



Heat exposure increases circulating fatty acids but not lipid oxidation at rest and during exercise



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ABSTRACT

Alterations in lipid oxidation during exercise have been well studied, but limited data exists on the effects of passive heat exposure and exercise in the heat on changes in lipid oxidation. This study was designed to examine: (1) the effects of heat exposure on lipid metabolism during passive heating and subsequent exercise in the heat by focusing on changes in whole-body lipid oxidation and plasma lipid concentrations, and (2) the effects of extended passive pre-heating on exercise performance in the heat. Male participants ($n=8$) were passively heated for 120 min at 42 °C, then exercised on a treadmill in the same temperature at 50% $\dot{V}O_{2max}$ for 30 min (HEAT). This same procedure was followed on a separate occasion at 23 °C (CON). Results showed that lipid oxidation rates were not different between HEAT and CON during passive heating or exercise. However, non-esterified fatty acid (NEFA) concentrations were significantly higher following passive heating (618 μM , 95% CI: 479–757) compared to CON (391 μM , 95% CI: 270–511). The same trend was observed following exercise (2036 μM , 95% CI: 1604–2469 for HEAT and 1351 μM , 95% CI: 1002–1699). Triacylglycerol, phospholipid and cholesterol levels were not different between HEAT and CON at any point. Four of 8 participants could not complete 30 min of exercise in HEAT, resulting in a 14% decline in total external work. Rate of perceived exertion over the final 5 min of exercise was higher in HEAT (9.5) than CON (5). We conclude that: (1) heat exposure results in increased circulating NEFA at rest and during exercise without changes in whole-body lipid utilization, and (2) passive pre-heating reduces work capacity during exercise in the heat and increases the perceived intensity of a given workload.

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1. Introduction

Research investigating heat-induced changes in metabolism has generally been focused on changes occurring during exercise, specifically with regards to carbohydrate (CHO) metabolism. This focus on CHO metabolism is not surprising as this fuel plays an important role in sustaining energy demands under various conditions while representing only ~1% of total reserves (Weber and Haman, 2005). In contrast to CHO, lipids can account for as much as ~95% to 98% of total energy reserves and play a substantial role in sustaining ATP production during prolonged aerobic activity (Jeukendrup, 2003). Despite the important role of both of these fuels, very little is known about changes in lipid metabolism

during passive heating or exercise in hot conditions. During passive heat exposure, non-esterified fatty acids (NEFA) have been shown to increase above baseline values or thermoneutral conditions, suggesting that lipid metabolism is altered (Eddy et al., 1976; Yamamoto et al., 2003). Exercise in hot conditions have been demonstrated to lead to an increased reliance on CHO oxidation, based on repeated observations of accelerated muscle glycogen utilization (Febbraio et al., 1994; Fink et al., 1975; Jentjens et al., 2002; Starkie et al., 1999), higher plasma and skeletal muscle lactate concentrations (Dolny and Lemon, 1988; Febbraio et al., 1994; Fink et al., 1975; Hargreaves et al., 1996), and a higher respiratory exchange ratio (Dolny and Lemon, 1988; Febbraio et al., 1994; Hargreaves et al., 1996; Young et al., 1985). In addition, it has been suggested that this increase in CHO metabolism during exercise is associated with a decrease in the contribution of lipid oxidation (Febbraio, 2000; Fink et al., 1975). Therefore, during prolonged passive heat exposure and subsequent exercise, the purpose of this study was to determine the effects of heat

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exposure on changes in oxidative fuel selection and plasma lipid concentrations. More specifically, this study sought to quantify whole body lipid, CHO and protein oxidation rates, as well as triglyceride (TG), NEFA, phospholipid (PL) and cholesterol (CHOL) levels in non-heat acclimatized men exposed passively to 42 °C for 2 h before exercising at the same temperature for 30 min at 50% $\dot{V}O_{2\max}$. Based on non-published preliminary experiments from our laboratory, we predicted that heat exposure would lead to an increase in CHO contribution while levels of circulating lipids would be increased in comparison to levels observed under normothermic conditions. In addition, as a secondary objective, this study will assess the effects of 2 h of passive heat exposure on exercise performance. It is established that performance during endurance exercise at a specific intensity is reduced during heat exposure (Arngrimsson et al., 2004; MacDougall et al., 1974; Morris et al., 2005). In addition, active or passive pre-heating to a core temperature of ~37.8 to 38.0 °C prior to exercise also reduces time to exhaustion (Arngrimsson et al., 2004; Gregson et al., 2002). Here, we anticipated that heat exposure (passive and active) will impede the completion of a 30 min run at 50% $\dot{V}O_{2\max}$ even if water loss is compensated.

2. Methods

2.1. Participants

Eight healthy, non-heat acclimatized active males volunteered for this study, which conformed to the standards set by the latest revision of the Declaration of Helsinki and was approved by the Health Sciences Ethics Committee of the University of Ottawa. Written informed consent was obtained from all participants. Exclusion criteria were; heat acclimatized (e.g. outdoor workers), smokers, and/or physically active less than 3 days/week. Anthropometric measurements (height, weight, percent body fat) and maximal oxygen consumption (Bruce Ramp treadmill protocol) were obtained prior to the first experimental session (Table 1).

