times approximated 90 min regardless of the state of training.

The ability of short-term aerobic training of unfit individuals to replicate the decreased physiological strain and elevated exercise-heat tolerance observed in individuals with a high level of aerobic fitness is of direct occupational interest. In many occupational settings, workers may be required to work in hot environments with minimal preparation time to significantly increase aerobic fitness or facilities to perform heat acclimation through heat exposures. However, our cross-sectional analyses suggests that, when wearing protective clothing, short-term aerobic training is not an adequate substitute for a high level of aerobic fitness resulting from habitual exercise and training [14]. We observed that, following two weeks of aerobic training, cardiovascular and thermoregulatory strain during uncompensable heat stress were similar in individuals of low to moderate fitness compared with those of high fitness. However, the range of core temperature that could be tolerated during the heat exposure remained significantly lower in the less fit individuals, as did the final T_{re} before the onset of voluntary exhaustion and overall tolerance time.

Based on the findings from these studies [11, 12, 14] we would have to conclude that aerobic training programs lasting from 2 to 8 weeks offer little benefit to work performance in the heat for previously unfit subjects when protective clothing is worn. However, the cross-sectional analyses presented above support the importance of aerobic

fitness to improve work performance when a very hot and humid microenvironment is created with the wearing of protective clothing.

Therefore, other factors, besides elevations in sweat rates that accompany improvements in aerobic fitness, must be involved in explaining the differences in heat tolerance between aerobically fit and unfit subjects. Certainly, the lower resting core temperature [8, 10] and the higher core temperature tolerated at exhaustion for subjects with higher aerobic fitness levels [10] are factors that will increase the work time when the protective clothing is worn. In addition, differences in body fatness between fitness groups [10] could account for some of the differences in response during the heat-stress exposure [15].

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PAPER 13: THE EFFECTS OF WEARING SUNSCREEN LOTION ON THERMOREGULATORY RESPONSES DURING EXERCISE IN THE HEAT IN ADULT AND ADOLESCENT MALES.

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INTRODUCTION

In recent years within Australia, there have been major public health campaigns encouraging the wearing of sunscreens when outdoors in order to avoid major skin damage and disease. Although such practice reduces skin damage, it is not clear whether the wearing of sunscreen compromises the capacity of exercising individuals to maintain core temperature within safe limits.

Two previous studies have investigated the effects of sunscreen on thermoregulatory responses during exercise [1,5]. The studies had conflicting results, with one study suggesting a favourable thermoregulatory effect of sunscreen [1] and the other study indicating an inhibitory effect of sunscreen during exercise in the heat [5]. Both of the previous studies were conducted on adults. Younger populations are also encouraged to wear sunscreen during exercise in the heat. Thermoregulatory responses in adolescent populations during exercise remain unclear in the literature.

Compared with adults, pre-adolescents have less efficient sweat loss mechanisms [2,4]. Uncertainty surrounds the exact stage of puberty at which sweating responses during exercise mature. Sweating rates appear to be higher at the end of puberty than at the beginning. Falk et al. [3] investigated the thermoregulatory responses of pre-, mid- and late pubertal males to exercise in dry heat (42°C and 20% relative humidity). Ten (aged 12.2 years), 13 (aged 13.6 years) and 8 (aged 16.7 years) males represented pre-, mid-, and late pubertal stages, respectively in this study. These participants performed three bouts of 20 minutes of cycling at 50% $\dot{V}O_2$ peak with 10 minutes rest between each bout. In this study, differences were noted in the population density of heat activated sweat glands (HASG). A higher number per cm^2 of HASG were observed in the pre-pubertal group compared with the late pubertal group. Importantly, sweat rate ($mL.min^{-1}.m^{-2}$) was lower ($p<0.05$) in the pre-pubertal group compared with the late pubertal group. Therefore the application of sunscreen may produce a different thermoregulatory response in adolescents who exercise in the heat when compared with adults.

The aim of the study was to examine the effects of wearing sunscreen on the thermoregulatory and cardiorespiratory responses in adolescent and adult males who performed 60 minutes of cycling at 60% of their maximal effort under hot conditions.

The study recruited 9 adult and 8 adolescent well-trained males. Participants were training and competing regularly in either cycling or triathlon events. They were also non-acclimatised and needed to have a peak oxygen uptake equal to or greater than 55 $mL.kg^{-1}min^{-1}$.

Following a familiarization trial, participants performed a peak O_2 consumption ($\dot{V}O_2$ peak) test. It was conducted using an incremental workload protocol (25 watt per minute) on an electronically braked Lode, Excalibre Sport cycle ergometer (Groningen, The Netherlands) at a cadence range of 80 – 100 rpm. Twenty-five watt increments were imposed per minute until volitional fatigue or a >30% decrement in cadence occurred over approximately 30 seconds duration. Expired air was analysed on-line using open circuit spirometry (Applied Electrochemistry S-3A and CD-3A analysers, Ametek, PA, USA) and

expired volumes were measured with a Pneumotach spirometer. Heart rates were monitored in all testing with a transmitter/receiver telemetry unit (Polar).

During the two randomly assigned environmental trials (36°C and 50% RH), participants cycled for 60 minutes at 60% of their previously determined peak $\dot{V}O_2$. Measurements of rectal temperature, skin temperatures, heart rate, and oxygen uptake were taken prior to exercise and continued on-line throughout the trials. Experimental trials were terminated when a participant completed the exercise task or when any of the following criteria were observed; a rectal temperature greater than 39.5°C, or any observation or report of nausea, weakness, or dizziness. To avoid hypohydration during exercise, participants consumed 400 ml of water 20 minutes prior to exercise and 150 mL of water every 10 minutes during the trial. Before the sunscreen trails, the lotion was applied liberally to all exposed body parts in the amount of $30ml \cdot m^2$ of exposed BSA parts (Connolly, 1996). Estimates of sweat loss were made by weighing the participants before and after the completion of the testing and correcting for water consumed and respiratory water loss. Evaporative sweat loss was calculated by subtracting the non-evaporative (drip) loss from the total sweat loss. Evaporative heat loss index ($g.hr^{-1}.W^{-1}$) was derived from the evaporative mass loss per unit of heat production.

RESULTS

Differences ($p<0.05$) between the adolescents and adults were noted in age, body mass, height and BSA (Table 1). No differences ($p>0.05$) were reported in the maximal effort characteristics of peak $\dot{V}O_2$, heart rate max and RER max (Table 2).

	Adults (N = 9)	Adolescents (N = 8)
Age (yr)	21.5	15.1*
	± 0.8	± 0.9
Body Mass (kg)	77.0	57.2*
	± 1.9	± 3.8
Height (cm)	180	168*
	± 3	± 3
BSA (m^2)	1.95	1.64*
	± 0.04	± 0.07
BSA:Mass Ratio (cm^2/kg)	255	293*
	± 2.5	± 7.8

Mean ± SEM, * denotes difference between groups ($p<0.05$)

Table 1: Descriptive Characteristics

	Adults	Adolescents
$\dot{V}O_2$ max ($ml.kg^{-1}\ min^{-1}$)	58.55	61.10
	± 3.44	± 2.40
Heart Rate Max ($b.min^{-1}$)	193	190
	± 2	± 3
RER Max	1.21	1.17
	± 0.02	± 0.03

Mean ± SEM

Table 2: Maximal Effort Profile

Statistical analysis revealed no interaction between the effects of sunscreen and age groups ($p>0.05$) on all thermoregulatory and cardiorespiratory measurements in this study. A selection of these responses is listed in Table 3.

	Sunscreen (N=17)	No sunscreen (N=17)
Body mass loss (kg)	1.27 ± 0.05	1.31 ± 0.07
Relative body mass loss (%)	1.90 ± 0.09	2.03 ± 0.18
Rectal temperature range (°C)	1.30 ± 0.10	1.37 ± 0.10
Mean skin temperature (°C)	35.20 ± 0.13	35.5 ± 0.17
Mean heart rate ($b.min^{-1}$)	155 ± 2	155 ± 2

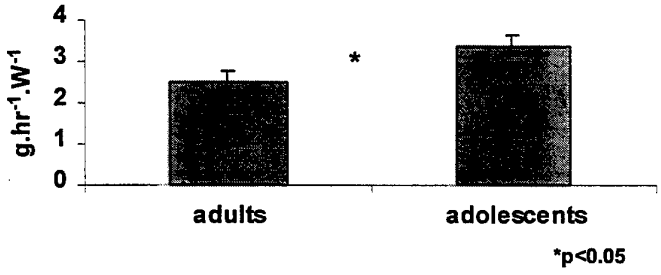
Mean ± SEM

Table 3: Results from Sunscreen and No Sunscreen Trials when Both Groups Combined

The data from the two trials in the heat were subsequently combined and a main effect for age was examined. Some age-related differences ($p<0.05$) were evident during the heat trials. For example, the mean absolute body mass loss was similar for adults (1.30 ± 0.06 kg) and adolescents (1.28 ± 0.05 kg). Relative body mass loss was however, lower for adults (1.68 ± 0.10 kg) when compared with adolescents (2.31 ± 0.13 kg) ($p<0.05$).

Differences ($p<0.1$) were observed in the respective adult and adolescent means for percentage of the total sweat loss attributed to the non-evaporative sweat (drip) loss (16.10 ± 1.55 and 7.78 ± 1.23%) and the evaporative sweat loss (83.69 ± 4.15 and 92.21 ± 1.23%). The calculated evaporative heat loss index presented in Figure 1 demonstrates the higher evaporative heat loss ($p<0.05$) in adolescents compared with adults. The higher mean skin temperature of adolescents compared with adults (35.59 ± 0.14 and 35.14°C ± 0.14 °C, respectively) may reflect the greater evaporative heat loss efficiency of the younger participants.

Figure 1: Evaporative Heat Loss Index



Differences ($p<0.05$) occurred in the mean values for rectal temperature (37.8 ± 0.11°C and 38.2 ± 0.09°C for adults and adolescents, respectively). The two age groups were not different however, when the delta temperature (rest to peak temperature) during the trials was calculated (1.37 ± 0.11 and 1.30 ± 0.10°C, respectively). Differences were also not apparent in the mean exercise heart rates (159 ± 5 and 153 ± 4 $b.min^{-1}$) of the two age groups during the two trials.

Table 4 presents total sweat loss in absolute and relative terms. Once again the adolescents appeared to be more efficient in sweat produced per unit of body surface area as well as in the amount of sweat produced per unit of heat produced.

Sweat index	Adults	Adolescents
Absolute rate ($L.hr^{-1}$)	1.25 ± 0.06	1.26 ± 0.05
Rate per BSA ($g.hr^{-1}\ m^2$)*	642 ± 40	778 ± 37
Sweat per heat produced (%)**	2.98 ± 0.24	3.65 ± 0.23

Mean ± SEM BSA = body surface area, * = $p<0.05$, ** $p = 0.054$

Table 4: Absolute and Relative Total Sweat Loss

CONCLUSION

The application of sunscreen lotion was not detrimental to thermoregulatory and exercise performance of the adult and adolescent athletes who participated in this study. The results suggest somewhat different thermoregulatory mechanisms in adolescents than adults during cycling in hot conditions. This was demonstrated in a lower relative non-evaporative sweat loss, and greater evaporative heat loss efficiency in adolescents compared with adults.

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PAPER 14: WHOLE-BODY PRE-COOLING: THERMAL, CARDIOVASCULAR AND METABOLIC CONSEQUENCES.

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INTRODUCTION

Elevated cutaneous blood flow, in response to exercise in a hot environment, causes profound changes in the central circulation, culminating in competition between the skin and muscle for the available cardiac output [1, 2]. Substantial mobilisation of the central blood volume, and partitioning of this volume between active muscle and the cutaneous circuit, eventually reduces cardiac filling pressure and stoke volume, requiring an elevated cardiac frequency to maintain cardiac output and blood pressure [1, 2]. To compensate for peripheral pooling of blood, vasoconstriction is elicited within the compliant splanchnic and renal beds, translocating a considerable volume of blood in an attempt to defend a decline in mean arterial pressure (MAP) and blood flow to both the active tissue and the cutaneous vasculature.

However, during prolonged exercise in the heat, stroke volume continues to decrease, and once a maximum cardiac frequency is attained, cardiac output fails to increase further, resulting in a MAP decline, which activates baroreceptor reflexes [1, 3]. Baroreceptor unloading attenuates active cutaneous vasodilation, eliciting a plateau in skin blood flow. Insufficient dissipation of endogenous and exogenous heat ensues, causing a progressive rise in core temperature (T_c), and impending hyperthermia [3, 4]. This pronounced rise in T_c and muscle temperature (T_m) can also evoke metabolic pertubations, which themselves may impair work performance. An increase in tissue temperature has been linked to increased anaerobic adenosine triphosphate production [5], increased muscle glycogen utilisation [6], and decreased lipid metabolism [7].

Recent research has indicated that whole-body pre-cooling can maintain exercise performance in the heat by reducing thermal strain and delaying metabolically induced hyperthermia during uncompensable thermal loading [8, 9]. Furthermore, when exercise is performed in a cool environment, both the rise in T_c and T_m, and the metabolic disturbances are less pronounced [10]. Whole-body pre-cooling likewise attenuates the exercise-induced rise in body temperature, and increases endurance during exercise in a hot environment [9]. This increased exercise endurance has primarily been attributed to a reduction in thermoregulatory and cardiovascular strain. Nevertheless, it may also be related to an influence of pre-cooling on muscle metabolism.

The purpose of this study was first to examine the impact of whole-body pre-cooling on the thermal and cardiovascular responses to exercise in hot, humid conditions. Second, we tested the hypothesis that exercise in the heat, following whole-body pre-cooling, would be accompanied by marked reductions in T_c and T_m, possibly resulting in reduced muscle glycogen utilisation and muscle lactate production, with a concomitant increase in lipid oxidation.

MATERIALS AND METHODS

Eighteen males performed two separate 35-min cycling trials (60% peak aerobic power) in hot conditions (35°C, 50% r.h.), either with or without (control) whole-body pre-cooling. Pre-cooling was achieved by whole-body water immersion to the axilla (29°C-24°C).

Stroke volume (Q) was measured using impedance cardiography. The first derivative of transthoracic impedance (dZ/dt) was collected at 100 Hz for 15 s. Stroke volume was

determined from inspection of individual waveforms, and reported as a single mean for each measurement period. Cardiac frequency (f_c) was recorded from electrocardiograph analyses and cardiac output ($\dot{Q}$) was derived from the product of Q and f_c.

Forearm blood flow ($\dot{Q}_F$) was measured using venous occlusion plethysmography, with a mercury-in-silastic strain gauge. The venous cuff was inflated to 50 mmHg for 10 s of every 30 s, during the first 15 min of exercise. Thereafter, $\dot{Q}_F$ was measured for 2 min, at 5-min intervals. Circulation to the hand was occluded with a wrist cuff inflated to >200 mmHg, which was released periodically between data collection, then reinflated. Forearm blood flow measurements commenced at least 30 s after wrist cuff inflation. The increase in $\dot{Q}_F$, during dynamic leg exercise, has been shown to be restricted to the skin [11, 12].

Muscle (T_m: *vastus lateralis*) and oesophageal temperatures (T_{es}) were measured continuously. Blood samples were collected from an antecubital vein throughout each trial, with blood glucose, free fatty acid ([FFA]), and lactic acid concentrations determined enzymatically. Muscle biopsies, collected from the *vastus lateralis* prior to immersion and after exercise, were analysed for muscle glycogen and muscle triglyceride contents, with muscle lactate determined fluorometrically.

RESULTS

Significant treatment effects were observed for T_{es} and T_m. Throughout the trial, the mean pre-cooled T_{es} and T_m were 0.5°C and 1.6°C lower (respectively) than for the control condition. Whole-body pre-cooling resulted in significant reductions to both Q and $\dot{Q}$, relative to control ($P<0.05$). Similarly, $\dot{Q}_F$ was markedly attenuated during exercise ($P<0.05$). Since the mean body temperature at the vasodilatory threshold flow following pre-cooling was raised by 0.59°C (±0.19; $P<0.05$), this implies that pre-cooling acted to delay cutaneous vasodilation.

Pre-cooling did not significantly alter plasma [FFA], glucose or lactate concentrations throughout exercise ($P>0.05$). Intramuscular triglyceride utilisation was not altered by pre-cooling. Whilst muscle glycogen utilisation tended to be lower following pre-cooling ($P>0.05$), the muscle lactate concentration was not significantly different at the end of exercise.

DISCUSSION

Pre-cooling evoked a marked decrease in all cardiovascular variables measured. However, the reduction in cardiac output observed in this study is somewhat contrary to that which may be expected. Initially, whole-body pre-cooling should augment venous tone, eliciting an increase in Q, and possibly $\dot{Q}$. This apparent disparity may be accounted for if whole-body pre-cooling allowed for a better maintenance of MAP, thereby lessening the need to either elevate $\dot{Q}$, or to recruit blood from splanchnic and renal reservoirs to defend blood pressure. Pre-cooling therefore permitted $\dot{Q}$ to remain lower throughout exercise, relative to the control condition. This implies that pre-cooling reduces cardiovascular strain during subsequent combined exercise and thermal stress, which may help account for previous observations of improved physical performance associated with pre-exercise cooling.

Whilst whole-body pre-cooling evoked markedly lower T_{es} and T_m during exercise in the heat, there was a limited affect upon exercise metabolism. In contrast to previous work [6], attenuating the body temperature elevation appeared not to result in a preferential use of lipids. Moreover, equivalent muscle lactate and blood lactate responses indicate that the anaerobic contribution to adenosine triphosphate production was not altered following whole-body pre-cooling. Whilst there was a tendency for muscle glycogen utilisation to be

reduced in the pre-cooled condition, carbohydrate depletion is not generally recognised as a limiting factor to exercise endurance in a hot environment [13]. With the similar pattern of metabolism observed for the control and pre-cooled trials, it is unlikely that, following whole-body pre-cooling, altered muscle metabolism can explain the increased endurance previously noted during exercise in the heat. Rather, reduced thermoregulatory and cardiovascular strain, associated with marked reductions in T_{es} and T_m, might be more appropriate possibilities.

CONCLUSION

This study has demonstrated that whole-body pre-cooling can substantially reduce cardiovascular strain, possibly due to reduced competition between intramuscular and cutaneous vascular beds for the available blood volume. Such a strain reduction may help account for the observation that, during exercise in hot, humid conditions, physical performance is improved following whole-body pre-cooling.

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All experiments were approved by the University's Human Research Ethics Committee.

PAPER 15: ENHANCED CUTANEOUS BLOOD FLOW AND HEAT OF SORPTION AFTER THE ONSET OF SWEATING DURING HEAT LOAD

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INTRODUCTION

When a highly hygroscopic fabrics such as cotton absorbs moisture, heat is evolved. The more hygroscopic the fiber, the greater the amount of heat evolved. Some studies have been conducted on the relationship between thermal responses and moisture absorption of fabrics during ambeint humidity transition. Heat changes in hygroscopic fabrics due to relative humidiy changes were of a sufficient magnitude to be readily perceived [1]. There were several studies which measured the effects of heat of sorption induced by sweating on thermoregulatory responses during exercise, when subjects wore some of layers of clothing in a constant environment. A tendency of higher mean skin temperatures in hygroscopic fabrics was observed, but not significantly [2].

Gavhed et al. [3] demonstrated that mean skin temperature was significantly higher in hygroscopic fiber (wool) than poor-hygroscopic (synthetic) fiber, indicating a higher degree of vasodilation. However, they have not determined cutaneous blood flow. There is no direct evidence for the effect of heat of sorption *per se* on cutaneous vasodilation during heat stress when subjects wore clothing ensemble. The effects of heat of sorption *per se* on these studies using two or three layers of clothing, might be masked by some other factors such as air layer between clothing layers [3]. Therefore it is necessary to clarify the effects of heat of sorption with a single layer of hygroscopic fabrics on thermoregulatory cutaneous blood flow responses.

Cutaneous blood flow in the nonacral regions is under dual sympathetic nervous control, by the noradrenergic vasoconstrictor system and by the active vasodilator system [4]. It has been postulated that active vasodilation is linked to sudomotor nerve activity [5]. Recently, it has been reported that cutaneous vasodilator responses are synchronized with sweat expulsions suggesting sudomotor activity [6]. We have proposed that vasoconstrictive and active vasodilative activities may be separated by plotting skin blood flow as a function of local sweating rate [7]. Moreover, our recent study showed that changes of microclimate vapor pressure was good index of local sweating rate during heat load in clothed subjects (unpublished data).

The aim of this study was to investigate the effects of heat of sorption per se on the relationship between cutaneous blood flow and microclimate vapor pressure change as an index of local sweating rate during heat load, under the influences of hygroscopic and poor-hygroscopic fabrics with a single layer of clothing at constant temperature and humidity environments.

MATERIALS AND METHODS

Age (yrs)	Height (cm)	Weight (kg)	Body Surface Area (m^2)	Body Fat (%)
21.9 ± 0.6	160.6 ± 2.3	55.4 ± 2.3	1.52 ± 0.04	23.5 ± 1.5

Table 1. Physical characteristics of subjects.

Two experiments (Expt 1 and 2) were conducted in the present study. Seven female subjects participated in the expt 1, their physical characteristics were as shown in Table 1. They participated at the same time of the separate two days in the same phase of their menstrual cycle. The subjects ingested a fixed light meal 3 hours before the experiment

start, and after that no food or water was taken until the end of the experiment. Subjects wearing clothing ensembles made of either hygroscopic 100% cotton (C) or weakly hygroscopic 100% polyester (P), immersed their lower legs and feet in a water bath at a temperature (T_w) of 35 for 10 min in a climatic chamber at an ambient temperature (T_a) of 27.20.5, relative humidity (RH) of 503% and air velocity of 0.2 m/sec. And T_w was raised from 35 to 41 taking 15min, and then kept at a constant of 41 for 45min until the end of the experiment. Rectal (T_{re}) and skin temperatures (T_{sk}) were measured using thermistors and forearm skin blood flow (SBF) was measured by a laser-Doppler flowmeter. Clothing microclimate temperature and vapor pressure (VP_{cm}), and clothing surface temperature (T_{cs}) and vapor pressure were measured at the three sites of the chest, arm and thigh using temperature-humidity sensors. Handmade frame-spacers (3 x 3 x 2 cm) were inserted between the skin and the clothing in the three skin sites, to keep constant clothing ease. Subjective thermal and comfort sensations were recorded every 5 min.

In expt 2, to simulate the clothing microclimate environment after the onset of sweating, C- and P-clothings were exposed from 50% to 95%RH at a constant T_a of 27.2 for 60min. T_{cs} in both clothings were measured via a thermograpy system.

RESULTS AND DISCUSSION

In expt 1, changes of SBF and mean T_{sk} were significantly higher in C- than in P-clothings ($P<0.05$) after the onset of sweating suggested by sudden increase of VP_{cm} during heat load. Also these responses were accompanied with significantly warmer and more uncomfortable sensations in C than in P, despite a significantly smaller increase of T_{re} in C comparing with P and identical changes of mean body temperature in both clothings.

Figure 1 shows an example of the relationship between SBF and VP_{cm} in both C- and P-clothings. In the relationship between SBF and VP_{cm}, two temporal phases were confirmed durng lower-legs water immersion, 1) an increase of SBF without an increase in VPcm, 2) the proportional increase of SBF and VP_{cm}.

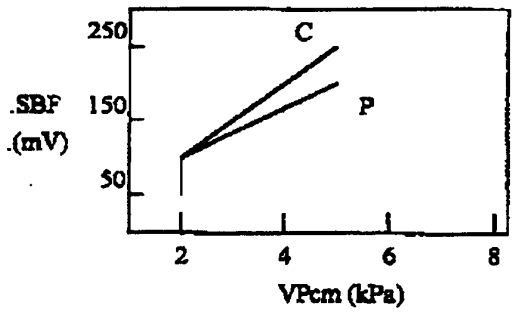

Figure1: An example od the relationship between skin blood flow (SBF) and clothing microclimate vapor pressure (VP_{cm}) in cotton (C)- or polyester (P)-clothed subjects during heat load.

In expt 2, T_{cs} rose by 2.2°C for C and by 0.5°C for P during humidity transients.

These results clearly showed that C-clothing produced greater amount of heat of sorption comparing with P after the onset of sweating during heat load. The heat of sorption in C enhanced rise in SBF through probably active vasodilator system. Because the slope of the line showing the relationship between SBF and VP_{cm} was higher in C than in P (Figure 1).

CONCLUSIONS

These results suggested that heat of sorption *per se* in hygroscopic fabrics after the onset of sweating enhanced thermoregulatory cutaneous blood flow through active vasodilator system.

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INVITED LECTURE 9: NUTRITIONAL NEEDS IN THE HEAT

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INTRODUCTION

Since many important competitive events in recent times have been conducted in hot climates, and most sports require year-round conditioning, it is now common for athletes to undertake considerable periods of training and competition in the heat. Although these conditions are not typically conducive to optimal performance, nutritional strategies play an important role in assisting the athlete to perform as well as possible. Exercising in the heat creates special nutritional needs, which must be balanced against its lesser known effects on appetite and voluntary intake of fluid and food. This paper will highlight the major issues in nutrition for sports performance in the heat, summarising some practical recommendations for achieving nutrition goals. Strategies will be considered for chronic periods of exercise in the heat (i.e. training needs) as well as acute strategies for optimising performance (i.e. competition strategies).

SUMMARY OF EXERCISE METABOLISM IN THE HEAT

The most notable effect of exercise in a hot climate is to increase fluid losses. In a hot climate, the chief method of dissipating body heat produced by the exercising muscles or absorbed from the environment is via the evaporation of sweat. In hot environments, sweat losses during sustained moderate intensity, or intermittent high intensity exercise may be as high as 2-3 L per hour. Despite the benefits of ingesting fluid to offset dehydration, there is a gradual rise in body core temperature, which causes a premature onset of fatigue, or a reduced performance, compared with exercise in cooler conditions. - Exercise in a hot environment is associated with a shift in substrate utilisation towards increased reliance on carbohydrate (CHO). This is manifested as increases in respiratory exchange ratio, muscle glycogenolysis, liver glucose production, and lactate accumulation [1]. Although strategies to enhance CHO availability during exercise in the heat may be useful, particularly during exercise of lower intensity or intermittent high-intensity performance where there is some opportunity for heat dissipation, thermoregulatory issues are typically the limiting factor in determining exercise performance.

There is some evidence that exercise in the heat causes an increase in protein degradation during exercise, in the form of an increase in ammonia accumulation from the oxidation of branched chain amino acids [2]. There is also indirect evidence of greater oxidative damage to muscle cells, perhaps via increased generation of free radicals.

FUEL REQUIREMENTS

Since exercise in the heat is likely to produce a greater CHO fuel cost, we might speculate that athletes have a greater general requirement for dietary CHO, especially when undertaking an intensive training program. Strategies to enhance recovery of muscle glycogen between training sessions should be undertaken, including timely intake of carbohydrate after the training session, and focus on CHO-rich eating patterns which supply a daily CHO intake of 7-10 g/kg body weight/d. Intake of CHO-containing fluids during prolonged (> 60 min) training sessions may enhance the performance of each work-out as well as add to the total daily CHO intake. Since appetite can be depressed in hot weather and by the fatigue of an increased training stress, the athlete may need practical guidance to choose an eating pattern based on palatable meals and snacks. Fluid-rich CHO foods (e.g. fruit, flavoured yoghurts), and CHO-containing beverages (juices, sports drinks,

fruit smoothies) are useful for their appetite appeal as well as a nutrient profile that provides fluid and carbohydrate simultaneously.

Competition nutrition, particularly in the case of prolonged events that typically deplete body CHO stores, should include dietary strategies that promote CHO availability. These include tapering training and consuming CHO to normalise muscle glycogen stores in the days before an event (or CHO loading in the case of events over 90 minutes duration), consuming a CHO-rich meal or snack in the hours prior to an event, and consuming CHO-containing fluids during the event. Most studies of these interventions have been undertaken in moderate environments; there is little specific research to document how well the performance benefits transfer to exercise in hot conditions, where the dual issues of increased CHO utilisation and an increased problem of thermoregulatory concerns co-exist. It is unlikely that strategies to enhance CHO availability will detract from performance in the heat, since they do not compromise fluid availability. At the worst they may fail to be as effective as when practised in moderate climates, since thermoregulation may limit performance before CHO availability becomes an issue.

FLUID NEEDS

Being hypohydrated, caused by the failure to replace sweat losses during exercise, increases the rise in body core temperature, reduces cardiovascular function, increases the perception of effort and impairs performance. Fluid intake during exercise can overcome or reduce these problems, and its importance as a nutritional strategy for sport is greater during exercise in hot conditions where sweat losses and fluid deficits are increased. Indeed, it provides an additional benefit of reducing muscle glycogen utilisation, probably by attenuating both circulating epinephrine concentrations and muscle temperature [3]. Since fluid needs take priority over CHO availability in many exercise situations in the heat, it has been suggested that plain water might be sufficient as an exercise drink. However, CHO ingestion may be of benefit to some events and in such situations the benefits of fluid and CHO are independent and additive [5]. Even if CHO intake is unnecessary, the ingestion of sports drinks providing 4-8% CHO and electrolytes does not impair fluid delivery. More importantly, the palatable taste of these drinks has been shown to increase voluntary intake of fluid during and after exercise. This is an important consideration since, typically, athletes voluntarily do not replace fluid needs fully across a range of sporting events and should implement a range of behavioural and stimulatory strategies to promote greater fluid intake.

Just as thirst does not guarantee adequate fluid intake in a single situation of exercise, there is a time lag of several days before voluntary intake of fluid increases to meet the increased daily fluid requirements of a hot climate. Athletes who suddenly move to a hotter environment should implement deliberate strategies to increase fluid intake. Hydration strategies need to be implemented in a cyclical fashion: to drink as much as is comfortable and practical during the exercise session, to rehydrate aggressively after the session, and to top-up just prior to the next session. Recovery of significant fluid deficits after exercise is assisted by simultaneous replacement of electrolyte losses.

In competitive events where the build-up of a large fluid deficit is unavoidable, there may be benefits from hyperhydrating prior to the event. This can be achieved by drinking a large quantity of fluid in the hours leading up to the event, but the athlete will have to cope with the disadvantages of an increased and perhaps untimely urine loss. Recent research has investigated the use of glycerol as a hyperhydrating agent. When a small amount of glycerol is consumed with a large volume of fluid in the hours prior to exercise the additional osmotic pressure allows the retention of an additional 500-600 mL of fluid. Some studies have shown this to benefit the performance of exercise undertaken in the heat [4], but the mechanism of action remains elusive. Since there are some conflicting reports

and a possibility of side-effects, glycerol hyperhydration should remain a strategy undertaken under supervision and tested before important events.

OTHER ISSUES

There is preliminary evidence that exercise in the heat increases protein oxidation and cellular damage due to generation of free radicals. Insufficient research has been undertaken to determine whether this translates into additional requirements for protein, anti-oxidant vitamins and minerals, or other micronutrients. There may be some advantages to increasing protein intake and supplementing with anti-oxidant vitamins in the early days of exposure to a hot climate, however these strategies have not been tested.

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PAPER 16: EXERCISE NORTHERN AWAKENING: NUTRITION STUDY

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INTRODUCTION

The question of vitamin status has been raised following a recent survey of the general population showing that up to 25 percent of apparently healthy people have a vitamin B deficiency [1]. This result has reinforced the need for a much closer monitoring of nutritional status. This is of particular relevance to the Australian Defence Force, which requires a high standard of fitness amongst its personnel.

Prompted by high rates of infectious disease amongst Ranger students, a detailed evaluation of the nutritional status of soldiers undergoing the US Army Special Forces Selection (Ranger) Course was conducted during 1991 [2]. Insufficient energy and extreme environmental conditions experienced by these soldiers resulted in decreased protection against infection (decreased T-lymphocyte function). This was an alarming outcome given that even minor infections can greatly reduce a soldier's operational effectiveness.

There is rising international defence concern regarding the safety of long term CRP usage. The major outcome of three years of deliberation by Action Group 16 of Group Human Resources and Performance of The Technical Cooperation Program (TTCP), is an agreement that Australia will be the lead nation for a series of detailed nutrition studies.

Australian and international field evaluations of the use of combat ration packs (CRP) have been reviewed [3]. That review, which provided the background rationale for the present study, highlighted that few international studies and even fewer Australian studies have documented the effects of long-term CRP usage. In fact, there have been no evaluations of the effects of long term consumption of Australian CRP since the 1960's. A common feature of long-term CRP usage is weight loss. In most cases these weight losses are believed to be tolerable. However, some alarming findings of US evaluation studies revealed measurable decrease in muscle strength and immunocompetence as well as weight loss after one month of CRP consumption as sole nutrition.

The aim of the present study was to evaluate the nutritional adequacy of Australian CRP as sole source of nutrition over two to three weeks by use of dietary, biochemical, physiological and psychological means.

MATERIALS AND METHODS

Subjects: Volunteers were Airfield Defence Guards from 2nd - Air Force Defence Squadron, RAAF Base Amberley, Queensland. Subjects were allocated to three treatment groups; Full CRP (15000 kJ, n = 10), Half CRP (7500 kJ, n = 10) and Freshly-Prepared Meals (15000 kJ, n = 13). The subjects were involved in a ten-day training exercise as part of Exercise Northern Awakening, conducted during April – May 1999 at RAAF Base Scherger, Far North Queensland.

Dietary intake was measured by recording all food discards. Strict portion control was used for issue of CRP and fresh foods.

Energy expenditure was estimated by use of the doubly-labelled water method [4]. Four subjects in each CRP group, who were chosen to represent the range of body weights, were studied.

Body composition: Bioelectrical impedance, skin-fold (four sites), weight measurements and D_2O dilution were used to estimate changes in body composition.

Muscular strength & endurance was estimated by use of the Military Physical Fitness Test and measurement of hand grip strength.

Nutritional status: Changes in skeletal muscle turn-over was estimated by measurement of urinary 1- and 3-methylhistidine. Visceral protein status was estimated by measurement of plasma insulin-like growth factor and fibronectin. Micronutrient status was estimated by measurement of plasma total homocysteine and total antioxidant capacity as well as measurement of vitamin status (vitamin K, PIVKA, folate, vitamin B-6, thiamin and riboflavin). Immune status was monitored by use of the delayed cutaneous hypersensitivity tests and measurement of salivary IgA and plasma cytokines (IL2, IL2R, IL6).

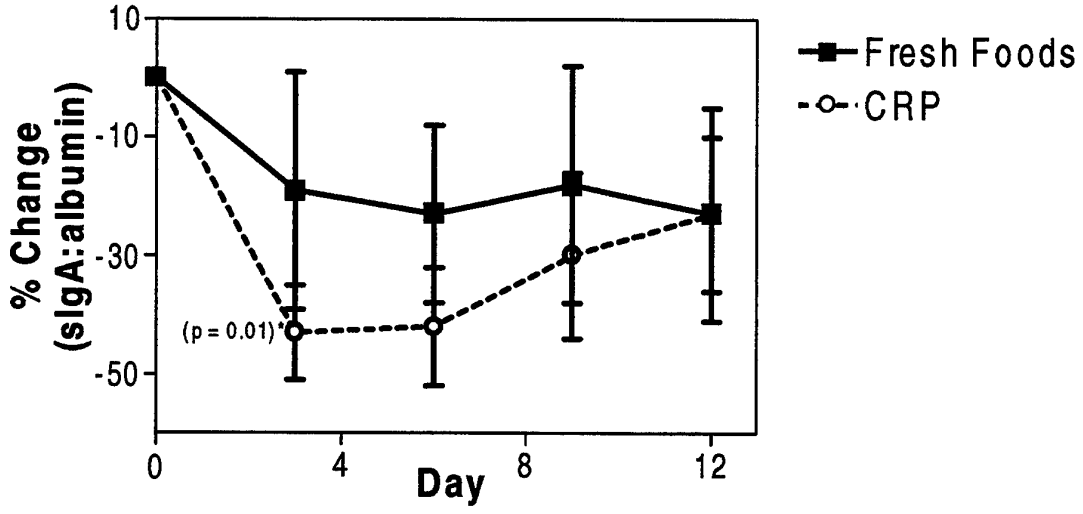
Psychological status: Cognitive function was determined by use of PC-based programs developed by the US Army Research Institute of Environmental Medicine (USARIEM). Changes in mood were monitored by the Profile of Mood States questionnaire and symptoms related to the harsh environmental conditions were monitored using the Environmental Symptoms Questionnaire. Sleep quality was estimated using Actigraph® Loggers worn on the wrist.

RESULTS AND CONCLUSIONS

The CRP nutrition study highlighted the problems with CRP design and usage. Subjects in the Full CRP group had 15,000 kJ of food energy available, but on average only managed to eat 9,000 kJ. Subjects in the Half CRP group consumed an average of 6,500 kJ of energy. The average total energy expenditure of these two groups was 15,500 kJ (no significant difference between CRP groups). This finding indicates that the military performance of some soldiers subsisting on CRP is at risk of being severely compromised. The nutrient most at risk - due to subjects discarding CRP items - was carbohydrate. This is the macronutrient of greatest value to active people such as soldiers on operations. Clearly, more acceptable high carbohydrate foods need to be found for inclusion in CRP.

Although the biochemical analyses are not yet completed and the collected data not yet fully analysed, some conclusions can be drawn from this study. In general, the control subjects, who received freshly prepared meals (15000 kJ) maintained their body weight and body composition and their nutritional and immune status was maintained or even slightly improved in some cases. The status of the subjects fed CRP tended to decline (not all results were $P < 0.05$) with no significant difference between Full- and Half-CRP groups. CRP-fed subjects lost weight in the form of fat tissue after ten days of a patrol exercise. Although all subjects showed signs of stress (psychological and biochemical), the CRP-fed subjects coped less well. All subjects experienced a highly disruptive and poor sleep quality with no apparent effect of dietary treatment.

As shown in the Figure, all subjects displayed reduced salivary IgA concentrations ($P < 0.05$) and the reductions were more pronounced in subjects receiving CRP ($P < 0.05$ for days 3, 6 and 9 of the 10 day patrol exercise).



Before commencing Exercise Northern Awakening 12 out of 33 subjects had elevated total plasma homocysteine (Hcys > 10 μmol/L). On completion of the exercise 22 of the subjects had elevated Hcys (P = 0.025). Although not reaching significance, the group receiving Half–CRP had the greatest increase > Full-CRP group > Freshly-Prepared Meals group.

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Medical ethics approval was granted by the Australian Defence Medical Ethics Committee for this study (ADMEC protocol 134/98).

PAPER 17: DO CAFFEINE AND EPHEDRINE HAVE A BENEFICIAL IMPACT ON HUMAN PERFORMANCE DURING PROLONGED EXPOSURE TO A COLD, WET AND WINDY ENVIRONMENT?

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INTRODUCTION

We have previously demonstrated significant cold strain in inadequately dressed individuals during prolonged intermittent walking in a simulated cold, wet and windy environment [1]. The influence of these environmental conditions on psychological and physical performance is not known, although it is likely that performance will be degraded. Physiological procedures that could enhance cold tolerance and performance have important survival and operational implications for military personnel exposed to prolonged cold stress. One such strategy—the ingestion of caffeine and ephedrine (C+E)—has been shown to enhance energy expenditure and retard the fall in deep body temperature during sedentary cold air exposure [2]. Furthermore, C+E has been reported to enhance intense exercise performance in a thermo-neutral environment [3]. Consequently, the present study investigated the potentially beneficial impact of this thermogenic drug combination on physiological strain and performance during rest and exercise in a cold, wet and windy environment. The cold strain data have been published elsewhere [4], and this paper reports the cognitive and physical performance data.

MATERIALS AND METHODS

In standard combat clothing (~1 clo), eleven men (age 28.1±1.3 (SEM) yr; height 1.82±0.02 m; body mass 81.8±2.8 kg; body surface area 2.03±0.04 m^2; body fat 13.6±0.8 %; $VO_{2\ peak}$ 45.4 $mL \cdot kg^{-1} \cdot min^{-1}$) undertook a protocol (lasting up to 330 min) in an environmental chamber on four occasions. On two occasions, the ambient air temperature was 5°C, the clothing was wetted every 15 min, and the subjects faced a ~5 $m \cdot s^{-1}$ direct air current (COLD), whereas on the other two occasions the temperature was 15°C, the clothing was not wetted, and the air speed was <0.2 $m \cdot s^{-1}$ (NEUTRAL). The protocol incorporated three consecutive elements: 1) 60-min seated rest (REST); 2) 120-min treadmill walking (5 $km \cdot h^{-1}$, 0% gradient) (WALK); and cycling at 85% of peak oxygen consumption ($\dot{V}O_{2peak}$) until exhaustion (CYCLE). On one occasion in both COLD and NEUTRAL, a single, combined dose of caffeine (5 $mg \cdot kg^{-1}$ body mass) and ephedrine (1 $mg \cdot kg^{-1}$ body mass) (C+E) was consumed 5 min prior to chamber exposure, whereas on the other a placebo was ingested (PLAC). The condition order was randomised, and the C+E and PLAC presented in a double-blind fashion. Between REST and WALK (Session 1), and WALK and CYCLE (Session 2), the subjects undertook a 25 min battery of computer-based tasks to determine the following indices of cognitive function: simple reaction time (SRT, both fixed and variable foreperiod); attention (focused and categoric search, [5]); visual vigilance [6]; and verbal reasoning [7]. Hand grip strength (HGS) was measured in the dominant hand after cognitive testing, and the time to exhaustion during CYCLE was assessed after Session 2 (TE). The subjects were familiarised with all the performance measures a few days before the start of the study, and on each trial day prior to chamber exposure they completed baseline assessments of cognitive function and HGS. Cognitive performance data were analysed using repeated-measures analysis of covariance with baseline performance as a covariate. Physical performance data were analysed using repeated measures analysis of variance. The independent variables included drug (D), environment (E), and session number (S). Significant effects were analysed further using Bonferroni's *t* test.

RESULTS

Body temperatures

During Sessions 1 and 2 in COLD, rectal and mean skin temperatures were, respectively, 0.4-0.8°C and 10°C lower than the initial starting values.

Cognitive performance

Influence of Caffeine + Ephedrine

C+E increased the speed of performance in several tasks (Table 1). Drug-induced decreases in reaction time (mean of Sessions 1 and 2) were evident in SRT (fixed and variable), the categoric search task, and the vigilance task. Reaction time during the focused attention task was also decreased by the ingestion of C+E during Session 1.

	PLAC		C+E		
	Session 1	Session 2	Session 1	Session 2	Significance
SRT (fixed)	215.1	243.6	193.1	213.0	D effect ($p<0.05$)
SRT (variable)	298.1	313.7	272.9	278.9	D effect ($p<0.001$)
Focused Attention	385.8	386.8	374.2*	389.9	D x S int. ($p<0.05$)
Categoric Search	489.9	499.9	471.7	481.4	D effect ($p<0.01$)
Vigilance	536.9	583.9	513.7	531.2	D effect ($p<0.05$)
Verbal reasoning	3444.9	3218.2	3337.1	3171.5	ns

ns represents no statistical significance, * indicates statistical significance at Session 1 ($p<0.05$), and *int.* denotes an interaction

Table 1. Mean reaction times (ms) during PLAC and C+E

There was no evidence that the reduced reaction times observed reflected a trade-off between the speed and accuracy of performance. Indeed, performance accuracy was enhanced by C+E in the focused attention ($p<0.05$), and categoric search tasks ($p<0.05$). Furthermore, in the vigilance task, C+E enhanced signal detection rate ($p<0.001$).

Influence of Environment

COLD increased reaction time during the variable foreperiod SRT, whereas there were no differences between NEUTRAL and COLD in the remaining tasks (Table 2). The accuracy of performance in the verbal reasoning task was also impaired by cold exposure ($p<0.01$).

Interactions between C+E and environment were evident in the focused attention task ($p<0.05$) and in the categoric search task ($p<0.01$). Further analysis indicated that, in NEUTRAL, C+E shortened reaction time ($p<0.05$ in the case of the focused attention task, and $p<0.01$ in the case of the categoric search task).

Physical performance

Neither HGS nor TE was influenced by C+E. However, HGS was lower in COLD compared with NEUTRAL (Session mean: 52.7±2.6 vs 56.9±2.2 kg) (T effect, $p<0.01$). TE was also lower in COLD compared with NEUTRAL (7.6±1.9 vs 12.0±2.6 min) (T effect, $p<0.01$).

