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RESEARCH ARTICLE

Running in the heat similarly reduces lipid oxidation and peak oxygen consumption in trained runners and inactive individuals

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Abstract

This study compared oxygen consumption and substrate oxidation while exercising in hot and temperate conditions in individuals with different physical activity statuses (i.e., inactive individuals vs. trained runners). Ten inactive individuals (IA: 26 ± 6 yr; 79.1 ± 14.1 kg; 40.7 ± 5.1 mL·kg^{-1}·min^{-1}) and 10 trained runners (TR: 25 ± 6 yr; 69.5 ± 9.1 kg; 63.1 ± 5.1 mL·kg^{-1}·min^{-1}) completed two incremental exercise tests (4-min stages) until exhaustion in temperate (TEMP: 18.7 ± 0.1°C; 43.2 ± 4.1% relative humidity) and hot (HOT: 34.4 ± 0.2°C and 42.6 ± 1.6% relative humidity) conditions. Expired gas and blood lactate concentrations were measured at the end of each stage. Peak oxygen consumption similarly decreased in HOT compared with TEMP for IA and TR [−13.2 ± 4.5% vs. −15.2 ± 7%; $P = 0.571$; effect size (ES) = 0.25]. In HOT compared with TEMP, lipid oxidation, from 30% to 70% of peak oxygen consumption ($\dot{V}O_{2peak}$), was reduced for both groups (IA: $P = 0.023$, ES = 0.43; TR: $P < 0.001$, ES = 0.72), whereas carbohydrate oxidation was increased for TR ($P = 0.011$; ES = 0.45) but not for IA ($P = 0.268$; ES = 0.21). Core temperature was different between conditions for TR (higher in HOT, $P = 0.017$; ES = 0.66) but not for IA ($P = 0.901$; ES = 0.25). Despite reduced physiological capacities in IA, both populations demonstrated reductions in lipid utilization and peak oxygen consumption in hot compared with temperate conditions. However, the increased carbohydrate oxidation in HOT for TR was not observed in IA, potentially explained by lower thermal strain.

NEW & NOTEWORTHY This study shows that lipid oxidation and oxygen consumption are similarly affected by heat exposure in trained runners and inactive individuals. Carbohydrate oxidation and core temperature are greater in hot conditions in trained runners but not in inactive individuals. A lower metabolic heat production in inactive individuals for a similar relative intensity compared with trained runners could explain these differences in core temperature.

metabolic flexibility; substrate oxidation; temperature; training status

INTRODUCTION

Substrate oxidation during exercise can be influenced by exercise intensity, environmental conditions, and fitness (1–3). With increasing exercise intensity, the contributions of carbohydrate and lipid are altered (1). At low intensities, lipids are the main fuel, but decrease in contribution as exercise intensity increases (4) becoming null when the respiratory exchange ratio exceeds one (i.e., carbon dioxide production exceeds oxygen consumption). Substrate utilization can also be affected by environmental conditions (2, 5). In hot conditions compared with temperate conditions, carbohydrate and glycogen utilization are increased during both prolonged, matched-intensity (2, 6, 7) and incremental exercise (5, 8), although the core temperature does not necessarily increase. Specifically, heat exposure leads to an earlier shift (i.e., at lower intensity) from lipid to carbohydrate use in healthy, trained athletes (5).

In addition, the fitness status of participants may also affect substrate contribution (3). Increased training status improves metabolic flexibility (i.e., capacity of individuals to switch between lipids and carbohydrates), increasing the ability to oxidize lipids even at relatively high intensities (3, 9), therefore reducing glycogen utilization during prolonged exercise. In healthy/trained individuals, these factors [increased lipid oxidation (4), lower reliability on carbohydrate metabolism (i.e., reduced glucose and glycogen utilization), and a delayed lactate accumulation at low intensity (10)] are attributed to an improved mitochondria density and function (1). In sedentary individuals, previous studies have demonstrated lower/impaired metabolic flexibility (11) often observed through insulin resistance, hyperlipidemia, decreased clearance of dietary lipids, and reduced fasting and post-prandial lipid oxidation, resulting in greater carbohydrate oxidation (11). In the long term,

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there is evidence of mitochondrial dysfunction and poor glucose uptake by muscle cells (12) often characterized by metabolic diseases such as type 2 diabetes mellitus and metabolic syndrome (13). Therefore, in less-active people (with or without metabolic syndrome), the switch between lipid and carbohydrate utilization can occur at lower intensities resulting in greater carbohydrate use at a given work rate (3).