2.2. Experimental Protocol

Each subject participated in two experimental trials, separated by at least 7 days. The trials consisted of a 90 min baseline period in ambient temperature, followed by 120 min at rest and 30 min exercise at 50% $\dot{V}O_{2\max}$ at either 42 °C (HEAT) or 23 °C (CON). The order of the trials was randomly assigned in a balanced, cross-over design. Experiments were conducted between 8 h00 and 13 h00. Participants were asked to refrain from consuming caffeine or alcohol for 12 h, and to avoid heavy physical activity for 48 h prior to the experiments. The last evening meal was standardized (~900 kcal, ~51% CHO, ~27% lipids, ~22% proteins). Participants were instructed to drink at least 1 L of water the evening before the trial, and to continue drinking water the morning of the trial to ensure they were well hydrated prior to the start of heat exposure. Participants reported to the laboratory at 8 h00 after a 12–14 h fast. Care was taken to minimize thermal stress between awakening and the start of the experiment (avoiding exposure to heat or cold, only very low-intensity exercise when traveling from home to the laboratory).

Upon arrival at the laboratory, participants were instrumented with skin temperature transducers, an esophageal thermocouple and heart rate monitor while wearing shorts and a t-shirt. Participants were then asked to void their bladder. To start the experimental trial, participants sat quietly for 90 min at ambient temperature (24 ± 0.5 °C, $36 \pm 4\%$ RH) for baseline measurements. At the end of the baseline period, participants again voided their bladder and nude weight was recorded. Participants were then

Table 1
Physical characteristics of subjects ($n=8$)

Age (years)	25 (23–27)
Body mass (kg)	76.2 (71.1–81.4)
Height (cm)	181 (177–185)
Body surface area (m ²)	1.96 (1.86–2.05)
Percent body fat (%) ^a	12 (10–15)
$\dot{V}O_{2\max}$ (ml · min ⁻¹ · kg ⁻¹) ^b	56 (52–60)

Values are means with 95% confidence interval (CI) in parentheses.

^a Underwater weighing.

^b Bruce Ramp treadmill protocol.

transferred for 120 min to a thermal chamber ($t=0$) at either 42 ± 0.3 °C, $24 \pm 3\%$ RH (HEAT) or at 23 ± 0.4 °C, $35 \pm 5\%$ RH (CON). During HEAT, participants also donned a sauna suit with elastic waist, wrists, neck and ankles (Training Sauna Suit, TKO Sports Group, Houston, TX, USA) to minimize evaporative heat loss. Throughout the passive period, participants consumed 1.5 L of water to replenish fluids lost through sweating. Heart rate and thermal responses were measured throughout the baseline and passive periods. Metabolic and ventilation measurements were recorded every 30 min. Blood samples were drawn prior to (Baseline), midway (T_{60}) and after (T_{120}) the passive period to determine changes in NEFA, TG, PL and CHOL concentrations. After 120 min, participants removed the sauna suit and towed off, then exited the thermal chamber for a maximum of 5 min while nude weight was recorded. Participants then returned to the thermal chamber and walked on a treadmill for 30 min or until exhaustion. Speed was set at 3.5 miles per hour and the treadmill incline was adjusted to a pre-determined level equivalent to 50% of the participant's $\dot{V}O_{2\max}$. Metabolic data, heart rate, thermal response and ventilation were recorded throughout the exercise period and a 10-point category scale (Borg, 1982) was used every 5 min to determine the participants' rate of perceived exertion (RPE). At the end of exercise, nude weight was again recorded and a final blood sample was drawn.

2.3. Thermal response

Changes in heat production (\dot{H}) were calculated by indirect calorimetry and corrected for protein oxidation (see below). Esophageal (T_{es}) and mean skin temperature (T_{skin}) were monitored continuously throughout the baseline period and experimental trial. Core temperature was measured using a pediatric probe (Mon-a-therm general purpose, Mallinckrodt Medical Inc., St. Louis, MO, USA) and skin temperature was measured using skin transducers (Concept Engineering, Old Saybrook, CT). Measures of skin temperature were collected from 12 skin sites, and average skin temperature was calculated based on the following proportions: forehead 7%, chest 9.5%, biceps 9%, forearm 7%, abdomen 9.5%, lower back 9.5%, upper back 9.5%, front calf 8.5%, back calf 7.5%, hamstrings 9.5% and hand 4% (Hardy and Dubois, 1938). The esophageal probe was inserted through the nose and the tip of the thermocouple placed at the level of the left atrium, or to a depth of one-quarter the standing height of the subject (Mekjavic and Rempel, 1990).

2.4. Cardiorespiratory response

Heart rate (HR) was measured using a Polar heart rate monitor (Polar FS2C Fitness Heart Rate Monitor System, Polar USA, Lake Success, NY, USA) and was recorded every 5 min during the baseline and passive periods, and every 1 min during exercise.

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