	NEUTRAL		COLD		
	Session1	Session2	Session1	Session2	Significance
SRT (fixed)	202.7	231.1	205.6	225.5	ns
SRT (variable)	270.7	276.2	300.2	316.5	T effect ($p<0.05$)
Focused Attention	382.0	388.0	378.1	388.7	ns
Categoric Search	485.4	493.9	476.1	487.4	ns
Vigilance	529.9	550.8	520.7	564.3	ns
Verbal reasoning	3337.8	3228.6	3444.2	3161.1	ns

Table 2. Mean reaction times (ms) during NEUTRAL and COLD

CONCLUSIONS

The ingestion of a combined dose of caffeine and ephedrine facilitated a general increase in the speed of psychological performance. This was not at the expense of accuracy; indeed, accuracy in the selective attention tasks and the signal detection rate in the vigilance task were enhanced by this drug strategy. Psychological performance was fairly resilient to the effects of prolonged exposure to cold, wet and windy conditions in which substantial cold strain was induced. However, where cold-induced impairments in psychological performance were observed, there was no evidence that the ingestion of caffeine and ephedrine ameliorated these effects. Caffeine and ephedrine did not enhance hand grip strength and time to exhaustion, although these indices of physical performance were appreciably degraded in the cold environment. Given the negligible effect of caffeine and ephedrine on thermoregulation during prolonged cold exposure [3], it is unlikely that this pharmacological strategy would have significant survival or operational benefits in this scenario.

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INVITED LECTURE 10: AN EVALUATION OF THE CONCEPT OF LIVING AT MODERATE ALTITUDE AND TRAINING NEAR SEA LEVEL

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INTRODUCTION

Many endurance athletes spend part of each year living and training at altitude in an attempt to induce physiological adaptations that may benefit competitive performance at sea level. The hypoxic environment is believed to stimulate renal release of erythropoietin (EPO), with consequent increase in total red blood cell volume (RCV) and sea level maximum oxygen uptake ($\dot{V}O_{2max}$) [1] . However, the scientific literature on altitude training is equivocal, with most controlled studies failing to observe a positive effect [2 to 6]. One possible reason is that even moderate hypoxia may substantially compromise training intensity, leading to gradual loss of specific neuromuscular fitness. This may offset any advantage produced by positive adaptation in other aspects of physiology [7].

Recently, Levine and Stray-Gundersen [8] suggested that an optimal effect of altitude might be gained by living at approximately 2500 m but training as close as possible to sea level. They subsequently demonstrated that 4 weeks of "living high, training low" (LHTL) produced performance outcomes superior to those obtained through conventional altitude training [9].

Geographically, there are few places in the world where LHTL is practical. This problem has been overcome by the construction at sea level of facilities that allow athletes to sleep and rest under conditions of simulated altitude [10]. Since 1997, a small "Altitude House" has operated at the Australian Institute of Sport in Canberra. It produces normobaric hypoxia by adding nitrogen to the ambient air, and has permitted studies aimed at evaluating both the rationale for and the effectiveness of the LHTL approach. We hypothesised that moderate hypoxia would compromise the routine training intensity of national-level athletes. We also used the Altitude House to investigate the nature and time course of physiological adaptation to LHTL

METHODS

1. Moderate Hypoxia and Training Intensity

This issue was addressed in two separate studies.

1a) Cross-country skiing – graded exercise

Nine male cross-country skiers underwent two tests on a motorised treadmill. One test was conducted under sea level conditions and the other at a simulated altitude of 1800 m. The order of the tests was counter-balanced and a double-blind research design was employed. Each test involved a series of 3-minute work bouts with progressively increasing speed and gradient. There were 1-minute intervals between work bouts to allow collection of arterialised capillary blood samples for measurement of lactate concentration [La] and pH.

Oxygen uptake, pulmonary ventilation ($\dot{V}_{ESTPD}$) and heart rate (HR) were measured throughout the test. Ratings of perceived exertion (RPE) [11] were obtained at the end of each work bout. The test continued until volitional exhaustion.

1b) Cycle ergometry – endurance and sprint training

Eight members of the Australian Women's Road Cycling Squad each completed 2 different training sessions under both ambient Canberra conditions (altitude 610m) and at a

simulated altitude of 2100 m. One training session involved 3 repetitions of 10 minutes on a cycle ergometer with 5-minute recovery intervals. The other entailed 3 sets of 6 x 15-s sprints. The recovery period between sprints was 45 s for the first set, 30 s for the second, and 15 s for the third. The recovery period between sets was 3 minutes. For all sessions, the cyclists were instructed to achieve the highest possible average power output. HR and blood [La] were measured regularly during each session. The cyclists were blinded to the inspiratory oxygen concentration and, for each type of training session, the order was counter-balanced between normoxic and hypoxic conditions.

2. Process of Adaptation to LHTL

Four separate groups of athletes were studied – female road cyclists (n=12), kayakers (n=11; 6 females, 5 males), male triathletes (n=13) and male middle-distance runners (n=11). Each group was subdivided into a LHTL and Control group.

The LHTL group slept for 8-11 hours night in the Altitude House, while the Control group slept under ambient Canberra conditions. The simulated altitude for the LHTL group was 2650 m except in the case of the triathletes, who were exposed to 3000 m. The road cyclists slept in the Altitude House for 12 consecutive nights, the kayakers for 11, and the triathletes for 23. The middle-distance runners completed 3 blocks of 5 nights in the House, separated by recovery intervals of 3 nights under ambient Canberra conditions. Within each sport, Altitude House and Control subjects trained together in the normal Canberra environment.

Athletes sleeping in the Altitude House were monitored for arterial oxygen saturation (SpO_2) at 30-minute intervals throughout each night, using pulse oximetry. All except the runners also underwent monitoring during 1-2 nights of sleep in the Altitude House set to ambient, normoxic conditions.

Early morning venous blood samples were collected from all Altitude House and Control subjects before, during and after the period of differential sleeping conditions. The schedule for blood collection differed between sports with the first sample collected after 1 night for the runners, 3 nights for the triathletes, and 5 nights for the cyclists and kayakers. The samples were analysed for haemoglobin concentration [Hb], haematocrit (Hct), the numbers and characteristics of erythrocytes and reticulocytes, and serum erythropoietin concentration. In one group (female road cyclists), a resting arterialised capillary blood sample was collected in the early morning on 2 occasions before commencement of LHTL, 5 occasions during the LHTL period, and once afterwards. The samples were analysed for pH and pCO_2.

Before and after the experimental period, the road cyclists and triathletes underwent measurement of total haemoglobin mass (Hb_{mass}) through a carbon monoxide rebreathing technique [12]. Athletes from all sports completed initial and final ergometer tests involving measurement of $\dot{V}O_{2max}$ and of maximum work output over 4 minutes. An exception occurred with the runners, who completed $\dot{V}O_{2max}$ tests on a treadmill but underwent performance assessments through a 1500-m time trial conducted in the field.

Statistics

Values are reported as mean ± SEM. Data were subjected to repeated measures analysis of variance (ANOVA) followed where appropriate by Tukey post-hoc comparisons or Student's t-test for independent samples.

RESULTS

1. Moderate Hypoxia and Training Intensity

1a) Cross-country skiing – graded exercise

At exercise intensities of up to ~55% of sea level $\dot{V}O_{2max}$, moderate hypoxia had no measurable effect on HR, blood [La], $\dot{V}_{ESTPD}$ or RPE. However, at higher intensities, it was evident that hypoxia increased the physiological stress. At a workload requiring a $\dot{V}O_2$ of ~80% of sea level $\dot{V}O_{2max}$, the skiers had a mean blood [La] of 5.7±0.4 mmol.L^{-1} at 1800 m simulated altitude, compared with 3.9±0.2 mmol.L^{-1} in normoxia ($p<0.01$).

There were also substantial increases in $\dot{V}_{ESTPD}$ (99.6 ±5.2 vs 92.5±4.6 L.min^{-1}; $p=0.05$), HR (182±3 vs 178±3 bpm; $p=0.08$) and RPE (14.9±0.6 vs 13.7±0.6 Borg units; $p=0.03$). Time to exhaustion was significantly reduced at simulated altitude (27.27±0.90 vs 29.88±0.58 min; $p<0.01$).

1b) Cycle ergometry – endurance and sprint training

At 2100 m simulated altitude, average power output during the 3 x 10-minute efforts was 226±6 W, compared with 244±6 W when the session was performed in normoxia ($p<0.05$). Simulated altitude also compromised performance in the repeated 15-second sprints. For the first set of 6 sprints, average power output was 459±11 W in hypoxia and 477±18 W in normoxia ($p<0.05$). A significant negative influence of hypoxia persisted in the second and third sets (429±17 vs 452±20 W and 373±15 vs 403±19 W; $p<0.05$ in both cases). There was no significant effect of moderate hypoxia on blood [La], HR or RPE during either type of training session.

2. Process of Adaptation to LHTL

Physiological Responses to Sleeping under Hypoxic Conditions

The average of all SpO_2 values recorded between midnight and 6 AM during our studies was 97.7% for athletes sleeping under ambient, normoxic Canberra conditions, 93.7% for those sleeping at a simulated altitude of 2650 m, and 91.0% for the triathlete group that slept at 3000 m. The SEM was less than 0.1% in each case. When the data from the groups exposed to 2650 m and 3000 m were combined to examine changes between 11 successive nights of simulated altitude, it was evident that SpO_2 gradually increased for the first 4 nights, and then stabilised (night 1=91.4±0.7%, night 4=93.3±0.4%, night 11=93.2±0.4%).

The pCO_2 of arterialised capillary blood (measured only in the female cyclists) showed no significant difference between LHTL and Control groups at baseline (38.4±1.4 and 40.1±1.3 mm Hg respectively), but was significantly lower in the LHTL group during the treatment period (eg, 36.2±1.6 vs 42.3±0.8 mm Hg for the first measurement – taken after 4 nights). Values for the two groups were again similar one day after cessation of treatment (41.5±1.3 vs 41.2±1.3 mm Hg). The resting blood pH of the cyclists sleeping in the Altitude House increased at the onset of the LHTL period. The mean value for the first sample obtained during this period was 7.448±0.008, compared with 7.421±0.011 for the Control group ($p = 0.02$). The values for the LHTL group then decreased and except on one occasion were not significantly higher than those recorded for the Control subjects.

Effect of LHTL on Haematological Parameters

Serum EPO rose from an initial level of 6.9±0.6 IU.L^{-1} to a peak value of 11.0±0.7 IU.L^{-1} for the LHTL group. The peak was usually observed in the first blood sample collected after 1-5 nights of hypoxic exposure. By the end of the exposure, the level had declined to 8.2±0.8 U.L^{-1}. Control subjects who slept under normal conditions recorded a baseline EPO concentration of 6.8±0.7 U.L^{-1}, a peak concentration (regardless of the day of

occurrence) of 8.1±0.7 $IU.L^{-1}$, and a final value of 6.8±0.7 $IU.L^{-1}$. Repeated measures ANOVA revealed a significant group x time interaction, and post-hoc comparisons revealed that the peak EPO readings differed significantly between the LHTL and Control groups.

The percentage of reticulocytes was not different between LHTL and Control groups before, during or after hypoxia. Athletes sleeping at simulated altitude had a baseline level of 1.0±0.1%, a peak of 1.3±0.1%, and a level of 1.1±0.1% at the end of the treatment period. The corresponding values for the control group were 0.9±0.1%, 1.2±0.1% and 0.9±0.1%. Similarly, erythrocyte count, [Hb], Hct and mean cell volume before commencement of the treatment period and 24-72 hours after its completion were not different between the LHTL and Control groups.

Total Hb_{mass} showed no change over time. For those who slept in the Altitude House, the value was 854±50 g at baseline and 845±46 g at the end of the treatment period. Initial and final values for the Control group were 899±53 g and 908±50 g, respectively.

Effect of LHTL on $\dot{V}O_{2max}$ and Performance

The LHTL group showed a trend toward slight reduction of $\dot{V}O_{2max}$ (from 4.49±0.16 to 4.40±0.14 $L.min^{-1}$) over the treatment period, whereas the Control group registered little change (4.46±0.15 to 4.48±0.15 $L.min^{-1}$). The group x time interaction ($p=0.07$) approached the conventional criterion for statistical significance. The largest reductions in $\dot{V}O_{2max}$ after exposure to moderate hypoxia occurred in the triathletes, who had both a longer program (23 nights) and a higher level of simulated altitude (3000 m) than any other group.

Since the method of assessing performance and the units of measurement differed between sports, the pooled analysis of the effect of LHTL was based on percentage change. In a task requiring ~4 minutes of supramaximal effort, the performance of the Altitude House group improved by 1.0±0.4%, while that of the Control group remained stable (improvement of 0.1±0.4%; $p=0.13$ for group x time interaction).

DISCUSSION

Our findings provide partial support for a fundamental tenet underlying the LHTL concept – that training intensity is compromised by moderate hypoxia [9]. Female cyclists self-selected lower workloads for high-intensity interval training when inspiratory pO_2 was reduced to simulate an altitude of 2100 m. Since [La], HR and RPE during interval training were similar for both normoxic and hypoxic conditions, it seems that the choice of workload was strongly influenced by physiological and perceptual feedback. However, the data from the progressive tests on the cross-country skiers indicate that moderate hypoxia reduces the workload associated with given levels of physiological and perceptual stress only when the exercise intensity exceeds a certain level. Power outputs during training sessions that elicit heart rates of less than ~140 bpm and blood lactates of less than ~1.5 $mmol.L^{-1}$ may be unaffected by moderate hypoxia. Therefore, athletes attending altitude camps may need to "train low" only when intensive work-outs are required – usually not more than 2-3 times per week.

The levels of SpO_2 recorded for athletes sleeping in our Altitude House are similar to those reported to occur at equivalent natural altitudes [13]. The gradual increase in mean SpO_2 over the first 4 nights indicates that merely sleeping at simulated altitude, with daytime hours spent in normoxia, rapidly induces a degree of acclimatisation. As with natural altitude, this probably involves hyperventilation, since morning blood samples collected from our athletes showed decreased pCO_2 and increased pH.

We have confirmed that sleeping under conditions of moderate hypoxia elicits a significant increase in serum EPO. The mean peak level of the LHTL group was 59% above the baseline, which is consistent with the findings of other researchers [14,15]. In our studies, the increase in serum EPO did not produce an increase in erythrocyte production, since reticulocyte percentage, erythrocyte count, Hct, [Hb], and Hb_{mass} were always similar for the LHTL and Control groups. Other researchers have reported mean increases of 5-9% in RCV (a parameter closely related to Hb_{mass}) and/or >8% in [Hb] during LHTL programs [9, 15, 16].

The failure of moderate hypoxia to stimulate erythrocyte production in our studies might be explained by an insufficient duration of daily exposure. Our athletes spent 8-11 hours per day in hypoxic conditions, compared with 12-16 hours per day for the subjects of Rusko et al [15] and longer still for participants in other LHTL research [9, 16]. However, we have previously reported that no increase in total haemoglobin mass occurred in a group of cyclists who lived and trained for a month at a natural altitude of 2690 m [17], and who thus had a continuous stimulus for EPO release. We suggest that in athletes with a long history of endurance training – which of itself may be erythropoietic [18] – there is little scope for further natural increase in total haemoglobin mass [17]. In general, our athletes have been of a higher competitive standard than those employed in other controlled studies, and may represent a different population.

Since we observed no increase in total haemoglobin mass, it is entirely consistent that we also measured no increase in $\dot{V}O_{2max}$, but the tendency toward a reduction of $\dot{V}O_{2max}$ after a period of sleeping in moderate hypoxia was unexpected. Chronic exposure to very high altitudes (>5000 m) leads to loss of skeletal muscle mitochondria [19] and reduction in the activities of mitochondrial enzymes [20, 21]. Our results raise the possibility that a degree of mitochondrial degradation might occur even at altitudes as low as 2650-3000m. Alternatively, the trend toward reduced $\dot{V}O_{2max}$ after exposure to moderate hypoxia may reflect improved efficiency of oxidative phosphorylation, with a given yield of ATP produced at a lower oxygen cost [22].

Although sleeping in moderately hypoxic conditions did not increase Hb_{mass} or $\dot{V}O_{2max}$ in our studies, it was associated with a mean performance improvement of 1.0% for an effort lasting ~4 minutes. This is very similar to the 1.1% gain in 3000 m track performance reported by Stray-Gundersen et al [16] for a group of elite runners following a 4-week LHTL program. In the latter group, the enhanced performance was ascribed to significant increases in $\dot{V}O_{2max}$ and Hct. Our data cast doubt on this explanation, by demonstrating that such increases are not essential to the performance effect. We believe that any performance improvement probably stems from either increased anaerobic capacity or greater efficiency of aerobic metabolism.

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PAPER 18: CLOTHING INSULATION AND THERMAL COMFORT OF TENT OCCUPANTS AT HIGH ALTITUDE

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INTRODUCTION

High mountain environments provide demanding test conditions for cold climate clothing and equipment. Mountaineers and trekkers rely on a limited set of functional garments to provide them with warmth and protection in different conditions ranging from heat to cold and during periods of high and low metabolic activity. The available clothing is applied in layers during the day and also at night to supplement sleeping bags. Field experiments studying functional clothing and the resulting wearer's sensations are by their nature less precise than well controlled tests in climatic chambers (Gonzalez and Cena, 1986), but provide important realism and direct applicability.

This paper presents a field study of clothing insulation used by trekkers to achieve satisfactory thermal conditions during sleep in tents at high altitude. This project also tests a simple protocol that can be used during trekking and mountaineering expeditions to obtain information about performance of cold weather clothing and sleeping bags in real conditions.

METHODS

Two groups of trekkers participated in this study. The longer (19 nights) trekking expedition of 10 participants took place in November 1998 in the Everest region of the Himalayas (in the valleys leading to Gokyo Ri and Kala Pattar) where subjects slept between 2640 and 5170 metres above sea level. A heavy snowfall forced the group to spend 5 nights in unheated lodges. The shorter (18 nights in tents) expedition of 7 participants, across Snow Lake and Hispar Pass (5150 m) in the Karakoram in August 1998, involved more physically demanding trekking, on the Biafo and Hispar glaciers, and 5 nights spent on ice at 4900 m. Three-season A-frame tents (with flies) were used in the Everest region and four-season dome tents (with flies) were used in the Karakoram. The tents were shared by two persons who usually slept on a combination of a 1 cm closed cell foam mat and a 2.5 cm self-inflating mattress. The Everest region trekkers comprised of 5 women (age 28 to 45 years, average weight 55 kg, average height 163 cm) and 5 men (31 to 59 years, 81 kg, 177 cm). The Karakoram expedition consisted of 2 women (24 and 33 years, 55 kg, 164 cm) and 5 men (27 to 59 years, 85 kg, 179 cm).

Subjects were asked individually, each morning, about the inventory of clothing and bedding they used at night. They also rated their overnight thermal sensation on a 7-category psychophysical scale used routinely in thermal comfort studies (Cena and de Dear, 1999). The discreet categories on the scale were: -3 (cold), -2 (cool), -1 (slightly cool), 0 (neutral), 1 (slightly warm), 2 (warm), 3 (hot). Subjects indicated their sensations on the scale at or between these categories.

Clothing insulation was calculated from an empirical formula developed by McCullough and Kim (1996) who found that both the static and dynamic insulation provided by cold weather clothing assemblies can be estimated with confidence from the number and thickness of garment layers on the arms and calves. Clothing insulation I_c (in clo) for static conditions is

$$I_c = 0.0198\, d_{arm} + 0.0149\, d_{calf} + 0.191\, n_{arm} + 0.242\, n_{calf} + 0.556\,,$$

where d_{arm} and d_{calf} denote thickness (in mm) of garment layers on the arms and calves, and n_{arm} and n_{calf} are respective numbers of garment layers. The clothing insulation unit is

defined as 1 clo = 0.155 m^2 K W^{-1}. The thickness of individual garments was estimated from the measurements by Kim (1995) included in his detailed catalogue of cold weather clothing. Accuracy of calculations using the above equation is better than 95%. Precise determination of garments' thickness has a smaller influence on I_c than the number of layers because of the weighing coefficients in the formula. Clothing worn by the subjects inside sleeping bags included mostly polypropylene 'thermal' underwear (typical thickness 0.9 mm) and polyester 'fleece' garments (5.3 mm for '200' type). On rare occasions, down jackets (45.8 mm) were also used. Sleeping bag liners were regarded as additional layers. Hats, neck warmers and socks are not counted when using the above formula.

Insulation of sleeping bags in a compressed state (i.e. when used by a reclining subject) was estimated, on the basis of their construction and weight, from the data by McCullough and Gonzalez (private communications). Insulation values for fully closed (zipped-up) sleeping bags were used and any adjustment of zips by individuals on warmer nights could affect only the lower limit of the total insulation. All subjects (but one) of the Everest trek used the same type of sleeping bag (total weight 1920 g, filled with 720 g of 100% duck down). The insulation of this bag was assessed to be 5.7 clo. The other sleeping bag (1100 g down fill) was 7.1 clo. The sleeping bags used by the Karakoram mountaineers were slightly warmer at 6.3 (± 0.7) clo.

Air temperature and relative humidity in tents (or lodges) at night was recorded every 12 minutes with miniature data loggers positioned at the head level of 20 cm in between two reclining subjects.

RESULTS AND DISCUSSION

The simple protocol used in this study produced valid results under realistic field conditions at high altitude. The choice of the rest period as focus for this study limited the variability of activity and exposure that would be present if the study was performed outside the tents. The assessment of clothing insulation was not significantly affected by changes in altitude because hypobaria has only a small effect on the intrinsic clothing insulation (Chang and Santee, 1996).

Figure 1 presents thermal sensations and total insulation of clothing and sleeping bags for the Everest region trekkers. The late autumn was relatively cool and the mean air temperature inside tents dropped below 0°C above 3000 m. The mean temperatures rose to 4-6°C when snowfall forced the trekkers into lodges, then decreased to –5 and –7.4°C at 4600 and 4940 m when subjects were back in tents. In between the latter episodes, the subjects spent another night in a lodge at 5170 m. These significant temperature variations in tents and lodges were compensated by the subjects who adjusted the clothing worn inside sleeping bags in a relatively narrow range of 0.8 clo. The total insulation (including sleeping bags) ranged from about 7.0 to 7.8 clo with an average of 7.4 (± 0.7) clo. During the whole trek, the individual minimum and maximum values were 6.3 and 9.3 clo indicating also that some subjects were 'warmer' or 'cooler' sleepers. All subjects, however, always achieved a high degree of thermal comfort and usually assessed their overnight thermal sensations between 'slightly warm' and 'warm' with an overall average of 1.7 (± 0.7) on the scale. These high ratings indicate that the subjects were able to predict effectively (and amply) their overnight insulation requirements on the basis of the previous night's experience and evening outdoor conditions.

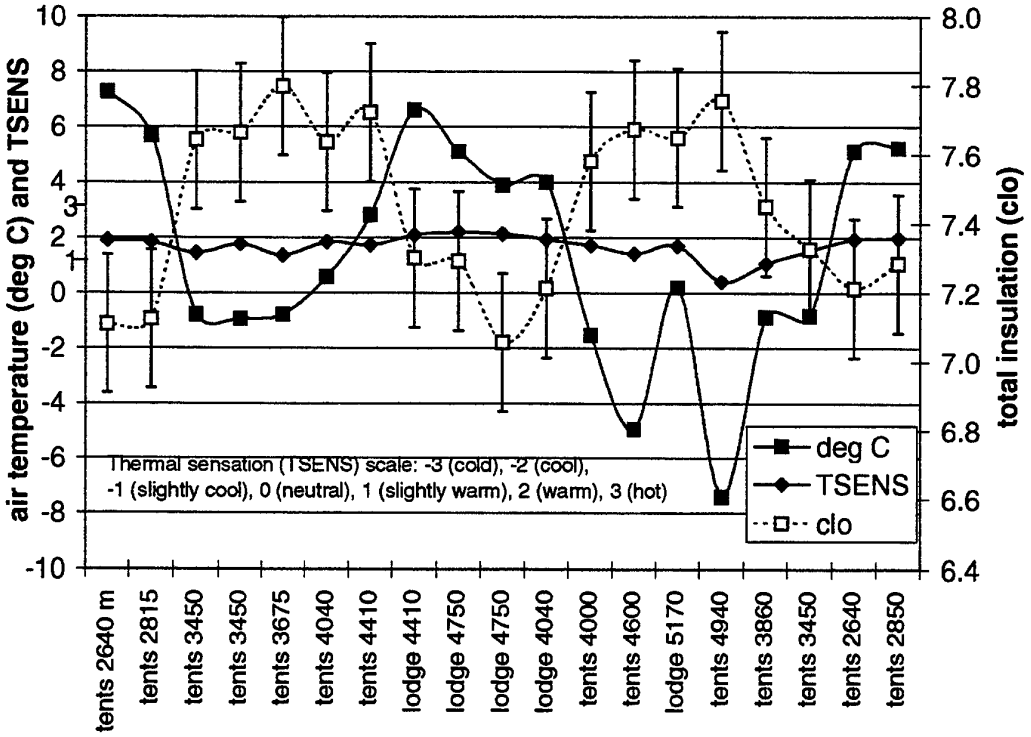

Figure 1. Thermal sensations and total insulation of clothing and sleeping bags for a group of 10 trekkers when staying overnight in tents and lodges during a 19 day trek in the Everest region between 2640 and 5170 metres above sea level at different air temperatures recorded in tents (or lodges) at the head level of reclining subjects. Bars depict standard errors for 'clo'.

The group of trekkers in the Karakoram reacted in a similar manner although the temperatures inside their tents were generally higher (between 18°C at 3200 m and –2°C when camping on a glacier at 4900 m). They also adjusted their clothing insulation within a similar small range of 1.0 clo. The average total insulation was 7.8 (± 0.7) clo. The seven subjects rated their average thermal sensations as 0.5 (±0.7) between 'neutral' and 'slightly warm'. This confirms the conclusion that both groups of subjects preferred to be warmer by wearing more clothing in their sleeping bags rather than risk any cold discomfort.

Further research allowing predictions of thermal sensations in tents and other shelters on the basis of thermal comfort theories (Cena and Clark, 1987) is suggested.

CONCLUSIONS

Two groups of high altitude trekkers wore up to 1 clo of clothing inside their sleeping bags providing the total insulation of about 8 clo when the air temperature inside their tents ranged between –8 and 18°C.

The subjects usually exceeded their subjective thermoneutral conditions and preferred to be warmer by wearing more clothing in their sleeping bags than risk any cold discomfort.

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PAPER 19: EFFECTS OF COLD ON MANUAL PERFORMANCE IN SUBJECTS WITH RAYNAUD'S PHENOMENON

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INTRODUCTION

Raynaud's phenomenon (RP) is characterised by episodic vasospams of the digital vessels. RP consists of attacks of an at least biphasic discoloration (white, blue or red) of fingers provoked by cold and/or emotional stress. White finger symptoms were subjectively reported by 18% of Finnish conscripts during their military service. Although cooling of the hands is known to decrease manual performance [1], less attention is paid to the effects of cold on manual dexterity and sensitivity in subjects with RP. Therefore, the aim of this study was to compare the effects of whole body cooling on manual performance in healthy subjects and subjects suffering RP, and to test different methods for evaluating manual performance.

MATERIALS AND METHODS

A total of 116 male conscripts, who claimed white-finger symptoms in a questionnaire study, were examined. Twelve of them (RPG) were diagnosed to suffer RP according to Maricq test [2]. Control measurements were performed in 19 healthy male conscripts (HG) without any cold or white-finger symptoms in the hands. Subjects were lightly clothed and rested in 5°C air for 60 min. Skin temperatures on 12 sites (body and fingers) were measured. Subjects performed several sensor and motor tests before and during the cold exposure at 15-min intervals.

Finger dexterity was assessed by the Purdue Pegboard (32020 Lafayette Instrument Co.) test. The subject was allowed 15 sec to place the metal pegs into the holes. After this the subject placed thin metal washers onto the pins in 15 sec. The score for the tests was the number of pegs and washers in the correct places. Maximal pinch strength of the fingers was performed by pressing a dynamometer by forefinger and thumb. Rate of abduction and adduction of fingers was determined by a motion analysis system (MacReflex, Qualisys AB). The subject performed five maximal abduction/adduction movements as quickly as possible.

Tactile sensitivity of the fingertips was tested by monofilaments (Semmes-Weinstein Monofilaments). Vibration perception threshold (VPT) test was determined on the tip of the forefinger with a frequency of 125 Hz (Brüel & Kjæl 4810).

RESULTS

Finger skin temperatures were 2°C lower ($p<0.05$) in RPG than in HG at the beginning of the cold exposure and stayed lower during the exposure ($p<0.05$, Fig. 1).

Tactile sensitivity ($p < 0.05$) and VPT ($p < 0.05$) were impaired already at the beginning of the exposure in RPG compared with HG. Pinch strength ($p = 0.08$), and dexterity (NS) showed a similar tendency.

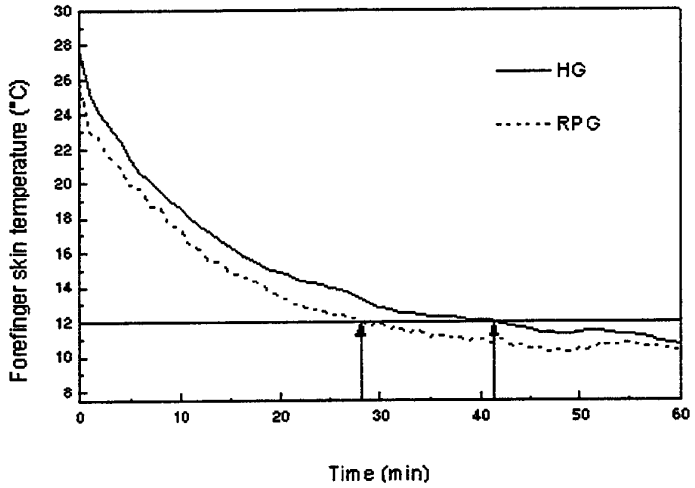

Fig. 1. Skin temperature of forefinger during the cold exposure. Arrows indicate the time when finger skin temperature reached 12°C. Data are means for 19 (HG) and 12 (RPG) subjects.

Cooling of the fingers and hand decreased manual performance more in RPG than in HG. After the exposure finger dexterity was decreased by 22% ($p < 0.05$) and 12% ($p < 0.05$) from the baseline measurements in RPG and HG, respectively. Dexterity was significantly lower in RPG than in HG at the end of the exposure. A significant decrease in pinch strength was noted at 45 min in RPG in comparison to HG. No differences were detected between RPG and HG in the abduction/adduction test. Tactile sensitivity was significantly ($p < 0.01$) impaired after 15 min and 45 min of exposure in RPG and HG, respectively. VPT was 140 ± 3 and 132 ± 1 dB ($p < 0.01$) in RPG and HG, respectively, at the end of the exposure.

A critical finger skin temperature for a marked decrement in manual performance seemed to be about 12°C for pinch strength (Fig. 2) and dexterity (Fig. 3). Noting 12°C as the threshold for reduced manual performance, this temperature was reached 15 min earlier ($p < 0.05$) in RPG than in HG (Fig. 1).

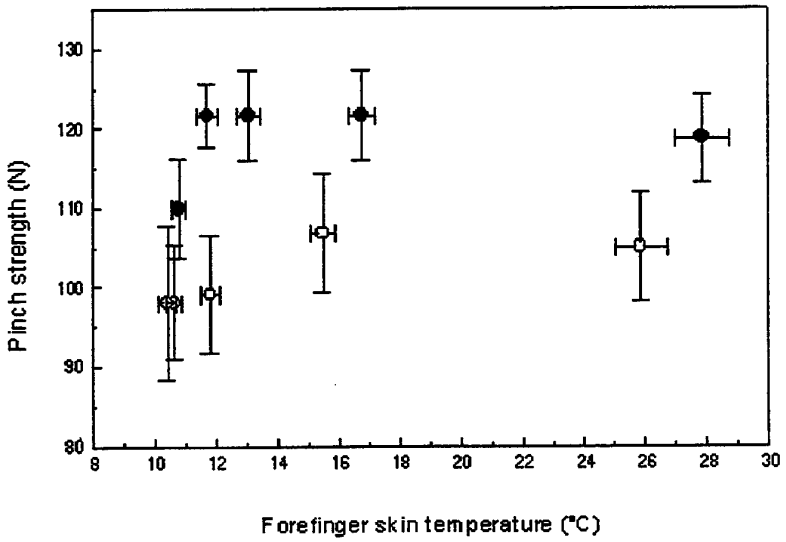

Fig. 2. Pinch strength in the relation to the forefinger skin temperature. ● = HG, O = RPG. Values are means and SE.

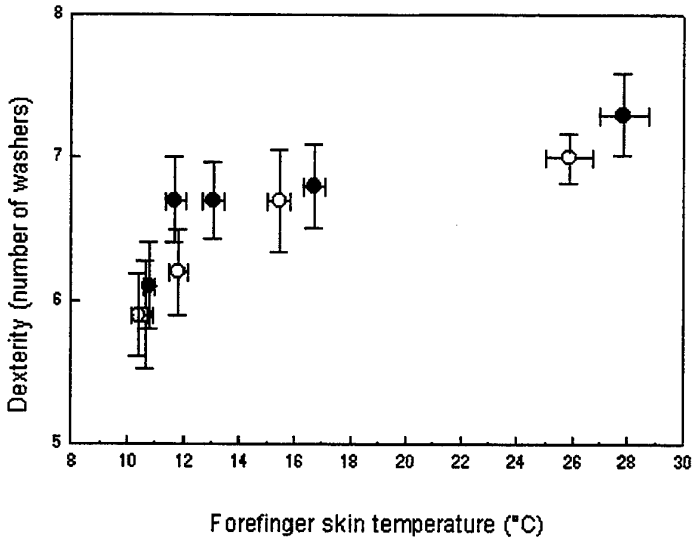

Fig 3. Number of washers in the relation to the forefinger skin temperature. ● = HG, O = RPG. Values are means and SE.

CONCLUSIONS

In the present study the best discrimination between the two groups was obtained in pinch strength from the motor tests. In the sensor tests the VPT showed the greatest discrimination. Greater impairment for manual performance was provoked in subjects with RP than in a healthy control in cold conditions. Furthermore, sensory and performance decrements were associated with critical finger skin temperature of approximately 12°C. This threshold was reached earlier in the RPG.

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The experimental protocol was approved by the Ethics Committee of the Institute of Occupational Health.

INVITED LECTURE 11: EXERTION-INDUCED FATIGUE AND THERMOREGULATION IN THE COLD

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INTRODUCTION

When humans are exposed to the cold, the first and most effective responses elicited for defending body temperatures involve behavioral strategies such as wearing warmer clothing or remaining in heated shelters. However, participants in outdoor sports and recreational activities may be unable to employ those behavioral strategies effectively enough to prevent body heat loss during cold weather, in which case, two major physiological responses act to maintain thermal balance [1,2]. A peripheral vasoconstriction decreases peripheral blood flow, thereby reducing convective heat transfer between the body's core and shell, effectively increasing insulation. However, body heat is conserved at the expense of a decline in peripheral temperature, since heat lost from the exposed body surface is not replaced, and skin temperature declines. Cold exposure also elicits muscular shivering which increases metabolic heat production. Shivering, which consists of involuntary repeated, rhythmic muscle contractions, may start immediately, or after several minutes of cold exposure, usually beginning in torso muscles, then spreading to the limbs. Shivering intensity increases and more muscles are recruited to shiver as cold stress becomes more severe.

Individuals vary greatly in their capability to maintain normal body temperature during cold exposure, primarily due to anthropomorphic differences [1,2]. A large body surface area facilitates heat loss, but a large body mass favors maintaining a constant temperature by virtue of a greater heat content, thus, persons with a large surface area to mass ratio generally cool faster than those with smaller surface area to mass ratio [1,3]. Both fat and non-fatty body tissues insulate against heat loss, but thermal resistivity of fat is greater than that of skin or muscle [1]. As a result, fatter persons usually shiver less and experience smaller declines in body temperature during cold exposure than leaner persons [1]. Human thermoregulatory responses to cold are also influenced by aerobic fitness and acclimatization status, and vary with age and gender, but those factors have considerably less influence than anthropomorphic factors for defense of normal body temperature [2].

EXERCISE DURING COLD EXPOSURE

Acute exercise can affect thermal balance during cold exposure, depending on a complex interaction among factors related to exercise intensity, environmental conditions, and mode of activity. Physical activity or exercise can increase metabolic heat production even more than shivering, so voluntarily increasing activity can increase heat production sufficiently to obviate the need for shivering. However, exercise also increases conductive and convective heat loss from the skin by two mechanisms. Limb movement during exercise disrupts the stationary boundary layer of air or water that develops at the skin surface in a still environment which tends to reduce insulation. More importantly, exercise increases blood flow to the skin and muscles thereby facilitating convective heat transfer from the central core to peripheral shell. Thus, while metabolic heat production increases progressively as exercise intensity increases, so too does heat loss, and the influence of these competing effects on overall thermal balance will be further modulated by anthropomorphic factors.

Cold exposure can also affect physiological responses to exercise. For example, oxygen uptake during exercise at a given intensity can be different in the cold than in warm

conditions, depending on the exercise intensity [4]. At low exercise intensities in the cold, metabolic heat production is not high enough to prevent shivering, thus oxygen uptake is increased. Cardiac output is elevated to satisfy the increased systemic oxygen transport requirement for shivering, but when comparisons are made at a given oxygen uptake, cardiac output is the same during exercise in cold and temperate conditions [5]. As metabolic heat production rises with increasing exercise intensity, core and skin temperatures are maintained warmer, shivering declines, and the difference in oxygen uptake becomes smaller. At high intensities, exercise metabolism is high enough to completely prevent shivering, and oxygen uptake during exercise is the same in cold and warm conditions. Similarly, cold exposure may also affect muscle energy metabolism during exercise [4]. When cold exposure is severe enough to lower core temperatures and cause shivering, blood lactate concentrations during exercise are higher than temperate conditions, but if cold exposure has no effect on core temperature and there is no shivering, then there is also no difference in the lactate accumulation during exercise. This observation has been interpreted as indicating that shivering-induced glycogenolysis accounted for the additional lactate accumulation during exercise. This is consistent with observations that muscle glycogen use was observed to be more pronounced in cold than temperate conditions during low intensity exercise [6], but not different during high intensity exercise [7]. However, in non-exercising subjects exposed to cold for 3 hours, muscle glycogen levels remained unchanged, despite the fact that subjects were shivering vigorously and their body temperatures declining throughout [8]. Thus, shivering per se is not likely the cause of accelerated lactate accumulation and glycogenolysis occasionally observed during exercise in the cold.

EXERCISE-INDUCED FATIGUE AND RESPONSE TO COLD

Prolonged exercise can lead to exertional fatigue. An anecdotal association between exertional fatigue and susceptibility to hypothermia has long been suspected [9,10]. Two recent experimental studies [11,12] attempted to demonstrate that an acute bout of prolonged fatiguing exercise impaired maintenance of thermal balance in the cold. Both reports showed that as fatigue develops, the intensity of exercise that can be sustained declines, thus, metabolic heat production declines and thermal balance is compromised. However, neither study could discern whether exertional fatigue directly affected thermoregulatory responses to cold. More recently, our laboratory investigated how physiological responses to cold were affected by more chronic exertional fatigue. Responses to cold were measured in 8 men who had completed an arduous nine-week military training course, throughout which participants perform very strenuous physical activity and daily sleep is limited to about four hours [13]. Daily energy expenditures exceeded 4,100 kcal per day, while daily energy intakes averaged only about 3,300 kcal per day. The subjects completed a standardized cold air exposure within two hours after finishing this regimen (no rest). The standardized cold exposure was repeated following a short (48 hours) recovery period for rest and refeeding, and again following a longer (16 weeks) recovery period. Those experiments demonstrated that cold tolerance and ability to maintain normal body temperature during cold exposure, was compromised during the trial performed without rest and remained compromised even after 48-hr of recovery. Two factors appear to account for the impaired capability to defend thermal balance exposed to cold in chronically fatigued and underfed persons. A 10% loss in body weight during the training course included both fat and non-fatty tissue which reduced tissue insulation and compromised body heat conservation during both the first and second trials, but was no longer apparent following 16 weeks rest and recovery of body mass. Also, the set point for shivering thermogenesis appeared reduced and the sympathetic nervous response to cold was blunted during the first trial, but these impairments resolved after 48 hours rest and recovery.

Those experiments demonstrated that the combined effects of exertional fatigue, negative energy balance and sleep loss reduced cold tolerance and compromised maintenance of normal body temperature during cold exposure. However, the relative importance of individual factors underlying exercise-induced fatigue on thermoregulation during cold exposure remains in question. For example, exercise-induced depletion of muscle energy substrates has been suggested to impair shivering and metabolic heat production during cold exposure, but experimental findings indicate that this is not the case either during [14] or following [8] heavy exercise. In recent studies from our laboratory employing well-fed subjects who were not sleep deprived, neither a single 60-min bout of heavy exercise [15] nor a 3-day regimen of exhaustive (6-hours/day) [16] had any effect on shivering thermogenesis during subsequent cold exposure, but peripheral heat losses were greater when cold exposure was preceded by exercise than by rest.

SUMMARY

Our research suggests that the ability to increase insulation by reducing peripheral blood flow in response to cold exposure may become impaired following exercise. It remains unclear whether this effect was due to a fatigue of the vasoconstrictor response to cold perhaps associated with central or peripheral nervous mechanisms. The shivering response to cold appears to be resistant to the effects of several hours or even several days of exhaustive exercise, but when extremely high levels of exertion are sustained for many weeks, shivering does become impaired.

DISCLAIMER

The views, opinions and/or findings contained in this report are those of the author and should not be construed as an official Department of the Army position, policy or decision, unless so designated by other official documentation. Approved for public release; distribution is unlimited.

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PAPER 20: RELATIONSHIP BETWEEN MANUAL PERFORMANCE, EXTREMITY TEMPERATURES, AND RATE OF BODY HEAT STORAGE DURING COLD EXPOSURE

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INTRODUCTION

Recently, it has been reported that active torso heating can be used to indirectly warm bare hands during exposure to -15°C air [2] . During that study, finger comfort was maintained despite a calculated rate of body heat storage ($\dot{S}$) of -48±33W. In contrast, Goldman [5] did a similar torso heating experiment in which he found that extremity comfort could not be maintained despite a calculated $\dot{S}$ of 84 W. In both experiments, a thermometric approach was used to calculate $\dot{S}$ and this may explain the apparent contrasting results. Partitional calorimetry may provide a better estimation of $\dot{S}$. Therefore, the purpose of the present experiment was to use partitional calorimetry to examine the relationship between $\dot{S}$, mean finger (T_{fing}), mean toe skin temperature (T_{toe}), and manual performance during active torso heating while subjects were exposed to -25°C air. It was hypothesized that the overall body heat gain during cold exposure must be greater or equal to the overall body heat loss ($\dot{S} \geq 0$ W) to keep the extremities comfortable and to maintain optimal dexterity.

MATERIALS AND METHODS

Eight, healthy, non-smoking male volunteers with the following mean±S.D. characteristics were recruited: age 32.8±7.4 years, height 176.4±6.3 cm, weight 82.4±7.5 kg, and body surface area 1.99±0.11 m^2. Body surface area was calculated using the formula of Dubois and Dubois [4] .

The subjects were exposed to four randomly assigned conditions. Condition 1, HI(bare), involved torso heating using an electrically heated vest (EHV) while the subjects wore heavy insulation (HI: 3.6 Clo Arctic clothing ensemble) and the hands were bare. Condition 2, LI(bare), was similar to condition 1 except the subjects wore lighter insulation (LI: 2.6 Clo). Condition 3, HI(g+m), was similar to condition 1 except the subjects wore contact gloves and Arctic mitts during the test. Condition 4, HI(g+m)NP, was similar to condition 3 except the EHV was not powered during the test. The conditions were defined to create a range of body heat storage values from positive (ex. HI(g+m)) to negative (ex. HI(g+m)NP) with some intermediate conditions near zero (ex. HI(bare)). The tests were done one week apart over a time period spanning from January to July. Subjects were exposed to an ambient temperature of -25°C for three hours during all tests. A description of the EHV and the methodology used to maintain the skin under the EHV at 42°C for the first 3 conditions is described in detail in Brajkovic et al. [2] .

T_{fing} and T_{toe} were measured using thermistors that were taped with double-sided tape on the "ring" finger of each hand on the tip of the finger and on the side of the large toes, respectively. Rectal temperature (T_{re}) was measured using a thermistor placed 15 cm beyond the opening of the anus. Finger dexterity was measured every 30 min by using a Purdue Pegboard (PP) test [1] or a C-7 Canadian Forces rifle disassembly and assembly test. The PP test was done at time 0 (at room temperature), 30, 90 and 150 minutes. Three, one-minute PP tests were done at each of these times. The C-7 rifle task is a timed task in which the subject takes apart a C-7 rifle into 12 pieces and puts it back together again. The rifle task was done at time 0 (at room temperature), 1, 60, 120 and 180 minutes.