Exercising in hot environments (≥30°C) is becoming increasingly common due to global warming (14), increasing the exposure of less-fit individuals to exercise-heat stress. Indeed, poor training status could compound the effects of hot environmental conditions, combining to exacerbate the impairment of metabolic flexibility. A reduction in lipid utilization and greater reliance on carbohydrate may contribute to an increased risk of metabolic diseases such as obesity or diabetes (11). Although existing research has primarily focused on the effects of heat exposure on energy metabolism in active individuals and athletes, there is a notable gap in understanding the impact on inactive individuals, who may be increasingly exposed to hot conditions due to global warming. To our knowledge, no studies have investigated the effects of exercising in hot environments on substrate use across a wide range of intensities in this population. Understanding these effects could provide valuable insight into the potential health implications of climate-induced changes in exercise conditions.

The purpose of the present study was to compare oxygen consumption and substrate utilization in individuals with different training statuses (i.e., trained runners vs. inactive individuals) across a range of exercise intensities in both temperate and hot environmental conditions. It was hypothesized that exercising with heat exposure combined with a poor training status would greatly reduce lipid oxidation and oxygen consumption in inactive individuals compared with trained runners.

MATERIALS AND METHODS

Participants completed a preliminary trial (PRE) followed by two experimental trials, consisting of an incremental test until exhaustion in either a temperate environment (TEMP: 18.7 ± 0.1°C and 43.2 ± 4.1% relative humidity; RH) or a hot environment (HOT: 34.4 ± 0.2°C and 42.6 ± 1.6% RH).

Participants

Ten trained non-acclimated runners (TR; 2 females) and 10 inactive non-acclimated participants (IA; 3 females) took part in this study. TR participated in running sessions at least three times a week and were classified as at least "Tier 2" on the Participant Classification Framework (15). IA did not meet World Health Organization minimum activity guidelines [i.e., completing <150-min moderate-intensity activity or <75 min of vigorous-intensity activity a week; (16)]. Females were tested during the follicular phase (i.e., *days 1–13* from the start of menstruation, as determined by a menstrual cycle questionnaire) to minimize potential variations in core temperature and substrate utilization (17, 18). All participants gave verbal and written informed consent and were healthy and free from metabolic diseases as assessed by a health screen questionnaire. The study received ethical approval from the Loughborough University Ethics Reviews Sub-Committee (LEON 16408). The sample size was estimated using data from previous experiments, investigating similar interventions with reported effect sizes ≥1.3 and involving participants with different training statuses (3, 9) and different environmental conditions (5). With an α of 0.05 and a statistical power of 0.8, it was estimated that at least 10 participants per group would be required to detect differences in substrate oxidation between trials.

Preliminary Visit (PRE)

During the first visit, body mass (AFW-120K, Adam Equipment Co., Milton Keynes, UK), height (Seca 216, Hamburg, Germany), and skinfold thickness (Harpenden Skinfold Caliper, Baty International, Burgess Hill, UK) at biceps, triceps, sub-scapula, and supra-iliac were measured (19). Participants completed a submaximal incremental test, on a motorized treadmill (mercury h/p/cosmos, Nußdorf, Germany), composed of 4-min stages at progressive running speeds until a heart rate (HR) >160 beats·min^{-1} was elicited (measured using Polar M400, Polar, Kemple, Finland). Expired gas was collected into Douglas bags during the final minute of each stage. After a short period of rest (~10–15 min), participants ran to volitional exhaustion at the final speed of the submaximal test using a ramped protocol (incline increased 1%·min^{-1}). An expired gas sample was collected during the final minute of the exercise.

Experimental Visits

In the 24 h preceding the first trial, participants recorded all food and drink consumed and physical activity performed and then replicated this prior to the second trial (verbally checked). They arrived at the laboratory at the same time of day, 3 h after consuming a standardized meal, providing 1.5 g carbohydrate·kg body mass^{-1} and 8 mL fluid·kg body mass^{-1} (consisting of Nutri-grain cereal bars, Kellogg's, UK; orange juice, Tesco, Welwyn Garden City, UK; and water). Participants sat for 10 min in the environmental chamber, with expired gas and blood lactate measured in the last 2 min. Participants then walked on the treadmill for 10 min at 3 km·h^{-1} (IA participants) or 4 km·h^{-1} (TR). Expired gas and blood glucose and lactate concentrations were measured at the end of this period.