During the HI(g+m) and HI(g+m)NP conditions, the knitted gloves and Arctic mitts were removed every 30 min so that the tasks could be performed barehanded. Open-circuit spirometry was used to determine $\dot{V}O_2$ and carbon dioxide output ($\dot{V}CO_2$ in $l \bullet \text{min}^{-1}$ STPD) every minute during the 3 hour exposure, but no metabolic data was collected during the dexterity tests. $\dot{S} = \dot{M} + \dot{W} - (\dot{R} + \dot{C} + K) - \dot{E}_{sk} - \dot{E}_{resp} - \dot{C}_{resp}$ was calculated using partitional calorimetry, using the equations outlined in McLellan et al. [6] , with the exception of the summation of the radiative, convective and conductive heat transfer ($\dot{R} + \dot{C} + \dot{K}$) equation which instead was measured directly using 12 heat flux transducers distributed over the body. $\dot{E}_{sk}$ was estimated from a model developed by Cain and McLellan [3] . A two-way ANOVA for repeated measures with heating condition and time as the independent variables was used to compare the four conditions for the dependent variables C-7 rifle time, PP score, T_{fing}, T_{toe}, T_{re} and $\dot{S}$ from 0 to 180 min. Results were considered statistically significant at $p < 0.05$ (using the Greenhouse-Geisser adjustment for repeated measures). A Newman Keuls post-hoc test was used to determine if there was a significant difference in any of the dependent variables from 0 to 180 min. All values are presented as mean±SE.

RESULTS

Extremity temperatures and T_{re} at the start of the tests averaged 33.0±0.4°C and 37.25±0.07°C, respectively, with no difference between conditions. During conditions HI(g+m), HI(bare), and LI(bare), $\dot{S}$ remained stable and statistically different for the three conditions at 13±5 W, -11±5 W (not different from 0), and -46±8 W, respectively. At the end of the 3-h exposure, T_{fing} were 34.9±0.4, 31.2±1.2, and 18.3±3.1°C, and T_{toe} were 33.2±0.8, 28.2±1.8, and 16.2±2.1°C. During HI(g+m), T_{re} increased 0.23±0.04°C during the first hour of cold exposure and then gradually decreased back down to its original value (observed at 0 min) at 180 min. During HI(bare), there was no significant change in T_{re} from 0 to 170 min and then a slight decrease (0.1°C) in T_{re} occurred during the last 10 min of the exposure (relative to the value observed at 0 min), whereas during LI(bare), T_{re} followed the same T_{re} response observed during HI(bare) for the first 154 min, after which time there was no data available for LI(bare). During condition LI(bare), four subjects were pulled out of the cold chamber at 70 min, 141 min, 154 min, and 178 min, respectively, because T_{fing} reached 6°C in each case. During condition HI(g+m)NP, $\dot{S}$ increased from -65±5 W to -19±7 W from 0 to 180 min due to an increase in shivering, but paradoxically, T_{fing} decreased from 32.4±0.4 to 12.1±0.5°C, T_{toe} decreased from 32.4±1.1 to 9.1±0.2°C from 0 to 180 min, and T_{re} decreased 0.57±0.08°C by 180 min.

During the 3-h exposure, finger dexterity decreased on average for the two dexterity tests by 0 (not significant), 0 (not significant), 25 (significant), and 39% (significant) for conditions HI(g+m), HI(bare), LI(bare), and HI(g+m)NP, respectively.

DISCUSSION

In examining conditions HI(g+m), HI(bare), LI(bare), there was a direct relationship between extremity temperatures, manual dexterity and $\dot{S}$; that is, extremity temperatures and manual dexterity were higher in those EHV conditions which had a higher overall $\dot{S}$. It should also be noted that in these 3 conditions, T_{re} was at or above thermal neutrality for the entire 3-h cold exposure. Rapaport et al. [7] also found that, in general, extremity comfort could be maintained if $\dot{S}$ was ≥ 0 W. The similar findings between the present study and the Rapaport et al. study may be attributed to the fact that both studies measured $\dot{S}$ using partitional calorimetry, whereas Goldman [5] and Brajkovic et al. [2] used

thermometry. The present findings also suggest that $\dot{S}$ may have been overestimated in Goldman's experiment and underestimated in the Brajkovic et al. study.

However, during condition HI(g+m)NP, the direct relationship between extremity temperature and $\dot{S}$ did not exist over time. The reason for the paradoxical decrease in extremity temperature is that extremity temperature is not only related to $\dot{S}$, but seems to be also related to core temperature. During HI(g+m)NP, Tre decreased significantly from 0 to 180 min. It is hypothesized that the extremities are comfortable only when $\dot{S} \geq 0$ W and the core temperature is at or above thermal neutrality. These two criteria were met during conditions HI(g+m), HI(bare), and LI(bare), but the criteria were not met during condition HI(g+m)NP.

In comparing HI(bare) and HI(g+m), the only difference between the two conditions was that during HI(g+m), knitted gloves and Arctic mitts were worn over the hands, whereas during HI(bare), the hands were bare. However, covering the hands (which are only 5% of the body surface area) resulted in a significantly greater $\dot{S}$. Comparing these two conditions shows the important role the hands play in regulating the body's state of heat balance.

CONCLUSION

Torso heating can be used to keep an individual's bare hands and insulated feet warm (T_{fing} and $T_{toe} \geq 28°C$) during exposure to -25°C air for 3 hours when Arctic clothing is worn. Extremity comfort is directly associated with a $\dot{S} \geq 0$ W, but only when the core temperature is at or above thermal neutrality.

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PAPER 21: THE EFFECTS OF WIND ON THERMAL RESPONSES DURING LIGHT AND MODERATE EXERCISE IN THE COLD

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INTRODUCTION

The wind chill index (WCI) [1] is a widely used method to predict the combined effects of low temperature and wind. However, the thermophysiological variables of human, such as cold induced vasoconstriction and muscle work induced heat production, are not included in the WCI. Also, since it is not possible to express the effects of heat loss from the person without referring to the clothing being worn, WCI should only be applicable to bare skin [2].

While working in cold, muscle activity produces heat and stimulates circulation, but at the same time, body movements reduce body and clothing insulation and increase convective heat loss. Thermoregulatory responses during work in the cold are dependent on the work level and the environmental conditions, of which the wind is an important component. Face cooling by stream of cold air during exercise has been shown to cause an increase in blood pressure, a reduction of heart rate, and an increase of energy expenditure [3,4], but the effects of cold wind exposure to the whole body during exercise has not been comprehensively investigated. The aim of the present work was to study the effects of cold and wind on thermoregulatory responses of subjects working in two different exercise intensities.

MATERIALS AND METHODS

Eight young and healthy men served as test subjects. Their mean age was 23 years, height 179 cm, weight 73 kg, and body fat 14%. During preconditioning the subject sat in a net chair for 60 minutes at 20°C, dressed in standard Finnish military winter clothing (basic insulation ca. 2.2 clo). Hat, gloves, jacket, and boots were not worn in order to avoid heat load. After preconditioning, the fully clothed subject entered the wind tunnel, where the temperature was at -10 °C and wind speed 0.2 (still air, NoWi), 1.0 (Wi1), or 5.0 (Wi5) m/s. During a 60 min exercise, subjects walked on the treadmill at a speed of 2.8 km/h, while the exercise intensity was adjusted by changing the gradient of the treadmill between 0° (light exercise level, energy expenditure 138W, L) and 6° (high exercise level, energy expenditure 232W, H). By that way the walking speed and the body movements were kept the same during both exercise intensities.

Skin temperature (15 sites) and rectal temperature (T_{re}) (YSI 400-series, Yellow Springs Instrument Co., Yellow Springs, USA) were measured. Heat flux from the skin was measured with heat flux transducers (HFT-A Model HA 13-18-10-P(C), Thermonetics Corp., San Diego, USA) from 8 sites. Oxygen consumption ($\dot{V}O_2$) was measured with an open-circuit system (Medikro 919 Ergospirometer, Finland) during the last 5 minutes of both the preconditioning and the exposure to the wind. Heart rate was also continuously measured (Polar sport tester, Finland).

RESULTS

Rectal temperature was increased during H at all three air velocities, but there were no significant differences of T_{re} between different air velocities at H (Table 1). At the end of L, T_{re} was lower ($p<0.01$) under Wi5 than under NoWi and Wi1. Mean skin temperature (T_{sk}) was significantly higher at the end of H under NoWi ($p<0.01$) and Wi1 ($p<0.001$) in comparison to T_{sk} recorded in L under the same air speed. These skin temperatures tended

to be higher than those recorded under Wi5 at their corresponding exercise intensity. Heat flow was significantly increased in Wi5, but was unaffected by the exercise intensity (Table 1). Oxygen consumption was increased ($p<0.05$) under Wi5 in L when compared with NoWi and Wi1, and similar trend was also observed in H (Table 1). However, heart rate was unaffected by increased air velocity.

	Light exercise			High exercise		
	NoWi	Wi1	Wi5	NoWi	Wi1	Wi5
T_{re}	37.1 ± 0.1	37.2 ± 0.1	36.9 ± 0.1	37.5 ± 0.1	37.6 ± 0.1	37.4 ± 0.1
T_{sk}	29.3 ± 0.2	28.7 ± 0.2	26.4 ± 0.3	30.2 ± 0.2	29.8 ± 0.3	27.1 ± 0.3
Heat flux	144 ± 4	158 ± 7	200 ± 9	157 ± 4	167 ± 5	196 ± 4
$\dot{V}O_2$	9.4 ± 0.6	9. 4 ± 0.2	11.8 ± 0.9	15.0 ± 0.3	15.2 ± 0.4	16.0 ± 0.5
Heart rate	73 ± 2	75 ± 4	73 ± 3	89 ± 4	92 ± 4	85 ± 5

Table 1. Rectal and mean skin temperatures (°C), mean heat flux (W/m^2), oxygen consumption ($\dot{V}O_2$: ml/min/kg) and heart rate (beats/min) at the end of cold exposure at different air velocities. Values are mean ± SE, n=8.

For local skin temperatures, the cooling effects of 5 m/s wind were seen mainly on anterior and peripheral parts of the body. Skin temperature of the hand is presented in Fig. 1. At the end of H the hand temperature was higher ($p<0.001$) under NoWi and W1, than the temperature of corresponding wind speed in L, but this difference was not observed under Wi5. A similar pattern of temperature change was seen on the finger, but not on the upper parts of the arm.

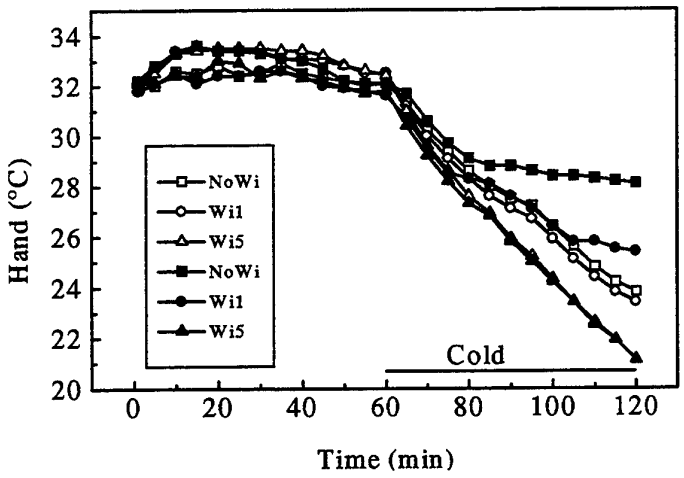

Fig. 1. Hand skin temperature. Open symbols represent the light and solid symbols the high exercise intensity. Values are mean, n= 8.

CONCLUSIONS

The high exercise intensity was accompanied by an increase in T_{re} under all three wind velocities.

Increased heat production during H resulted in a higher T_{sk}, even though none of the exercise intensities could totally prevent superficial cooling. Larger negative heat balance was seen as a faster cooling rate during L in NoWi and Wi1, in comparison to H. Under

Wi5, T_{sk} decreased markedly at both exercise intensities. It appears that exercise intensity is a less determining factor for superficial cooling when wind speed becomes predominant.

Peripheral parts of the body exhibit larger vasomotor responses to cold air than proximal parts, and due to peripheral vasoconstriction skin temperatures decreased more markedly on the hand and finger. High exercise intensity induced heat production partly offset this decrease in NoWi and Wi1 conditions, but not in Wi5.

Wi5 increased mean heat flux. The heat transfer was unaffected by the exercise intensity, even though the exercise intensity affected the skin and body temperatures. These changes were relatively small, and thus did not affect the amount of convective heat loss, particularly when there was no differences in the walking speed.

Increased oxygen consumption during Wi5 was most probably due to a decrease insubmaximal endurance performance caused by the increase of environmental stress and also due to increased muscle tonus and shivering.

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The experimental protocol was approved by the Ethics Committee of the Finnish Institute of Occupational Health.

PAPER 22: EVIDENCE OF SHIVERING FATIGUE: VERIFICATION OF A PREDICTION MODEL

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INTRODUCTION

Search and Rescue authorities in Canada use a prediction model of survival time for cold exposure developed at DCIEM [1, 2]. This model predicts body cooling rates based on anthropometric, environmental, and clothing protection inputs. In circumstances where the cold stress overwhelms the individual's capacity to generate sufficient metabolic heat to offset heat loss, survival time essentially depends on how quickly the body cools to the point of lethal hypothermia (assumed to be a core temperature of 28°C). Often, the cold stress is less severe and the body attains a stable core temperature, usually at a value less than normal. In this circumstance, survival time depends on how long shivering metabolism can be maintained to offset the steady state rate of heat loss.

The survival model uses a prediction of shivering endurance developed by Wissler [3], yet this prediction has not been verified. The present study was undertaken to determine the onset of decline in steady state shivering during continuous exposure to cold and to test the shivering endurance prediction. The first phase of the study determined maximum shivering intensities and the second phase addressed shivering endurance.

MATERIALS AND METHODS

Twelve fit male (n = 9) and female (n = 3) subjects (mean ± SD: age = 24.8 ± 6.3 yr; mass = 71.7 ± 13.2 kg; height = 1.75 ± 0.10 m; body fat = 22.7 ± 7.4%, and $\dot{V}O_{2max}$ = 52.8 ± 6.1 $mL \cdot min^{-1} \cdot kg^{-1}$) participated in the experiment after providing an informed consent. In the first phase, subjects began neck-level immersion in 8°C water for up to 70 min to decrease their core temperature. Thereafter, while still immersed, the water temperature was gradually increased to 20°C to invoke a maximal shivering response. Core temperature (T_{es}) was measured with an esophageal thermocouple. Skin temperatures were measured at 12 sites and weighted [4] to obtain a mean value ($\overline{T}_{sk}$). Oxygen consumption was measured continuously from expired minute ventilation and gas concentrations. The maximal $\dot{V}O_2$ reported ($\dot{V}O_{2shiv\,max}$) was the highest value measured during the entire immersion.

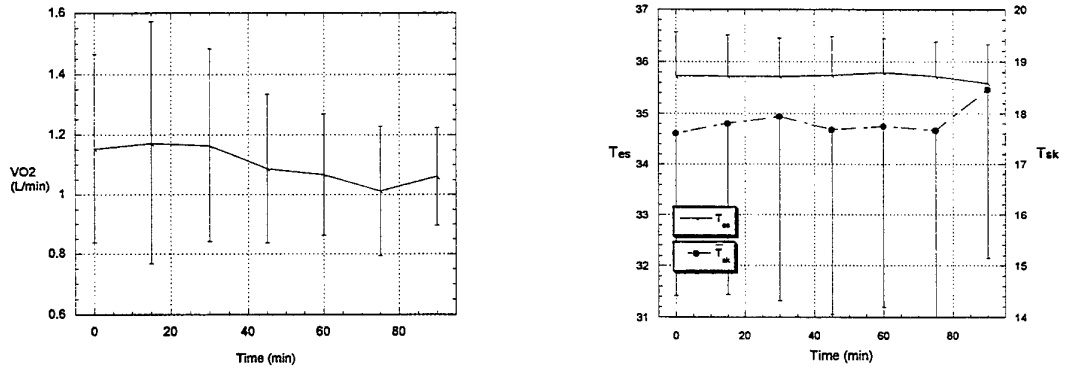
In the second phase, subjects were immersed in water at 8°C for the first 15 min and then at a warmer temperature (but less than 20°C) to invoke a submaximal shivering response. Subjects remained immersed until 6 h elapsed, by request, or by discretion of the investigator.

According to Wissler [3], the end of shivering endurance (t_{end}) is predicted when SUM, defined by $\sum L \cdot \exp^{4 \cdot L} \cdot \Delta t$, equals 18, where L is the relative shivering intensity given by $(\dot{V}O_2 - \dot{V}O_{2rest}) / (\dot{V}O_{2shiv\,max} - \dot{V}O_{2rest})$ and Δt is the time step (in h) based on the interval of the measured oxygen consumption rate. Shivering thermogenesis is considered to be driven by decreases in the core and skin temperatures [5]. Thus, any change in the

metabolic rate due to shivering must be adjusted to compensate for changes in body temperature before any trend analysis on shivering intensity is attempted. The normalization chosen for this purpose was to divide the measured shivering metabolism by a predicted value based on core and mean skin temperatures [6]. The resultant variable was designated as NSHIV, which under ideal circumstances should equal unity. Data (1 min values) were grouped according to the last 30 min before t_{end} (pre30), the 30 min period following t_{end} (post30), and the last 60 min of immersion (last60). These data were then linearly regressed and slopes were tested for significance ($p < 0.05$). All mean data are reported with ± SD.

RESULTS

The subjects' oxygen consumption rates during rest and at maximal shivering were 0.330 ± 0.055 and 1.533 ± 0.238 $L \cdot min^{-1}$, respectively. The latter represents a 465% increase in metabolism over resting values. The accompanying figures show the subjects' oxygen consumption rate, and esophageal and mean skin temperatures during the last 90 min of immersion.



Figures of subjects' mean ± SD rate of oxygen consumption, and esophageal and mean skin temperatures during the last 90 min of immersion.

The predicted end of shivering endurance during the second phase immersion was 113 ± 47 min in contrast to the total immersion time of 215 ± 79 min. The immersion was terminated in 6 cases due to the discretion of the investigator, in 4 cases due to subject request, and in 2 cases because the maximum immersion period of 6 h was achieved. During the immersion, the value of SUM was 38.4 ± 16.8, representing an excess in the predicted endurance time by 113%. The average rate of oxygen consumption was 1.043 ± 0.209 $L \cdot min^{-1}$ or 68% of $\dot{V}O_{2shiv\,max}$. The slopes of NSHIV were +25.8, -37.8, and −25.3%$\cdot h^{-1}$ for pre30, post30, and last60, respectively, and all were significant.

CONCLUSIONS

The nearly 5-fold increase in shivering metabolism over resting values is in close agreement with the results reported by Iampietro et al. [7], and were deemed valid for the analysis of shivering endurance. That shivering continued well beyond the predicted end of endurance suggests an underprediction in the Wissler endurance function [3]. However, shivering intensity began to decrease significantly after t_{end}, indicating that perhaps this variable expresses not the abrupt end of shivering, but the start of shivering fatigue. Indeed, Wissler [3] acknowledged that t_{end} may more accurately reflect the end of steady state shivering. Accordingly, this study supports the Wissler shivering endurance function, although the underlining mechanism of shivering fatigue has not been verified.

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INVITED LECTURE 12: PSYCHOPHYSIOLOGICAL ASPECTS OF COGNITION: TOWARDS AN UNDERSTANDING OF PERFORMANCE IN EXTREME ENVIRONMENTS

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INTRODUCTION

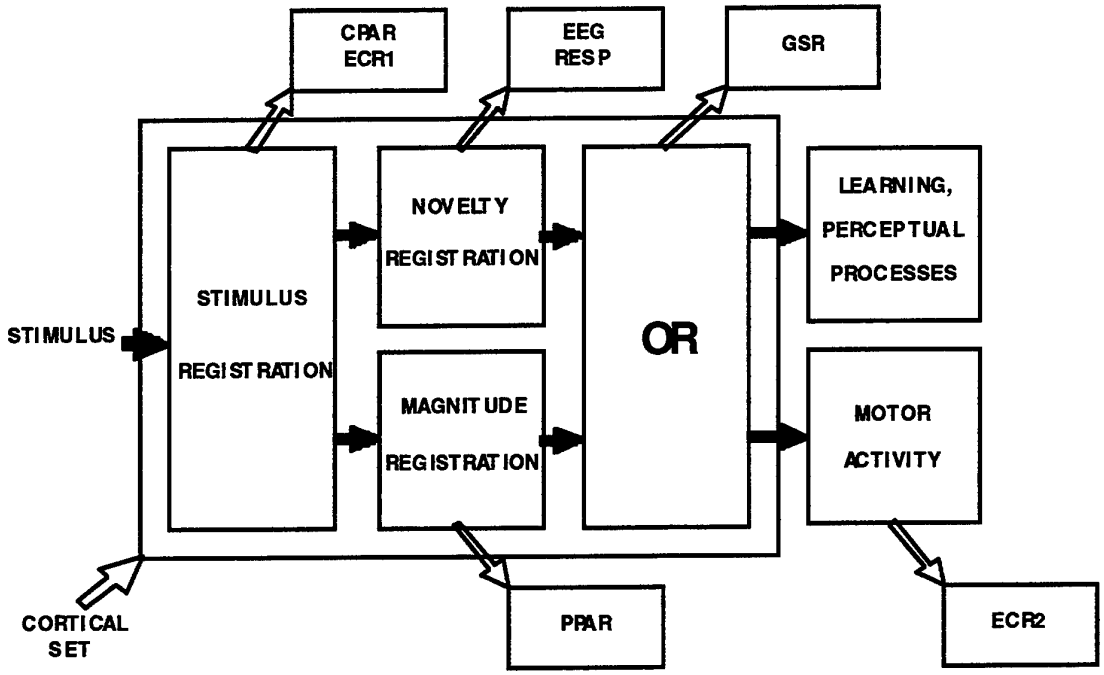
This paper presents an overview of a psychophysiology of attentional processing which incorporates a number of tonic physiological measures, commonly, but perhaps inappropriately, conceptualised in "activation" terms. Although largely ignored in my field, these tonic measures are differentially affected by temperature. A consideration of the nature of these effects holds implications for the understanding of cognitive processing in extreme environments.

THE ORIENTING REFLEX

A major foundation stone in Psychophysiology is the study of the Orienting Reflex (OR), a reflexive orienting of attention to a novel stimulus. This reflex is useful as a model of fast-acting changes in attention - its phasic aspect - as well as a broader model for our interactions with the environment. The OR was first noted as a nuisance variable in Pavlov's work on conditioning of the salivary reflex in dogs. This behavioural orienting of the animal towards a novel stimulus in its environment was named originally as the "What is that?" reflex by Pavlov, and began to be studied in its own right. The most influential Russian worker in the OR context is Evgene Sokolov [1], who made a systematic study of the determinants and physiological correlates of the OR. Sokolov viewed the OR as a unitary reflex, in which a number of physiological components occurred together, and covaried with changes in the parameters of the eliciting stimulus. For example, to the first presentation of an innocuous novel stimulus, such as a 1 sec 1000 Hz tone at 60 dB in the laboratory, human subjects produce brief phasic changes in a number of physiological parameters. These include a rapid heart rate (HR) deceleration (ECR1) of 5-10% beginning within 200 ms and lasting 3-5 sec, a cephalic vasodilation (CPAR) of similar parameters, an increase in skin conductance (GSR) of a similar duration but with an onset latency of about 2 sec, a slow peripheral vasoconstriction (PPAR) peaking at about 8 sec poststimulus, a rapid brief pupillary dilation (PDR), a slowing of respiration (RESP) evident in the respiratory cycle containing the stimulus onset, and a rapid desynchronisation of EEG alpha activity. This response complex was seen by Sokolov as *the OR*, a unitary complex which had a functional role to facilitate stimulus intake and processing. Because the OR marks a shift in attention to the eliciting stimulus, it came to be recognised as the unit of attentional processing. It serves as a model system in the study of how we relate to environmental events. Today, the OR is not accepted as a unitary process. I have proposed instead a sequential attentional processing model, which links various phasic response components to specific preliminary processes in a chain which leads to the shift in attention [2,3,4].

PRELIMINARY PROCESS THEORY

The figure below shows a widely-published schematic of the sequential processing model inherent in Preliminary Process Theory (PPT), emphasising mainly phasic autonomic response components. In recent years I have been integrating more central measures of phasic responding into this theory, particularly event related potential components, and attempting to clarify the role of more slowly- changing (tonic) measures of state. This latter aspect recognises the importance of the state of the organism in modulating the links described above between stimulus determinants and the OR outcomes.

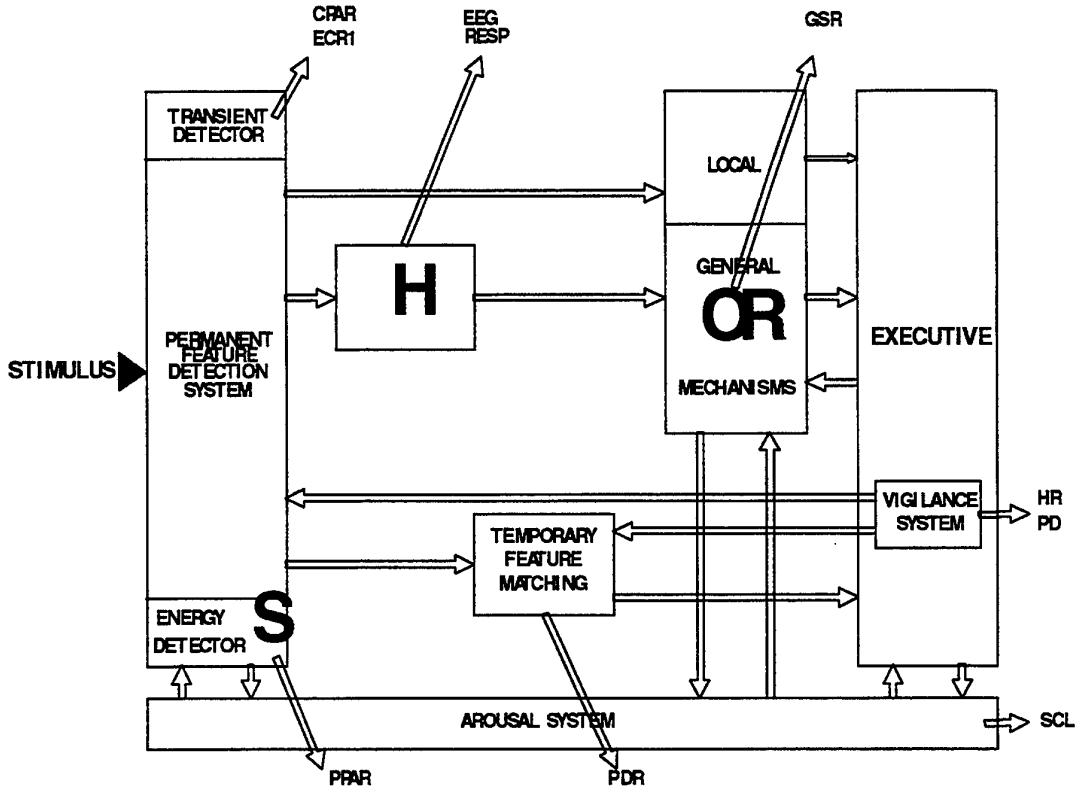


Arousal has long been used in Psychology to describe behavioural outcomes in terms of the individual's state. For example, the inverted-U hypothesis reflects the expectation that the intensity and efficiency of a behaviour will increase with increases in the arousal level of the individual up to some optimal state, after which further increases in arousal will result in reduced performance outcomes. In this sense arousal or activation can be seen as the amplifier of behaviour. Two state measures of particular interest are skin conductance level (SCL) and HR. As measures of arousal or activation, these often, but not always, increase together. For example, in expectation of an oral presentation at this conference, many will experience parallel increases in HR and sweat gland activity. In some other situations, e.g., when engaged in a vigilance task - such as a sonar operator on a submarine in wartime, sweat gland activity will again increase, but HR will decrease. While this has been referred to as "fractionation" of arousal [5], and taken to decrease the usefulness of the arousal concept, an alternative view seems advantageous. I prefer to separately identify these two tonic measures, SCL and HR, with the overlapping but separable concepts of arousal and vigilance. The former refers to the behaviour-amplifying traditional notion of activation/arousal, while the latter refers to an active, often anticipatory, attentional process, which facilitates cognitive processing.

I will present two examples of this vigilance process. Richards and Casey [6] found that infants allowed to watch Sesame Street engaged their attention for a limited period on the video. This was accompanied by a regular decrease in HR, which then plateaued until the infant stopped attending. This "disengagement" was marked by a rapid rebound in HR back to the pre-attentive level. A field study from my laboratory, with expert pistol shooters, found a remarkably similar cardiac response profile. Commencing up to 15 sec before the shot, HR began to drop, and continued to do so until the shot, after which it rebounded to the initial levels. In general, this was accompanied by similar changes in electrodermal activity. However, the systematic nature of the cardiac deceleration was greater in best compared with worse shots, with a slower "disengagement" rebound after the shot. The electrodermal response profile did not differ with the shot outcome. We interpret these data as indicating that the expert shooters quieten their somatic activity prior to the shot, apparent in decreased electrodermal activity. This is a general preparatory

stilling and does not predict performance at this level. In parallel with this somatic quietening, expert shooters narrow and enhance their attentional focus on the sight/target nexus, accompanied by HR deceleration. The extent of this atttentional focussing, also marked by a slower HR rebound at the shot, predicts the level of performance.

The figure below shows a recent version of PPT [4] which incorporates these state processes and facilitates the inclusion of ERP components. In this form we essentially have a model of the psychophysiology of attentional processes, including voluntary switching of attention and higher cognitive processes. This is a long way from the reflexive turning towards a novel stimulus observed by Pavlov, but that remains the core process.



IMPLICATIONS FOR COGNITIVE PROCESSING IN EXTREME ENVIRONMENTS

Because most Psychophysiological research occurs in air-conditioned laboratories, the role of temperature extremes in this context has had little attention. But consideration of the two state measures discussed here suggests that this area is worth pursuing. Differences between the electrodermal and cardiac systems, particularly in terms of cardiac effects on cortical functioning [7,8] suggests that efficient cortical processing in vigilance-type tasks may be adversely affected by extreme temperature changes. Such impairment may occur not solely as a correlate of activation or stress changes from the unpleasant environment, but as a more direct outcome of baroreceptor activity accompanying HR changes occurring in thermoregulation. This possibility appears open to investigation using the model of sequential stimulus processing presented here, and serves to demonstrate the benefits of a systematic approach to the Psychophysiology of attentional processes in the understanding of performance in extreme environments.

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PAPER 23: ANS AND CNS EFFECTS OF LIMB IMMERSION IN ICE COLD WATER

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INTRODUCTION

The cold pressor test (immersion of a limb in ice cold water) is a standardised model of tonic pain. The cold pressor evokes substantial changes in the sympathetic division of the autonomic nervous system (ANS) with increases in heart rate (HR), systolic and diastolic blood pressure (SBP and DBP) being well documented [1]. However because the cold pressor test also influences thermoregulatory and somatosensory mechanisms in the ANS [2] it is difficult to separate the cardiovascular responses produced by cold and those produced by pain. Peckerman et al. [2] concluded that this overlap in responses is seen primarily in arterial blood pressure, with HR increases primarily related to pain.

Quantitative-electroencephalogram (Q-EEG) analysis has also been used over the past decade to investigate EEG frequency changes during the cold pressor test. Q-EEG studies have shown increases in delta activity [3; 4; 5] which has been interpreted as reflecting pain [3]. Increases in beta activity [3; 4; 6] and decreases in alpha activity have also been reported [4; 5] with one study [6] reporting augmentation of alpha activity during the cold pressor test.

The present research was designed to clarify ANS and Central Nervous System (CNS) responses during the cold pressor test. Experiment 1 investigated skin conductance level (SCL), respiration rate (RR), heart rate (HR) and systolic and diastolic blood pressure (SBP and DBP). In accordance with previous literature, it was predicted that the cold pressor test would evoke increases in the cardiovascular system. It was also predicted that the cold pressor test would produce a general increase in sympathetic activity thus producing increases in SCL and RR. Experiment 2 examined Q-EEG responses to the cold pressor (delta, theta, alpha and beta). It was predicted that cold stimulation would bring about increases in delta activity and beta activity and decreases in alpha activity with eight frequency bands examined in the response.

MATERIALS AND METHODS

Subjects

Participants were fifty-seven female subjects in Experiment 1 and thirty-six female subjects in Experiment 2. Subjects were undergraduate Psychology volunteers from the University of Wollongong.

Procedure

In both experiments, subjects were seated in a testing booth for a physiological adjustment period averaging 15 minutes. The last 3 minutes of this physiological adjustment period was recorded and used as a baseline stage. The cold pressor test required subjects to immerse their non-dominant hand up to the level of their mid forearm in an insulated container for a maximum period of 3 minutes.

Data recording and transformation

In Experiment 1, SCL in microSiemens, RR in breaths per minute, HR in beats per minute and SBP and DBP in millimetres of mercury were recorded continuously over the baseline and cold pressor stages. In Experiment 2, electroencephalogram (EEG) and electro-oculogram (EOG) were recorded continuously from the midline sites (Fz, Cz, Pz) referenced to linked ears. The signals were digitised at 246 Hz and stored to hard disk for

later off-line analysis. Equally spaced 2 second Fast Fourier Transforms (FFTs) were averaged over 15 second epochs with the data classified into eight frequency bands, delta-1 (0.5-1.5 Hz), delta-2 (2-4 Hz), theta (4.5-8 Hz), alpha-1 (8.5-10 Hz), alpha-2 (10.5-12 Hz), beta-1 (12.5-18.5 Hz), beta-2 (19-24.5 Hz) and beta-3 (25-30.5 Hz). These data were converted to relative power measures (%). Equally spaced 15-second epochs were analysed over the baseline and cold stages. Baseline effects were examined using repeated measures MANOVAs and any effects were controlled for in the response analysis by ANCOVA in Experiment 1 or a regression procedure in Experiment 2. Response patterns were analysed from the last 15 second time point of baseline to the first two 15 second points in the cold pressor cold using repeated measures MANOVAs or MANCOVAs.

RESULTS

Due to space restrictions only significant responses to the cold pressor test will be presented.

Experiment 1: Autonomic Nervous System Responses

A MANCOVA examining SCL from the end of baseline-1 to the first two points in cold, revealed linear, $F_{(1,54)} = 73.59$, $p < .001$ and quadratic, $F_{(1,54)} = 74.40$, $p < .001$, response components with the means indicating an increase in SCL over this period (6.20, 7.70 and 7.58 respectively). A repeated measures MANOVA examining HR showed linear, $F_{(1,54)} = 22.21$, $p < .001$, and quadratic, $F_{(1,54)} = 41.90$, $p < .001$, increases from the last point in baseline (72.21) to the first two points in the cold pressor test (83.35 and 78.03). A repeated measures MANOVA from the last point of baseline to the first two points in cold showed a linear effect of time, $F_{(1,54)} = 23.27$, $p < .001$, with the mean DBP showing an increase from the end of baseline (77.64) to the first two points in cold (81.27 and 83.92).

Experiment 2: Q-EEG Responses

Delta-1 activity

The response analysis revealed significant linear, $F_{(1,33)} = 7.50$, $p < .05$, and quadratic, $F_{(1,33)} = 5.18$, $p < .05$, increases in delta-1 from the last point of baseline to the first two epochs in the cold pressor test, as indicated in Figure 1.

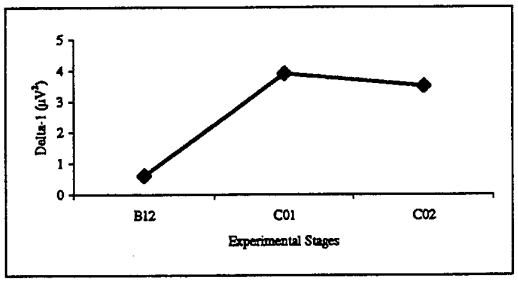

Figure 1. Delta-1 across experimental epochs.

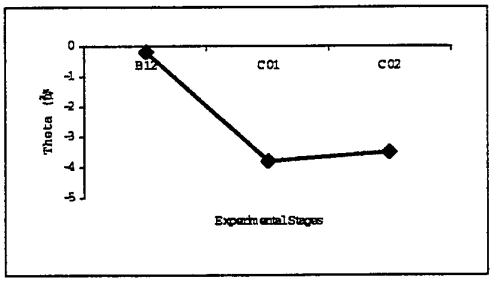

Figure 2. Theta across baseline and cold stages.

Theta activity

The response analysis revealed linear, $F_{(1,33)} = 10.20$, $p < .01$ and quadratic, $F_{(1,33)} = 11.70$, $p < .01$, response components, and this decrease in theta is shown in Figure 2.

Alpha activity

Figure 3 shows alpha-1 and alpha-2 activity from the last point of baseline-1 to the first two points in the cold pressor test. There were significant linear, $F_{(1,33)} = 19.84$, $p < .001$, and quadratic, $F_{(1,33)} = 37.50$, $p < .001$, response components in alpha-1 from the last epoch of the baseline stage to the first two epochs in the cold pressor test; this effect on alpha-1 is

shown in Figure 3. There was a significant decrease in alpha-2 from the last epoch of baseline-1 to the first two epochs in the cold pressor, apparent as linear, $F_{(1,33)} = 4.69$, $p < .05$, and quadratic, $F_{(1,330)} = 12.32$, $p < .01$ trends over the data points. This is indicated in Figure 3.

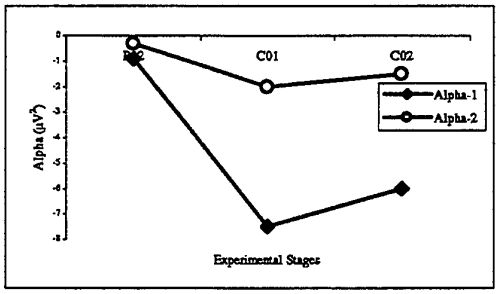

Figure 3. Alpha-1 and alpha-2 across epochs

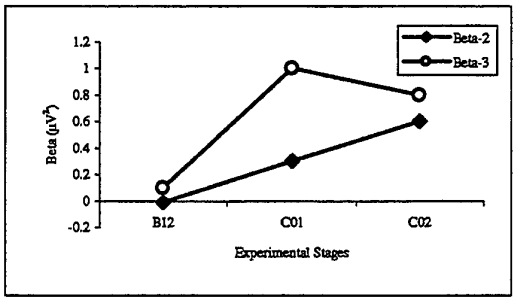

Figure 4. Beta-2 and beta-3 across time

Beta activity

The response analysis of beta-2 showed a significant linear increase of beta-2 in the cold pressor test, $F_{(1,33)} = 11.44$, $p < .01$. The response analysis of beta-3 showed greater beta-3 revealed significant linear, $F_{(1,33)} = 9.18$, $p < .01$, and quadratic, $F_{(1,33)} = 4.09$, $p = .05$ response components; these effects on beta-2 and beta-3 are shown in Figure 4.

CONCLUSIONS

The cardiovascular results were consistent with previous research [1] and from [2], it can be concluded that the increase in DBP is a result from the additive effects of cold and pain, with HR being primarily related to pain. Because SCL is interpreted as reflecting heightened autonomic arousal, the increase in SCL in the present study is also interpreted as reflecting pain. Q-EEG analysis showed increased delta and beta activity, which was as predicted and decreased theta and alpha. Increased delta has been interpreted as reflecting pain [3] and from the present study, this is primarily reflected in delta-1. Beta activity has also been thought to reflect pain [3] but because this increase was seen in only high frequency beta, this increase may be due to increased muscle artifact from clenching of the facial and jaw muscles [6]. The finding of alpha desynchronisation concurs with previous studies, with alpha-1 interpreted as an arrest reaction of arousal of the subject and alpha-2 explained as reflecting increased pain. Like alpha, the decrease in theta is also interpreted as a non-specific increase in arousal.

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NOTE: This protocol was approved by the Human Research Ethics Committee of the University of Wollongong.

PAPER 24: HEART RATE VARIABILITY AS A MEASURE OF COGNITIVE WORKLOAD

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INTRODUCTION

As complex human-machine systems have developed, the importance of cognitive workload and its assessment have become increasingly significant. Increasing automation and the introduction of new technologies have seen a reduction in physical workload requirements and a marked increase in cognitive demands [1, 2, 3].

Arising from this shift towards cognitively-based activities is the realisation that traditional operator workload measurement techniques are less successful in complex domains. In order to distribute and schedule tasks efficiently and safely in the military cockpit or flight-deck, the extent to which an operator is loaded is of great importance. The shift from physical to cognitive demands has made it increasingly difficult to quantify the effects of task requirements on operators. Modern day military aviators for example, are presented with enormous amounts of data. The management of these data can place excessive demands on aircrew cognitive resources. Moreover, as the role of the operator shifts toward monitoring and information management the observation of overt behaviours becomes an increasingly inadequate measure of workload [4].

This paper addresses the use and suitability of heart rate variability (HRV) as an index of cognitive processing requirements, reporting on a laboratory-based experiment investigating the extent to which variations in cognitive workload are reflected in operator HRV.

The basis for the analysis of HRV in the operator workload context stems from the work of Kalsbeek and Ettema [5,6]. They reported a 'gradual' reduction in HRV arising from increased levels of perceptual load and task difficulty. While some have identified relationships between the physiological and the cognitive [7,8]; it should be noted that the timing of the heart beat is also influenced by factors unrelated to cognitive demand and efforts must be made to exclude these factors. Spectral analysis of the cardiac interbeat interval (IBI) time series (providing a measure of HRV) has made it possible to partition out the unwanted effects of several homeostatic feedback loops [9]. More precisely, frequency bands corresponding to thermoregulation (0.02-0.06Hz) and respiration (0.15-0.50Hz) have been decomposed from a middle frequency band associated with the regulation of arterial blood pressure (0.07-0.14Hz) [11,12]. It is around the 0.1Hz region that has been found to be most associated with variations in operator perceived task load [11, 13, 14, 16].

Through the collection and comparison of objective performance measures, subjective workload estimates and HRV measures this study examined the suitability of HRV as a metric of operator cognitive workload during aviation-related tasks.

METHOD

Eight participants (7 males and 1 female, 22-41 years of age, with a mean age of 28 years), all employees of DSTO, took part in the research. Their basic task was to perform a 2-axis visual tracking task (manipulating a joystick to keep a pair of intersecting lines centred on a computer screen) and respond to a series of auditory warnings by pressing one of eight function keys corresponding to the type of threat indicated by the warning. Workload was further manipulated by adding tasks to this base task. In one set of conditions an additional

visual/manual task required the participants to monitor two moving gauges on the left and right hand side of the screen, and respond by pressing the appropriate button on the joystick whenever a gauge went out of range (which would then re-set the gauge). A second workload condition involved an additional auditory task with a significant cognitive component. A series of air traffic control (ATC) messages was played to the participants as they carried out the base task. They were to monitor the messages and remember any that were associated with their call sign. A third workload condition combined the gauge and ATC message monitoring tasks with the base task. Four main workload conditions were therefore employed: (1) tracking task and auditory warnings (TW); (2) tracking task, auditory warnings, and gauges (TW+G); (3) tracking task, auditory warnings, and ATC messages (TW+M); and (4) tracking task, auditory warnings, gauges, and ATC messages (TW+G+M). The combination of these tasks was meant to simulate some of the workload experienced when flying an aircraft.

Each participant completed seven test sessions: an initial practice session to familiarise them with all the tasks, and six experimental sessions. Each experimental session involved four short practice trials followed by eight test trials, each of 150 seconds duration. Over the 150 seconds participants tracked continuously, responded to eight auditory warnings, and depending on the workload condition, monitored gauges (performing eight re-sets), and/or monitored ATC messages (with a maximum of four messages to be remembered). At the end of each trial, they rated their workload on four scales (input demand, central demand, output demand, and overall workload on a 0-100 scale). The eight test trials in an experimental session consisted of two repeats of each of the four workload conditions. The order of conditions was balanced across trials, sessions, and participants.

Finally, during the test trials each participant's heart rate was recorded using a Polar R-R Recorder™. The recorder is a digital, 24-hour precision ambulatory recording device that measures, stores, and post-processes real-time R-R interval data (inter-beat intervals – IBIs - in milliseconds). The data from each experimental session was downloaded and corrected for any artifacts. The mean of the IBIs for each trial was calculated, together with the standard deviation of the IBIs, which was used as an overall measure of heart rate variability (HRV). The IBIs for each trial were then converted to a periodic time series, the nonstationary low frequency trends were removed, the data were smoothed and the detrended time series was passed through a Fast Fourier Transform. The power (ms^2) within the 0.07 Hz to 0.14 Hz band was calculated and the natural logarithm of this measure was used as the 0.1 Hz component measure of HRV.

RESULTS

The following measures were obtained on each trial: Tracking performance (root mean square error – RMSE – in pixels – with scores closer to zero indicating greater accuracy); Auditory warning performance (mean correct response latency to warning); Subjective workload ratings; and the three heart rate measures described above. The impact of the addition of the gauge and/or ATC message monitoring tasks on each measure was assessed by a 2 (gauge versus no gauge) by 2 (ATC messages versus no ATC messages) repeated measures analysis of variance ($\alpha = .05$).

For the tracking task (see Figure 1) the addition of the gauge (G) and ATC message monitoring (M) tasks led to more error ($F_{(1,7)} = 5.11$, $p<.05$), however, the combined G+M workload condition did not impair performance beyond that obtained with the addition of a single workload task. For the auditory warning task, response times were slowed down by an average of 100 milliseconds with the addition of the message monitoring task ($F_{(1,7)} = 15.7$, $p<.01$). The gauge task, however, had no impact on these response times. A similar pattern was obtained with the *overall* subjective workload ratings: Workload was rated 30-points higher with the addition of the message monitoring task ($F_{(1,7)} = 40.3$, $p<.001$), but

the gauge task was not rated as adding significantly to the overall load. However, when the workload ratings were broken down to input, output and central demands, both input ($F_{(1,7)}$ = 14.9, p<.01) and output ($F_{(1,7)}$ = 7.0, p<.05) demand were rated 5-points higher with the addition of the gauge task. The message monitoring task increased rated input load by 20-points ($F_{(1,7)}$ = 108.2, p<.001) and central processing demand by 34-points ($F_{(1,7)}$ = 67.9, p<.001).

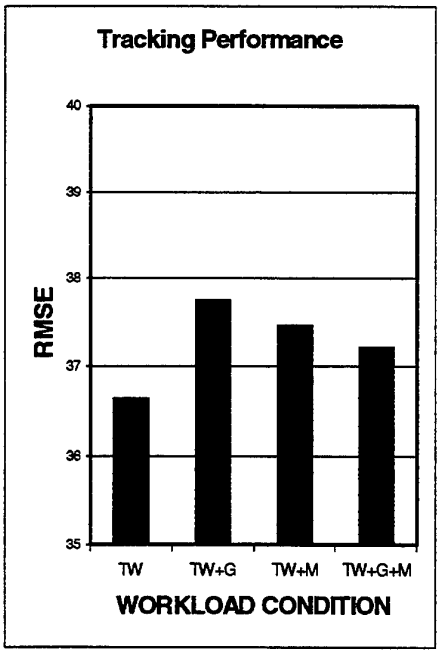

Figure 1. Workload Effects on Tracking Performance.

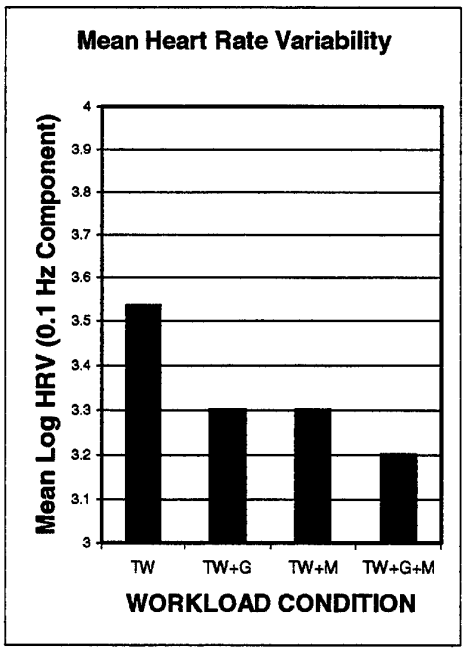

Figure 2. Workload Effects on Heart Rate Variability (0.1 Hz component).

For the heart rate measures, neither the mean IBI nor the overall HRV measure was affected by workload conditions. There was however a significant reduction in the 0.1 Hz component of HRV (see Figure 2) with the presence of the message monitoring (M) task ($F_{(1,7)}$ = 6.1, p<.05). The gauge task did not significantly reduce this HRV component, although the trend (p=.08) was in this direction.

DISCUSSION

The results from this research have confirmed the utility of heart rate variability as a physiological index of mental workload. In particular, it has demonstrated that a spectral analysis of HRV provides a sensitive measure of cognitive workload. This measure was sensitive to manipulations that had been clearly rated as increasing workload, and that had impacted on the performance of the primary tasks (tracking and auditory warning responses). The other heart rate measures were unsuccessful in capturing these effects. It appears that partitioning out the influence of factors such as respiration and body temperature regulation on heart period has left a cardiac index that is well suited to measure *overall* cognitive workload. What the measure does not capture is moment-to-moment fluctuations in workload. The spectral analysis process requires a time series of sufficient length to enable the partitioning of unwanted sources of variation. The measure also lacks a diagnostic capability – it may index level of workload, but it does not provide an indication of the underlying source of that workload. These limitations suggest that a combination of workload measures, including subjective and performance-based measures in addition to this physiological measure, should always be employed to get a complete workload picture. What the spectral HRV measure does offer is an objective on-line measure (there is no impact of operator biases and expectations), that is relatively

unintrusive (no overt response is required by the operator, and the Polar system used here was light-weight, and did not restrict performance of the tasks). The Polar system, with its simple download capability, together with a suite of software to carry out the spectral analysis, means that a spectral measure of workload can be made available almost immediately following any recording session.

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PAPER 25: PSYCHOMETRIC ASSESSMENT OF THE EFFECTS OF THERMAL STRAIN ON COGNITION

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INTRODUCTION

While the physiological responses to environmental heat have been well established, its impact on cognition is less clear. A review [1] of the effects of thermal environments on vigilance concluded that thermal stress would not cause deterioration unless external conditions are severe enough to perturb the deep core temperature. There appears to be a general consensus that thermal strain will compromise cognition, but the level of performance deterioration is dependent on the severity of heat strain and the complexity of the task.

In a military context, it is important to understand the limitations of human performance imposed by environmental heat, as so many strategically important areas lie in hot regions [2]. Australia's Strategic Guidance [3] indicated that the focus of future military operations would be in the north. This means that soldiers would have to operate in tropical conditions, and the likelihood of heat illness, if soldiers are unprepared, would be high. While physiological deterioration can certainly impair an individual's ability to accomplish specified tasks involving physical work, the inability to assess situations and make rational decisions due to cognitive deficit could have profound effects that may compromise the mission at a higher level. Communications, command and control may break down, leading to failure in mission accomplishment. It is therefore imperative to develop appropriate methodology for assessments of cognitive performance in the heat so that thermal stress management strategies can be improved.

This study used psychometric testing to investigate the impact of thermal strain on cognition. The psychometric testing can be used as a package for assessing cognitive performance.

MATERIALS AND METHODS

Eleven males underwent testing under three levels of thermal stress: (1) 25°C/65%RH, (2) 35°C/65%RH, and (3) 35°C/65%RH with raised core temperature (>38.5°C).

Subjects changed into their combat uniform (DPCU) and were then instrumented. This involved attachment of a Polar heart rate monitor, followed by placement of the rectal thermistor (self-administered after instruction) for the purpose of measuring core temperature. For the raised core temperature condition (35°C+NBC), subjects then dressed in the Nuclear Biological and Chemical warfare (NBC Mk. IV) suit.

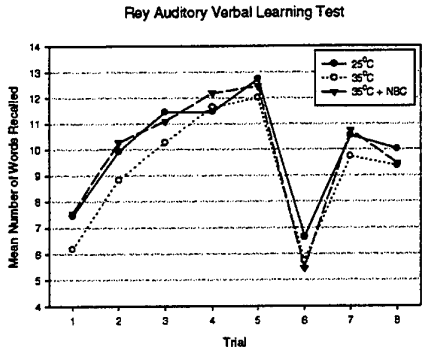
For the 25°C and 35°C conditions, subjects began walking at a slow pace (1.8 $km.hr^{-1}$), which was maintained during the recording session. For the 35°C+NBC condition, subjects began walking at a higher rate (5 $km.hr^{-1}$) and on an incline of 5-12% in order to raise their core temperature. The work-rate was individualised and was maintained (under close supervision of heart rate and subjective ratings of well-being) until core temperature reached 38.5°C, at which point the treadmill was set to 0% gradient and 1.8 $km.hr^{-1}$, in preparation for the beginning of the testing session. Subjects were tested on the Rey Auditory Verbal Learning Test, Digit Span, and Inspection Time.

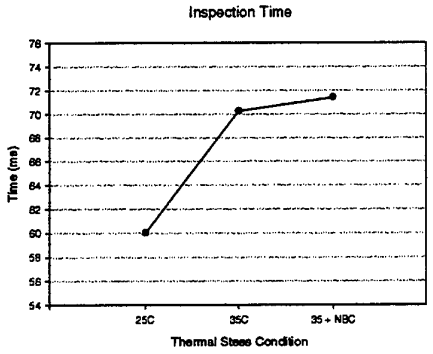
RESULTS

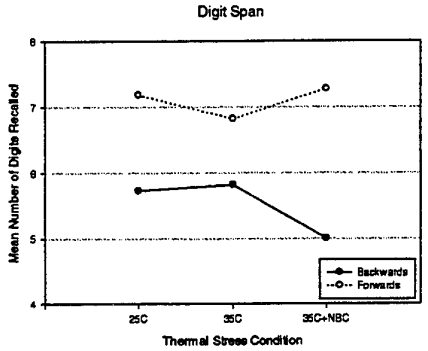
The relevant behavioural data (accuracy) for each test was analysed using a within subjects design repeated measures analysis of variance (ANOVA). For the psychometric tasks (RAVLT, Digit Span and Inspection Time), three measures showed significant differences across the three treatment conditions (25°C, 35°C and 35°C+NBC).

Trial 2 of the RAVLT showed a significant difference across the three thermal stress conditions $\{F_{(2,9)}=6.09, p=0.021\}$, but simple contrasts (which compare each of the thermal conditions to the others and indicate which differences are significant) showed that the 35°C condition was significantly different from both the 25°C condition $\{F_{(1,10)}=5.714, p=0.038\}$ and the 35°C+NBC condition $\{F_{(1,10)}=8.101, p=0.017\}$, while there was no significance difference between the thermally neutral (25°C) and 35°C+NBC condition. This tends to suggest that an elevation of thermal strain may not be linked to performance deterioration in RAVLT.

Digit Span backwards showed a trend towards significance $\{F_{(2,9)}=4.262, p=0.05\}$ and contrasts revealed that performance in the raised core temperature condition (35°C+NBC) was significantly lower than in the 25°C $\{F_{(1,10)}=7.11, p=0.024\}$ and 35°C $\{F_{(1,10)}=5.400, p=0.042\}$ conditions, suggesting that there may have been an effect of thermal strain.







Inspection Time was also significantly different across the three conditions $\{F_{(2,9)}=9.387, p=0.006\}$, but the contrasts revealed that both the 35°C $\{F_{(1,10)}=19.582, p=0.001\}$ and 35°C+NBC $\{F_{(1,10)}=6.539, p=0.029\}$ conditions were significantly different from the 25°C condition. Performances in the 35°C & 35°C+NBC conditions were not significantly different from one another $\{F_{(1,10)}=0.137, p=0.719\}$, suggesting that there may have been an effect of raised ambient temperature.

DISCUSSION

There was no clear indication of performance decrements with increased core temperature, suggesting that thermal strain had no effect on the ability of subjects to perform the tasks. However, the decrement for Inspection Time during the high ambient temperature (35°C and 35°C+NBC) conditions was a promising result, as was the trend towards a significant difference observed for Digit Span Backwards, which was achieved with a very small subject pool (n=11). These results possibly warrant further investigation, given that these changes may have reached statistical significance had there been a larger number of subjects.

A review of the literature on the effects of thermal stress on sustained attention [1] suggests that mild hyperthermia can improve performance, but only if the increase in temperature does not disturb homeostasis (i.e., the mildly hyperthermic state of the subject is static). "These conditions are similar to those observed during the sequential increase in temperature associated with the circadian rhythm. Under these circumstances, in which the relative rate of change of body temperature is small, performance also improves with ascending temperature level." [1, p.278].

However, in conditions where deep body temperature is perturbed, stress acts to drain attentional resources, thereby leaving less attentional resources left to perform a task [1]. The thermal stress experienced by the subjects did perturb their core body temperature away from homeostasis, and the data for Inspection Time and Digit Span Backwards are promising preliminary results, given that they were achieved with a very small subject pool (n=11). It is conceivable that these changes would have achieved statistical significance had there been a larger number of subjects.

Consistent with attentional resource capacity explanations of the effects of thermal stress on vigilance [1], it is suggested that in this study, whilst task performance was not affected by thermal stress, this was achieved by the use of more attentional resources or effort by subjects to compensate for the 'draining' effect of thermal strain.

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INVITED LECTURE 13: MARKSMANSHIP AND SENTRY DUTY PERFORMANCE UNDER EXTREME ENVIRONMENTS

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INTRODUCTION

The Weaponeer, a U.S. Army M16 rifle marksmanship simulator and training device, has been adapted for assessment of soldier performance in the laboratory. The Weaponeer permits evaluation of both marksmanship speed (ability to detect and to hit rapidly appearing pop-up targets) and accuracy (the variability, or "tightness," of the shot group). Performance on the Weaponeer has been shown to be predictive of live fire performance on the rifle range [1]. This report briefly reviews key experimental studies utilizing the Weaponeer, which show how marksmanship varies with the wearing of protective clothing, the exposure to ambient heat, the administration of drugs (nerve agent antidotes, antihistamines), the administration of a mild stimulant (caffeine), and requirements for sustained attention.

MARKSMANSHIP AND PROTECTIVE CLOTHING

In the first study [2], the rifle marksmanship of 30 soldiers was assessed under three U.S. military clothing configurations of increasing bulk: (a) the battle dress uniform (BDU); (b) the fighting load which consists of the BDU plus helmet, web gear, and full canteen; and (c) the chemical protective clothing ensemble (MOPP-IV). Soldiers were given 5 days practice on the Weaponeer by repeatedly firing at 32 randomly presented pop-up targets at simulated distances of 100 and 250 meters. Rifle marksmanship was assessed on the fifth day. Rifle marksmanship was significantly poorer ($p<0.05$) under the MOPP-IV condition (M = 22.1 hits) than under either the fighting load condition (M = 26.0 hits) or the BDU condition (M = 27.4 hits). Impairments while wearing MOPP-IV were attributed to the awkwardness in obtaining a rapid and proper sight alignment while wearing the chemical protective mask.

MARKSMANSHIP, AMBIENT HEAT AND NERVE AGENT ANTIDOTE

In a comprehensive study of the effects of ambient heat and nerve agent antidote on sensory and psychomotor performance [3,4], the rifle marksmanship of 15 soldiers was assessed in a 2 x 2 design: (a) 35°C vs. 21.1°C ambient temperature, and (b) nerve agent antidote (600 mg 2-PAM chloride, 2 mg atropine sulfate) vs. placebo (saline). Marksmanship speed (as measured by the ability to hit rapidly appearing pop-up targets) was impaired by nerve agent antidote while marksmanship accuracy (tightness of the shot group for a stationary target) was impaired by 35°C ambient heat. Compared to the placebo condition, a single dose of nerve agent antidote significantly impaired rifle marksmanship for pop-up targets such that marksmanship speed was 3% poorer. Compared to the 21.1°C condition, the 35°C ambient condition significantly impaired rifle marksmanship for stationary targets such that the tightness of the shot group was 13% less accurate. Nerve agent antidote and ambient heat did not interact to further impair rifle marksmanship performance. Since ambient heat and nerve agent antidote each impair separate components of marksmanship (accuracy and speed, respectively), the soldier's overall marksmanship performance is likely to be degraded more if the soldier has to operate in a hot environment while also under the influence of nerve agent antidote.

MARKSMANSHIP DURING SIMULATED SENTRY DUTY

Mackworth 's classic work on vigilance [5] showed that the ability to detect infrequent and brief (less than one second) stimulus changes in the visual field deteriorated after only one-half hour and remained deteriorated for the remainder of a two hour test session. While Mackworth's task was modeled after that of a sonar operator, the task is analogous to that of a soldier on sentry duty who must scan a visual field and detect the appearance of enemy targets. The soldier on sentry duty, however, must not only detect the appearance of a visual target but must also pick up a rifle, aim, and fire accurately at the target. In our first sentry duty study [6], the rifle marksmanship of 8 soldiers was assessed during 3 hours of simulated sentry duty during which time the soldier had to respond to the infrequent appearance of a target at a simulated distance of 250 meters (12 presentations per 30 minute period). When the target appeared, the soldier's task was to pick up the rifle, aim, and fire at the target. In accordance with Mackworth's findings, target detection time deteriorated with time on sentry duty; impairments were not evident within the first hour but were clearly evident by 1.5 hours. The ability to hit the target remained constant over time; soldiers were just as accurate at the end of the 3 hours of sentry duty as they were at the beginning (M = 9.6 hits out of a possible 12). The results of this study suggest that sentry duty performance may be optimized if appropriate duty intervals are assigned.

EFFECTS OF ANTIHISTAMINES ON SENTRY DUTY PERFORMANCE

The first in a series of studies of the effects of drugs on sentry duty performance evaluated two antihistamines, terfenadine and diphenhydramine [7]. Unlike terfenadine, diphenhydramine crosses the blood brain barrier and is likely to cause drowsiness. The sentry duty scenario was chosen as the model for assessment of the relative sedative effects of the drugs. The rifle marksmanship of 12 soldiers was assessed during 3 hours of simulated sentry duty during which time the subject had to respond to the infrequent appearance of a target (12 per 30 minute period) by picking up the rifle, aiming, and firing at the target. Prior to the study, the subject received either a clinical dose of antihistamine (either 60 mg terfenadine or 50 mg diphenhydramine), a placebo, or no pill (control). Speed of target detection was impaired by time on the task. Both speed of target detection and marksmanship accuracy were further impaired by diphenhydramine. Neither measure of marksmanship was impaired by terfenadine, an antihistamine which does not cross the blood brain barrier.

EFFECTS OF CAFFEINE ON SENTRY DUTY PERFORMANCE

In an attempt to improve sentry duty performance, by administration of a mild stimulant, we used the sentry duty model to evaluate the effects of caffeine (an over-the-counter stimulant commonly used to maintain mental alertness) on the sentry duty performance of 12 male soldiers [8]. The results showed that 200 mg caffeine (equivalent to about 2 cups of coffee) markedly improves the sentry's speed of target detection while leaving M16 rifle firing accuracy unimpaired. In a subsequent study, the efficacy of 200 mg caffeine was replicated with both male and female soldiers [9]. However, while the men's rifle firing accuracy again remained unimpaired during the entire 3 hours, the women's rifle firing accuracy became impaired after 1.5 hours on the task.

While the results of these first two caffeine studies are consistent with what is generally accepted about caffeine being a mild stimulant and about time on a task leading to impaired vigilance performance, their generalizability to the combat soldier is limited. That is, these studies were conducted with all targets being foe (meaning that the sentry fires at any and all targets). This is of vital importance since soldiers on the battlefield must routinely discriminate friend from foe. Wartime fratricide rates indicate that friend-foe discrimination decisions are not always correct. Our latest study [10] evaluated sentry performance when friend-foe discrimination is required. Eleven men and 11 women

participated in four simulated sentry duty sessions lasting 3 hours each: (a) 200 mg caffeine, foe-only; (b) 200 mg caffeine, friend-foe; (c) placebo, foe-only; and (d) placebo, friend-foe. Participants monitored the target scene of the Weaponeer Rifle Marksmanship Simulator with instructions to fire only at enemy targets. Without impairing marksmanship, 200 mg caffeine reduced friend-foe discrimination errors and eliminated the decrement in target detection speed associated with time on the task. Men were likely to commit friendly-fire errors (shoot at friendly targets) and women were likely to commit fail-to-fire errors (fail to shoot at enemy targets).

CONCLUSIONS

The Weaponeer M16 Rifle Marksmanship Simulator was used for assessing soldier performance under environmental extremes and under procedures designed to protect the soldier from environmental threats. Experimental data indicate that rifle marksmanship is impaired by exposure to ambient heat (35^{o}C), the wearing of combat chemical protective clothing (MOPP-IV), medications (antihistamines and nerve agent antidotes), and sentry duty (vigilance) conditions. Speed of target detection and the accuracy of friend-foe discrimination during simulated sentry duty is improved by caffeine.

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PAPER 26: COGNITIVE PERFORMANCE AND PHYSICAL STRESSORS IN EXTREME ENVIRONMENTS

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INTRODUCTION

Deterioration of performance is a serious problem in extreme environments especially in the battle field. The individual soldier faces many stressors while trying to accomplish his assigned missions. Heat stress [1] can degrade performance and reduce mission effectiveness particularly when combined with carrying heavy loads [2] over long distances, wearing chemical protective clothing [3,4] and sustained mental and physical work load [5,6]. This raises a necessity to develop a standardized, simple and comparable method for measurement of performance in field conditions. The assessments require the use of well-defined laboratory performance tests, their applicability validated in field studies conditions. This study reports the results of four experiments, investigating the effects of several stressful conditions on subjects performing specific military-relevant laboratory tasks [7], designed to evaluate a range of psychological processes under extreme environments [8].

MATERIALS AND METHODS

Subjects: Healthy, young male volunteers, aged 18- 28 years.

Procedure: Four experiments were conducted. In the first lab experiment 12 subjects walked for two hours on a treadmill, consisting at a constant speed of 5 km/h pace with a 5% grade, in a climate chamber with temperature set at 20°C and RH at 50%. Experimental design involved two levels of exercise dictated by carrying backpack loads of varying weight (20 and 35 kg). The second lab_experiment involved an exposure to a combined stressful condition of heat stress and physical exercise. Eight heat-acclimatized subjects walked for two hours on a treadmill, at a constant pace speed of 5 km/h and 1% grade, in a climate chamber of 35°C and RH of 50%. Eight full different chemical defense ensembles, consisting of monopack, bipack, foam cloth or carbon fiber cloth were used. The third experiment was a field study designed to evaluate performance of 13 subjects, tank crews, following an exercise of 1-2 hour which involved an engagement in operational-tactical tasks. The temperature climate ranged from 20°C to 25°C and RH from 20% to 40%. Two stressors were employed: a newly Israeli developed chemical protective mask (with standard Battle Dress Uniform + helmet) and the same mask fitted in a full chemical protective gear (including an ensemble buttoned up, hood, helmet and nomex gloves + chemical protection gloves). In the last field study, 11 subjects were randomly divided into 2 teams, and were monitored during continuous operation lasting 72 h. Subjects rested during the day but were active at night. Temperature and RH climate ranged between 20°C and 75% in the morning and 37°C and 20% at noon.

Behavioral testing: Quantitative assessment of performance and behavior was made using a computerized battery of brief, standardized and comparable laboratory tests, tapping attentional, perceptual, psychomotor and cognitive functions, as well as motivational and affective processes. The tests represented elemental skills generally regarded as underlying real-world tasks. Specific tests used in the study are: 1. Four Choice Serial Reaction Time task, evaluating information processing resources dedicated to stimulus encoding and categorization, response selection and execution [9]. 2. Number Facility Test, measuring the subject's ability to sum simple addition problems [10]. 3. Alpha-Numeric Visual Vigilance Test, characterizing the observer's ability to attend and respond to small

stimulus changes over long, unbroken periods of time [6]. 4. Tracking test, evaluating the ability to execute rapid and accurate manual responses in a model of measuring the dynamics of manual control behavior [11]. A profile of mood states questionnaire is reported too [12]. Prior to experiments, subjects were trained eight times until they reached a baseline. Data were analyzed by MANOVA followed by simple main effects contrasts analysis.

RESULTS

Four Choice Serial Reaction Time: No significant effect on reaction time (RT) was indicated following the carrying of a backpack load of 20 kg. Carrying a backpack load of 35 kg , while walking on the treadmill, resulted in a significant moderate increase (~ 10%) in RT for worst-10% correct-responses (lapses) (see Fig 1). Wearing protective clothing, including wearing mask + gloves alone, while walking on a treadmill in the heat, increased RT for lapses (22 to 27%), although specific ensembles degraded performance more than others (see Fig 2).

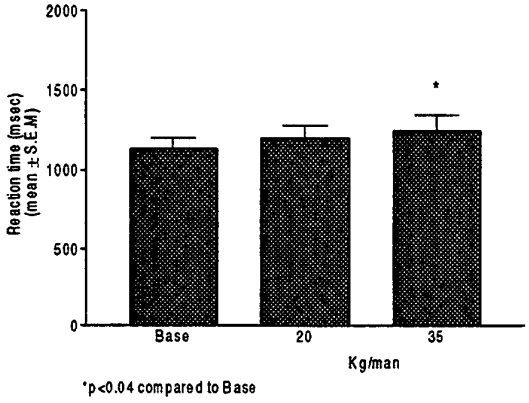

Fig1: Four choice reaction time test- Exp. 1.

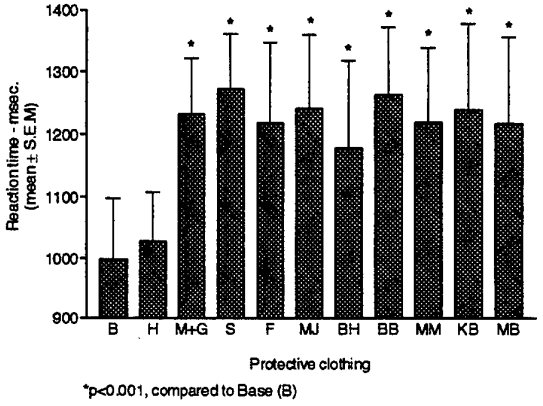

Fig 2: Four choice reaction time test- Exp. 2.

RT was not affected by heat stress only (without protective clothing). In the third experiment RT for lapses significantly increased by approximately 50% in subjects wearing a full protective gear, while an increase of only about 25% was observed in subjects wearing the newly developed mask. (see Fig 3). A similar increase in choice RT (15-20%) was observed in team 2 subjects engaged in continuous operation, specifically at 4:00 a.m. or following a night of sustained physical workload activities (see Fig 4).

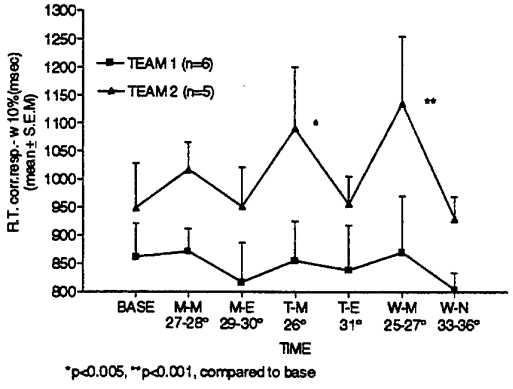

Fig 3: Four choice reaction time test- Exp. 3.

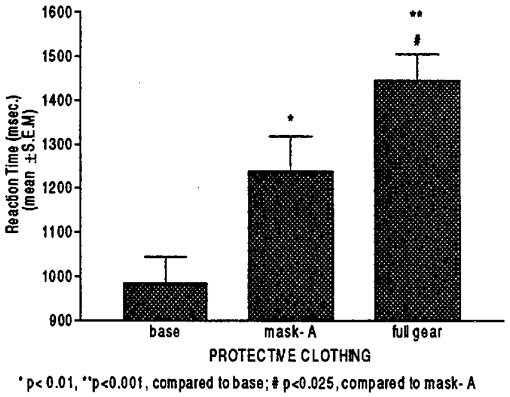

Fig 4: Four choice reaction time test- Exp. 4

Number Facility Test: In the second experiment, number of corrections significantly increased (150-300%) only in subjects wearing 3 specific ensembles while walking on a treadmill (see Fig 5). In the third experiment, RT for correct responses increased by 20% in subjects wearing full gear, while an increase of only 10% was observed in subjects wearing only the newly developed mask (see Fig 6). No other effects have been demonstrated.

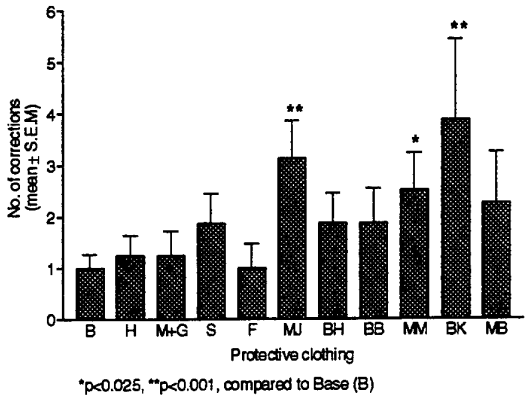

Fig 5: Number facility test- Exp. 2.

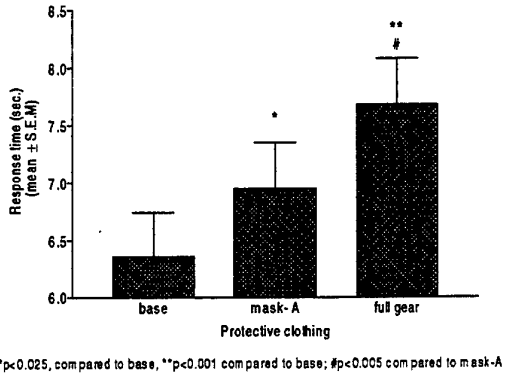

Fig 6: Number facility test- Exp. 3.

Alpha-Numeric Visual Vigilance Test: A significant increase in RT for correct target detection was demonstrated in team 2 subjects during continuous operation, specifically at 4:00 a.m. (170%) or following nights of sustained physical activity (75%) (see Fig 7). No other effects were observed.

Tracking Test: A significant increase in number of losses of control was shown in team 2 subjects exposed to sustained operation, either at 4:00 a.m. (40%) or following a night of continuous physical workload activities (80%) (see Fig 8). No other effects were observed.

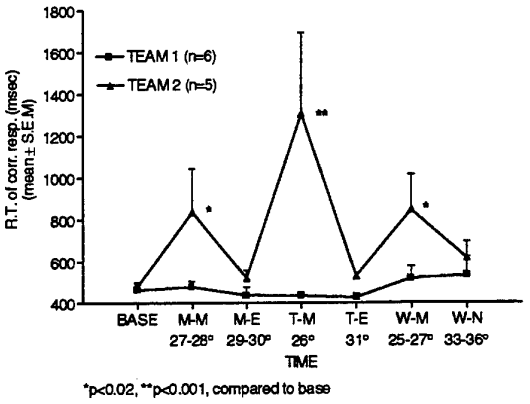

Fig 7: Visual vigilance test- Exp. 4

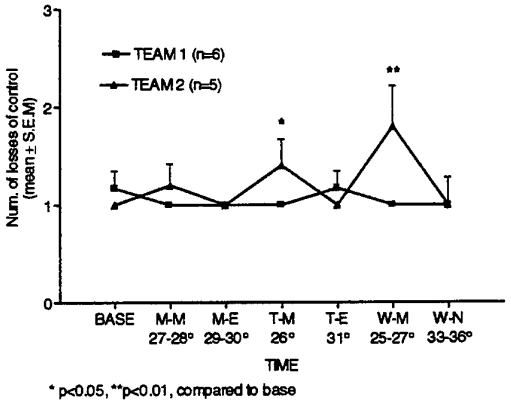

Fig 8: Tracking test- Exp 4.

Profile Of Mood States: A decrease in self-reported activity and friendliness, a general deterioration of mood and a substantial increase in fatigue were noted in Exp. 2, when subjects were wearing 3 foam-cloth ensembles, although there were no evidence of cognitive deficits. During continuous operation, fatigue and activity changed concomitantly with cognitive deficits but emotional changes did not.

DISCUSSION

Psychomotor functioning was sensitive to the effects of several stressors including load carry, physical exercise activities, wearing protective clothing and sustained physical and mental workload. Mathematical-symbolic information integration requiring working

memory was sensitive to short period of physical exercise and wearing protective clothing. Sustained attention (vigilance) and tracking ability were specifically sensitive to continuous workload and circadian rhythm. Heat stress degraded cognitive performance only when interacted with other stressful conditions such as physical discomfort as a result of wearing protective clothing and prolonged physical exertion. Otherwise, deterioration in motivation and mood were the only deficit observed. Finally, sustained physical workload exertion and circadian rhythm interacted with individual variables (motivation, intellectual level, etc.) of the subjects in their effect on performance. The results show that the psychometric tests used enable a differential detection of various behavioral processes and characterization of a precise profile of performance degradation. They also prove the potential benefit of computerized test batteries for differential identification of the effects of stressful conditions on different cognitive demanding tasks in extreme environments.

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All experiments were approved by the Ethical Committee of the Medical Corps.

PAPER 27: BRAIN ELECTRICAL ACTIVITY MAPPING AND THE EFFECTS OF THERMAL STRAIN ON A SPATIAL WORKING MEMORY TASK

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INTRODUCTION

A wide range of environmental factors can influence human performance. Of these, high heat and humidity are particularly debilitating, as elevated environmental heat can impose an enormous strain on the cardiovascular system to maintain a consistent body temperature. Failure to dissipate excessive body heat will lead to a rise in deep core temperature, resulting in physiological dysfunction, heat exhaustion and, if unchecked, death. While the physiological responses to environmental heat have been well established, it is less clear about its impact on cognition.

Hancock (1986) [1] concluded that thermal stress would not cause deterioration unless external conditions are severe enough to perturb the deep core temperature. Razmjou (1996) [2] argued that deficit of mental performance in the heat could be offset by an increase in arousal, and that only tasks of a more complicated nature (secondary task) resulted in a significant performance deficit. There appears to be a general consensus that thermal strain will compromise cognition, but the level of performance deterioration is dependent on the severity of heat strain and the complexity of task. In hot and humid conditions, the cognitive functions most vulnerable are probably the maintenance of vigilance and short-term working memory.

While conventional psychometric test batteries can provide a broad understanding of cognitive performance under specific conditions, recent advances in brain-imaging techniques have increased the capabilities for visualising the working brain. These techniques are potent tools to uncover the functional areas of the brain responsible for various cognitive tasks, leading to a better understanding of the neural architecture of mental abilities [3].

Silberstein et al (1990) [4] have developed an EEG paradigm known as Steady State Probe Topography (SSPT), which utilizes the steady-state visual evoked potential (SSVEP). It involves using a probe or task-irrelevant stimulus (a visual flicker), and then observing the changes in amplitude and phase of the potentials evoked by that probe stimulus as a cognitive task is undertaken. The SSPT technique provides a measure of rapidly changing brain processes in a continuous fashion and with a relatively high temporal resolution (seconds) [4].

This study used functional brain electrical activity imaging to investigate the impact of thermal strain on cognition. The SSPT technique was employed to investigate regional changes of brain activity and information processing speed while subjects performed specific tasks under thermal stress.

Recording Difficulties

Testing under conditions of increased temperature and humidity creates unique problems for recording EEG, including overheating, condensation and increased sweating by subjects, which causes 'bridges' of sweat to electrically connect scalp electrodes. The problem with sweating is that it may be non-uniform across the scalp, leaving some electrodes to perform as expected whilst others are "bridged", leading to differences in the EEG signal due to recording conditions rather than due to differing regional brain activity. This was overcome by applying a low-conductive gel to the entire head, which had the

effect of "sweating for the subject" and removing sweat-induced variations in inter-electrode resistance.

Another difficulty in recording EEG was the fact that to maintain the subject's temperature at or above 38.5°C, the subject had to continue exerting himself during the recording period, leading to an electrically "noisy" environment and production of large voltage, undesirable signals (artifact) that masked the extremely low voltage, underlying cortical activity of the brain.

One of the properties of the Steady-State Probe Topography technique is the ability to filter out unwanted electrical signals. Electronic 'noise' (50 Hz interference) was further reduced by using a filter that cut off any frequencies higher than 40 Hz. Perhaps the biggest problem caused by movement is "clipping", a situation whereby the electrical signal exceeds the dynamic range of the amplifier. In such a situation, all information conveyed by the EEG signal is lost, and to minimise loss of signal produced by movement of the subject, the speed of the treadmill during recording was held at 1.8 $km.hr^{-1}$, and an amplifier with a large dynamic range (16-bit) was utilised.

MATERIALS AND METHODS

The study was designed to look at the effects of thermal strain on brain electrical activity under three different levels of thermal stress: (1) 25°C/65%RH, (2) 35°C/65%RH, and (3) 35°C/65%RH with raised core temperature (>38.5°C).

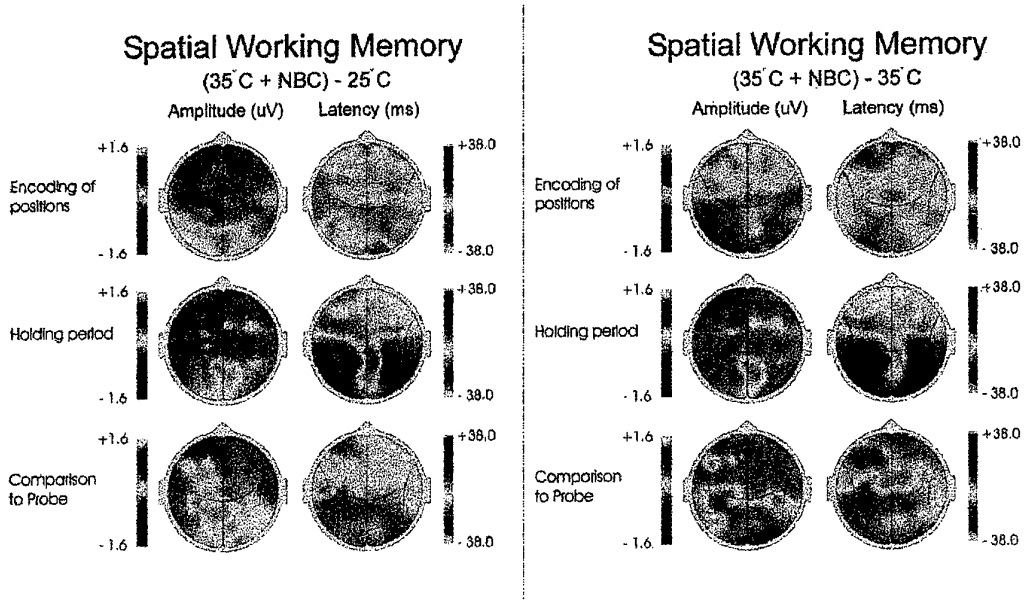
Eleven male participated as subjects. Subjects changed into the standard Australian Army Disruptive Pattern combat uniform (DPCU) before instrumentation was carried out. This involved attachment of a Polar heart rate monitor, followed by placement of the rectal thermistor (self-administered after instruction) for the purpose of measuring core temperature. For the raised core temperature condition (35°C+NBC), subjects then dressed in the Nuclear Biological and Chemical warfare (NBC Mk. IV) suit.

Preparation of reference and ground electrode sites was then carried out, followed by application of the electrically conductive gel to the scalp. EEG was recorded from 64 sites using standard electrophysiological techniques, with the nose serving as ground and linked references attached to each earlobe. Brain electrical activity was amplified and bandpass filtered prior to digitisation to 16-bit accuracy at a rate of 500 Hz, and stored for later offline analysis.

Subjects then stepped onto the stationary treadmill and each electrode was checked to ensure a clean signal. For the 25°C and 35°C conditions, the treadmill was then started and subjects began walking at a slow pace (1.8 $km.hr^{-1}$), which was maintained during the recording session. For the 35°C+NBC condition, subjects began walking at a higher rate (5 $km.hr^{-1}$) and on an incline of 8-12% in order to raise their core temperature. The work rate was individualised and was maintained (under close supervision of heart rate and subjective ratings of well-being) until their core temperature reached 38.5°C, at which point the treadmill was set to 0% gradient and 1.8 $km.hr^{-1}$, in preparation for the beginning of the recording session.

RESULTS

The topographic brain electrical activity maps below are a birds-eye view of the head, and show differences between the raised core temperature condition (35°C+NBC) and 25°C and 35°C conditions. Warmer regions indicate increased brain activity for the raised core temperature condition.



DISCUSSION

The results show that whilst the performance on the Spatial Working Memory task was not significantly different across the three temperature conditions $\{F_{(2,4)}=0.932,\ p=0.426)\}$, there were marked changes in the SSVEP. Compared to the 25°C and 35°C conditions, the raised core temperature (35°C+NBC) condition showed increased SSVEP amplitude and decreased latency, notably in frontal regions. It is suggested that these changes during the raised core temperature condition were reflective of increased brain activity associated with performance of the tasks, and reflect a greater utilization of neural resources or effort by subjects to maintain performance at the same level as when they were not thermally stressed.

This interpretation is suggestive of the existence of a 'cognitive reserve', whereby subjects have at their disposal a certain amount of neural resources that can be allocated to the performance of tasks and activities. Performance of these tasks and activities will deteriorate when the amount of resources is insufficient to deal with both the tasks and thermal stress, such that subjects will be able to maintain their performance level until the resources are overloaded.

Such a construct is consistent with attentional resource capacity explanations of the effects of thermal stress on vigilance [1], in which stress is viewed as draining attentional resources. In the case of this study, whilst task performance was not affected by thermal stress, the SSPT results suggest that this was achieved by the use of more attentional resources or effort by subjects to compensate for the 'draining' effect of thermal strain.

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INVITED LECTURE 14: THE MEASUREMENT OF THERMAL STRAIN IN SOLDIERS OPERATING IN NORTHERN AUSTRALIA

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INTRODUCTION

Australia's strategic priorities are focused on the Defence of Australia and in offering assistance in regional stability. Most of this activity occurs in tropical Northern Australia or in similar environments. High temperatures, humidity and solar load, associated with the tropics contribute to the development of heat related injuries in military personnel. Consequently, thermal stress can limit performance with respect to physical work output, cognitive function and long-term effectiveness. The exposure to high levels of thermal stress arising from physical work, clothing, environmental conditions can potentially reduce the operational capability and effectiveness of the Australian Defence Force (ADF) [1,2,3,4,6].

It has been clearly demonstrated both scientifically and anecdotally that heat related illnesses are a major concern for the ADF during operations [5]. The implications of poor management of heat strain could therefore have a dramatic negative impact on mission specific operations and personnel well being. The development of a general-purpose personal monitor to assess and inform ADF combatants and their commanders of their current state of physiological strain would enable the implementation of better strategies for heat stress management. One possible answer would be the development of a personal device to monitor the "*vital signs*" of personnel during most circumstances.

Vital Signs Monitoring

In man, physical, environmental or psychological stress can significantly contribute to physiological strain, consequently limiting physical and cognitive performance [7,9]. In the protected state our bodies may tolerate large variations in environmental temperature but the range of deep core temperature (T_{core}) variations are generally within 4°C [8]. Beyond this limit optimum physical and cognitive performance could be severely affected. The consequence of disrupting the thermoregulatory processes affects many physiological systems; principally, temperature-dependent processes and physical mechanisms that take place during normal body functions [9,10]). Although thermo-regulation and its control have been thoroughly investigated [9 to 13], physiological strain generally manifests itself as cardiovascular and thermal (T_{core} and sweat rate) strain. Thus, the development of a "*vital signs*" monitor for the detection, control and prevention of the debilitating impact of physiological strain, with particular emphasis on heat related issues, is currently being pursued.

A wide spectrum of devices are focused on monitoring critical care patients in stable, controlled environments or intervened field situations [14,15]. They rely on both obtrusive and invasive measures to record " vital" parameters of a patient's physiological state. These particular measures, such as blood pressure, pulse oximetry, core temperature (rectal, esophageal, intravascular, tympanic), ECG etc. are either highly invasive or easily disengaged by physical movements. In the case of bedside monitoring, these logistical considerations are not significant issues. Hence many companies have developed specialised instrumentation to perform bedside monitoring (Compumedics, Keller, Alaris, Kenquest, Mediconf) that is inappropriate for field situations.

Advances in miniaturisation have enabled the recording, logging and telemetry capabilities of current bedside instrumentation to be used in the field and highly portable monitoring

devices for personal use have recently taken advantage of these changes. For instance, a data logger for recording of core temperatures (rectal, skin and auditory canal) during exercise has been successfully miniaturized while maintaining sensor accuracy ±0.04°C [16]. Similarly, vital signs monitors, such as VITSEM 200, have been developed for field use on military casualties [14,15]. The physiological parameters measured were heart rate, rectal temperature, pulse oximetry and resting blood pressure. Filtering improvements and RF shielding, have significantly improved their capability to withstand noisy environments, such as in helicopters and moving vehicles.

The requirement for such types of personal monitors has lead to the development of personnel status monitors that have been used to successfully monitor physiological parameters during training [17 to 20]. The configuration used by Hoyt et al. [17] contained an elaborate array of sensors to include HR, metabolic cost of locomotion, exercise intensity ($\%VO_{2max}$) and core temperature by radio pill. The sophisticated device, Windows CE palmtop computer, recorded metabolic energy expended in locomotion, core temperature (T_{core}) (CorTemp and BCTM), heart rate (HR) (polar) and location. They suggested that the relationship (slope) between HR and metabolic cost of locomotion may be used to monitor aerobic fitness and heat acclimation, and nutritional information could be obtained by the EEM (expended energy monitor). Although this complicated monitor successfully recorded various physiological parameters, it provided no feedback to soldier or scientist in real-time and was only applicable to unrealistic activities such as walking on level terrain. Cotter et al. [19] and Lau et al [20] have used separate commercially available miniature devices to record HR, T_{core} (gastrointestinal temperature by radio pill) and skin temperatures and found the techniques suitable for field applications.

An enhancement to basic physiological recorders has been the inclusion of a telemetry system. Used in a similar fashion to most portable, miniaturized monitors/recorders, these devices have the added potential to relay images and data, such as soldier position and physiological status to remote sites for analysis [21,22].

The present state of technology allows for adequate storage and telemetry of real-time data but the selection and suitability of appropriate vital physiological indicators depicting heat-related problems for field use is still unclear. Hence the quest for appropriate markers of physiological status that can be applied with minimal disruption and discomfort.