Participants then started the incremental exercise on a treadmill to maximal volitional fatigue. The skin test was composed of 4-min stages (to allow a steady state to be reached in both groups, (20), interspaced by 30-s rest for capillary blood sampling. The starting speed for IA was set to 4 and 5 km·h^{-1} for females and males, respectively, with an increment of 1 km·h^{-1} every 4 min (21). TR started at 5.5 and 7 km·h^{-1} for females and males, respectively, with an increment of 1.5 km·h^{-1} every 4 min (21). Expired gas was collected during the final minute of each stage. HR, core and skin temperature, and blood lactate and glucose concentrations were measured at the end of each stage. Rating of perceived exertion [RPE; 6–20 scale (22)] and thermal sensation [RTS; −10 to +10 scale (23)] were recorded at exhaustion. Appropriate convective cooling was applied to participants

using two fans (one aimed at the torso, one aimed at the lower body) positioned 1 m in front of participants. To ensure appropriate airflow, as treadmill velocity increased, the wind speed generated by the fans (0–5.5 $m \cdot s^{-1}$) was increased.

Gas Exchanges

Expired gas collected into Douglas bags was analyzed for oxygen and carbon dioxide concentrations (Servomex 1400 Oxygen and Carbon Dioxide Gas Analyzer; Servomex, Crowborough, UK). Gas volume (Harvard dry gas meter; Harvard Apparatus Ltd., Edenbridge, UK) and temperature (RS Pro digital thermometer; RS Components, Corby, UK) were measured and volume was corrected to standard temperature and pressure, dry. To correct oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) values, ambient air was measured simultaneously with expired gas samples (24).

Core and Skin Temperature

Participants ingested a radiotelemetry pill (BodyCap, e-Celsius Performance, Hérouville Saint-Clair, France), before going to bed (trial start time of 8:00–11:00 AM) or upon waking up (trial start time of 1:00–3:00 PM), to continuously record gastrointestinal temperature (T_{core}). Skin temperature measures were collected from the upper arm, chest, thigh, and calf using programmable iButtons (DS1922L, iButtonLink Technology, WI). Mean skin temperature (T_{skin}) was subsequently calculated using the weighted average of the four sites (25).

Blood Measurements

Blood lactate and glucose concentrations were analyzed (Biosen C-Line Glucose and Lactate Analyzer, EKF Diagnostics, Cardiff, UK) from 20 μL fingertip capillary samples collected pre-exercise (i.e., after rest period and warm-up) and at the end of each stage.

Substrate Calculations

The respiratory exchange ratio (RER) was calculated using the following equation:

$$\text{RER} = \dot{V}CO_2(\text{L} \cdot \text{min}^{-1})/\dot{V}O_2(\text{L} \cdot \text{min}^{-1}). \quad (1)$$

Carbohydrate and lipid oxidation rates were assessed using *Eqs. 3* and *4* (respectively) proposed by Frayn (26):

$$\text{Carbohydrate oxidation}(\text{g} \cdot \text{min}^{-1}) = 4.55 \cdot \dot{V}CO_2(\text{L} \cdot \text{min}^{-1}) - 3.21 \cdot \dot{V}O_2(\text{L} \cdot \text{min}^{-1}) - 2.87 \cdot 0.01 \quad (2)$$

$$\text{Lipid oxidation}\ (\text{g} \cdot \text{min}^{-1}) = 1.67 \cdot \dot{V}O_2(\text{L} \cdot \text{min}^{-1}) - 1.67 \cdot \dot{V}CO_2(\text{L} \cdot \text{min}^{-1}) - 1.92 \cdot 0.01. \quad (3)$$

Substrate partitioning via non-protein respiratory quotient (NPRQ) was calculated using equations proposed by Zarins et al. (27):

$$\text{NPRQ}(\%) = \left[\dot{V}CO_2(\text{L} \cdot \text{min}^{-1}) - (0.01 \cdot 4.89)\right] / \left[\dot{V}O_2(\text{L} \cdot \text{min}^{-1}) - (0.01 \cdot 6.04)\right] \quad (4)$$

$$\%\text{Carbohydrate oxidation}\ (\%) = \left[(\text{NPRQ} - 0.707)/0.293\right] \cdot 100 \quad (5)$$

$$\%\text{Lipid oxidation}\ (\%) = (100 - \%\text{Carbohydrate oxidation}). \quad (6)$$

The last RER value considered for Carbohydrate oxidation, Lipid oxidation, %Carbohydrate oxidation, and %Lipid oxidation analyses was the first value ≥1.0 but lower than 1.05 (3). For RER values ≥1.0, Lipid oxidation and %Lipid oxidation were considered to be null.