Critical indicators of thermal strain

There are numerous studies that have measured many indicators of thermal strain, but the selection of parameters that are practical for field situations has not been clearly defined. Current physiological parameters for the determination of thermal strain are generally based on body core temperature and heart rate measurements. However, there is some evidence to suggest that one or a combination of indices of physiological strain should be adopted to predict impending hyperthermia. There is however a lack of consensus on the critical physiological limits at which performance of the individual is affected. Wyndham, Strydom et al [23] found that acclimatised men were judged as performing easy if their rectal temperature was below 38.1°C (onset of linear rise) and excessive if it exceeded 39.4°C (saturation). Similarly specific heat stress guidelines [12] suggest that 39.0°C should not be exceeded and propose that a 1°C rise in core temperature will increase cardiovascular strain by 33 $b.min^{-1}$, a very conservative estimate. Kamon, Benson et al. [24] used limits of 110 $b.min^{-1}$ and 38°C as suitable thresholds for thermal strain but indicated that these were daily averages that an individual should not exceed.

Many other approaches have been attempted by using a single index for prediction of heat strain. Studies have used heart rate recoveries as indices of impending thermal strain [25, 26]. Fuller & Smith [25] used 1-min HR recovery points as an indicator of thermal strain and suggested that 110 $b.min^{-1}$ was an acceptable threshold for 1 min post recovery.

However accuracy of prediction was poor under various ambient conditions and the technique could only be applied upon cessation of physical exercise. These findings were later supported, since there was concern over the generation of false positive results using this approach [7]. Others have used sweat rate as the index of physiological strain [28,29]. The former indicated that T_{rec} was not a reliable measure of body temperature due to its slow dynamics and based their prediction on the inverse relationship between sweating and skin temperature. However, they did not include the effect of hydration status on sweat rate and heat strain, and considered sweat loss by weight as the indicator of dehydration and hence physiological strain. Consideration was given only to conditions where evaporative cooling occurred and heat strain indices were adjusted for each individual. Hence there was no consideration for acclimatization status or its applicability in other scenarios. Pandolf, Sawka et al. [29] found HR and T_{rec} correlations with total sweat rate, onset of dripping, and ratio of forearm blood flow to dripping sweat rate and hence concluded that the level of thermal strain while exercising in the heat may be determined by skin blood flow and sweating. However they failed to control for relative work load ($\%VO_{2max}$) in subjects under test. Since it is well known that repeated heat exposures decrease sweating threshold [10], this approach may produce misleading results.

In summary, the major determinants of heat tolerance appear to be acclimatisation status, physical fitness, obesity, age and clothing [30,31]. Hence, since similar adaptive processes occur during physical training and heat adaptation, the suggested variables to monitor physiological strain are T_{core}, HR, sweat rate and skin blood flow. Contrary to previous studies, there is insufficient evidence to indicate that convergence of T_{core} and T_{skin} indicates subsequent hyperthermia [32].

Modelling thermal strain

The development of predictive models of thermal strain (T_{core}) have led to the implementation of complex algorithms requiring vast numbers of independent predictor variables. The USARIEM model [33] has been used extensively to predict thermal strain during space operations and in soldiers wearing different military clothing ensembles with a high degree of accuracy, within 1 RMSD of observed value. As expected, it found that dehydrated unacclimated subjects exhibited the greatest heat strain. The LUT25 model [34] generated similar results but failed to correctly predict T_{core} under various states of acclimation and intermittent work intensities. Because of the complex nature of these models they are difficult to apply and the calculated parameters are delivered in retrospect.

Physiological strain indices

The latest and probably the most realistic predictor of physiological strain is the Physiological Strain Index (PSI; [35]). It includes HR and T_{rec} terms normalised across their functional range, rest to maximal values determined by workplace guidelines. This study compared PSI with three other previously developed indices, the Integral Stress Index (ISI), Cumulative Heat Stress Index (CHSI) and the Heat Strain Index (HSI). The ISI and CHSI correctly described the rise in T_{rec} and HR over 120 min, but failed to represent the recovery period correctly; whereas HSI used too many parameters and produced misleading results. However a major disadvantage was that the indices could only be solved after data collection. Moran, Shitzer et al. [35] therefore developed the PSI for real-time calculations. It assumes a T_{rec} range of 36.5-39.5°C and a HR range of 60-180 $b.m^{-1}$.

$$PSI = 5(T_{rec} - T_{rec0}).(39.5 - T_{rec0})^{-1} + 5(HR_t - HR_0).(180 - HR_0)^{-1}$$

The PSI is scaled from 0 to 10 and contains strain categories synonymous with Borg's RPE scale. The flexibility of this index allows either T_{es} or T_{rec} to be used depending on required kinetics. Subsequent validation of the PSI has been made at various levels of hydration [36]. Furthermore, the PSI correlates to exercise intensity and hypohydration level and

inversely to sweat rate, correctly ranking each combination in order of strain severity. The PSI also appears sensitive enough to reveal gender differences based on aerobic fitness (VO_{2max}) [37], with aerobic fitness being the most important variable for matching genders when heat exposed.

Surrogate indicators of thermal strain

Core temperature measurements generally involve the use of standard intrusive methods using devices such as rectal and oesophageal thermistors. The very nature of these procedures induces not only a psychological objection but also can be quite uncomfortable for a subject. Hence, a less intrusive procedure appeals not only to scientific investigators in practical terms but is more likely to be acceptable to the subjects. Thus monitoring of thermal strain by insulated skin is a novel, but by no means new, concept for the prediction of body core temperatures or thermal strain. The rationale for using a surrogate measure of core temperature, such as insulated skin, is based on evidence suggesting that T_{skin} is influenced largely by T_a at low-medium work-rates and to a lesser extent by exercise intensity [30]. At higher work rates, they hold similar importance. When compared with T_{esoph}, T_{skin} appears to have a delay and continues to rise for approximately 5 min after exercise, in a manner similar to T_{rec}. Thus, skin blood flow is a major determinant of T_{skin}, which is relatively independent of local muscle temperature. From the current literature [30] it appears that the four major factors governing T_{skin} are exercise intensity, total heat production, cutaneous adiposity, and skin blood flow.

IR Tympanic temperature

Infra-red (IR) thermometry which samples tympanic membrane temperature (T_{ty}) rapidly via an otoscope-like probe in the ear canal [40] is a possible alternative to T_{re} monitoring in the field. Significant correlations between T_{re} and IR T_{ty} have been noted in diverse subject groups, including firefighters exercising in 25°C conditions [49] and fun-runners who collapsed in 16°C conditions [41]. Hansen and Amos [42] concluded that IR T_{ty} measures can provide useful estimations of exercise core temperature in warm and hot environments, provided corrections are made for the effect of ambient conditions in the field and provided best-fit regression equations for predicting T_{re} from T_{ty}. Amos et al [3] concluded that the IR T_{ty} technique showed considerable promise as a surrogate measure of T_{re} during a field trial on soldiers conducting routine patrol and reconnaissance operations in the tropics. The correlation between T_{re} and T_{ty} was such that T_{ty} could predict T_{re} with a standard error of estimate of 0.3°C. [$T_{re} = 0.39\ T_{ty} + 23$]

Personal heat strain monitors

Fox and Solman [38,39] attempted to exteriorise core temperature by an active insulated skin thermistor. The concept was based on using zero-heat transfer between the insulated environment and skin, hence exteriorising T_{core}. The technique, however, was found to degrade with progressive increases in environmental temperature [38] and was invalid at temperatures <25°C and >40°C. The device had limited use since it appeared to be applicable only to subject groups in non-extreme environments under low physical demands [43]. Bernard & Kenney [44] theoretically suggested using insulated skin temperature (chest) and heart rates to predict core temperature, with a covariate of clothing type. Adjustments for work demand ($\%VO_{2max}$) based on an individual's heart rate range and endurance times could also be used to improve the prediction. However, complex averaging techniques and numerous parameters complicate the use of this procedure in real time and limited its practicality in most situations. Prior to this work the same authors developed a personal heat stress monitor, based on insulated skin temperature (chest) and heart rate. Bernard et al found that the environmental condition, age, clothing, fitness level and gender did not influence the accuracy despite correct prediction in only 85% of the subjects. This success rate was quite high considering that their experimental protocol

involved 5-min work periods, discounting the phase delay between core (rectal) and subcutaneous insulated skin temperatures.

An recent investigation found that, from a practical point of view, an insulated skin temperature measured over the spine at the level of t2-t4 offers the best solution [30,47 ,48]. However, the core prediction equations used data epochs where core temperatures (T_{esoph}) increased, no recovery or resting periods were used in prediction. The authors did not control for ambient temperature, rather three separate simple linear equations at 25, 33 and 40°C were derived. Insulated skin temperature and T_{es} converged in heat but remained divergent at thermoneutral temperatures. Nevertheless, strong correlations (r>0.8) between T_{esoph} and insulated spine temperature were observed at the various ambient temperatures [39].

Future developments

The prediction of thermal strain requires sophisticated computer models and is of general applicability to groups of soldiers. However the development of an individual monitoring device would be of inestimable value in measuring thermal strain during military operations. Primarily, the parameters measured need to be good indicators of physiological strain, such as HR and an acceptable measure of core temperature either by radio temperature pill or by a surrogate measure such as insulated skin temperature. While radio pill temperature measures are accurate, the equipment, especially the radio pill, is expensive for routine use; hence the interest in surrogate measures for core temperature. Coupling core temperature, however derived, with heart rate in a single device should be able to monitor the Moran PSI in real time to give a ready indicator of overall physical strain.

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PAPER 28: THERMAL STRESS AND PERSONAL COOLING SYSTEM

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INTRODUCTION

Large areas of the Australian subcontinent are either hot or hot and humid. Defence personnel are required to operate routinely in these environments where heat stress is a major health concern. The use of combat body armour and / or Nuclear Biological Chemical (NBC) protective suits exacerbates this heat strain considerably, by encapsulating the wearer from the environment and prevents the dissipation of body heat. Heat stress and related injuries can significantly restrict the efficiency of military personnel operating in these conditions. The impact could result in either a reduction in the operational tempo, or a significant risk of heat casualties. Most of the defence equipment available to the ADF are designed specifically for cooler climatic conditions. NBC suits from US or UK for example are more suitable for operations in colder climatic conditions. Microclimate cooling systems employing circulation of ice water or air-cooling have been developed to cool personnel operating in hot conditions. Such systems require sophisticated logistic support and heavy battery power. For military operations, particularly for infantry, such technologies are not practical.

There is no effective method of cooling individuals to reduce heat strain in circumstances where air-conditioning cannot be set up in confined areas or inside a vehicle. This applies particularly to dismounted combatants, fire-fighting and damage control parties, and all members wearing protective ensembles that fully or partially isolate them from the environment.

CSIRO and DSTO have an ongoing interest in developing personal, readily man-portable personal cooling system (PCS) which would provide sufficient cooling to enable full operational performance to be maintained for extended periods while the combatants are fully protected.

PERSONAL COOLING SYSTEM DESIGN REQUIREMENTS

For the continuation of a physical activity at a given level of work, while wearing a NBC suit, the quantity of heat rejection through the NBC barrier must be greater or equal to the metabolic heat generation rate, while the skin and the core temperature are less than prescribed maximum values.

Heat rejection estimation

In order to obtain a realistic measure of increased combatant performance, the alleviation of thermal strain by PCS has been modelled using a modified USARIEM heat strain predictive model [1]. Figure 1 shows the predicted body core temperature of combatants wearing two different protective clothing systems assuming 150 W or 300 W heat extraction from the body by the PCS. When wearing combat body armour, a combatant, exercising hard (800 W) and without cooling, would reach the thermal strain limit (core temperature 40^0C) in approximately 80 minutes. For the same exercise, the PCS would lower the core temperature by 0.7^0C with 150 W heat extraction and 1.3^0C with 300 W heat extraction.

The operational time would be increased approximately three-fold assuming 150 W heat extraction and further with 300 W extraction, with a reduced level of thermal strain. The simulation also predicts core temperature reduction of 0.7°C for a person exercising at a high work rate wearing DPCU and body armour for 80 minutes and 1.6°C for a moderate work rate with a US NBC protective ensemble for 170 minutes in 40°C heat and 30% relative humidity when cooling system can extract 150 W of body heat to the ambient air.

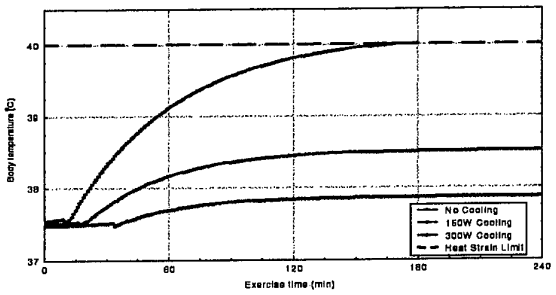

Figure 1. The effect of cooling on body temperature during heavy work at 40°C in US NBC Ensemble

Majority of the heat production is in the trunk and leg [2] and for this study it is assumed that the heat transfer device will collect heat from the trunk and reject it to the ambient air. The trunk is assumed to be 0.5 m^2 and all of this surface has the same efficiency of heat transfer .

Available temperature potential estimation

Figure 2 shows ambient conditions for 4 different locations in Australia on the psychrometric chart, their corresponding wet-bulb temperature and the skin temperature (assumed 36°C). It can be seen that the difference between the skin temperature and the dry-bulb temperature is less than 4°K (and for Uluru negative) which suggest that a reasonable rate of heat transfer from the body to the ambient air is not possible. However if the sink temperature is taken as the wet-bulb temperature of the ambient air then the potential is over 10°K except for Snake Bay in NT where it is 7°K.

Figure 3 represents a thermal network of heat transfer from a body through a NBC barrier to the ambient air using a personal cooling system. Heat is collected from the skin surface through a layer of wet singlets and is rejected through a wet cooling pad to the ambient air. As long as the outer cooling pad is kept moist and sufficient air flow over it is maintained, the temperature of the pad will approach the wet-bulb temperature of the ambient air.

Thermal resistances and temperature drop across various components of the personal cooling system for the assumed thermal load of 300 W are summarise in Table 2. It can be seen that temperature drop across the four components is 2.7°K and assuming the 7°K as the available temperature potential, the temperature drop across the heat transfer device is required to be less than 4.3°K for a thermal load of 300 W.

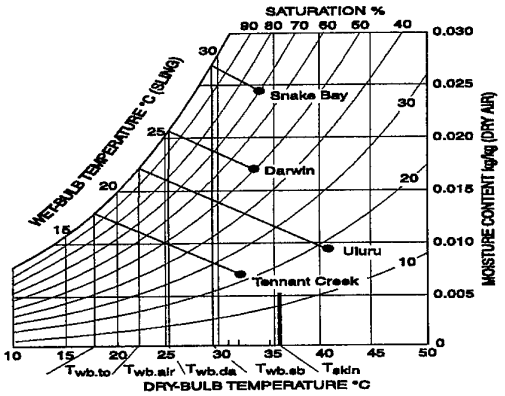

Figure 2. Ambient conditions of 4 typical locations and their corresponding wet-bulb temperatures

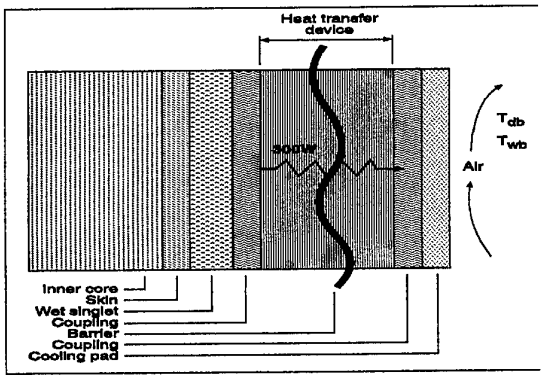

Figure 3. Thermal network model of a cooling device with wetted outer surface

Component	thickness mm	surface area m^2	k W/m K	Resistance K/W	ΔT (K) for 300 W
wet singlets	1.0	0.50	0.60	3.33×10^{-3}	1.0
inner coupling	0.1	0.50	0.25	8.0×10^{-4}	0.24
external coupling	0.1	0.25	0.25	1.6×10^{-3}	0.48
cooling pad	0.5	0.25	0.60	3.33×10^{-3}	1.0
				TOTAL	2.74

Table 2. Thermal resistance and temperature drop across various components of the personal cooling system for a thermal load of 300 W.

A possible solution

Heat pipe is a device which is a closed system containing only one liquid. For a given pressure the liquid and its vapour can only exit in equilibrium at one temperature. Any heat entering the system changes its internal pressure and its respective temperature, thus producing a constant temperature device capable of transferring large quantity of heat by evaporation at the hot end and condensation at the cold end [3]. CSIRO has been developing low temperature heat pipes [4, 5, 6]. These heat pipes can be flexible and transfer 300 W of heat through a barrier with temperature drop of approximately 2K. These low temperature heat pipe systems are ideally suitable as a heat transfer device for the personal cooling system. Work in developing working prototype of these devices is in progress at CSIRO.

CONCLUSIONS

A cooling system capable of removing 150 W from the skin can triple the time a person can safely work in a NBC suit at 40°C db and 30%Rh. A personal cooling system, for effective personal cooling in NBC applications, in most parts of Australia, should use wet-bulb temperature as the sink temperature and use an efficient heat pipe as the heat transfer device between the skin and the outer ambient air.

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PAPER 29: EVALUATION ON THE COOLING SYSTEMS OF AIRTIGHT SUITS USED IN THE CLOSED ECOLOGY EXPERIMENTAL FACILITIES

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INTRODUCTION

In order to control the closed ecological systems properly, little or no heat and moisture exchange between the Closed Ecology Experimental Facilities (CEEF) and outside the CEEF is allowed. Caretakers who enter the CEEF for daily facility care are most likely to cause heat or moisture gain to the systems. Therefore, wearing airtight suits with closed circulation systems becomes mandatory. Once they wear airtight suits, however, they will suffer form the heat stress due mainly to suppressed heat dissipation from the skin without properly designed cooling systems. This demand on cooling systems has prevailed for design of chemical protection suits [1], pilot suits [2] and astronaut suits [3]. According to the literature, water circulation systems and /or air circulation systems were basically incorporated into the almost all types of protective suits. In this study, the cooling capacity of the suits used in the CEEF was evaluated.

MATERIALS AND METHODS

Subjects

Six male subjects, aged from 19 to 23 year old, participated in the experiments. Their physical characteristics are listed in Table 1.

Sub.	Age (yr.)	Height (cm)	Weight (kg)	A_D (m^2)	Body Fat (%)	Adiposity* (N.D.)
A	19	172.9	68.2	1.80	20.42	0.27
B	21	177.9	72.7	1.88	31.62	0.38
C	20	160.0	54.6	1.56	21.78	0.32
D	23	172.4	85.0	1.95	40.48	0.46
E	19	170.8	73.2	1.83	27.39	0.26
F	21	173.4	57.9	1.69	26.65	0.44
Av	20.5	171.2	68.6	1.79	28.05	0.35
±SD	1.4	5.5	10.2	0.13	6.67	0.08

*Adiposity = m_{ad} / weight, m_{ad} = (ZFAT×5.85+25.6)/(170.18/HT)^3
FAT = (SFAT×(170.8×HT)-116.79)/34.8 [mm]
where SFAT: sum of skinfold thickness of six sites [mm]

Table 1. Subjects' physical characteristics

Suits and its life supporting systems

The overall suits, consisted of a hood, made of polyvinyl sheets with thickness of 2 mm, were designed for caretakers working in the closed ecology experimental facilities. A water air circulation system and an air circulation system were incorporated into the suits for heat extraction from the human body. Fine tubes, of which inside were filled with cool water (10°C) supplied at 2.8 *l*/min from the chiller, were attached to the underwear. Cool

air of 18°C was continuously supplied at 200 *l*/min from the blower connected to a dehumidifier. The system diagram is depicted in Fig.1.

Experimental protocol

All the experiments were carried out in the artificial climatic chamber where air temperature and relative humidity were set at 28°C and 40% respectively. Air movement was controlled to be less than 0.02m/s. Subjects wearing the suits exercised on a bicycle ergometer for 40 min at 30% their maximum capacity under the following three different cooling conditions; cooling with the air circulation system (*cond.1*), cooling with the water circulation system (*cond.2*) and cooling with the both circulation systems (*cond.3*).

Oxygen consumption and carbon dioxide production were simultaneously measured with the flow meter and the gas analyzer (AE-280S, Minato Med. Co.) on a breath-by-breath basis. Metabolic heat production was then calculated from the following equation; $M=\{(0.773+0.128\times RQ)\times 4.34\times \dot{V}O_2\times 60\}/A_D$ [W/m^2], where RQ=respiratory quotient [N.D.], A_D=body surface area [m^2], $\dot{V}O_2$=minute oxygen consumption [*l*/min STPD], EEG was continuously monitored with a EEG monitor (OEC-6301, Japan Koden Co.) for heart rate (HR) calculation. Tympanic temperature was also continuously measured with a tympanic sensor (Sensa-technica Co.) Skin temperatures and the amount of local dry heat exchange were measured with C-C thermocouples and heat transducers (HFM-ER6, Kyoto Densi Co.) at head, chest, upper arm, front thigh and front calf. Mean skin temperature ($\bar{T}_{sk}$) and mean dry heat exchange were then calculated with the weighting factors. Microclimate volume of the test suits was estimated from volume measurements with and without subjects. In addition, subjective evaluation on heat stress was recorded after each trial.

As a useful heat stress index, the rate of body heat storage ($\dot{S}$) was calculated from the Kakitsuba's equation incorporating body composition [4]; $\dot{S}=0.72\times m/A_D\times\{0.38\alpha\times\Delta\bar{T}_{sk}+(1-0.63\alpha)\times\Delta T_c\}$ [W/m^2], where α=adiposity [N.D.], $\Delta\bar{T}_{sk}$=change in mean skin temp.[°C/h], and ΔT_c=change in core temp. [°C/h]

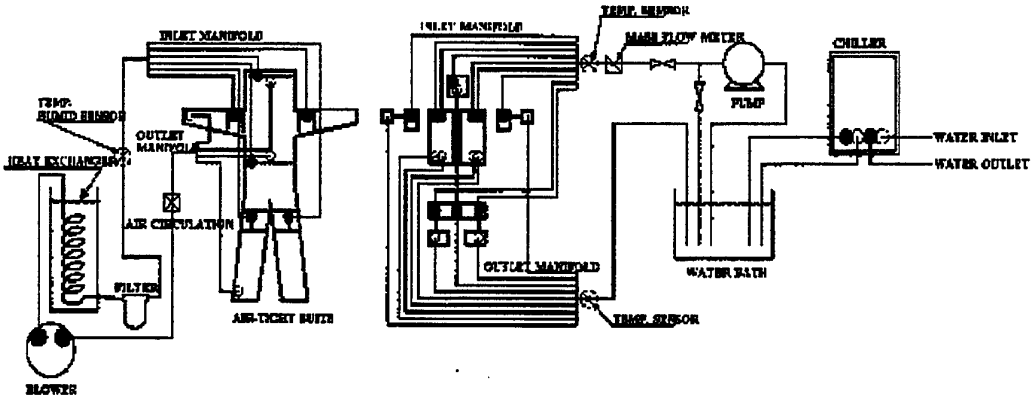

Fig. 1. Systematic diagram of the water cooling and air circulation systems

RESULTS AND CONCLUSION

Mean skin temperature during exercise under cond.1 was significantly higher ($p<0.05$) than that under the other two conditions as a result of a higher cooling capacity of the water circulation system. Tympanic temperature remained stable under cond.2 and cond.3. However, it increased progressively under cond.1, resulting in an increase of >1°C at the end of exercise as compared with the initial value.

As seen in Fig. 2, average heart rate under cond.1 was significantly higher ($p<0.05$) than those under the other two conditions. Fig.3 showed that the amount of heat loss from the skin (HL) was about one third the metabolic heat production (M) in cond.1 whereas HL were slightly smaller than M in the other two conditions. The rate of body heat storage calculated in cond.1 was significantly greater ($p<0.05$) than those calculated in the other two conditions. Therefore, it can be concluded that heat extraction by the water circulation system was sufficient to reduce heat strain under the given conditions. However, the subjective evaluation showed a substantial benefit of using both circulation systems because the air circulation system can dehumidify air trapped in the suits. For this reason, the optimally designed suits should be equipped with both cooling systems.

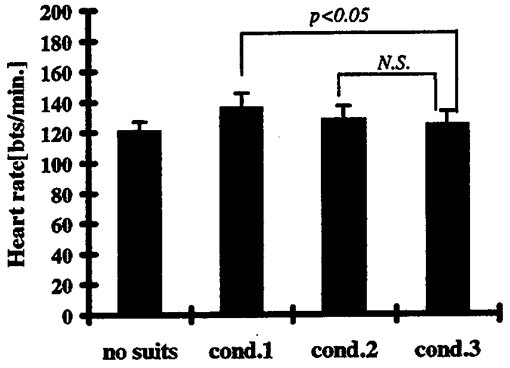

Fig.2. Comparison of heart rates

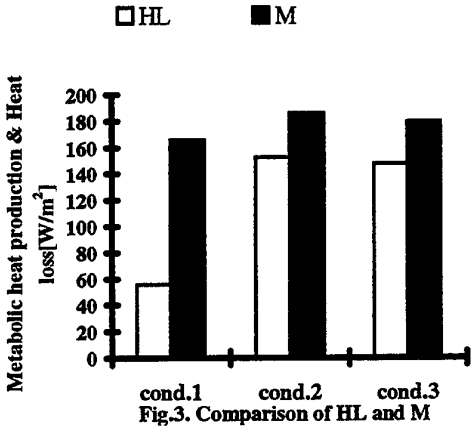

Fig.3. Comparison of HL and M

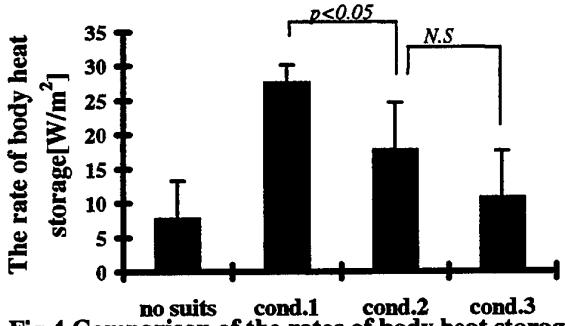

Fig.4 Comparison of the rates of body heat storage

In regard to required cooling capacity of the airtight suits, the minimal amount of heat loss from the human body is estimated to be approximately 130 W/m^2. To meet this requirement, however, a weight of the cooling systems will be over 50 kg that causes a great burden for caretakers, particularly female caretakers. From the practical point of view, the current engineering technique should cope with difficulties in the design stages to make airtight suits compact and usable.

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INVITED LECTURE 15: PREDICTIVE MODELING: ITS USE IN FORECASTING HUMAN RESPONSES TO THE ENVIRONMENT

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INTRODUCTION

This report discusses the development, thermal attributes, and use of two heat strain models that describe user effectiveness while wearing protective clothing. Because experimental settings with human subjects are restricted to finite limits to protect the individual, modeling often fulfills the requirements to test performance at environmental extremes. In general, mathematical models of thermal strain incorporate essential variables active in thermoregulation. These often describe proportionality coefficients active in the heat balance equation. Models describing steady-state responses apply appropriately when quasi-heat balance exists and are useful in the prediction of a wide assortment of physiological effector responses particularly when a given metabolic activity stays constant over time.

MATERIALS AND METHODS

Passive State and Active Controlling System in Thermal Models: A regulating system is regarded in two distinct ways: that of a passive or controlled system and an informational or controlling system. In physiological terms, the controlled system is considered as the body with its inclusive anatomical features, heat capacities, energy fluxes from various tissues: core, muscle, adipose, and skin sites, whereas the controlling system encompasses the complete central nervous system transmitting information in a network manner [1,2,3,4]. The classical approach to generate a thermal model is to describe the passive state and build algorithms to validate, as closely as possible, information integrating physiological controls of the controlling system. General numerical methods include finite difference or finite element methods to generate code. The most complete description of the passive system to date is from the work of Stolwijk and Hardy [1] that describes body heat exchange from six segments, further subdivided into 25 compartments or nodes. During the last 30 years, research attention to the mode of operation of the controlling system has taken precedence over description of the passive heat flux between various segments of the body [3,4]. As a result significant progress in the modeling of controller activity exists. The thermoregulatory controlling system has been considered as existing with linear and nonlinear control operations with and without a discrete reference (set) point [1,2,5]. Use of feedforward and feedback neural networks are being used more frequently [5]. A thermoregulatory model has been developed [4] that reliably incorporates new schemes for calculating changes in blood flow to muscle, visceral areas and skin, and changes in stroke volume, heart rate and cardiac output along with previous depictions for control of core temperature and sweating rate.

Empirical (Operational) Models: Early operational models of human performance include the formulation of a Heat Stress Index (HSI) [6]. Modern advances have allowed the incorporation of appropriate coefficients from studies involving human research, thermal manikins, and biophysical devices that measure clothing properties. These studies have led to the implementation of paradigms that forecast tolerance limitations to heat stress [7] and generated a series of predictive equations [7,8]. Others [9] have used an empirical approach which gives a reasonable scaling of heat strain based on core temperature and heart rate responses of individuals exposed to a wide environmental range.

RESULTS AND DISCUSSION

Useful information comparing operational (empirical) and servo loop thermoregulatory models have been generated [10]. A database has been garnered as part of the Technical Cooperation Program (TTCP). An example of the utility of the USARIEM operational model output features [8] is shown in Table 1.

				UKArmy MK4 +Fatigues			UK Rnavy (Romeo3)			Canada TOPP High		
Ta (°C)	Ta (°F)	WBGT (°C)	Heat CAT	VL	L	M	VL	L	M	VL	L	M
27.8	82	25.5	2	NL	NL	57	NL	NL	51	NL	NL	65
30.6	87	27.8	3	NL	172	52	NL	126	48	NL	NL	58
32.8	91	30	4	NL	113	48	NL	100	45	NL	240	53
35.6	96	32.2	5	NL	86	44	NL	82	42	NL	110	48
37.8	100	34.4	5	NL	73	41	NL	72	39	NL	87	44
41.7	107	37.8	5	111	59	35	121	60	35	162	65	38

VL=very light work; L=light work; M= moderate work; Fully acclimated, wind speed=2m/s; 50% RH; NL= no limit to work activity. Heat Category= Heat CAT.

Table 1. USARIEM Heat Strain Model prediction of maximum work times (min) during daylight operations with various TTCP ensembles.

As shown in Table 1, evaluations of heat stress and strain risk analysis of a soldier completely encapsulated in chemical protective clothing (CP) often necessarily become more empirical by the nature of the fact that definitions to operational activities (such as prediction of heat casualties, status of water requirements, work:rest cycles, and tolerance times) are statistical events. With heavy protective clothing, extensive work intensity is not as effectively tolerated. The particular level of CP requires the soldier to don individual protective equipment that coincides with chemical threat, work intensity, and environmental stress. Because more individuals with heat casualties are likely to recover from the hyperthermia, this decision often dominates provided that the troops are heat acclimated, well-trained, and fully hydrated. In such cases, application of operational models that predict limits to work and water requirements is especially crucial at the field site.

Results from SCENARIO Model (Figure 1) [4]: A recent feature added to this model is the incorporation of effects of progressive dehydration based on studies by Sawka *et al.* [11] and Montain *et al.* [12]. In the latter study, changes in central and peripheral coefficients during progressive dehydration were studied. They found that the gain in the peripheral control of sweating decreased and the central control increased with each percentage drop in body weight due to water loss (%ΔW). These changes have been incorporated into the code of the above model. The detrimental effects of elevated core temperature (T_c) and T_{sk} during dehydration are considered as well. As T_{sk} and T_c rises, basal stroke volume (SV_n) is diminished because of displacement of central intravascular volume into cutaneous veins. This aspect of SV control has also been modeled. As energy expenditure and SV_n

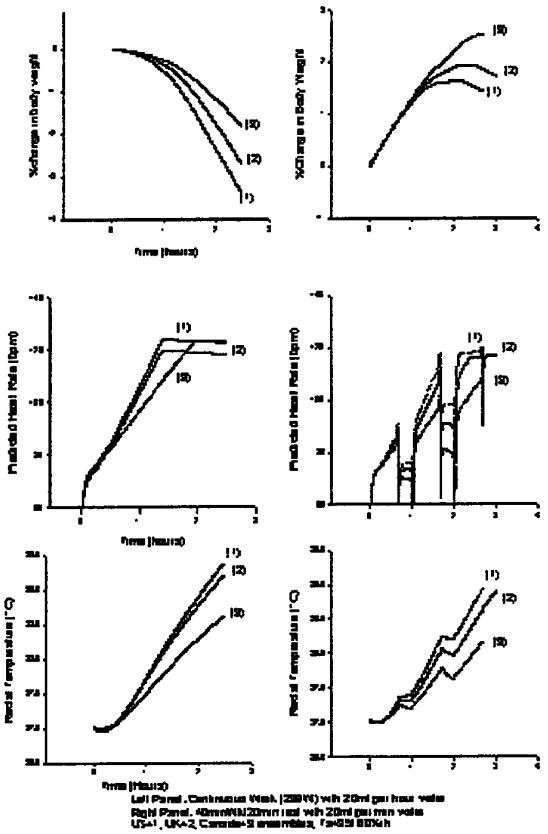

Figure 2. Model simulations with TTCP CP clothing during progressive dehydration.

increase, the impact of increasing T_{sk} becomes more pronounced; SV can be as much as 25 mL lower than when T_{sk} is cool. Figure 1 shows the data output from the model predicting progressive dehydration when a person is dressed in various CP ensembles from representative TTCP countries (US BDO+BDU; UK MK4+fatigues and Canada TOPP high) in a warm/humid environment (T_a=35°C; rh= 60%). Work rate was set at 233W executed over a time period of 2.5 hours and the individual is allowed to drink only 20ml of water each hour (*Left Panel*). This simulation is especially interesting because it often represents 'real world' effects observed in many training missions. The simulation shows the physiologic consequences of inadequate water replacement with the various ensembles (T_{re} reaching some 39.5°C and almost 4% ΔW with the US garment). Figure 1 shows that subjects progressively dehydrate much more rapidly over the time period when water replacement becomes inadequate and skin evaporation is impeded by CP clothing. A person can typically lose about 5% of body weight (3.5 kg or liters) in such cases. The model output in the *Right Panel* of Figure 1 shows simulations when 20 mL/min H_2O is supplied (1.2 L/h) (i.e., an amount the gut handles easily such as from a 'Camel Bak Portable Hydration System') coupled with 3 bouts of intermittent work of 40 min (M=233W) with rest periods of 20min after each bout. It is clear that rectal temperatures do not reach dangerous heat casualty levels and cardiac output was maintained. The model simulation shows that although cardiac output and sweating rate were maintained, there was a greater physiological cost in terms of heart rate and core temperature elevation with the US and UK ensembles, but not using the Canada ensemble. Therefore, efficacy of garment evaluation is both provided by model simulation and supported by actual heat transfer and physiological data [11,12].

SUMMARY

The utility of either operational or servo-control simulation approaches depends on the validity and application of a particular model to ample laboratory or field data. When precise clothing heat transfer coefficients are available (e.g., via a manikin analyses), the use of a prediction model is effective in forecasting work/rest cycles, water requirements, and tolerance times in the heat with CP clothing. Simulation models provide key data for situations in which risks involved preclude use of humans or in which cost of direct field testing is prohibitive. Finally, model analyses over wide scenarios allow the tactical commander an ability to oversee military operations and assess capabilities of all troops.

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PAPER 30: THE CONTRIBUTION OF SOLAR RADIATION TO HEAT STRESS AND HEAT STRAIN DURING WORK IN ENCAPSULATING PROTECTIVE SUITS

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INTRODUCTION

A report was presented to the NSW Fire Brigades giving findings and recommendations for safeguarding of personnel against heat strain created by carrying out activities, outdoors in sunlight, wearing fully encapsulating, impermeable protective suits supplied from enclosed Self-Contained Breathing Apparatus (SCBA) [1]. The reasoning applied to the method of study was based on previous experience of the difficulty of applying then-available indices of heat stress to outdoor workers, in the varied and often changeable ambient conditions, with the range of personal characteristics to be expected in the operatives, and having regard to the difficulty of assessing the metabolic heat component of the stress in a wide variety of tasks [2], even when work was done in "normal work clothing".

This implied the use of physiological monitoring methods, rather than the use of any applicable heat stress index, as the underlying guidance, if not the routine surveillance method, for the management for the NSW problem. Preliminary assessment of then-available devices for real-time, on-the-job, monitoring of heart rate and (surrogates for) core temperature, carried out with the help of the Australian Institute of Sport, showed that although heart rate was reliably assessed, practicable methodology for core temperature in that situation was not yet available, as was confirmed by trials in the study. A study by Pandolf and Goldman [3] had shown that convergence of skin temperatures with core temperature appeared to be a factor of major importance in similar suit use, rather than raised core temperature. The NSW study indeed suggested that severe strain, not indicated by core temperature rise, even in mild weather was the result of rapid creation of saturated in-suit air in occupied suits at work and showed that even in an inflated unoccupied suit in sunlight the in-suit air temperature was related to the ambient Globe Thermometer Temperature (GT) rather than the external dry bulb (DB) air temperature or the WET Bulb Globe Temperature index (WBGT), or to any other currently used heat stress index. Subsequent reports appear to have supported that conclusion and indeed [4] showed the importance of radiant heat loads; the suggestion has been made [5] that a type of "glass house effect" might require investigation, outdoors in sunlight.

Prior to study of the use of more informative physiological monitoring methods a suggestion was made that a study of allowable exposure times (AET) devised for mine rescue workers [6] in saturated atmospheres in SCBA might provide a basis for relating AET to GT in suit work. Together with the need to verify the resulting criteria [7] in outdoor situations in sunlight, methods were required for relating in-suit conditions to the measurable external environment, for the choice, by study, of the most appropriate physiological parameters that might be useable for on-the-job monitoring of suit users.

METHODS

The Victorian Country Fire Authority provided a suit which was inflated and suspended in a "standing" posture in a grassed area remote from any obstruction to solar radiation throughout a winter day notified by meteorologists as forecast to be cloudless. External GT (standard 150m globe), Assman powered psychrometer DB and WB, and in-suit thermocouple temperatures at head, thorax and crutch were continuously logged, and manual sling psychrometer WB and DB recorded at half hour intervals together with the

best estimates for those times of the air movement rates, from a continuous record from a thermometer. Closest available meteorological reports of solar azimuth and altitude, and suit-orientation to sun, are on record with the experimental layout.

RESULTS

Individual temperatures of thermocouples derived from smoothed traces logged, at the times of the periodic sling psychrometer and air movement readings, are listed in the Table (Appendix 1). WBGT values are calculated from the sling readings using Natural Wet Bulb values (NWB) deduced from a nomogram prepared from [9].

DISCUSSION

In addition to the indications in the Table that the in-suit values are more closely related to the concurrent GT rather than to either the ambient air DB or calculated WBGT (= 0.7 NWB + 0.2 GT + 0.1 DB), comparison of the smoothed traces of the suit interior values with GT shows them to follow the pattern of changes in GT rather than those of DB or WB. The Assman psychrometer figures are thought to be affected somewhat by its air intake being closer to the grassed surface than the approximate thorax-height at which the sling was used.

The effect of sunlight entry via the transparent suit face piece at 1030 is very apparent, though likely to be of relatively minor practical importance to the overall solar radiation component to suit conditions throughout actual daylight work operations.

In view of the apparent importance of the external GT it seems possible that a significant practical benefit may be found by using white material for suits.

Provisional setting of AET for one level of work, on the expected saturation of an occupied suit interior [7] was based on the essentially sealed interior being ventilated only by the small release via the suit exit valves of the amount of air from SCBA use. In other (non-open air) situations use of suits fed by a cooled external air supply via, eg, Vortex coolers [10], has been practicable and acceptable. But for open-air activities other issues must be considered [11] before such use can safely be employed to override the solar radiant heat load indicated by GT.

If intended subsequent work in occupied suits confirms the convergence of skin and core temperatures suggested [3], it will require verification of the proposed [12] (36°C) skin temperature where heat balance maintenance is difficult, and the 37°C temperature at which work should stop. Reduced tolerance times with raised skin temperatures have been found in subsequent studies [13].

CONCLUSION

It seems essential that the present findings are further developed by the carrying out, in summer conditions, of known work-levels, eg by step rates [14], in occupied suits in the open air, while the wearers are monitored for appropriate physiological responses in parallel with recording, similar to that now reported, of ambient environmental conditions. The physiological data suggested are of heart-rate, core temperature by rectal thermometer, and skin temperatures at an adequate number of locations (which may indicate whether skin temperature alone, at a single site – as has been suggested [11,15] – will in fact be sufficient in real-life work situations, without any separate measure of core temperature being needed). Such study could usefully be combined with any reliable assessment of the actual water vapour pressure and mean temperature of the atmosphere of a suit occupied by a sweating worker using SCBA. Clearly such experiments can only be ethically carried out by volunteers under experienced clinical physiological supervision, but the findings and observations may then prove of value in work situations other than those here considered.

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APPENDIX 1.

THE CONTRIBUTION OF SOLAR RADIATION TO HEAT STRESS AND HEAT STRAIN DURING WORK IN ENCAPSULATING PROTECTIVE SUITS:

RECORDED ENVIRONMENTAL AND IN-SUIT CONDITIONS

		ASSMAN		SLING							
TIME, 5 JULY	APPROX. AIR VELOCITY, m/s	WET BULB TEMP., ^{0}C	DRY BULB TEMP., ^{0}C	WET BULB TEMP., ^{0}C	DRY BULB TEMP., ^{0}C	(CALC.) NATURAL WET BULB, ^{0}C	WBGT, (USING SLING DATA) ^{0}C	GLOBE THERM. TEMP. ^{0}C	SUIT HEAD TEMP. ^{0}C	SUIT THORAX TEMP. ^{0}C	SUIT CRUTCH TEMP. ^{0}C
10.00	1	7.75	7.75	9.25	11.0	11.0	12.05	16.25	22.25	17.5	17.0
10.30	1	8.5	8.5	9.5	11.0	11.0	12.85	20.25	23.75	18.25	17.25
11.00	1	9.0	9.0	9.75	11.75	11.75	13.8	22.0	21.5	18.5	18.0
11.30	1	9.5	9.5	10.25	12.5	12.25	14.5	23.25	21.25	20.5	19.25
12.00	0.9	10.0	10.25	10.0	12.25	12.25	14.8	25.0	22.25	21.25	20.25
12.30	1.5	10.5	10.75	10.0	12.5	12.5	15.5	25.25	23.0	21.25	19.5
13.00	1.5	10.75	11.5	10.5	12.5	12.5	14.7	23.5	22.0	20.0	18.5
13.30	0.8	10.75	11.75	11.0	13.25	13.0	15.1	23.5	21.5	20.0	18.5
14.00	1.0	11.0	12.0	11.25	13.75	13.75	16.0	25.0	23.0	21.0	19.5
14.30	1.5	11.0	12.25	11.0	14.0	13.75	15.9	24.25	22.25	21.0	19.5
15.00	1.9	11.0	12.25	13.75	13.75	13.25	15.4	23.25	21.75	20.0	19.25
15.30	Turbulent	11.0	12.25	11.57	14.0	13.75	15.5	22.5	21.25	19.25	18.5
16.00	2	10.75	12.0	11.0	13.75	12.25	13.8	18.75	18.75	16.5	16.25
16.30	2	10.5	11.5	11.0	13.75	11.25	12.0	13.5	14.5	13.5	13.5

PAPER 31: THE DISCOMFORT INDEX PREDICTS THE PHYSIOLOGICAL STRAIN ASSOCIATED WITH INDOOR SPORTS HEAT STRESS

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INTRODUCTION

While information exists concerning the heat stress involved in a range of outdoor exercise pursuits, there is little data on the combination of environmental and metabolic heat stress associated with indoor sports. This is surprising, as many indoor sports are known to involve considerable workloads, and the metabolic heat production must therefore be high. In addition, several sports, including basketball and squash, are often played, in Australia, in non-airconditioned stadiums. This implies that the environmental heat loads must be high when hot weather prevails.

The Discomfort Index (DI; the mean of wet and dry bulb temperatures) is a simple, readily obtained means of defining environmental conditions. This project addressed the related questions:

- can the DI predict players' physiological responses in two popular indoor sports? (basketball and squash); and
- can weather forecasts predict heat stress in non-airconditioned stadiums?

METHODS

General protocol

Core temperature, heart rate and sweat rate responses associated with match play in several environmental conditions were obtained from 27 elite male basketballers and six competition level male squash players. These subjects participated in two studies which have been previously reported [1, 2]; these reports provide detailed information on the physiological and environmental monitoring and data analysis employed. The physical characteristics of the athletes are summarised in Table 1. The basketballers were participating in a "round-robin" competition held over a summer long weekend in Canberra, A.C.T. The squash players were monitored during several matches involved in their normal team training and competition throughout a typical year.

Group	Age (y)	Height (cm)	Mass (kg)	$\dot{V}O_{2\,max}$ (ml/kg/min)	Comp. Level
Squash players	37±5	180±8	77±16	44±4	C to F grade
Basketballers	15-18	179-207	63-106	not measured	Elite juniors

Values are means ± SD for squash players and ranges for basketballers

Table 1. Characteristics of subjects

Environmental conditions

Court DI was obtained by measuring dry bulb (T_{db}) and wet bulb temperature by sling psychrometer, and court air movement was measured by either a low velocity flow analyser (Model 54N50, Dantec Electronik, Denmark) or a katathermometer (British Standard BS3276). On each occasion when physiological data were obtained (16 squash matches; 7 basketball matches), both indoor and outdoor environmental observations were

taken, to assess the influence of local weather on court conditions. In addition, approximately 50 *ad hoc* observations were taken at the squash courts, and a datalogger (Smart Reader, ACR Systems Inc., Canada) was placed in the basketball stadium for a two-week period. This device recorded indoor T_{db} and relative humidity, permitting comparison of 9 a.m. and 3 p.m. stadium data with corresponding outdoor shaded observations at a local Bureau of Meteorology station.

RESULTS

Outdoor versus indoor conditions

Court DI ranged from 14 to 29°C for the squash matches, and 17 to 26°C for basketball. Low court air movement values (<0.25 m/s) were consistently observed for both sports.

Court DI values were highly correlated ($p<0.001$) with outdoor observations, for both squash and basketball. Variability in local weather conditions, as reflected in outdoor DI values, accounted for approximately 90% of the variability in court DI values. The relationship between court and outdoor data for the basketball stadium which was extensively monitored is given in Figure 1. The datalogger results indicated that court T_{db} typically reached a maximum at around 18:00 hrs, with this maximum consistently agreeing to within 1.5°C of outdoor (Bureau of Meteorology) maximum T_{db}.

Court DI and players' responses

Court DI correlated with the core temperatures ($p<0.01$), heart rates ($p<0.05$) and sweat losses ($p<0.0001$) of the squash players. The basketball players' sweat losses also correlated with DI ($p<0.05$), such that a 10°C increase in DI predicted an increased sweat loss of 0.93% of body weight/h for the basketballers and 0.8% body weight/h for the squash players. Figure 2 summarises the relationship between court DI and sweat losses for both sports.

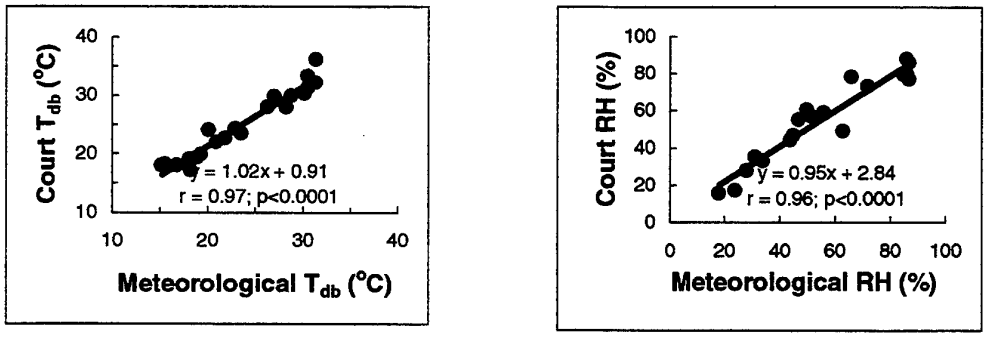

Figure 1. Court versus outdoor (Bureau of Meteorology) T_{db} and relative humidity (RH) values for the basketball stadium.

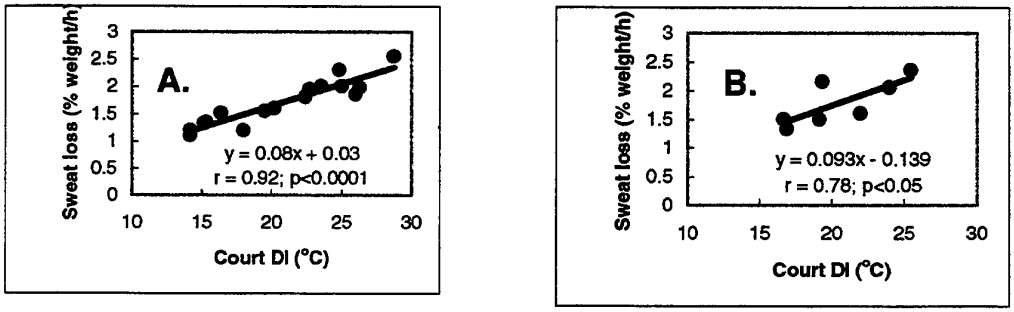

Figure 2. Sweat losses versus court DI for individual squash players (A.) and for groups of 4-7 basketball players (B.).

In the hottest conditions (a squash match, DI=28.8°C), considerable heat strain was evidenced: core temperature was 40.1 °C on completion of the 75 minutes of match play, heart rate was188 beats/min, and the player experienced a sweat loss of 2.6% of body weight/h. Several basketball players experienced sweat losses in excess of 2% of body weight/h when the DI was >24°C.

DISCUSSION & CONCLUSIONS

These data clearly show that considerable physiological strain can be experienced by both squash and basketball players in non-airconditioned stadiums, especially in summer. The data also show that environmental conditions in such stadiums can be very heavily influenced by local weather. Court DI values were often almost identical to outdoor values. This suggests that weather forecasts can be successfully used to predict playing conditions, thence heat strain, in non-airconditioned stadiums.

The overall degree of environmental heat stress observed in these studies should be interpreted in light of the significance of DI values as proposed by Sohar et al [3], bearing in mind that the metabolic demands of these sports are high, and that airflow is reduced indoors, potentially limiting players' heat losses [1, 2]. Under these conditions, DI values >24°C represent *high* heat stress, and the squash court conditions were *severe* on some summer days (DI >28°C).

The significant correlations between indoor DI and players' physiological responses indicate that the DI can indeed be used to predict the thermoregulatory and cardiovascular strain involved in these two popular sports. The strong relationship between DI and sweat loss is particularly important, as adequate fluid replacement is essential if performance is to be maintained in lengthy matches or throughout tournaments [4]. The regression coefficients can be used to predict sweat loss, so that fluid intake is adjusted to prevailing conditions. For example, the data suggest that in warmer court conditions (DI~24°C), sweat losses of approximately 2% body weight/hour for both squash players and basketballers will occur, while in cooler conditions (DI~18°C), the average loss will be some 1.5% body weight/hour.

In summary, these studies show that local weather forecasts can be used to predict conditions in non-airconditioned sports stadiums, and that the DI is a useful, practical means of predicting the physiological strain involved in elite basketball and competition level squash.

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The protocols followed in these studies were approved by the Human Ethics Committee of the University of Sydney, and all subjects gave their informed consent prior to measurement.

PAPER 32: OCCUPATIONAL HEAT ILLNESS: AN INTERVENTIONAL STUDY

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INTRODUCTION

Australia is the world's hottest, driest continent. Ambient dry bulb temperatures exceeding 40^0C are common for several months of the year in inland areas and, in the hot, humid, tropical regions, workplace temperatures exceeding 28^0C WB are also frequent.

The historical approach to controlling heat stress and heat illness in occupational settings has been to apply a shortened shift when certain thermal limits were exceeded.

One of Australia's most important industries is the minerals industry. For various reasons, more mines in Australia are developing as underground rather than surface operations. Moreover, mines are increasingly being based on 12 hour shifts and "fly in, fly out" rosters with 14 or more days on site before taking a break. Long term trends towards lower commodity prices are forcing mines to become more productive and to continually lower their operating costs.

Hot working conditions in mines are common. The reasons include the hot surface climate, "autocompression" of air as it enters deep mines, the very intense use of high-powered diesel equipment in confined spaces and the high moisture pick-up in the ventilating air, which increases the humidity and wet bulb temperature and consequently the heat stress on workers.

The changing business and social needs of mines, along with the increased heat stress, have raised a number of questions regarding working in hot environments:

At sweat rates of one litre per hour, mine workers can "turn over" their entire body weight within seven days. What are the short and long-term consequences of this?

- What is the most appropriate hydration/fluid protocol?
- How should the issue of acclimatisation be addressed?
- What are the physiological limits with respect to work intensity/work duration?
- Should physiological criteria be used to reduce the risk of occupational heat illness?
- How should the working environment be measured in terms of the strain it imposes? For example, most current indices are based on a constant work intensity. We know that this key assumption is not correct for many workplaces, which makes current indices difficult if not impossible to implement.
- Is a reduced shift length the most appropriate way to manage thermal strain in workers?

METHOD

In the summers of 1996/7/8, a major program of study was conducted in Australia's largest, deepest and hottest underground mine. These tests included:

- Continuous measurements of "core" (deep body) temperatures during the working shift and in the recovery period between shifts,
- Continuous measurements of heart rate during the shift,
- Measurement of fatigue levels before, during and at the end of the shift,
- Measurements of hydration status before, during and at the end of the shift,
- Measurements of wet and dry bulb temperature and wind speed at the work place during the shift.

At the conclusion of the tests, some results became clear:

- If thermal stress was excessive, then an exposure of even two hours was not safe, and certainly six hours (the typical shortened shift length) was not safe,
- If thermal stress was not excessive, then there was no reason to shorten the shift length, providing self-paced workers were healthy and started their shift well-hydrated and remained well-hydrated during their shift,
- Most heat illness was occurring where the wind speed was low (less than 0.5 m/sec); wind speed was largely ignored in the existing protocols at this work site,
- Dehydration was by far the biggest cause of heat illness in the workplace. Some of this dehydration was occurring at work, but much was occurring before workers started their shift.
- No suitable instrument was available to measure all the environmental parameters needed to assess heat stress [1].

After carefully reviewing the current methods used to assess thermal strain in the workplace (e.g. WGBT and ISO7933), it was clear that these would be inappropriate for workers who were undertaking different tasks at different metabolic rates in different thermal environments on a daily and even hourly basis [2]. An index which took all the necessary environmental and clothing parameters into account, and which was designed for self-paced work, was therefore formulated [3].

Four management zones and a comprehensive management protocol [4] for working-in-heat were developed based on this new index and the recognised deficiencies in the existing protocols. These zones were:

Unrestricted	No restrictions due to thermal stress apply
Acclimatisation	Special precautions apply, but only for unacclimatised workers
Buffer	Restrictions apply for all workers and conditions are closely monitored.
Withdrawal	No work allowed except for a safety emergency. Where work is required, a formal permit to work in heat must be authorised in advance by the senior company officer, and this work permit requires, amongst other things, formal work-rest cycling.

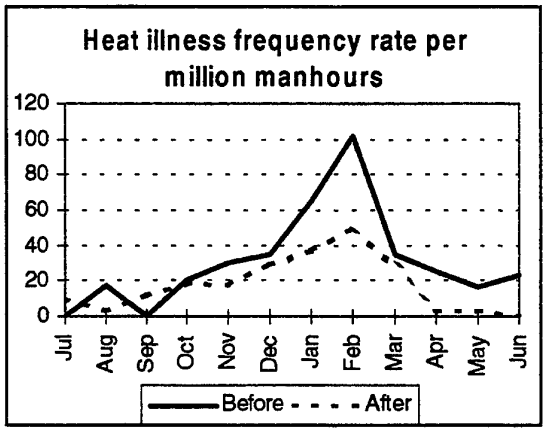
Over summer 1998/9, after the protocols had been implemented and the shortened shift removed, the test program was repeated and compared to the initial data.

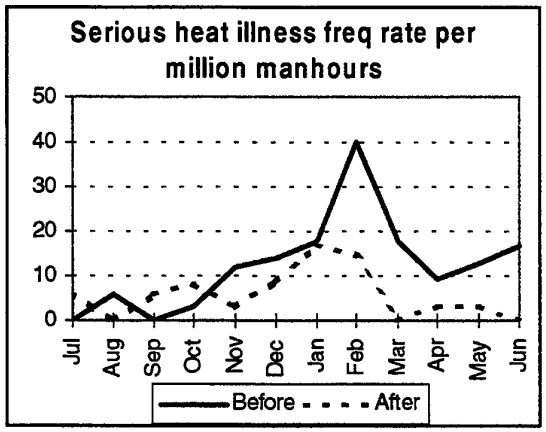
RESULTS

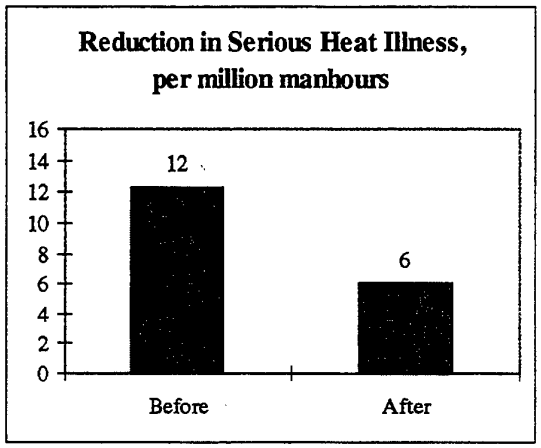
In summary, the total incidence of medically reported heat illness per million manhours worked has fallen from 31 under the old protocols to 18 under the new protocols, despite the abolition of the shortened shift. Likewise, the incidence of more serious episodes (based on symptoms and their severity) has fallen from 12 per million manhours to six.

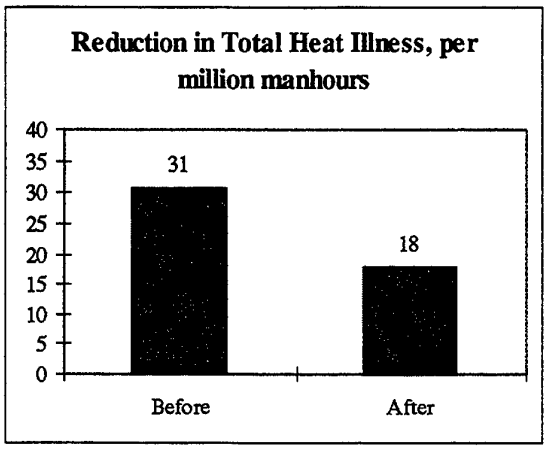
DISCUSSION

Whilst these protocols have been developed in the context of a large underground mine, the underlying physiological principles are equally applicable in most occupational settings - mines, smelters, bakeries, foundries and even outdoor workers - and should have a similar effect in terms of reducing heat illness, with its commensurate improvement on safety, productivity, cost and morale.









CONCLUSIONS

Heat illness is now an acknowledged occupational condition [5] that traditionally may have been under-diagnosed and under-reported. The lack of practical and realistic protocols and suitable environmental measurement instruments has prevented this significant problem being adequately addressed in the past. This study has shown that correct interventions, including a practical heat stress index for occupational settings, can lead to a substantial reduction in occupational heat illness.

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POSTER 1: NECK MUSCLE FATIGUE ISSUES RELATED TO NIGHT VISION GOGGLE USE

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INTRODUCTION

Symptoms of neck discomfort and general fatigue occur during Night Vision Goggles (NVG) use by Australian soldiers. Previous studies examining NVG use have found the weight of the goggle was related to differences in subjective ratings of comfort. Little (1997) described that significantly more aches and strains, specifically in the head and back regions, were reported by operators of the binocular-NVG than by the lighter monocular-NVG [1]. Bee (1992) noted that soldiers found the monocular-NVG "less uncomfortable" than the binocular-NVG [2]. Both Bee and Little note NVG operators also reported undesirable side-effects other than muscle pains; they commonly experienced headache, eye strain and nausea. These can be more than a simple discomfort and it is likely that they may, at times, interfere with concentration and operational performance. A previous study has shown that wearing the heavier binocular NVG produce greater complaints of discomfort, dizziness and nausea, generally referred to as *malaise* [1]. In no previous study has it been determined if the weight of the NVG is a contributing factor in the production of these symptoms. The current experiment was designed to obtain a measure of neck muscle fatigue following wearing the NVG. The aim was to determine if the influence of the NVG weight on neck muscle fatigue was significantly greater than not wearing the NVG and whether fatigue was related to reports of malaise and performance decrement.

MATERIALS AND METHODS

Ten healthy volunteers participated in the experiment. The NVG used were the International Technologies (Lasers) Ltd Mini N/SEAS. The monocular goggle weighed 552 grams. The binocular goggle weighed 880 grams. Both goggles were described as having a restricted field of view (FOV) of 40 degrees by the manufacturer. A 'dummy' monocular goggle was developed and used which weighed 10 grams but restricted FOV to 40 degrees. The activity of the posterior neck muscle (trapezius) and left lateral neck muscle (sternocleidomastoid (SCM)) were measured using surface EMG (the *Bagnoli-2 EMG System* from DelSys). The EMG output was collected and analysed using *EMGworks* by DelSys. The Median Frequency method [3] was used to obtain a measure of muscle fatigue. Symptoms of malaise were recorded using the Simulator Sickness Questionnaire (SSQ) [4] which was given before and after each data collection session.

Subjects provided a Maximal Voluntary Contraction (MVC) for the trapezius and left SCM muscles following procedures described by Phillips & Petrofsky (1986) [5]. Using the Median Frequency method, 20% of the MVC for each muscle was determined to be the *resistance weight* for each muscle during the 15 second isometric, isotonic resistance test (see below). This technique follows the guidelines of Median Frequency EMG data collection [3].

Each subject completed four sessions of data collection on separate days. Each session consisted of a target identification task. The subject sat in front of two VDUs placed at 45 degrees to the left and right of their forward facing. The subject's task was to identify stimuli that were presented on either of the two VDU screens at a random time interval of between 2 and 10 seconds. For each session the subject wore either the binocular NVG, monocular NVG, dummy goggle or no NVG (the control, called the *unaided* condition).

Subjects wore a different goggle type for each session, and the order in which the goggles were worn was randomised for each subject. Because the monocular, binocular and dummy goggles had a narrow FOV, the subject was forced to scan the VDUs with a lateral motion of their head in a 90-degree arc. Subjects were not required to actively scan for the next target in the un-goggled, control condition. There were four targets, the numbers 2, 3, 4 and 5. Subjects responded to detected stimuli by selecting the corresponding key on a keyboard. Subjects rehearsed the target detection task to eliminate any practice effects. Reaction Time (RT) was recorded. Each session was divided into six blocks of the scanning task. Each block was five minutes long. Between each block a resistance test was given and EMG was recorded. The resistance test was a 15-second isometric, isotonic exercise carried out by the Trapezius and SCM muscles separately. Whilst sitting the subject was required to keep their head level and still whilst resisting against their *resistance weight.*

RESULTS

The collected data were analysed and results illustrated in figures 1 to 3 below. Error bars for Figures 1 to 3 are one standard error of the mean. Figure 1 illustrates the amount of muscle fatigue (as measured by the 'median frequency' of the EMG) for each goggle type. The lower the median frequency value the more fatigued the muscle was. Using a paired samples t-test all goggle groups were significantly different from each other ($p < .05$) except the dummy vs binocular goggle. Figure 2 shows the mean reaction time on the target detection task for each goggle type. RT for all goggle groups were significantly different from each other ($p < .05$) except the monocular and binocular goggles. Figure 3 illustrates averaged scores on the SSQ obtained directly before (PRE) and following (POST) the 30 minute scanning task. A non-parametric test for multiple related samples (Friedman test) found that the POST SSQ scores were significantly different from the PRE SSQ scores ($X^2 = 14.777, p < 0.01$).

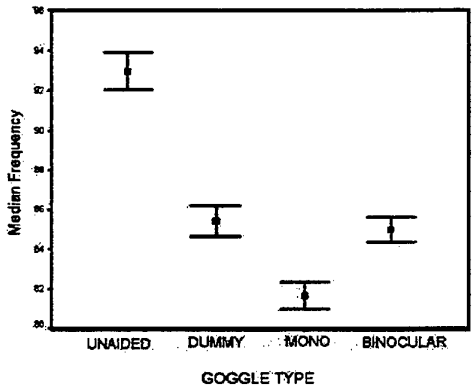

Figure 1: Median Frequency of EMG averaged for all subjects for each goggle type

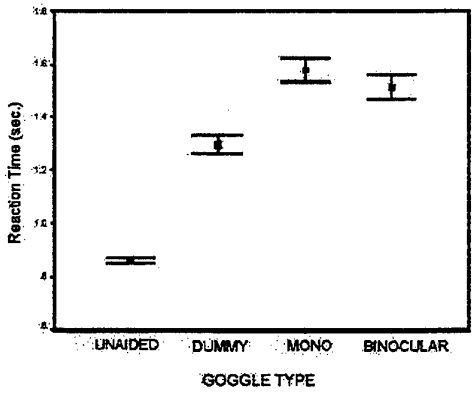

Figure 2: Reaction Time (in seconds) averaged across subjects for each goggle type

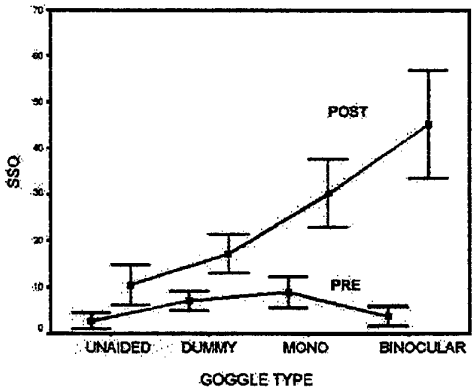

Figure 3: Simulator Sickness Questionnaire averaged across subjects. Pre test and post test results given

DISCUSSION

This study demonstrates that the addition of weighty goggles to the head increased the amount of neck muscle fatigue. This study also provides evidence of the common complaint of malaise by NVG operators. Scanning for targets without additional head weight (the dummy condition) increased muscle fatigue over not scanning (unaided condition). The monocular NVG increased muscle fatigue over the dummy condition, supporting the hypothesis that the weight of the NVG will increase neck muscle fatigue.

However, when wearing the binocular NVG (which weighs more than the monocular NVG) subject's neck muscle fatigue did not further increase over the monocular levels. Discussion with subjects following the experiment revealed a possible cause: the pupillary distance of the binocular NVG can be adjusted, allowing an increased FOV at a distance of 60-80 cm (the distance of the VDU from the subject) than the monocular NVG. This allowed the subjects to move their head through a smaller angle when scanning for targets. There was less head movement resulting in less muscle fatigue.

Not surprisingly, scanning increased RT on the target identification task compared to the un-goggled condition where no scanning was required. The monocular NVG resulted in a significantly slower RT than with the Dummy goggles alone. The mean RT whilst wearing the binocular NVG was slower than when wearing the monocular NVG but not significantly so. It is thought that this drop in RT in the binocular condition reflects the reduced scanning arc which influenced results on the median frequency score. The SSQ was significantly elevated after the 30-minute target detection task. The SSQ is an established measure of malaise as a result of artificial visual and movement cues. The NVG subjects the eye to an abnormal view of the world, not only the monochromatic green hue, but the reduced resolution and loss of depth cues.

Further research on neck muscle fatigue and target identification must also examine the scanning behaviour of the subjects, including head movement. The amount of head movement for each subject must be controlled for to isolate the effect of the goggle alone on neck fatigue.

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POSTER 2: HEAT STRAIN DURING COMBAT FITNESS ASSESSMENT OF SOLDIERS IN NORTHERN AUSTRALIA.

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INTRODUCTION

Heat stress during military operations in tropical northern Australia can exert a significant strain on personnel, due to the combined effects of heat, humidity, intense or prolonged work bouts, load carriage, inadequate fluid consumption and insulating effects of combat or specialised protective clothing ensembles. Activities in which high and sustained strain may be anticipated include the endurance modules of combat fitness assessments (CFAs). Such modules are often self-paced, in the sense that soldiers need to merely complete a given distance or task, sometimes within an allocated time. The incurred strain would be expected to vary across soldiers, but knowledge of its distribution, particularly its higher levels, is important. Trialing of a prototype CFA in northern Australia provided an opportunity to evaluate methodology and measure the extent of physiological strain experienced during combat training and assessment.

MATERIALS AND METHODS

The prototype CFA was trialed in October 1998, using 64 male soldiers of 3 Brigade, Townsville. The sample consisted of 32 Infantry, 8 Artillery, 13 Armour and 11 Engineer corps soldiers. Estimations of maximal aerobic ($\dot{V}O_{2max}$) and anaerobic (peak and 30-s mean) power were made for each soldier using Beep and Wingate tests, respectively. On the following morning, infantry soldiers undertook a 20 km march, aiming to finish within 4 hours whilst carrying 35 kg (mean load ±SD = 35.0 ±0.5 kg). All corps then undertook a 5 km march on the following morning, aiming to finish within 55 min whist carrying 20 kg (20.3 ±0.9 kg). Both marches were performed in combat uniform, ie. boots and disruptive pattern combat uniform (DPCU). The DPCU consists of cotton/polyester long-sleeved shirt and trousers. DPCU fabric has intrinsic resistances to air and water vapour of 0.02 $m^2 \cdot K \cdot W^{-1}$ and 2.0 $m^2 \cdot Pa \cdot W^{-1}$, respectively [1].

A sub-sample of soldiers was monitored for physiological and perceived strain during marches. Soldiers were selected to ensure a wide range of aerobic fitness in the sample. Heart rate was recorded (Polar SportTester, Finland) during marches and the Beep test. Gastrointestinal temperature (T_{gi}) was measured using three systems (BCTM2, PED Inc., USA; CorTemp, HTI Inc., USA; BFMS, PCD Inc., USA). All systems use a pill (HTI, Inc. or Koningsberg, PCD Inc.) that emits a low power, temperature-dependent radio signal. The major functional differences between systems were pill size, aerial configuration and download software. Pills were swallowed on the evening prior to participation or immediately after arriving in the morning. T_{gi} has been validated against oesophageal and rectal temperatures during heat stress [2], possibly being related more closely to the latter index during marching [unpublished observations from our laboratory]. Skin temperature was logged (Smart Reader, ACR Systems Inc., Canada) from thermistors (Edale Instruments, UK) at three sites. Mean skin temperature was calculated after Burton [3]. Oxygen consumption ($\dot{V}O_2$) was measured using a portable expiratory-gas analysis system (Metamax®, Cortex, Germany). Measurements recorded pre- and post-marches included temperature of the tympanum and auditory canal (T_{ty}, in triplicate, using a First Temp, Genius, USA), urine volume and composition (Combur[10] Test, Boehringer Mannheim,

Germany), body mass (±50 g), and perceptions of exertion, body temperature and thermal discomfort.

RESULTS

Soldiers' mean ±SD mass was 79.8 ±10.5 kg. Estimated $\dot{V}O_{2max}$ was 45.5 ±6.0 mL.min^{-1}.kg^{-1} (n=64). Peak heart rates of 194 ±8 b·min^{-1} (n=61) during this test indicated maximum efforts. Peak and 30-s mean power outputs were 12.7 ±1.9 W.kg^{-1} and 9.3 ±1.0 W.kg^{-1}, respectively (n=64).

The Wet Bulb Globe Temperature (WBGT) averaged 27.1°C and 27.6°C during the 5 and 20 km marches, respectively. Mean energy expenditures were 7.2 W·kg^{-1} (n=5) and 7.4 W·kg^{-1} (n=3); approximately one half of the calculated maximum aerobic power. The 5 km march was completed by all 51 participants, of whom 47 finished within the 55 min allocated. However, of 31 soldiers who began the 20 km march with 35 kg load, only 9 (29%) finished in the 4 hours allocated. Correspondingly, Table 1 shows that both real and perceived strain in these soldiers – whilst high in both marches – tended to be higher in the longer march. There were instances of urinary protein and erythrocytes that were attributable to marching, particularly for the 20 km march (25-30 mg protein·dL^{-1} in 3 of 11 soldiers sampled). The estimated $\dot{V}O_{2max}$ was modestly related to final T_{gi} (r=0.54, n=15, p=0.04), but not to HR or psychophysical strain at completion of the 5 km march. Table 1 appears to indicate that the mean $T_{ty'}$ was moderately indicative of mean T_{gi}, but this belies a poor agreement between indices within individuals (ie. r =0.04, n=15 after the 5 km march).

Mean pre-march urine production rates of 62 mL·hr^{-1} (5 km, n=29) and 56 mL·hr^{-1} (20 km, n=11) may indicate prevalent chronic hypohydration, since soldiers were instructed to drink 1-2 L additional fluid prior to arrival. Despite fluid availability and the limited duration of activity, 9 of 48 soldiers dehydrated by more than 1% of body weight during the 5 km march. Dehydration was not significantly greater during the 20 km march (520 mL or 0.65%) than during the 5 km march (230 mL or 0.26%, n=18, p=0.21).

	5 km march (20 kg load)			20 km march (35 kg load)		
	N	Mean ±SD	Min - Max	*N*	Mean ±SD	Min – Max
Duration	51/51	48:39 ±4:40	39:30 – 59:20	18/31	243:34 ±18:15	212:00-275:00
T_{gi}	15	38.6 ±0.6	37.7 – 40.0	5	39.1 ±0.5	38.5 – 39.7
$T_{ty'}$	39	38.4 ±0.6	37.2 – 39.6	15	38.6 ±0.6	37.9 – 40.3
$T_{mean\ skin}$	17	35.4 ±1.1	32.7 – 37.3	6	34.1 ±1.3	32.4 – 36.0
HR (b·min^{-1})	24	162 ±18	132 - 201	10	168 ±7	155 – 179
HR (%HR_{max})	22	82 ±10	68 – 99	10	86 ±3	82 – 91
PSI	8	7.6 ±2.5	5.0 – 12.9	4	8.0 ±1.6	6.4 – 10.2
Urine rate	17	45 ±45	8 - 166	8	20 ±11	8 – 37
RPE	34	14 ±2	11 - 18	9	18 ±1	15 – 20
Sensation	34	10 ±1	8 - 11	9	11 ±1	10 – 12
Comfort	34	3 ±1	1 – 4	9	4 ±1	3 – 5

Notes: Duration is in min:sec; Temperatures are in °C; PSI = Physiological Strain Index, calculated after Moran et al. [4], using their upper critical levels of heart rate (180 b·min^{-1}) and core temperature ([T_{gi}] 39.5°C); RPE = rating of perceived exertion: 11 = fairly light, 13 = somewhat hard, 15 = hard, 17 = very hard; Sensation: 8 = warm, 10 = hot, 12 = extremely hot; Thermal comfort: 1 = comfortable, 3 = uncomfortable, 5 = unbearably uncomfortable; Urine production rate is in mL·hr^{-1}.

Table 1. The magnitude of strain at completion of the endurance modules (5 and 20 km marches) of a prototype combat fitness assessment.

DISCUSSION

Physiological and psychophysiological strain was generally high, and was severe in some soldiers, during the endurance marches of a prototype CFA, conducted in mildly heat-stressful climatic conditions. Clearly, some soldiers experience very high heat strain during training. The present estimations of $\dot{V}O_{2max}$ were not closely related to heat strain, although this may reflect methodological limitations in this study, such as sample sizes and field estimations of $\dot{V}O_{2max}$, and the probability that endurance conditioning predicts exertional heat tolerance more appropriately than does $\dot{V}O_{2max}$ [5]. It might be expected that aerobic fitness is important for soldier capability in northern Australia because of the combined heat stress effects of the environment, clothing and load carriage. It is therefore recommended that aerobic fitness and heat strain should be examined further during phase 2 trialing of the CFA.

Methodologically, the use of urine composition and gastro-intestinal radio-pill thermometry might be considered for inclusion in field assessments of physiological strain, particularly during load carriage. T_{gi} seems to be a readily acceptable and valid [2] measure of core temperature. Nevertheless, potential problems with this index include cost, timing of ingestion (to avoid contamination of T_{gi} by drinking) and reliability of signal detection.

CONCLUSIONS

Some soldiers experience very high actual and perceived strain during training in tropical northern Australia. This can be monitored using gastro-intestinal radio-pill thermometry where necessary. The aerobic fitness of combat soldiers in north Australia requires further study, since it partially determines exertional heat tolerance, and preliminary indications are that it may be a limiting factor that is amenable to improvement.

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The generous loan of equipment and personnel from James Cook University is much appreciated, as is the commitment of the soldier volunteers of 3 Brigade, Townsville. These trials were conducted in accordance with Australian Defence Medical Ethics Committee approval (97/100).

POSTER 3: THE EFFECTIVENESS OF AN ICE VEST OR INTRAVENOUS ADMINISTRATION OF FLUID ON RECOVERY FROM HIGH HEAT STRAIN.

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INTRODUCTION

Physical exertion causes heat to be stored in the body, raising its temperature. The rise tends to be greater with higher climatic heat stress or duration of work, a lack of aerobic fitness, or the wearing of insulative clothing and/or equipment. The combined effect of these factors can lead to high core temperature (T_c), including hyperthermia ($T_c \geq 39°C$). Exertional hyperthermia is undesirable because it facilitates dehydration, cognitive deterioration, work intolerance, heat exhaustion, and heat stroke. Cooling during rest periods can reduce physiological strain - such as T_c - during subsequent work [1,2], thus conferring greater work capacity in the heat [3]. Therefore, determining the relative effectiveness of different methods of body cooling would be useful for both military performance and medical management in the tropics. We used the opportunity of having individuals already heat stressed by participation in another experiment, to examine the effectiveness of two cooling strategies of potential application to military operations.

METHODS

Protocol: Nine males (age = 32.4 ±3.6 yr, weight = 80.8 ±9.9 kg) provided informed consent of participation in these trials. Subjects were made hyperthermic by cycling in humid heat (35°C, 60% rh), on three occasions, and cooled by each of three methods, in balanced order:

CON: Seated rest with no intervention;

ICE: Donning an ice vest (Cooll©, NZ) on the torso, and water-cooled cuffs on the thighs;

IV: Intravenous administration of 1 L crystalloid (Hartmanns) saline as rapidly as possible ($3.1 \pm 0.5\ L \cdot hr^{-1}$) without pump-assisted pressurisation.

Hyperthermia was induced by cycling in humid heat (35°C, 60% rh) as part of the pre-cooling trials. Confounding effects of the three pre-cooling treatments were minimised by using nine subjects to balance the pre- against post-cooling treatments, and by encouraging subjects to cycle beyond the pre-cool protocol to reach T_c of 39°C or T_c equivalence between trials. After cycling, subjects were weighed, seated and configured rapidly for the cooling treatment, then monitored for physiological and psychophysical status at 0, 1, 3, 10, 20, 30 and 40 min.

Measurements: Core temperature (T_c) was measured using thermistors in the rectum (T_{re}) and oesophagus (T_{es}). Mean core temperature ($\bar{T}_c$) was taken as the unweighted average of T_{re} and T_{es}. Skin temperature was measured using thermistors positioned on the forehead, chest, abdomen, back, forearm, hand, front thigh, posterior leg and dorsal foot. Area weightings were used to calculate mean skin temperature ($\bar{T}_{sk}$). Heart rate was measured from the R-R interval of ventricular depolarisation. Forearm blood flow (FBF) was measured by venous occlusion plethysmography, using triplicate samples from a mercury-in-silastic strain gauge. Sensations of body temperature and thermal discomfort were recorded using 13-point and 5-point scales, respectively. A final body weight was obtained at 40 min for estimation of sweat loss.

Statistics: The experimental design is a fully-repeated, two-factor design, with three levels of factor one (treatment: CON, ICE and IV) and seven levels of factor two (time).

Dependent variables were analysed using 2-way repeated ANOVAs. Significant ($p<0.05$) differences were isolated using the Neuman-Keuls post hoc test. Data are reported as mean ± SEM.

RESULTS

The $\bar{T}_c$ at onset of cooling treatments was 39.0 ±0.2°C, and was equivalent across the three cooling treatments ($p=0.86$, Figure 1). Figure 1 illustrates that neither ICE nor IV hastened the rate of recovery of $\bar{T}_c$; remaining equivalent across treatments at 1 min ($p=0.41$), 30 min ($p=0.49$) and 40 min ($p=0.84$). However, the indices of T_c responded differently to cooling treatments. T_{es} fell more rapidly than T_{re} ($p=0.00$), particularly in ICE ($p=0.01$ for interaction of method · time · index). Mean skin temperature was equivalent between conditions at time 0 ($p=0.86$), but was 3°C lower in ICE by 1 min ($p=0.00$), and 6°C lower by 30 min ($p=0.00$). Heart rate appeared to drop more rapidly in ICE than in CON ($p=0.01$), but post-hoc comparisons were not significant. The sensations of body temperature and thermal discomfort dropped faster in ICE than in IV or CON (both $p<0.05$ by 1 min). There appeared to be no such impact of skin cooling on FBF (Figure 2, $p=0.30$ for method · time) or mass loss ($p=0.55$ for ICE versus CON, $N=5$). Therefore, skin cooling in ICE alleviated the perception of heat stress, whilst apparently having little inhibitory effect on thermolysis at high $\bar{T}_c$.

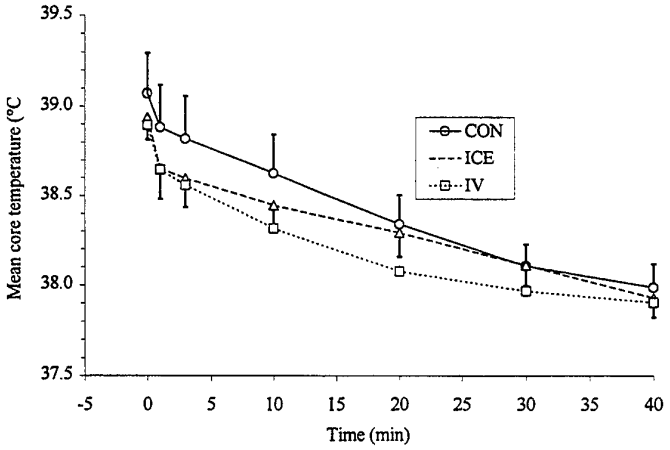

Figure 1. Mean (±SE) core temperature during cooling with ice vest and thigh cuffs (ICE), intravenous infusion (IV) or with no intervention (CON). N=9. Core temperature is taken as the average of T_{re} and T_{es}.

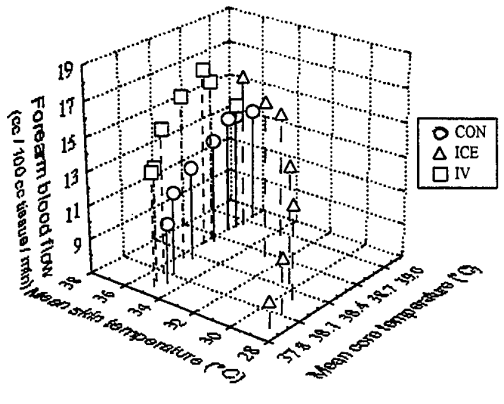

Figure 2. Mean forearm blood flow as a function of mean skin and core temperatures during cooling with ice vest and thigh cuffs (ICE), intravenous infusion (IV), or with no intervention (CON). Note the maintenance of high blood flow during the initial, rapid reduction in skin temperature at high core temperature. N=9.

DISCUSSION

There was apparently no effect of cooling by ice vest and thigh cuffs or by intravenous infusion on $\bar{T}_c$ recovery. Five issues are noted. First, the statistical power of these findings may be restricted because the treatments were applied after the pre-cooling trials. However, as explained above, it is unlikely that there was a systematic influence of those trials confounding these data. Second, the present data further illustrate the divergence that can occur between rectal and oesophageal indices of T_c [4]. Third, mean body temperature was not calculated for the purpose of estimating (negative) heat storage in these trials because of the disparate thermal conditions and the limitations of determining heat storage by thermometry [5]. However, it is suggested that T_{es} was more indicative of the body heat loss than was T_{re}. If so, ICE may be more effective than IV or CON in lowering body temperature and facilitating subsequent work. Fourth, at high T_c there appeared to be strong cutaneous cold sensitivities driving the perceptions of temperature and thermal comfort, whereas this may not have been the case for control of skin blood flow (Figure 2) [6,7]. Finally, the cooling treatments were applied to persons at borderline hyperthermia and in the absence of heat stroke. It can not be assumed that the cooling rates observed here would represent those under heat stroke, when skin cooling by ice vest might adversely affect recovery because of the potential for paradoxical thermoregulatory control [8].

CONCLUSIONS

There was no clear benefit of either an ice vest or intravenous administration of 1 L crystalloid solution for improving the rate of recovery of core temperature from mild hyperthermia. However, skin cooling increased the thermal heterogeneity of the body's core, presumably via its effects on mixed venous blood temperature. Thus, interpretation of the effectiveness of core cooling depends on the site at which core temperature is monitored. The ice vest was effective in rapidly reducing skin temperature, heart rate and the perceptions of temperature and heat discomfort, but may have had less impact on thermolytic processes. These findings should equate to individuals having a quicker perceived recovery from high heat strain if wearing an ice vest, while their actual thermal recovery should, at worst, not be impeded (if heat stroke is absent).

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These trials were conducted in accordance with Australian Defence Medical Ethics Committee approval. We wish to thank Lt Col S Rudzki for offering his input and advice concerning recovery by intravenous infusion, and also Dr Mark Febbraio for his advice and assistance.

POSTER 4: EFFECT OF ENVIRONMENTAL TEMPERATURE ON THE ANAEROBIC CAPACITY OF HEAT ACCLIMATISED ATHLETES

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INTRODUCTION

While the effects of warm and hot conditions on aerobic metabolism have been extensively researched, there is a paucity of investigation on the effects of these conditions on anaerobic metabolism. The increase in skin blood flow at the expense of exercising muscle perfusion during warm and hot conditions may result in a greater reliance on anaerobic metabolism. Although Dotan and Bar-Or [1] concluded that there was no detriment to anaerobic performance in warm and hot conditions, to our knowledge, this is the first study to investigate the effect of environmental temperature on the maximal accumulated oxygen deficit (MAOD).

The MAOD quantifies the limited amount of energy that is available from anaerobic sources and as such is a finite entity. Its magnitude should therefore not be affected by other variables, such as heat. It was hypothesised that the magnitude of the MAOD should remain unchanged, although due to the greater reliance on anaerobic metabolism in the heat it may be exhausted sooner.

MATERIALS AND METHODS

Six heat acclimatised subjects (25 ± 7 yr; 71.8 ± 4.4 kg; $\dot{V}O_{2peak}$ 56.8 ± 6.4 $ml.kg^{-1}.min^{-1}$) were recruited from Darwin triathlon and cycling clubs. The experimental treatments consisted of exercise in temperate (21.8 ± 0.5 °C; 52 ± 5 % humidity) and warm conditions typical of the local environment (29.6 ± 0.5 °C; 51 ± 9 % humidity).

Steady-state submaximal cycling economy and the $\dot{V}O_{2peak}$ of each subject were measured using an electromagnetically braked ergometer (Quinton Instruments) and a Medgraphics CPX/D gas exchange system. Individual regressions of $\dot{V}O_2$ on power output obtained from the cycling economy tests were extrapolated to predict the oxygen requirements and workload at 120% $\dot{V}O_{2peak}$. The anaerobic capacity test comprised constant intensity cycling at 120% of $\dot{V}O_{2peak}$ until exhaustion. The tests were conducted in temperate and warm conditions in random order. The MAOD was calculated as the difference between 120% $\dot{V}O_{2peak}$ and the actual oxygen consumption over the duration of the test.

Core (T_c) and skin temperatures were monitored using a rectal probe and skin thermistors (YSI 400 Series), respectively. Mean skin temperatures (T_{sk}) were calculated from a weighted mean of calf, forearm and chest sites. Electronic scales (AND) were used to measure changes in bodyweight. Heart rate was recorded every five seconds with a heart rate monitor (Polar Vantage NV). Capillary blood samples were taken from a hyperaemised ear lobe pre and post exercise and a lactate analyser (YSI 2300 Stat Plus) was used to determine the changes in concentrations of lactic acid.

Comparisons between the experimental treatments and between various points in time were made with repeated measures ANOVA. Tukey post-hoc comparisons were used in the event of statistically significant differences. Student's paired two-sample for means t-tests were used where appropriate for comparisons between experimental treatments.

RESULTS

There was no significant difference in MAOD between the two conditions. Mean values were 3.3 ± 0.9 and 3.5 ± 1.1 L (p=0.58) for temperate and warm conditions, respectively. Time to exhaustion (TTE) was also unchanged, being 175 ± 19 and 170 ± 18 seconds (p=0.56) for temperate and warm conditions, respectively. Post warm up T_{sk} averaged 3.0°C higher in the warm conditions. It remained relatively constant during the anaerobic capacity test and increased by the same amount for temperate and warm conditions during the first six minutes of recovery. T_c prior to the warm up was 37.3 ± 0.2 and 37.4 ± 0.2°C (p=0.83) for temperate and warm conditions, respectively. It continued to rise throughout the test by a statistically significant amount that was of the same order for both temperate and warm conditions. Post exercise core temperature peaked at 38.0 ± 0.2 and 38.0 ± 0.3 °C (p=0.71) for temperate and warm conditions, respectively. There was no significant difference between the 320 ± 112 and 416 ± 131 g mass loss (p=0.004) in the temperate and warm conditions, respectively.

Even though there was no significant difference in MAOD between the two conditions, there was a trend for greater reliance on anaerobic metabolism at all measured points, at least until 2.5 min of exercise in the heat (Figure 1). Peak post-exercise lactate values were 14.7 ± 3.8 and 14.4 ± 4.5 $mmol.L^{-1}$ (p=0.72) for temperate and warm conditions, respectively. Peak heart rates were 180 ± 6 and 180 ± 9 $b.min^{-1}$ (p=0.92) for temperate and warm conditions, respectively.

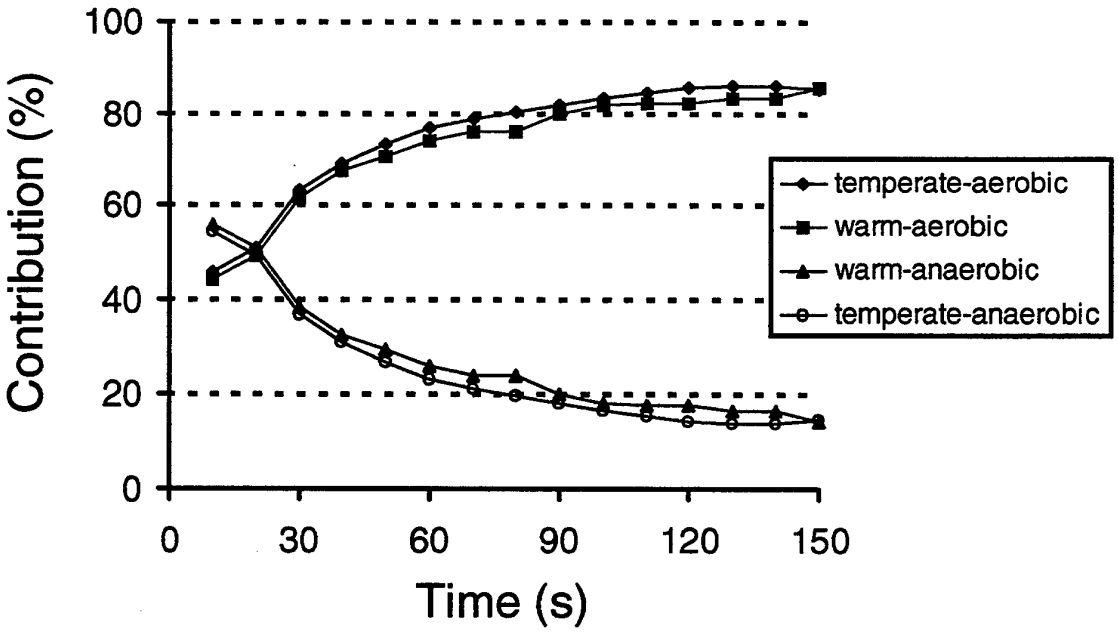

Figure 1: Anaerobic and aerobic energy contributions during the first 2.5 minutes of the anaerobic capacity test. The anaerobic contribution was the difference between the predicted 120% $\dot{V}O_{2peak}$ and the actual oxygen consumption during the anaerobic capacity test.

DISCUSSION

Dotan and Bar-Or [1] demonstrated, using the Wingate Test, anaerobic performance is not affected by environmental temperature. The MAOD Test varies from the Wingate Test in that it is able to quantify the aerobic and anaerobic contribution to performance. Claremont [2] used a similar test to investigate the effect of elevated body temperature on anaerobic

performance, however subjects ran for 6 min at $\dot{V}O_{2peak}$, and he manipulated body core rather than environmental temperature.

The MAOD is a quantity and not a rate. Since both the stores of creatine phosphate and the extent to which lactate can accumulate are limited then anaerobic capacity is finite and a separate entity from the aerobic energy system [3]. The current results support this concept. Despite the trend toward increased reliance on anaerobic metabolism in the heat as shown in Figure 1, the magnitude of the MAOD was not affected. This is similar to Linnarsson et al. [4] who observed unchanged MAOD values when measured in both normoxic and hypoxic conditions, in which there would also be an increased reliance on anaerobic metabolism. It is difficult to reconcile this trend toward increased reliance on anaerobic metabolism with no increase in blood lactate concentration in the heat. Blood lactate concentration reflects the balance between production and removal. Possibly elevated muscle temperatures during exercise in warm conditions may enhance lactate utilisation in heat acclimatised athletes and mask increased lactate production.

The expected performance decrement in a constant intensity test would be an inability to maintain the supramaximal power output, resulting in a decreased TTE. An increased reliance on anaerobic metabolism would also result in an earlier onset of fatigue. However, the heat acclimatised athletes suffered no performance decrement in the warm environment during a constant load anaerobic capacity test. This was despite a higher mean T_{sk} in the warm condition prior to and during exercise, and therefore an assumed increased skin blood flow. The similar heart rates between conditions during the anaerobic capacity tests may have been achieved by an increase in stroke volume or fluid shifts to maintain blood plasma volume, thus maintaining exercising muscle perfusion, and subsequently maintaining exercise performance.

CONCLUSIONS

It was concluded that the MAOD was not affected by environmental temperature in the ambient temperature range of 20-30°C and approximately 50% relative humidity in heat acclimatised athletes. Exercise performance (TTE) was also not affected despite the trend for increased rate of anaerobic energy production, and the higher mean skin temperature with assumed higher cutaneous blood flow and potentially reduced muscle perfusion. Further research to elucidate the response of non-acclimatised individuals is indicated. Due to less efficient cooling mechanisms, the non-acclimatised athletes may show that the anaerobic exercise performance can be affected under similar environmental conditions.

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This study was approved by the Northern Territory University Human Ethics Committee.

POSTER 5: COMPARISON OF TWO SYSTEMS OF WATER DELIVERY FOR USE ON MILITARY OPERATIONS

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INTRODUCTION

The significance of heat illness to military operations should not be underestimated: for example, it has been reported [1] that 29.6% of British soldiers engaged in operations in Iraq in World War II became heat casualites. Hypohydration is a major factor leading to, or exacerbating the risk of heat illness. Even relatively mild hypohydration (e.g. loss of 2-3% of body weight) will adversely affect physical performance in the heat [2]. As dehydration (the process that leads to a state of hypohydration) continues, cognitive function starts to deteriorate and physical performance will diminish further. A level of hypohydration equivalent to 5-6% of body weight will incapacitate most soldiers [2]. Further dehydration is associated with a high risk of heat stroke, a condition that has life-threatening consequences. It is essential that soldiers engaged in arduous physical work in the heat maintain adequate fluid intake to avoid dehydration.

The Australian Army uses water bottles (also referred to as 'canteens'), stored in pouches, as the primary means of carrying water. To obtain a drink the pouch must first be opened, the canteen removed and the lid unscrewed. Therefore, drinking cannot readily occur while the soldier is moving and it cannot be 'hands-free'. This may discourage drinking and hence may increase the risk of heat illness.

An alternative system of water carriage and delivery is now available that may encourage water consumption. It is based on a plastic bladder with a flexible tube. At the end of the tube is a valve that allows water to be drunk hands-free and while moving. This bladder/tube system shows promise as a means of promoting water intake while simultaneously improving operational effectiveness.

A study was conducted to determine the effectiveness, and acceptability to users, of the Roll-Top SportTank® (Ultimate Direction, USA) compared to canteens. Objective measures were made of hydration status and thermal strain of soldiers engaged in simulated operations in the heat. Acceptability was determined by questionnaire.

MATERIALS AND METHODS

Ten male soldiers performed the same military activities on successive days, while obtaining water from either the SportTank®, or from water canteens. As far as possible, the use of SportTank® and canteens was balanced across subjects and days.

Subjects were weighed in briefs ('nude weight'), in uniform, and in full patrol order (including any instrumentation). They were instrumented for measurement of body temperature and heart rate. Water consumption was monitored by weight change of water container. All voidings of urine were collected for measurement of volume and aliquots were retained for analysis of specific gravity (SG) and sodium. SG was determined by Unicon SG Urine Refractometer. Sodium concentration was measured using the Synchron CX5 System, Beckman Instruments Inc., Fullerton, California). Total sweat was estimated by weight loss according to the following equation:

Total Sweat = Initial nude weight - final nude weight + water intake + food intake - urine output.

Deep body temperature was measured rectally, using a flexible thermistor (YSI type 401, Yellow Springs Instruments, Ohio, USA) inserted by the soldier 10 cm beyond the external anal sphincter.

Skin temperature was measured using thermistors (Edale Instruments, Cambridge, U.K.), fastened by strapping tape (Leukoplast) at three shaved sites: scapula, forearm and calf. Area-weighted mean skin temperature was later derived according to Burton's formula [3]:

Mean Skin Temperature (T_{msk}) = $0.50 \cdot T_{chest} + 0.16 \cdot T_{forearm} + 0.34 \cdot T_{calf}$ (°C).

A Squirrel data logger (1206 series, Grant Instruments Ltd, U.K.) was used to collect and store temperatures in the field, each data point being the average temperature for a period of one minute. Heart rate (HR) was recorded at one-minute intervals from the R-wave frequency of ventricular depolarisation (Polar SportTester™, Electro Oy, Finland). The transmitter was fastened around the soldier's torso, and the receiver was insulated from physical harm, before being placed in a breast pocket of the soldier's uniform. HR data and temperatures were downloaded to a portable PC at the completion of each trial.

Questionnaires developed by the Defence Nutrition Research Centre (DNRC) on acceptability of the two systems were completed by all subjects.

RESULTS AND DISCUSSION

Table 1 shows the hydration results for subjects who obtained water from SportTank® compared to canteens. Although all hydration parameters showed a slight trend towards increased water intake when using the SportTank®, none of the differences between treatments were statistically significant (ie, $p>0.05$ for all differences).

During the patrolling phase, using different methods of water delivery did not statistically significantly affect thermal strain: no trend in core temperature was indicated ($p=0.53$); There was, however, a slight trend toward lower HR with the SportTank® ($p=0.17$). This result is consistent with the hydration results, indicating that the SportTank® does not exacerbate, and may even slightly reduce the thermal strain experienced by soldiers patrolling in tropical environments. In summary, there was little impact on thermal strain attributable to the use of SportTank® relative to water canteens.

All soldiers preferred the bladder/tube concept of water delivery, agreeing that the SportTank® is easier to use, more comfortable to carry and allows a drink to be obtained more easily than does the water canteen.

Water Delivery		Rate of Weight Change ($kg \cdot h^{-1}$)	Rate of Water Intake ($L \cdot h^{-1}$)	Total Sweat Rate ($L \cdot h^{-1}$)	Rate of Urine Production ($L \cdot h^{-1}$)	Final Urine Sodium ($mmol \cdot L^{-1}$)	Final Urine Specific Gravity
SportTank®	Mean	0.19	0.72	0.90	0.08	38.9	1.023
	SD	0.18	0.16	0.16	0.05	47.3	0.008
Canteens	Mean	0.22	0.63	0.84	0.07	30.4	1.023
	SD	0.27	0.20	0.16	0.03	28.6	0.004
Difference	Mean	-0.03	0.09	0.06	-0.01	-8.5	0.000
	SD	0.23	0.21	0.13	0.04	52.5	0.007
Probability		0.38	0.20	0.12	0.26	0.33	0.46

Table 1: Summary (Mean and SD) of hydration parameters for soldiers using water bottles or SportTank® (a positive value implies that the SportTank® led to the higher result)

CONCLUSIONS

It was concluded that the bladder/tube has advantages over water canteens through:

1) allowing 'hands-free' drinking while moving; and
2) being more acceptable to soldiers.

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Declaration: All subjects who took part in this study were volunteers who gave their free and informed consent to participate. All aspects of the study conformed to the requirements of the (Australian) National Health and Medical Research Council's 1983 'Statement on Human Experimentation'. The study protocol was approved by the Australian Defence Medical Ethics Committee.

POSTER 6: USE OF SENSORS TO STUDY THE MICROCLIMATE WITHIN A CLOTHING ENSEMBLE

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INTRODUCTION

Little is known about the microclimate within the clothing layers of soldiers in the field because until now, temperature and humidity sensors have been too large to lie unobtrusively within clothing ensembles. Data logging equipment has also been too bulky to carry around, restricting experiments to the controlled conditions found in environmental chambers. Temperature and relative humidity sensors have been developed by the DCTA, in collaboration with Leeds University, which are able to unobtrusively monitor the microclimate within the clothing ensembles of active subjects in the field. This will enable us to obtain new and vital information regarding microclimatic changes in the clothing layers, including how they are effected by external conditions, physiological status and different configurations of the clothing ensemble.

MATERIALS

The temperature and relative humidity sensors are wired to a data logging device, 21.6 × 61 × 106 mm in size and weighing approximately 150 g, which can be placed unobtrusively in the pocket. Each logger is attached to four pairs of sensors, each pair consisting of a temperature sensor (diameter 0.3 mm) and a R.H. sensor (12.8 × 6.8 × 3 mm in size). One pair of sensors is for monitoring the outside environment and the remaining three are for placing within the clothing ensemble. The temperature sensor is a thermistor that can give readings between -40 to 75 ± 0.2°C and the R.H. sensor is a ceramic printed circuit board that has a resistance proportional to relative humidity. This enables measurements to be taken between 0 to 100% R.H. with a sensitivity of ± 3% R.H. The data-logging device has a memory capacity of 3600 readings and the logging interval can be set for between 1 second and 4.5 hours. The battery has a life of 3 months.

DISCUSSION

A long-standing problem for land forces, particularly when operating in a cold climate, is the need to vary the number of clothing layers dependent upon their work rate. Multiple layers of clothing are required whilst at rest or stationary, with fewer or even single layers desirable whilst undertaking physical work. If too many layers are worn whilst working, there is a considerable heat stress with reduced capabilities and a paradoxical risk of hypothermia during subsequent rest because of the reduced insulating properties of sweat sodden clothing. The addition or removal of layers of clothing is impractical during operations. Furthermore, it is not always possible to adjust the layers of clothing whilst operating in an NBC environment. A technological solution to this problem is to provide clothing in which the insulation can be varied.

'Smart' materials are currently under development at the DCTA with properties such that their insulation and vapour permeability vary in response to the temperature and humidity of their immediate environment. It was thought that skin temperature might be a suitable activator for these materials but preliminary experiments using sensors in clothing have shown that although a subject may have an elevated core temperature, the actual skin temperatures may remain relatively constant if evaporative cooling is efficient enough to maintain a thermally neutral condition. Skin temperature may therefore be an unsuitable activator for smart clothing although it is felt that further research is required before this parameter can be dismissed completely. Exactly where in the clothing ensemble smart

materials will be employed and over which temperature and relative humidity ranges they will be engineered to act will be determined by the results of physiological trials using the new sensors to monitor the microclimate next to the skin and between clothing layers.

The sensors have been successfully used by Julie Gretton et al [1,2,3] from Leeds University to monitor the microclimate under moisture vapour permeable rainwear. Their results clearly reflected the different moisture vapour transmission properties for microporous, hydrophilic and bicomponent membranes that had been previously measured using standard laboratory tests. At the DCTA, our aim is to improve the comfort and hence the operational effectiveness of land forces. Using the sensors to monitor the microclimate occurring within the layers of the soldiers' combat clothing ensemble, we will be able to determine where smart materials should be employed and also identify areas for improvement in the current clothing issue.

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POSTER 7: IMPACT OF PERSONAL COOLING SUITS ON AN INFANTRY ATTACK SCENARIO

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INTRODUCTION

This paper examines the impact of a Personal Cooling Suite (PCS) on the thermal status of a soldier conducting an Infantry Attack Scenario. The effectiveness of the PCS is measured by its ability to keep soldiers' core temperature below a level that is commonly accepted as indicative of high thermal strain (38.5°C). The thermal strain predictive model adopted for this evaluation was originally developed by the US Army Research Institute of Environmental Medicine (USARIEM). It is based on a series of equations representing the response of body temperature and heart rate to a range of environmental factors, type of activity and clothing system. The model has been investigated previously by DSTO and was modified based on the data collected from a laboratory study [1]. This modified model was used in the current study.

SCENARIO

A generic attack scenario was constructed for this study with the assistance of military subject matter experts. The scenario is outlined in Table 1. During initial use of the model it was found that sensible results were not obtained above certain workloads. Hence some variables in Table 1 were constrained, such as gradient and velocity, to give reasonable results for soldiers carrying a standard load and wearing a standard combat uniform. The impact of PCS and other changes in clothing were then investigated.

Time (min)	Activity	Speed (m/s)	Gradient (%)	Comments
10	Admin	0	0	Admin prior to departure (resting for the majority of soldiers).
25	Walking	1.39	0.5	Initial movement for 50 minutes over two different gradients.
25	Walking	1.39	1.0	
10	Resting	0	0	Around 10 min in every hour of walking is spent resting[1]
25	Walking	1.39	1.5	Second hour of walking completes move to the FUP[2]
25	Walking	1.39	0.5	
15	Resting	0	0	Brief halt in FUP for Admin
3	Assault	1.67	5	Initial Assault
30	Fight Through	1.42	3	Fight through of the adversary position
5	Regroup	0.67	0.01	Move to 'reorg' location after fight through
27	'Reorg'	0	0	Conduct of Admin after the assault, resting for the majority of soldiers.

[1] Observations from field exercises [2]

[2] FUP = Forming Up Position, assembly area prior to an assault.

Table 1: Details of Attack Scenario

MODEL PARAMETERS

The values of other parameters required by the USARIEM model are given in Table 2. The sources of data are also indicated. Note that in some cases estimates have been used, as precise data are not available. For example the weight and cooling power of the PCS have been estimated based on discussions with staff associated with the PCS project. Further work could be conducted to examine the impact of these parameters on results.

Parameter	Value	Comment
Initial Heart Rate	65 bpm	Average value recorded in laboratory studies [2]
Naked Mass	74 kg	Average mass of soldiers observed during field exercises [3]
Height	180 cm	Average height of soldiers observed during field exercises [3]
Acclimatisation	14 days	Acclimatisation time required for reasonable agreement between model and experiment results [1]
Temperature	30 °C	Typical conditions
Relative Humidity	30%	Typical conditions
Wind Speed	1 m/s	Wind speed used during laboratory studies [2]
Patrol Order Weight	16 kg	Average load carried during field exercises [3]
Weight of NBC	3.3kg	Weight of new NBC uniform including gloves and hood.
Weight of Body Armour	8.3kg	Weight of body armour including vest and two ballistic plates.
Weight of PCS	2.5kg	Assumed weight of PCS.
Cooling Power of PCS	300W	Assumed cooling power of PCS
Terrain Type	Light Scrub	Scrub type used to calculate energy expenditure

Table 2: Parameters required by USARIEM heat model

The predicted cooling power of the PCS is 300 W. In the results presented in this paper a maximum (100%) heat extraction efficiency has been assumed and this energy is removed directly from the metabolic heat generated by the soldier. The actual cooling power of the suite will depend on its heat extraction efficiency, which is likely to be less than 100%, hence these results are an upper limit on the effectiveness of the PCS.

VARIABLES

The variables examined during this study relate to the clothing system being worn by the soldiers. They are the standard Australian Disruptive Patterned Camouflage Uniform (DPCU), the NBC suite[1], Body Armour and the PCS. For this study results are discussed in reference to the impact of the PCS on core temperature assuming procedures for the activity are not altered by changes to the clothing system.

Six clothing combinations were considered, they are:

- DPCU

[1] Current US system

- DPCU plus PCS
- DPCU plus Body Armour
- DPCU plus Body Armour plus PCS
- NBC
- NBC plus PCS

RESULTS

For each clothing combination the model has been used to generate data on core temperature as a function of time. Results for each of the configurations are given in Figure 1. Results indicated that all attacks involving DPCU and Body Armour were completed satisfying the measure of effectiveness described above (ie. staying below a core temperature of 38.5°C). Note that in these cases the cooling from PCS had negligible impact on the predicted core temperatures.

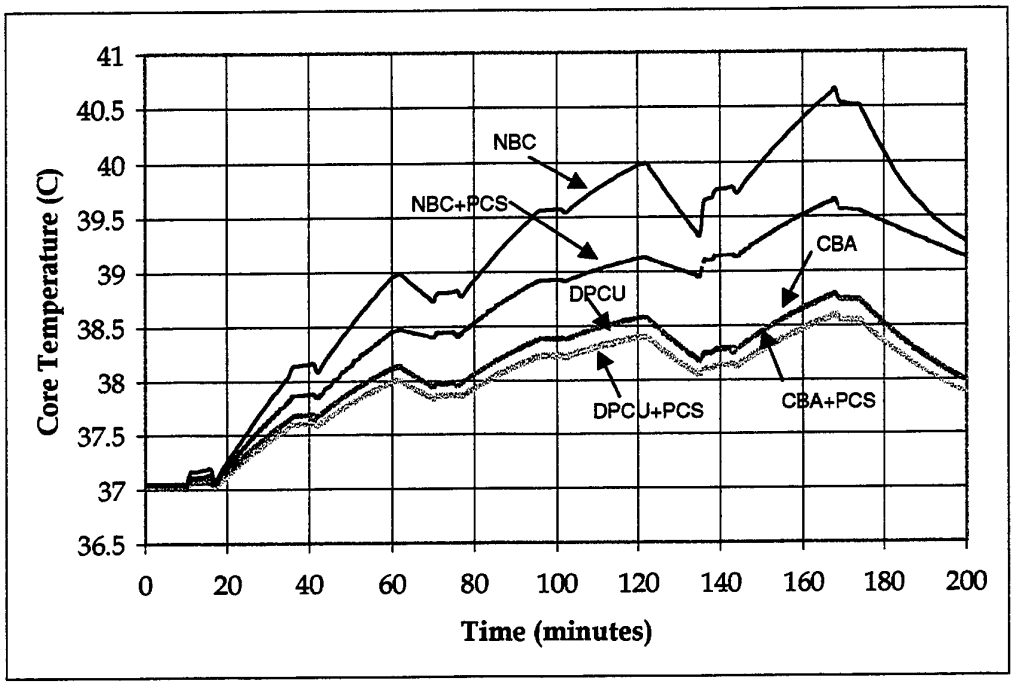

Figure 1: Core temperature against time for the generic attack scenario

Wearing the NBC suit had a significant impact on the predicted core temperature. Fifty minutes into the attack scenario the core temperature rose above the 'safe' limit. Using the PCS the 'safe' limit was exceeded after 80 minutes and the peak core temperature was 1°C lower than that reached without the PCS.

It should be noted that whilst a core temperature of over 40.6°C is predicted at the peak of activity when using NBC, heat stress or fatigue may force soldiers to halt before this temperature is reached. If we assume that soldiers whose temperature reaches 38.5°C require resting until a lower temperature is reached, such as 38.0°C, then the reduction in the required rest time required to achieve a specific period of work could be used as an alternative measure of performance for the PCS.

The time required to complete 100 minutes of work whilst staying below a core temperature of 38.5°C for several clothing configurations is given in Figure 2. The activity of soldiers in this example is walking at 1 m/s up an incline of 2% through light scrub. Ambient conditions of 30°C and 30% relative humidity were also used in this example.

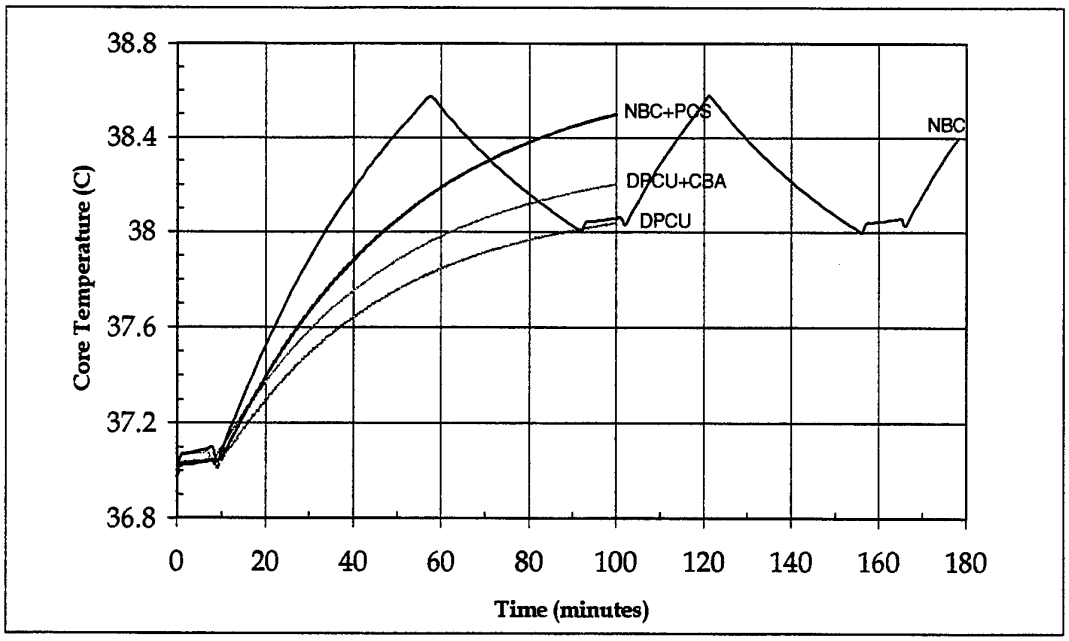

Figure 2: Time required completing 100 minutes of work.

The time required to complete 100 minutes of work whilst wearing NBC clothing is reduced from 178 minutes to 100 minutes when PCS is used (Fig 2), at this time the predicted core temperature is just below the 38.5°C threshold.

CONCLUSION

The PCS, assuming it is capable of delivering the cooling effect of 300 W, would have a significant impact on the performance of soldiers in an NBC environment. In situations when full encapsulation is not required, PCS would have a much smaller impact on the thermal load of the soldiers.

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POSTER 8: ORIGIN AND REGULATION OF METABOLIC HEAT

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INTRODUCTION

The origin of metabolic heat in a living organism is the biological work conducted. According to thermodynamic laws any work is accompanied by a release of heat. A living cell performs three kinds of work: chemical synthesis, transfer of ions against electrical and concentration gradients, and muscle contractions. An organism performs all these kinds of work both at rest and during muscular activity. Both at rest and during activity the amount of heat released depends on the quantity of work conducted and on the efficiency of work (η). The lower the value of η, the greater is the amount of heat released per unit of useful work. These basic ideas are often misunderstood. Let us explain the importance of this concept by use of specific examples.

At rest, a man of average weight and age releases about 7,500 kJ of energy per day. What biological work in an organism demands the highest energy expenditure and liberates the greatest quantity of heat? Almost all the energy that an organism receives from food is used to do work via the hydrolysis of ATP to give ADP and Pi. It is easy to calculate the energy cost of this, as under the so-called standard conditions, the efficiency of this work, according to Lehninger [1], is about 40%. Consequently, at the scale of the whole organism, e.g. an average man, only about 3,000 kJ of the daily energy expenditure of 7,500 kJ will be "accumulated" as ATP and 4,500 kJ will be lost as heat during ATP synthesis. The hydrolysis of ATP releases a very small amount of energy — about 34 kJ per mole of ATP. Consequently, to obtain energy from ATP at a rate of 3,000 kJ per day it is necessary to synthesize and subject to hydrolysis 3,000/34 = ≈90 moles of ATP. The mass of a mole of ATP is 506 g. Thus a human being synthesises and hydrolyses about 40-50 kg of ATP every day.

But the calculation of the heat balance does not end here. During ATP hydrolysis and mechanical work at least 50% of the energy released is converted to heat. Consequently, from 7,000 kJ of daily energy expenditure only ~1,500 kJ is available for biological work; the remainder is lost as heat. This is the main heat source in the living organism.

A very low η for biological work explains the relatively large energy consumption of humans. The population of the earth is now 6 billion. On average 2200 kcal or 9,200 kJ or ≈2.55 kWh is expended per person per day. The total amount of energy expended by humanity during a year is about $5.58 \cdot 10^{12}$ kWh. In 1993 all the thermal, atomic and hydroelectric power stations of the world produced $5.17 \cdot 10^{12}$ kWh [2].

Knowledge of the efficiency of biological work allows the energetics of an organism to be considered in a new way. Examples include: the physiological mechanisms of increasing and regulating the level of the heat production in a homeothermic organism under the threat of cooling; the mechanisms of increasing heat production after a prolonged adaptation to cold; the reasons for pronounced differences in the energy expenditure per unit of mass between homeothermic animals of different body sizes, between adult and new-born homeothermic animals, and between homeothermic and poikilothermic animals. These are major areas of interest in bioenergetics.

For many years the investigations of the laboratory of thermoregulation and bioenergetics of the I.P.Pavlov Institute of Physiology in St.Petersburg and of the laboratory of the physiology of thermoregulation of the Institute of Physiology in Novosibirsk were devoted

to investigating these relationships. We obtained results that allowed us to draw new conclusions about bioenergetics.

MATERIALS AND METHODS

To make a detailed quantitative analysis of the specific contractile activity in skeletal muscles (thermoregulatory muscle tone, shivering) we used powerful electromyographs (with sensitivity up to 5 μV/mm of the film), electronic integrators for summing the muscle electrical activity (S. μV · sec), microdevices which were introduced into a muscle and allowed us to record concurrently the electrical activity and temperature changes in the part of the muscle under study (sensitivity up to 0.005°C/mm of the tape of a self-recording potentiometer). To study the temperature effects of the contraction of an isolated muscle of a rat diaphragm we used a thermostatic control mechanism and measuring devices with sensitivity of up to 0.001°C/mm of the tape. We developed and used methods for measuring in situ the oxygen consumed by an isolated m. soleus of rats and by the femoral muscles of rabbits. We measured η of the work of isolated rat hearts after adaptation to cold and in control animals, and corresponding correlations were made. We measured the heat production of whole animals in a calorimeter and compared the total muscle electrical activity before and after adaptation to cold. Heat production and the total muscle electrical activity were measured in new-born animals (eg. rat and dog) at various environmental temperatures. Under supervision by physicians, similar observations were made on new-born babies (aged from several hours to several days) in an obstetric clinic, and also on cold-habituated sportsmen ("winter swimmers") in Novosibirsk.

RESULTS AND DISCUSSION

There is no longer a need to postulate the existence of special biochemical reactions of intermediary metabolism in cells, reactions whose sole purpose is increasing heat production. The evidence is now strong that the level of heat production in homeothermic organisms depends on the total volume of biological work, and on the efficiency of this work. Our experiments were aimed at verifying this concept in a range of animal species.

Muscles are able to increase quickly their energy expenditure and heat production at the expense of purposeful physical activity. Excessive cooling of the organism results first in an increase in thermoregulatory muscle tone and then, if cooling continues, in muscle shivering. According to our direct measurements, the increase in muscle tone leads to an increase in oxygen consumption and heat production in muscles by a factor of 2-3 compared to complete rest. The total increase in body heat production resulting from this is 25-50%. Muscle shivering causes oxygen consumption and heat production to increase by a factor of 8-12 (with total heat production by the organism increased by a factor of 2-2.5).

In the 1960s, many researchers concluded that after a prolonged adaptation to cold, animals acquire the ability to increase heat production in muscles, and in the whole organism, in response to cold without any specific contractile activity of muscles. A similar effect was also observed after injecting noradrenaline to cold-adapted animals. This phenomenon was called "nonshivering muscle thermogenesis" [3].

From the point of view of the origin of metabolic heat and its physiological regulation, the possibility of the existence of "nonshivering muscle thermogenesis" is of major significance. Hence we thoroughly studied this phenomenon. The electrical activity of muscles in our experiments was recorded using a powerful electromyograph. We showed that both after adaptation to cold and after noradrenaline injection, the total increase in heat production always coincides with an increase in the thermoregulatory muscle tone or in cold shivering. However, in cold-adapted animals, the level of muscle electrical activity during these reactions appeared to be 2 - 3 times lower than in control animals [4, 5].

Our hypothesis was supported by results obtained with a microdevice that allowed us to register simultaneously very small temperature changes (with a sensitivity of about 0.005°C/mm of the tape) and the total electrical activity (S, μV · sec) in the part of a muscle under study in situ in unanaesthesized and nonfixed rats. Each measurement lasted 10 sec. We carried out several tens of such measurements in every animal in the external temperature range 10-20°C. We tested 10 cold-adapted rats and 10 controls. The quantitative ratios between an increase in the total electrical activity and temperature changes in the same site of a muscle are given as the regression equations:

$$1)\ \Delta t^{\circ} = (41 + 0.85S) \cdot 10^{-2}$$

$$2)\ \Delta t^{\circ} = (24 + 1.16S) \cdot 10^{-2}$$

$$3)\ \Delta t^{\circ} = (45 + 1.66S) \cdot 10^{-2}$$

$$4)\ \Delta t^{\circ} = (68 + 2.50S) \cdot 10^{-2},$$

where Δt° is the change in temperature of the muscle in °C. Equation 1 - control animals, (air temperature 18-20°C); Equation 2 - control animals 15-20 minutes after injecting 200 μg/kg of noradrenaline into the blood; Equation 3 - animals adapted to cold (4 weeks in individual cages at air temperature 3-5 °C); Equation 4 – cold-adapted animals 15-20 min after injecting 200 μg/kg of noradrenaline into the blood. The differences between the angular coefficients of all the curves is statistically significant ($P_{1-2} < 0.05$; $P_{2-3} < 0.05$; $P_{3-4} < 0.01$).

These equations show: That even in animals not adapted to cold, the injection of noradrenaline results in a certain increase in heat production per unit of muscle electrical activity (i.e, an increase in thermoregulatory muscle tone and relatively weak shivering); that after adaptation to cold, the temperature effect per unit of muscle activity increases almost two-fold, and; that the injection of noradrenaline to cold-adapted animals increases the temperature effect per unit of muscle electrical activity by a factor of almost four compared to control nonadapted animals (a comparison between equations 1 and 4). The differences in the energetics of muscles in control and cold-adapted animals were evident only during contractile acts. At rest, their temperatures and the rates of oxygen consumption did not differ statistically significantly [6, 7].

We carried out more accurate thermometric studies on isolated rat diaphragm. The muscle was stretched over a special frame. It was directly electrically stimulated in the thermostat (1V; 0.1 sec). The isometric tension was measured. The temperature effect allowed us to calculate the amount of heat released per unit of muscle tension. We found that in control rats 0.502 ± 0.029 mJ (n=11; $P < 0.01$) was released per gram (g) of tension. After adding noradrenaline (0.008 μg/mL) to the nutrient solution, the increase in heat production per g of tension was 0.569 ± 0.021 mJ (n=11). The increase in heat production following addition of noradrenaline is not statistically significant ($P > 0.05$). The muscle of the diaphragm of cold-adapted rats increased its heat production per g of tension by 0.787 ± 0.050 mJ (n=11) in comparison with control animals $P < 0.01$). After adding noradrenaline to the nutrient solution in the same dose, the heat production of the diaphragm muscle of cold-adapted animals increased to 1.109 ± 0.033 mJ per g of tension, i.e. almost twice as much as the diaphragm muscle of control animals (n=11; $P < 0.01$) [6].

Isolated hearts of control (n=4) and of cold-adapted (n=5) rats had the same intensity of heat production, if they were arrested: 1.63 ± 0.08 and 1.63± 0.13 J / g · min respectively. However, during contractions, η of mechanical work of the heart of control animals was 8.00 ± 0.35% (n=8), and for the cold-adapted animals it was 5.10 ± 0.34% (n=2; $P < 0.001$). In other words, for the hearts of control animals to perform mechanical work of 1 J required ≈12.5 J of total energy expenditure. After adaptation to cold, performing

mechanical work of 1 J by the heart required ≈20 J of total energy expenditure. Thus, the heat production per unit of work increased by about 40%. The contribution of the heart to total heat production is not large: these results are presented simply to demonstrate the principles of regulating heat production [6, 7]. These principles were corroborated by studies of heat production of cold-habituated "winter swimmers" in Novosibirsk and St.Petersburg. Using a powerful electomyograph, we found that in new-born animals and new-born children, an increase in heat production in response to a decrease in temperature is also associated with an increase in muscular contractile activity [5]. In rats 3-4 days old, heat production per unit of electrical activity was shown to be almost twice as great as in rats 8-9 days old {11.9 ± 1.5 mW/g (n=7) and 5.4 ± 1.3 mW/g (n=9), respectively ($P < 0.01$)}. An increase in electrical activity in new-born animals and children was always accompanied by an increase in the activity of the brown fat [6, 7].

The uncoupling of oxidation and phosphorylation forms the basis of the decrease in η of ATP synthesis. We found this in the studies cited above. A particular mechanism of the decrease in η of ATP synthesis in the mitochondria consists of an increase in the proton leakage across the mitochondria inner membrane leading to "a futile cycle" [8].

CONCLUSIONS

The differences in energy consumption and in the volume of heat production per unit of mass and per unit of time between homeothermic and poikilothermic vertebrates, between large and small homeothermic organisms, between new-born and adult homeothermic organisms depend on: 1) the volume of the work of ATP synthesis, 2) the value of η of the work of ATP synthesis. In the outlined cases, η of the work of ATP synthesis is determined by the specific biological features of the structure and functions of mitochondria, the endocrinic status of an organism, and also other constant biological factors. The variations of the heat production, for the purposes of current thermoregulation or during a prolonged adaptation to cold, are regulated physiologically with the help of temporary changes in η of the current work of ATP synthesis in the cells of various organs by a temporary increase or decrease in the level of hormone and mediator secretion. These data suggest that a man and homeothermic animals have no special biochemical mechanisms for increasing the heat production. The source of heat for any living organism is current physiological work.

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POSTER 9: RESTORATION OF PHYSIOLOGICAL FUNCTIONS IN A COOLED ORGANISM WITHOUT REWARMING THE BODY

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INTRODUCTION

A man in 0°C water becomes helpless in 10-15 minutes and inevitably dies. An immediate cause of cold death is the arrest of pulmonary ventilation. However, in water of 18-20°C, death due to body cooling may occur in several hours. Accidental hypothermia is usually fatal upon the body core temperature decreasing to 27-25°C. This threshold differs between animals. In a white rat, pulmonary ventilation ceases at a brain temperature of 17-18°C. In homeotherms there is a paralysis or an abrupt decay of the physiological functions at tissue temperatures above the freezing point. Consequently, the reason for the paralysis and of the cold death is not mechanical destruction of the cells caused by the formation of ice crystals. What then, are the causes of cold paralysis of physiological functions and of cold death?

Hochachka [1] may have provided the first clear concept of the causes of disruption of physiological functions in homeotherms, and of their death from cold. According to this concept, the cold-induced disruption of function and cellular death result from an accumulation of calcium ions (Ca^{2+}) in the endoplasm of cells. Calcium ions play an important role in regulating various kinds of cellular metabolism. The physiological concentration of Ca^{2+} in the endoplasm is very small—approximately 10^{-8} M (i.e. about 5 ions in 1 μ^3 of the endoplasm). An increase to 10^{-6} M results in the "mismatching" of metabolism and the destruction of cell membranes through activation of intra-cellular lipo- and proteo-lytic enzymes. Since the Ca^{2+} concentration in the extracellular fluid (ECF) is several thousand times higher than that of the intracellular fluid (ICF), Ca^{2+} continuously moves into the cell by diffusion through calcium channels in the cell membrane. One of the physiological mechanisms of maintaining a low physiological concentration of Ca^{2+} in a cell is an active transport of Ca^{2+} from the ICF to the ECF, against a very large concentration gradient. According to Carafoli's calculations [2] the energy of the hydrolysis of one ATP molecule (i.e. about 33-50 kJ per mole of Ca^{2+}) is required to transport one calcium ion from the ICF to the ECF. These are large energy expenditures on the bioenergetic scale. A decreased tissue temperature in homeotherms destroys the quaternary structure of the ATPase [3], slowing the resynthesis of ATP and thereby causing an energy deficit. Since an energy deficit initially affects the most power-consuming processes, we can suggest that during hypothermia the maintenance of the ECF:ICF Ca^{2+} gradient is rapidly reduced. The accumulation of Ca^{2+} in the endoplasm leads to the blocking of various metabolic processes, cold paralysis of the cell, and ultimately to irreversible destruction of cellular membrane structure and function, causing its death. [1, 4].

The above theory appears logical, although we are unaware of experimental efforts to determine its validity. Some support for the theory may come from the observation that an organism's cold resistance can be increased if membrane permeability (via calcium channels) is reduced using hormones. However, it must be taken into account that there is considerable intra-cellular Ca^{2+} in the mitochondria and the reticuloendothelial system. If a deficit of energy arises, Ca^{2+} begins to pass into the endoplasm from these sources. Hence, if calcium channels in the cell membrane were blocked, Ca^{2+} would, nevertheless, accumulate in the endoplasm. Therefore we applied another method. We attempted to decrease the Ca^{2+} concentration in the plasma and, consequently, in the interstitial fluid

(ISF), We proposed that reducing the ICF:ISF Ca^{2+} concentration gradient would decrease the requirement for energy expenditure for Ca^{2+} transportation, thus decreasing the rate of Ca^{2+} accumulation in the endoplasm.

MATERIALS AND METHODS

We used the sodium salt of ethylenediaminetetraacetic acid (EDTA) to bind calcium ions in the blood. EDTA reacts with Ca^{2+} in the ratio 1:1. Using Ca^{2+} selective electrodes we determined the Ca^{2+} concentration in the blood of our test animals to be $1.8 \cdot 10^{-3} \pm 3 \cdot 10^{-3}$ mmol/ml in rabbits and $1.1 \cdot 10^{-3} \pm 0.1 \cdot 10^{-3}$ mmol/ml in rats. The dose of EDTA was based on the following measurements: the entire content of Ca^{2+} in the blood of a 3 kg rabbit was approximately 0.130 mmoles and approximately 0.016 mmoles for a 300 g rat. Introduction of EDTA into the rabbits' blood decreased the content of Ca^{2+} in the blood (and, presumably, in the ISF) by 15 ± 3% (the first injection) and by 27 ± 2% (the second injection). In rats, the content of Ca^{2+} in the blood after the first injection decreased by 22 ± 3% [5]. We did not obtain a second sample in rats because of the need to avoid decreases in blood volume within this comparatively small animal.

RESULTS AND DISCUSSION

Our first investigations were carried out on separate cold skin thermoreceptors of rabbits. A 6-7 cm^2 site of the nose back skin was cooled to 0-3°C and maintained at this level by a water thermode with melting ice, causing the biopotentials from separate thermoreceptors to disappear almost completely. In 4-7 minutes after beginning the infusion of EDTA (4 ml of the solution for 4 min) into the v. femorais (at the same skin temperature) biopotentials of the receptors were restored and attained high firing rates typical of those observed at a skin temperature of 12-15°C. Four minutes after the beginning of EDTA injection, the Ca^{2+} concentration in the blood decreased by 15 ± 3% ($P < 0.01$). When in the following 15 minutes the Ca^{2+} concentration recovered, for physiological reasons, to almost the initial level, the firing rate of the receptor biopotentials again decreased almost to zero. After repeated EDTA injection in the same dose the decrease in the Ca^{2+} concentration appeared to be greater: at 27 ± 2% ($P < 0.001$). This decrease coincided with a new increase in the firing rate of the biopotentials of thermoreceptors. After the second EDTA injection the decrease in concentration lasts for a longer period. Correspondingly, the excitation of the cold thermoreceptors also lasts for a longer time. Therefore, at a very low skin temperature, at which the cold thermoreceptors never work, they became excited without rewarming at the same skin temperature: 0-3°C [5, 6, 7, 8].

In subsequent experiments we tried to restore the cold-paralysed functions of the thermoregulation centre in the hypothalamus in rats by changing the Ca^{2+} concentration in the blood. Rats were submerged in water at 8-9°C up to the ears. Cold shivering resulted from cooling the animals, and attained maximum levels at a brain temperature of 28.5 ± 0.5°C and in the rectum at 25.8 ± 0.5°C. The intensity of cold shivering was estimated by integration of the biopotentials of the electromyogram (S, $\mu V \cdot sec$) with the help of an electron integrator. On further cooling, when the brain temperature decreased to 20.7 ± 0.3°C and in the rectum to 18.2°C, there was almost total cold paralysis of the thermoregulation centre and cold shivering ceased (according to the electromyogram). At 5-8 minutes after the beginning of EDTA injection the cold shivering returned and recovered to 60 ± 8% of the maximum ($P < 0.01$). Correspondingly, as in the preceding experiments, the Ca^{2+} concentration decreased by 22 ± 3% ($P < 0.01$). A physiological increase in the Ca^{2+} concentration at 14-16 minutes after the EDTA injection coincided with a new suppression of cold shivering. Repeated EDTA injection led to an almost complete (79 ± 7%, $P < 0.001$) re-attainment of the maximum intensity of cold shivering. Over the whole course of the experiment the body temperature remained constant at 19.2 ±

0.4°C. After the second EDTA injection, the effect of inducing the cold shivering lasted for 20-35 minutes, which corresponded to the period of the decreased Ca^{2+} concentration in the blood. By the end of this period the body temperature of the animals began to increase slowly [9, 10].

As was mentioned above, an immediate cause of death from cold in animals and man is ventilatory arrest, which never restores itself on its own at the temperature of the cold paralysis of the respiration centre. Hence, the restoration of the cold paralysed respiratory centre appears to be of special importance from both theoretical and practical points of view. In white rats a complete cold paralysis of the respiration centre occurs at a brain temperature of 17.5 ± 0.3°C or 18.5 ± 0.3°C (depending on the series of tests and the season). Even if the external cooling is stopped, the animals (in special control experiments) never restore their respiratory activity at a room temperature of 18-20°C. We were able to show that after a repeated injection of EDTA to decrease the Ca^{2+} concentration in the blood by 25-27%, the activity of the respiratory centre was restored and retained, despite the temperature of the respiratory centre being at the level of cold paralysis and even lower (15.5-16°C). All animals survived after the repeated EDTA injections [11, 12].

Another method of restoring and maintaining the activity of the cold-paralysed respiratory centre is an enhanced supply of cooled blood to the brain. Preservation of the arterial blood pressure at 70-80 mm Hg (as in hibernators) allows the respiratory centre to function at low brain temperatures. The respiratory centre actively functions at the medulla oblongata temperature of 17.2 ± 0.2°C, whereas, at an arterial pressure of 35-40 mm Hg, complete paralysis of the respiratory centre occurs when the temperature in the medulla oblongata decreases to 18.2 ± 0.2°C (the differences are significant: $P < 0.05$) [13].

CONCLUSIONS

Close examination of medical reports after many sea catastrophes convinced us that the techniques used to revive victims of severe hypothermia must be changed on the basis of new data. Treatment should be based on an adequate consideration of methods of recovery existing in nature. It is known that hibernators can preserve their body temperature of 2-4°C for several weeks. The physiological "warming up" of their bodies begins with an abrupt enhancement of ventilation, an enhancement of heart activity, and an increase in the arterial blood pressure whilst still at this very low body temperature. Only after such "preparation" will the mechanism of increasing heat production—the intensive muscle shivering—switch on. The process of "warming up" occurs under continuous physiological control. This excludes the development of an energy imbalance in the organism. As the possibility of stimulating physiological functions within a deeply cooled organism without rewarming the body develops, we must strive to save the victims of severe hypothermia using a technique that was long ago created by nature.

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All experiments on animal carried out during the last 15 years, had the permission for publishing.

POSTER 10: THE EFFECT OF AIR GAP ON THE FABRIC SURFACE APPARENT TEMPERATURE AND ITS THERMAL RESISTANCE

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INTRODUCTION

Thermal imaging (TI) technology has become more sophisticated and affordable. This has increased the possibility of a combat soldier being detected [1], resulting in a strong demand for developing TI camouflage materials to reduce the thermal IR (infra-red) signature of a soldier. The camouflage uniform must also possess thermal properties allowing efficient dissipation of body heat to the environment. In a theoretical analysis, Lee [2] indicates that the air gap between a heated surface and the test fabric has a significant effect on the fabric surface's apparent temperature. An optimum air gap of 9 mm was inferred. This paper investigates the effect of an air gap on the fabric surface's apparent temperature and its associated thermal resistance, and the implications of this for heat loss in both dry and wet conditions.

MATERIALS AND METHODS

The Disruptive Pattern Combat Uniform (DPCU) fabric was used as the test fabric in the investigation. The fabric sample was cut into a circular shape (0.02 m^2 in test area) and glued into a spacer that was just larger than the outer diameter of the heated surface of a sweating hotplate. The hotplate surface was set vertically and its surface temperature was measured using a radiometer and several Resistance Temperature Devices (RTD). Temperatures in the configuration of a bare plate and a plate with a layer of filter paper were measured separately. The ambient temperature of the chamber was set at 20°C, 25°C, 30°C and 40°C respectively with 60% relative humidity. Air gap thickness between the fabric and the hotplate was set between zero and 11.9 mm. Each condition was measured for about 1 hr. The hotplate was used in both dry and sweating configurations. In the wet configuration, a sweating rate of 0.3 $g.m^{-2}.s^{-1}$ was used, which is equivalent to a man of skin surface area of 2 m^2, sweating at the rate of 2,160 $g.hr^{-1}$. A constant sweating rate was used to ensure that the filter paper wicking layer remained wet throughout the test. A cellophane membrane was used to cover the filter paper during wet testing to ensure that only water vapour diffused through the test specimen. A radiometer (Inframetrics 760) was placed inside the environmental chamber (Figure 1) to measure the fabric surface's apparent temperature under different settings.

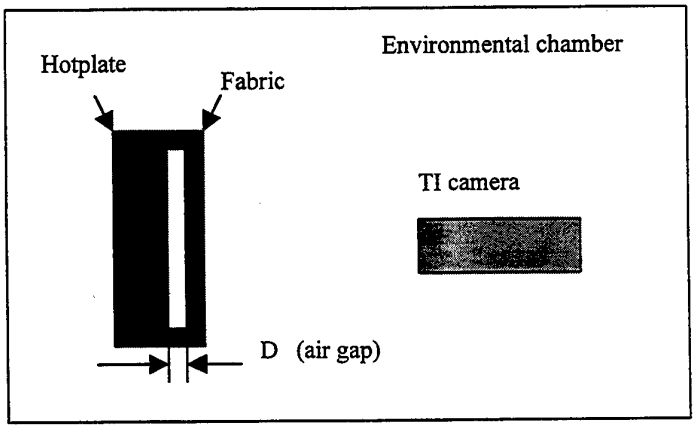

Figure 1. Schematic diagram of hotplate/fabric/environmental chamber set up

RESULTS AND DISCUSSIONS

Fabric surface's apparent temperature

Figure 2 shows the fabric surface's apparent temperature plotted against the air gap at different ambient temperatures, as determined by theory [2] and by experiment. The dotted lines refer to theoretical predictions [2] and solid lines indicate the experimental results. As shown in figure 2, most experimental points fit well into the theoretical predictions, although some data points are slightly lower than theoretical predictions at an ambient temperature less than 25°C and with an air gap less than 2.5 mm. This discrepancy probably arises from the effect of free convection associated with the temperature difference between the fabric surface and the chamber air temperature, which is not considered in the theoretical calculation. Figure 2 clearly demonstrates that the fabric surface's apparent temperature is a function of air gap, and that an air gap of approximately 9 mm will effectively reduce or increase the fabric surface's apparent temperature.

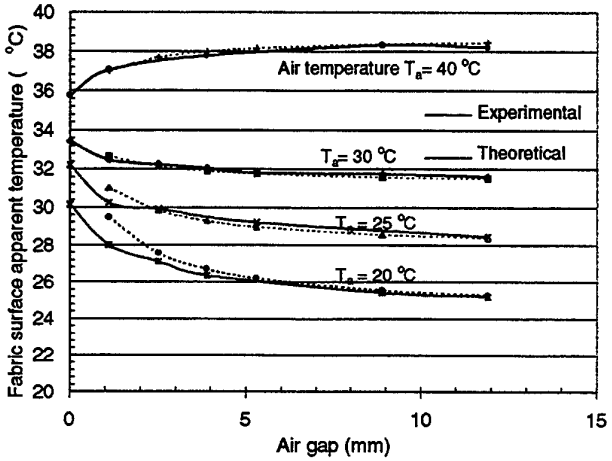

Figure 2. Effect of air gap on the apparent temperature of the fabric surface

Table 1 shows the fabric surface's apparent temperature measured at chamber air temperature of 20°C and 60% relative humidity. The hotplate was set in three different states: dry, wet and water vapour.

°C	Bare plate	0 mm	1.1 mm	2.5 mm	3.9 mm	5.3 mm	8.9 mm	11.9 mm
Dry	33.22	30.13	27.98	27.09	26.33	--	25.41	25.19
Wet	32.37	30.89	27.59	--	--	25.96	25.39	25.22
Water vapour	32.96	29.65	27.91	27.16	26.46	25.94	25.31	--

Table 1: Fabric surface's apparent temperature (°C) at 20 °C and 60% R.H.

When the hotplate was in the dry state no water was pumped from the reservoir. The electrical power consumption by the hotplate was due to the loss of dry heat to the surroundings through the fabric sample. When the hotplate was in the wet state its surface was covered with a layer of filter paper to provide a uniform wicking of the water across the surface. Throughout the course of the test, the filter paper was kept in a fully saturated state by maintaining a sweating rate of 0.3 $g.m^{-2}.s^{-1}$. Power consumption in this case was used to compensate for both the loss of dry heat and water vapour evaporation. When only water vapour transmission was required, the filter paper was covered by a cellophane

membrane. This was to prevent the fabric sample under test from coming in contact with the liquid water at the hotplate surface, while allowing water vapour to permeate through it and into the atmosphere. As shown in Table 1, although there are some slight variations in temperature in the case of bare plate and zero mm air gap among the dry, wet and water vapour states in the hotplate, no significant difference can be found among these three states. Table 1 also indicates that once an air gap exists between the fabric under test and the hotplate surface, the fabric surface apparent temperature appears unaffected by wet heat transfer.

Thermal resistance

The primary heat loss is the dry heat loss, which is a function of the temperature gradient from the hotplate to the environment. As shown in Table 2, when the ambient condition was at 20°C and 60% R.H., the thermal resistance increased with the increase of air gap between the hotplate and the test fabric. Dry heat loss would decrease as a result. However, in the case of sweating hotplate, the evaporating water carried away a substantial amount of heat energy as vapour into the environment. Thus, the intrinsic thermal resistance decreased. As shown in Table 2, the intrinsic thermal resistance at an air gap of 8.9 mm was only 34.4% of its thermal resistance when it was tested in the dry state. It should be noted that the intrinsic thermal resistance increased with the increase of the air gap.

Air gap (mm)	Bare plate	0	1.1	2.5	3.9	5.3	8.9	11.9
Thermal resistance (m^2k/w) (Dry)	0.070	0.094	0.135	0.143	0.162	-	0.195	0.203
Intrinsic thermal resistance (m^2k/w)(Vapour)	0.019	0.025	0.033	0.039	0.046	0.053	0.067	-

Table 2: Variation of thermal resistance due to different air gap settings and water evaporation

As the specific heat of evaporation of water is 2.43 $kJ.g^{-1}$, the corresponding additional heat losses due to water evaporation could be translated into the amount of water vapour transfer as shown in Table 3. (assuming a mean body skin surface area of 2 m^2)

Air gap (mm)	Bare plate	0	1.1	2.5	3.9	5.3	8.9	11.9
Vapour loss (g/hr) surface area 2 m^2	1780	1421	1070	886	737	-	467	-

Table 3: Variation of water vapour transfer due to different settings of air gap

It is apparent from Table 3 that the air gap can significantly influence the vapour transfer from the hotplate to the environment through the fabric. At the air gap of 8.9 mm, the rate of water vapour transfer was about one-third the rate that occurred at the air gap of 0.0 mm.

CONCLUSIONS

The following conclusions may be reached:

- The optimum air gap to effectively achieve the reduction in fabric surface's apparent temperature is about 9 mm;
- The presence of vapour evaporation did not affect the apparent surface temperature of the test fabric;
- The air gap increases the thermal resistance of the test fabric ensemble;

- The water vapour transfer from the hotplate to the environment reduced the intrinsic thermal resistance of the test fabric ensemble significantly;
- The resistance of vapour transfer loss increased with respect to the increase in the air gap.

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POSTER 11: MICROCLIMATE OF THE SM1 TANK IN STATIONARY CONDITION

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INTRODUCTION

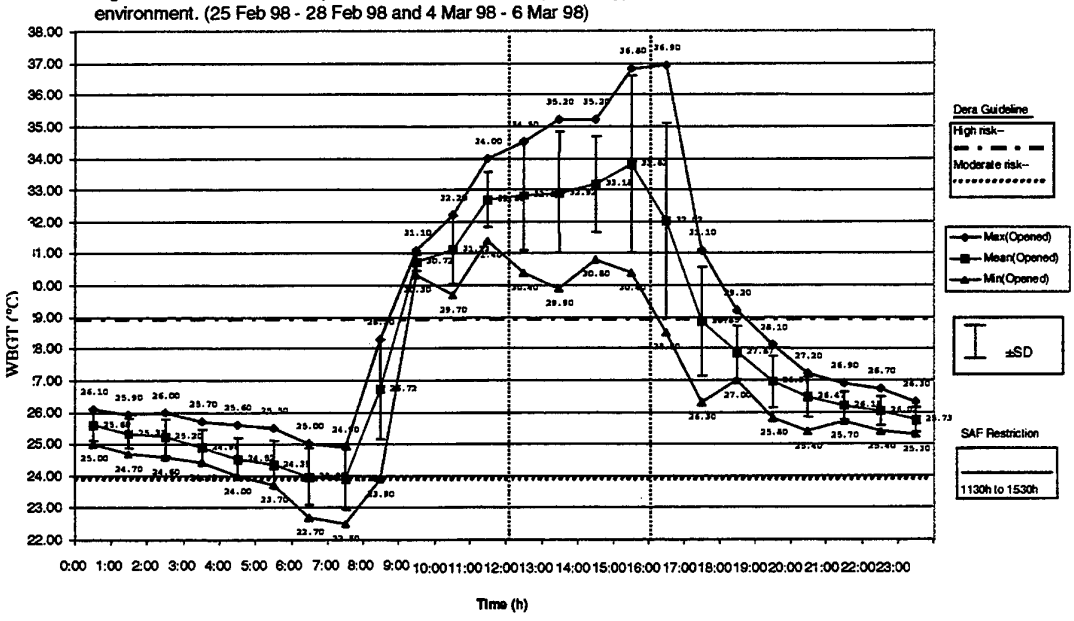
Soldiers operating in enclosed environments are exposed to high heat stress, which can be detrimental to performance in physical [1,2] and cognitive tasks [3]. To reduce this risk, climatic considerations are critical in the planning of training and operations. An understanding of the profile of the microclimates in these operational conditions would facilitate the design and implementation of measures to counter the effects of thermal stress on human performance. This paper describes the climatic profile of the SM1 tank in stationary condition. Trials were also conducted when the tank was in motion, but these trials are not described here due to space constraints.

MATERIALS AND METHODS

The climatic parameters measured include Dry Bulb Temperature (T_d), Wet Bulb Temperature (T_w), Globe Temperature (T_g), Humidity (Rh) and Wind Speed (ws). Wet Bulb Globe Temperature Index (WBGT) was based on the equation: WBGT= 0.7 T_w + 0.2 T_g + 0.1 T_d [3]. The climatic parameters were logged for the vehicle while it was parked in the open in an opened hatch condition and in a closed hatch condition. Data were logged continuously for several days for each stationary condition. The data were collected in the Commander (CmC) and Driver (DC) compartments of the tank and the ambient environment (ENV) of the parked vehicle, in February and March 1998.

RESULTS

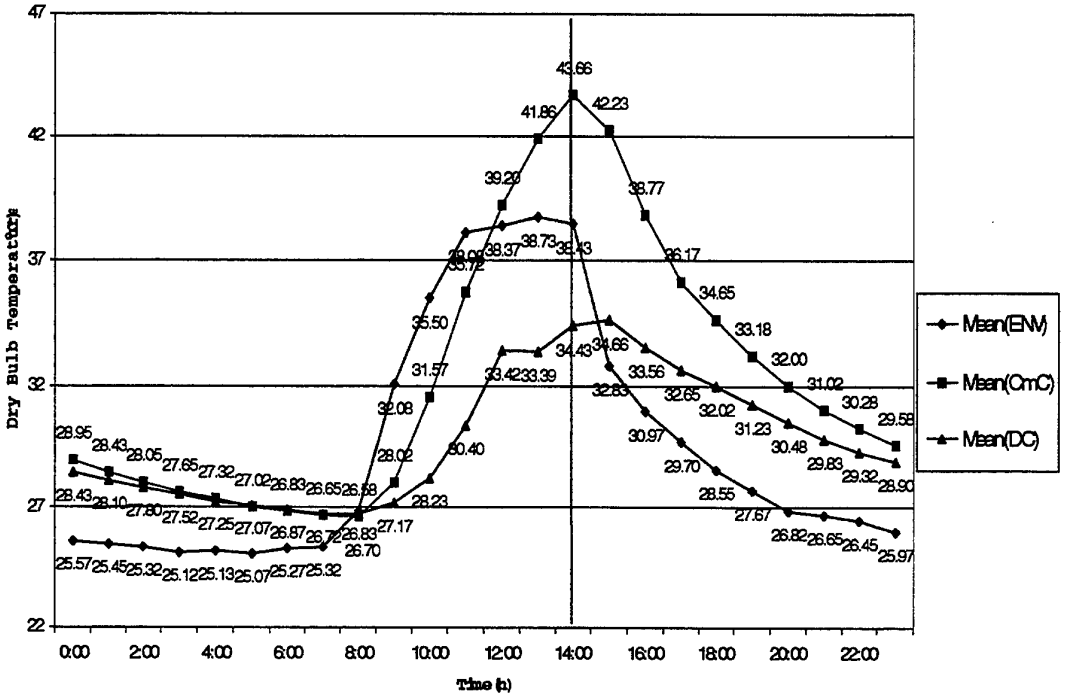
Fig 1: SM1 tank with hatch opened, monitored over 24h (stationary). Mean ± SD of WBGT in the environment. (25 Feb 98 - 28 Feb 98 and 4 Mar 98 - 6 Mar 98)



The highest and lowest climatic parameters of the ENV recorded are: Td = 22.6°C and 45.5°C; T_w = 22.4°C and 33.9°C; T_g = 22.6°C and 63.7°C; WBGT = 22.5°C and 39.3°C (Fig 1); Rh = 43.6% and 100%; ws = 0 and 3.3 $m \cdot s^{-1}$. The results showed that there was a

delay in the rise to peak hourly mean T_d between ENV and the compartments within the tank. The compartments had different profiles in terms of the rate of rise and final mean temperatures (Fig 2). Although the CmC and DC are located beside each other in the tank, there was a difference of 9°C between them for peak T_d (Fig 2). Prediction equations were developed from the data to predict internal conditions of the tank from environmental conditions (Table 1).

Fig 2: Graph of Dry Bulb Temperatures of Commander, Driver compartments and Environmental conditions of SM1 tank in closed hatch condition. Difference of 9°C between CMC and DC at peak Td.



	Condition (Opened Hatch)	Regression equations	N	R	r^2
a.	T_d (DC) from T_d (ENV)				
(i)	24h (0000h to 2359h)	y = 0.66x + 9.97	17327	0.88	0.78
(ii)	Day (0700h to 1859h)	y = 0.72x + 7.66	8686	0.82	0.67
(iii)	Night (1900h to 0659h)	y = 0.97x + 1.74	8640	0.96	0.93

Table 1: Example of prediction equations for climatic parameters of a stationary SM1 tank in opened hatch condition.

CONCLUSIONS

These differences were statistically significant and suggest that each compartment should be considered separately when designing cooling systems. Due to the difference in the response times between the compartments and ENV, conditions in the environment should not be used as a direct indicator of vehicle conditions. Regression equations could be used to predict internal conditions of the vehicle. The high heat load recorded in the compartments of the vehicles suggests that consideration should be given to fluid replacement and dress codes of personnel operating within it. When compared against the DERA guidelines on risk assessment of heat stress [5], the environment and vehicle compartments are considered to be at moderate to high risk for most part of the day. The study showed that the high heat load in the tank deserves more specific considerations in

the design and development of personal cooling systems and training regimes. Further study is recommended to provide detailed information, particularly on heat stress during operations.

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POSTER 12: IMPROVING THE MILITARY'S WET WEATHER GARMENT

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INTRODUCTION

Military personnel are required to wear clothing in more extreme environments than the average civilian does. The clothing they wear must be designed to protect them against the elements, yet still enable them to successfully complete their operational activities. Their basic uniform consists of a camouflage-printed jacket and pants with additional clothing being worn as required. One of the most important items of additional clothing is their raingear.

The major factors that contribute to the comfort of clothing ensembles are their ability to transport heat, air and moisture vapour to the environment. The amount of air or moisture vapour that can permeate any ensemble is dependent on the breathability of the most impermeable component, which is usually the rainwear.

There are two distinct mechanisms by which coated or laminated fabrics can achieve "breathability". One is by using a Microvapour Porous (MVP) material as the barrier layer; the other is by using a hydrophilic material.

AIM

The aim of this work is to identify the most suitable fabric from which to manufacture military rainwear for ADF personnel. The selected fabric must be not only waterproof but must also be breathable. Rainwear manufactured from the selected fabric must be water repellent, yet provide a high level of breathability.

METHODS AND MATERIALS

A sweating hotplate was used to test the thermal resistance and rate of moisture vapour transmission of a range of wet weather fabrics. The sweating hotplate is used in the dry mode with no water passing through to the hotplate surface when determining the resistance to the transfer of dry heat transfer, and with 0.3 $g \cdot m^{-2} \cdot s^{-1}$ of water passing through the glands when measuring the rate of transfer of moisture vapour. **Samples:** A number of fabrics were evaluated to determine their suitability for military rainwear and compared to the in-service item. A listing of the fabrics and their resistances to dry heat and moisture vapour transfer is given in Table 1.

FABRIC	RESISTANCE TO THE TRANSFER OF HEAT ($R_t = m^2KW^{-1}$)	RESISTANCE TO THE TRANSFER OF MOISTURE VAPOUR ($R_e = m^2PaW^{-1}$)	THICKNESS (mm)
DPCU (1)	0.084	8	0.9
In-service raincoat Polyurethane coated (2)	0.072	184	0.3
Knitted polyester laminated with a 30 μm MVP Poly(ether)urethane Membrane (3)	0.074	13	0.5
Goretex ™PTFE membrane	0.078	13	0.8
A 12μm Poly(ether)urethane laminated to a cotton-polyester oxford fabric	0.074	10	
A 30 μm Poly(ether)urethane MVP membrane laminated to a cotton-polyester oxford fabric	0.074	16	0.75

Table 1. Thermophysical properties of selected materials.

DISCUSSION

The coating or membrane used as the waterproof barrier in rainwear is thin compared to the fabric supporting it. Because of this the barrier material itself provides little resistance to the loss of dry heat.

Conversely, there is a difference in the rate of transmission of water vapour between materials. This is not so dependent on the thickness of the membrane but is related both its permeability and the mechanism by which it transports the moisture vapour through its cross-section and into the atmosphere. From Table 1 it can be seen that the in-service item has a high resistance to the transport of moisture vapour, whereas the other samples are have a much lower resistance and are more permeable. From the data provided in Table 1 it may be considered that a fabric laminated with the 12 μm membrane would be the best choice as a raincoat fabric. In Figure 2 the resistance to the transmission of water vapour of each membrane, measured as the equivalent resistance offered by a layer of "still" air, highlights the differences in the mechanisms of moisture vapour transmission between the hydrophilic membrane and the MVP membrane. Figure 2 clearly shows that the rate of diffusion of water vapour through the hydrophilic membrane is dependent on the vapour gradient. As the vapour gradient decreases the resistance to the transfer of water vapour given in an equivalent thickness of air increases. This demonstrates that the resistance to the transfer of moisture vapour for the MVP laminate is independent of the vapour gradient. As it is unlikely that a raincoat or any form of rainwear will be in direct contact with a wet surface over its total area, the 30 μm laminate is considered more suitable.

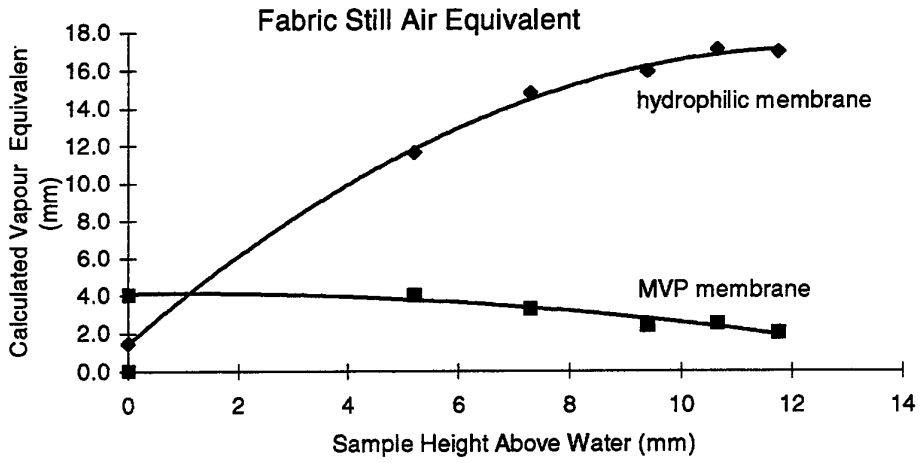

Figure 2. Resistance to water vapour transmission-equivalent thickness of still air

In an attempt to show the improvement in comfort a raincoat manufactured from the 30 μm MVP fabric the fabric parameters were fed into a computer model designed to simulate the physiological strain on soldiers operating under different scenarios. The model used was developed at US Army Research Institute of Environmental Medicine (USARIEM). Lau and Sanders [1] have studied the model and its application.

The following parameters have been fed into the model and the increase in body core temperature with increasing work time plotted. Figure 3 shows the improvement in comfort (reduced body core temperature) for a combatant operating under the same conditions wearing a raincoat manufactured from the MVP fabric compared with a combatant operating in the in-service, impermeable raincoat. In both cases the model assumes each raincoat is worn over the Disruptive Printed Combat Uniform (DPCU).

PARAMETERS:

- Environmental condition 35°C/90%RH
- Nude mass 75 kg
- Height 185 cm
- Additional load 10 kg
- Walking speed 1.5 $m \cdot s^{-1}$
- Wind Speed 1.0 $m \cdot s^{-1}$
- Terrain Dirt Road

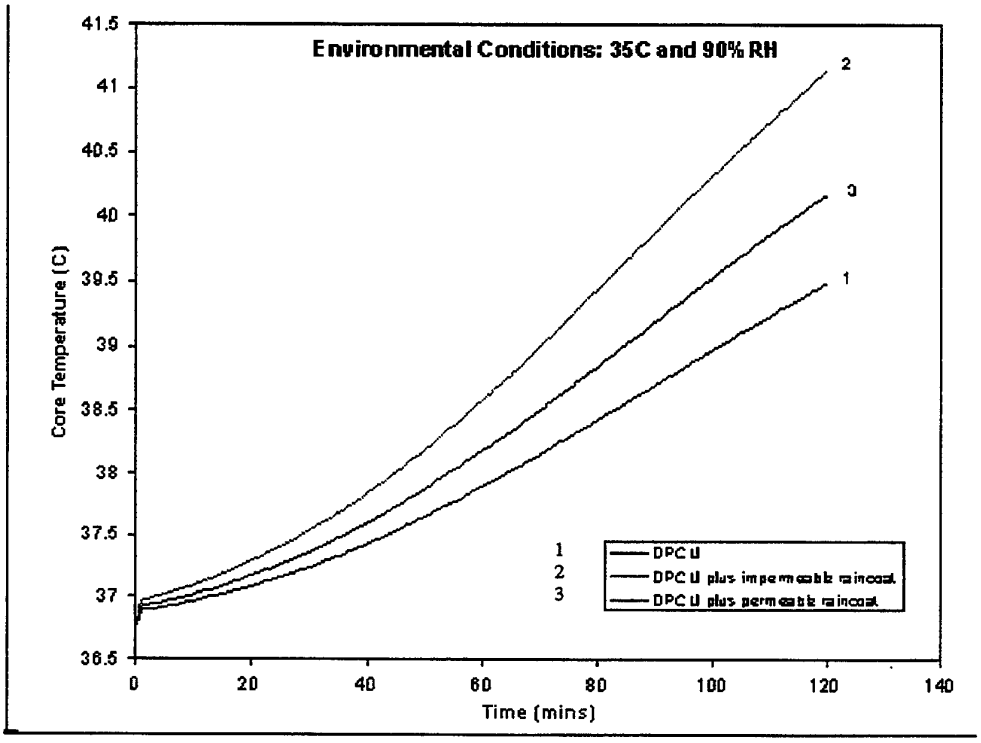

Figure 3. Theoretical physiological load when wearing different ensembles

CONCLUSION

A sweating hotplate was used to determine the thermal and moisture transmission properties of the fabric used in the current ADF rainwear, as well as a MVP and hydrophilic laminated fabric. These tests demonstrated that the MVP and hydrophilic fabric provided superior "breathability" compared with the in-service fabric.

Additional testing confirmed that the MVP fabric was preferred as its level of "breathability" was unaffected by changes in the vapour pressure gradient across it, whereas the "breathability" of the hydrophilic fabric was dependent on the vapour pressure gradient.

Theoretical calculations for the physiological load imposed by rainwear on a soldier showed that the MVP fabric imposed a physiological load that was significantly less than that of the in-service garment.

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POSTER 13: PREDICTION OF THERMAL STRAIN USING NEURAL NETWORKS

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INTRODUCTION

Computer programs have been developed over many years for simulation of physiological responses to exercise under differing conditions [1, 2, 3, 4, 5, 6]. An empirical heat strain computer program developed by the US Army Research Institute of Environmental Medicine (USARIEM) was used extensively during the Gulf War and during recent US peacekeeping missions in Somalia and Rwanda. Heat casualties reported in these military operations were substantially fewer than expected.

The development of suitable algorithms for the implementation of computer programs from statistical analysis of experimental data has required that the researcher first select a mathematical equation with due regard for known theory and then perform successive refinements by repeated regression analysis until an acceptable level of accuracy has been attained. In contrast, a Neural Networking technique starts with a number of sets of input parameters, the corresponding sets of output results and a large matrix of arbitrarily initialised numbers. To 'train' the network, each set of input parameters is used to calculate one set of output values, which will be in error to some degree. A portion of the output errors are then fed back into the network (back propagated) to adjust the values in the matrix of numbers. This process is repeated until some measure of success achieves its optimal level. The major difference from the regression technique is that there is no need for any advance knowledge of the nature of any relationship between input and output. The matrix is then said to be 'trained' on the input data and can be used to generate or 'generalise' output from parameters which were not part of the training process.

MATHEMATICAL BASIS

The Neural Network method which best lends itself to the production of linear outputs from linear inputs is called the Adaptive Linear Element (Adaline) with Supervised Back-Propagation as the learning rule [7]. The elements of this Neural Network consist of a vector [I] of input numbers, a vector [O] of output numbers; the weights matrix [W] and two vectors ([N] and [B]) the only purpose of which is to improve computational accuracy by 'normalising' data values. Therefore each N_i will approximate the reciprocal of the mean value of all I_i and each B_j will approximate the mean value of all O_j in the training data.

Processing begins by initialising all the weights in the matrix to be approximately 1. Outputs (G) are then calculated [6] using an equation similar to the following:

$$G_j = \frac{B_j}{n} \sum_{i=1}^{n} I_i N_i W_{i,j}$$

The generated output values will have some errors. The weights matrix will be adjusted by back-propagating an arbitrarily chosen fraction (F) of this error. The training may have to be repeated several times to enable an optimal value for F to be determined. Each weight then has a new value:

$$\left[W_{i,j} = W_{i,j}(1.0 - \frac{G_j - O_j}{B_j} F) \right]_{i=1,n}^{j=1,m}$$

Simultaneous with this back-propagation, some measures of the error levels are accumulated for later use in determining whether sufficient training has been conducted. This processing is repeated for each of the training sets. One epoch is said to be completed when the entire training data has been processed once. At the end of each epoch, the overall error level for the entire epoch is compared to preceding epochs and decisions as to whether to continue training and whether to modify processing constants (such as the back-propagation fraction) are made.

This single-layer Adaline often does not have sufficient complexity for subtle real-world problems, in which case up to three or even four matrices of weights may be employed. In these Multi-Layer Adalines, processing occurs one layer at a time as described above but now the outputs from the first layer become the inputs for the second layer (using a second weights matrix), the outputs from the second layer become the inputs for the third layer and so on. In the Multi-Layer Adaline, the normalisation vectors are not employed between layers, as this is unnecessary - they are only needed for the initial inputs and the final outputs.

METHOD

Laboratory trial data on soldiers exercising in the heat were used to train the Neural Network. Details of the trial objectives and conditions have already been reported [1].

A program to implement the Adaline was written in Gnu C++ on a 120 MHz Pentium computer running the Gnu Public Licence Unix 'Linux' but the resulting source code is not system dependent. The single-layer Adaline written initially was expanded to two and then three layers as the software matured.

The Adaline was provided with the following inputs:

- Subject details (nude weight; height; length of acclimatisation),
- Clothing properties (insulation; permeability and 'bagginess'),
- Environmental factors (ambient temperature and relative humidity; wind speed; terrain difficulty),
- Total metabolic rate (calculated from equation 4, Givioni & Goldman [3]).

The calculated outputs during the training were the 100 minute-by-minute body core temperature measurements.

The written program was set up so that initially 50% of the generalisation stage errors were used to correct the weights matrices until the most significant digit of the RMS collation of the errors had been unchanged for 10 epochs. The error factor was then divided by 10 and the next most significant digit of the RMS error was monitored in the same way. This algorithm was repeated a further four times which required less than 100 epochs or less than two minutes on the computer in use. This level of training accuracy is considerably more than the 0.1°C required to equal the accuracy of experimental data and may be decreased if larger data sets result in long training times.

RESULTS AND DISCUSSION

Table 1 shows the end-point (100 min) rectal temperature calculated by the generalisation stage of the three-layer Adaline and the measured temperatures from the 1995 AMRL laboratory studies. This table shows a mean error of 0.3°C at 30°C and 60% RH. Under warmer and more arid conditions (40°C and 30% RH), the discrepancy between the measured and calculated mean end-point rectal temperature changed to -0.2°C.

	Environment 30°C and 60% RH		Environment 40°C and 30% RH	
Subjects	Measured Temp (°C)	Calculated Temp (°C)	Measured Temp (°C)	Calculated Temp (°C)
A	38.4_a	38.8_b	38.4	38.2
B	38.3	38.4	38.7	38.4
C	38.7	38.4	39.0_a	38.3_b
D	38.3	38.7	38.5	38.4
E	38.1	38.6	38.5	38.2
F	38.4_a	38.4_b	39.0_a	38.6_b
G	37.6	38.3	nd_a	38.4_b
H	38.2	38.6	38.5	38.6
I	37.9	38.4	38.6	38.5
Mean	38.2	38.5	38.6	38.4

Note: $_a$ A full 100 minute set of measured data is not available in these instances because the subject exceeded the experimental safe core temperature threshold (39.0°C) or because of loss of good contact with the rectal temperature probe.

$_b$ These end-point values were calculated after the network had been trained. The parameter sets for these subjects were not part of the training process, hence these points are examples of the predictions that the network is capable of making.

Table 1: The Measured and Calculated End-Point (100 minute) Rectal Temperature under Two Simulated Tropical Conditions.

Pearson Correlation coefficients between the measured and predicted rectal temperatures were 0.19 at 30°C and 60%RH and 0.34 at 40°C and 30%RH. These results indicate that a positive but weak correlation existed between the predicted and the measured rectal temperatures in both experimental conditions.

The accuracy of prediction of Adaline-calculated end-point rectal temperatures can be further evaluated by analysing their Specificity and Sensitivity. The definitions of Sensitivity and Specificity are based on the criterion that a warning will be issued if rectal temperature reaches or exceeds 38.5°C, the level accepted by OH & S practitioners as an upper safety limit for heat strain for civilians. Specificity is defined as True Negative divided by (False Negative + True Negative)[TN/(FP+TN)]. Sensitivity is defined as True Positive divided by (True Positive + False Negative) [TP/(TP+FN)] [8]. Table 2 shows the resulting specificity and sensitivity values for 100 min end-point temperatures calculated by the Adaline.

	30°C and 60% RH	40°C and 30% RH
Specificity	0.75	1.0
Sensitivity	0	0.4

Table 2: Sensitivity and Specificity Values for the Three-Layer Adaline Calculated Rectal Temperatures Adopting an Upper Threshold of 38.5°C

A good prediction model would have high sensitivity and specificity and high correlation with the measured values. The lack of simultaneous high specificity and high sensitivity

and the generally low correlation between the measured and predicted values suggests that this neural networking technique has relatively low predictive precision. However, when the above results are compared with those obtained from the unmodified USARIEM program [5], it is found that this infant neural networking technique has achieved similar predictive precision to a program that required significant resources to develop. The potential now exists for further experimental data to be examined using this new technique.

CONCLUSIONS

The Adaptive Linear Element Neural Networking technique with Back-Propagation as the learning rule is worthy of further investigation as a tool for the prediction of human physiological responses to thermal stress. A data set of only nine subjects used for this study is insufficient to allow more definite conclusions to be drawn. It is recommended that further evaluation on the validity of the Neural Network program technique should be conducted against larger data sets measured under a variety of experimental conditions.

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POSTER 14: THE COMBINED EFFECT OF HEAT AND CARBON MONOXIDE ON THE PERFORMANCE OF THE MOTORSPORT ATHLETE

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INTRODUCTION

This research examined the combined effect of heat and carbon monoxide (CO) on the performance of Motorsport Athletes (MSA) competing in simulated NASCAR races. The recording of environmental conditions within a racecar have indicated that air temperature exceeds 50°C, and that CO levels can approximate 200 ppm [1]. Blood sampling of the MSA after a race has indicated that a 12-15% carboxyhemoglobin (HbCO) level develops [2]. Although there is limited research into the combined effect of heat and CO, individual examination of theses two stressors has indicated that they can both lead to deterioration in psychomotor performance [3,5].

MATERIALS AND METHODS

To safely examine the MSA under the combined stressors of heat and CO, a racecar simulator was developed within an environmental chamber. The simulator was not designed to create the sensation of racing, but create a medium through which to expose the MSA to the physical, environmental and psychomotor stressors that exist during a race. Psychomotor stress was induced through positioning a dashboard in front of the MSA, which required them to react to either a warning light (choice reaction) or a gauge warning (vigilance reaction).

Eight non-smoking, male subjects (mean age, weight and height, respectively, ± SD = 26 ± 6 y, 82 ± 13 kg & 179 ± 4 cm) were selected from the local population of elite MSA. Subjects underwent an orientation period prior to being randomly assigned to three test conditions, (with at least 48 hours rest between conditions), – Cool (20°C), Heat (50°C) and heat plus CO, designated Heat/CO (50°C and 10-12 % HbCO). For each condition subjects wore the required FIA standard race clothing (three-layer nomex suit, gloves, boots and helmet). The research procedure was divided into two stages: first, the subject performed a physical fatigue protocol consisting of riding a Monark cycle ergometer for 15 minutes at 125W in the environment being examined, followed by a simulated racing protocol consisting of three 20-minute racing segments. In the Heat/CO condition, a bolus dose of 3,000 ppm CO was administered to the subject in the five minute rest period prior to the commencement of the first simulated racing segment, followed by a maintenance dose of 200ppm CO for the duration of the simulation. During each simulation, changes in body mass, heart rate, body temperatures (rectum, calf, forearm & sternum) and oxygen consumption were recorded. Choice and vigilance reaction times were periodically recorded during each of the simulations. The driving performance of the MSA was recorded by monitoring lap times and the number of mistakes made. At the end of the CO, condition subjects were placed on a 100% oxygen protocol to remove residual CO from their system, before being allowed to leave the laboratory.

RESULTS

A summary of the results is contained in Table 1.

	Cool	Heat	Heat/CO
Stored Body Heat ($W \cdot m^{-2}$)	15.08 ±7.20	61.03 ±10.52[1]	72.45 ±6.54[2, 3]
Index of Thermal Strain	1.16 ± 0.22	2.55 ± 0.66[1]	3.05 ±0. 65[2, 3]
Change in body mass (kg)	0.35 ± 0.12	1.14 ± 0.28[1]	1.53 ±0.20[2, 3]
Oxygen Consumption ($l \cdot min^{-1}$)	0.40 ± 0.03	0.42 ± 0.04	0.39 ±0.06
RER	0.84 ± 0.04	0.85 ± 0.05	0.86 ±0.90
Change in T_{skin} (°C)	1.11 ± 0.32	3.67 ± 0.48[1]	4.10 ±0. 54[2]
Change in T_{rectal} (°C)	0.09 ± 0.30	0.73 ± 0.26[1]	1.06 ±0.39[2, 3]
Final Heart Rate (bpm)	88 ±9	126 ±20[1]	134 ±15[2]
Number of contact points	22 ±13	25 ±11	38 ±16[2, 3]
Gauge reaction time (s)	5.59 ±5.27	3.44 ±3.94	4.12 ± 5.59
Light reaction time (s)	0.99 ±0.21	1.06 ±0.14	1.11 ± 0.49

Significance at $p<0.05$: [1]Cool vs Heat [2]Cool vs Heat/CO [3]Heat vs Heat/CO

Table 1. Summary of results

DISCUSSION

The main measure of psychomotor performance was the 'tallying' of contacts that the MSA made with either another competitor or the racetrack wall. Although there was no significant change in the number of contacts between the Cool and Heat condition, a significant ($p<0.05$) increase in contacts occurred with the inclusion of CO into the hot environment. (see Figure 1). This may manifest on the race track as a decrease in the ability of the MSA to accurately determine the speed and position of their racecar in relation to other racecars [4].

One of the important tasks of a NASCAR MSA is the ability to draft a lead car to reduce aerodynamic drag. High levels of CO could impair the ability of the MSA to effectively and safely complete this task. Although this research was conducted under simulation, it still required subjects to utilise similar reaction, vigilance and tracking skills as required in controlling a real racecar. The limited change in psychomotor results between the Cool and Heat conditions is of interest. It would be expected that the optimum environment for psychomotor performance would be a cool condition. However, there was no change in performance between the Cool and Heat conditions. Questioning of subjects after the cool simulation indicated that there was a decrease in arousal due to the absence of the heat stress that is usually present during a race (ie. 'it just felt like we were playing a game'). It is proposed that the absence of the heat stress may limit the transfer of task learning from the simulator to the real environment.

Mixed results occurred in regards to the reaction lights and gauges. An examination of the gauge test results (vigilance reaction) indicated that the fastest reaction occurred in the Heat condition, with a non-significant increase in reaction time occurring in the Heat/CO condition. In regards to the light reaction test results (choice reaction), there was a non-significant increase in response time across the three conditions. The use of a larger subject group may have produced a significant difference in these psychomotor tests.

The Heat condition produced a significant increase ($p<0.05$) in physiological index of strain and stored body heat in comparison to the Cool condition. The addition of CO into the heat environment produced a further significant rise ($p<0.05$) in these two measurements (see also Figure 2). There is insufficient data to determine why there was a

further rise in these values with the inclusion of CO. The use of blood lactate measures may have provided more insight into the effect of CO on muscle metabolism. The increase in stored body heat in the Heat/CO condition also produced a significantly ($p<0.05$) higher sweat rate in comparison to the Heat condition. It is proposed that during races held in high CO environments, a larger volume of fluid should be consumed by the MSA to limit dehydration.

There was no significant change in oxygen consumption between the conditions. Therefore, it is suggested that the heart rate (88 bpm) recorded in the Cool condition reflects the strain placed on the MSA through the physical task of controlling the simulated racecar. With the exposure to heat, there was an insignificant change in oxygen consumption, but a significant increase ($p<0.05$) in heart rate (to 126 bpm) due to cardio-vascular drift as a result of the hot environment. Heart rate data collected during a race indicated that MSA heart rates are constantly above 160 bpm [6,7]. Therefore, the difference between the simulated and recorded heart rates is likely to be a result of the psychomotor stressors and the enhanced secretion of adrenaline due to increased sympathetic nervous activity [7]. A non-significant increase in heart rate occurred with the addition of CO to the hot environment, but this could be a result of anxiety due to subjects not being "blind" to the test conditions.

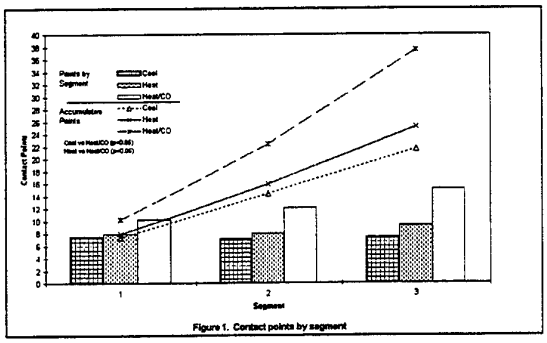

Figure 1. Contact points by segment

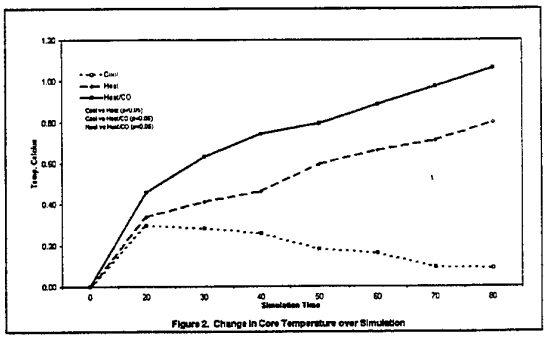

Figure 2. Change in Core Temperature over Simulation

CONCLUSION

The results indicated that a combination of heat and CO produced a larger decrement in performance in comparison to a heat only condition. However, it is difficult to determine whether the decrement occurred due to an increase in % HbCO or an increase in stored body heat. Due to the effect that CO has on the MSA's mental and physical performance, all International Motorsports Organizations should enforce rules that require competitors to carry equipment that reduces the CO inhaled by the MSA.

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This research protocol received approval from the Human Ethics Committee, The University of Western Australia.