Data and Statistical Analyses

Data are presented as means ± SD. To allow comparisons between participants with different absolute speeds, all data are presented according to relative exercise intensity, which is expressed as a percentage of each individual's maximal peak oxygen uptake (i.e., regardless of the condition), employing linear interpolation (i.e., to ensure that the data points align accurately with the specified intensities). Substrate utilization was measured between 30% and 70% $\dot{V}O_{2peak}$ as substrate equations may be compromised at intensities below 25% and above 75% $\dot{V}O_{2peak}$ (5). Statistical analyses were conducted using SPSS software (version 29, IBM, SPSS Inc., Chicago). Shapiro–Wilk tests were used to check the normality of the dependent variables. No violation of normality was observed. Linear mixed models (LMMs) were performed to examine the differences between groups and conditions. Fixed (group, condition, and intensity) and random (participants) effects for LMM were fit for each dependent variable. The most appropriate model was chosen using the smallest Hurvich and Tsai's criterion (Akaike information criterion corrected; AICC) in accordance with the principle of parsimony. Pairwise comparisons of estimated marginal means were performed using the Holm–Bonferroni adjustment to control for multiple comparisons (28). Hedges' *g* effect size (ES) was calculated and interpreted for differences as "trivial" (<0.20), "small" (0.20–0.59), "moderate" (0.6–1.19), "large" (1.20–1.99), or "very large" (>2.0). Statistical significance was set at $P < 0.05$.

RESULTS

Participants Characteristics

Physical activity (i.e., duration and number of sessions) during a week was higher for TR compared with IA ($P < 0.001$; ES = 2.54; Table 1). Height, age, and body mass were not different between TR and IA, but body mass index and sum of skinfolds were higher in IA compared with TR ($P = 0.014$; ES = 1.18 and $P < 0.001$; ES = 1.56, respectively; Table 1). $\dot{V}O_{2peak}$ (i.e., maximal value of peak oxygen consumption measured among the three sessions, which occurred during the preliminary session or TEMP) was higher in TR than in IA ($P < 0.001$; Table 1). The maximal speed reached during experimental trials (i.e., regardless of the condition) was higher in TR than in IA (17.2 ± 1.4 vs. 9.7 ± 1.5 $km \cdot h^{-1}$; $P < 0.001$; ES = 4.64). Regardless of the conditions, incremental test duration was greater for TR than for IA ($P < 0.001$). Regardless of the populations, incremental test duration was greater in TEMP compared with HOT; for

Table 1. *Participant characteristics*

Group	IA (n = 10)	TR (n = 10)
Age, yr	26 ± 6	25 ± 6
Height, m	1.74 ± 0.08	1.76 ± 0.11
Body mass, kg	79.1 ± 14.1	69.5 ± 9.3
Body mass index, kg·m^{-2}	26.0 ± 3.7	22.4 ± 1.9*
Sum of skinfolds, mm	70.7 ± 24.1	30.0 ± 10.8*
$\dot{V}O_{2peak}$[a], mL·kg^{-1}·min^{-1}	40.7 ± 5.1	63.1 ± 5.1*
Duration of moderate or vigorous-intensity aerobic physical activity per week, min	30 ± 24	504 ± 22*
Number of sessions of moderate or vigorous-intensity aerobic physical activity per week	1 ± 1	8 ± 2*

IA, inactive individuals; TR, trained runners. [a]$\dot{V}O_{2peak}$, maximal value of peak oxygen consumption measured among the three sessions. *P < 0.05: TR different from IA.

TR (HOT: 30.7 ± 3.2 min vs. TEMP: 34.8 ± 3.5 min; P < 0.001) and IA (HOT: 24.0 ± 7.6 min vs. TEMP: 26.8 ± 8.2 min; P = 0.001). RPE was not different at exhaustion between TEMP and HOT for TR (HOT: 19.4 ± 0.5 vs. TEMP: 19.3 ± 0.7) and IA (HOT: 17.8 ± 1.7 vs. TEMP: 17.6 ± 1.3).

Oxygen Consumption

Oxygen consumption, observed through $\dot{V}O_2$ slope relative to speed (Fig. 1), was reduced in HOT compared with TEMP (Condition effect; P = 0.005; ES = 0.62) and in TR compared with IA (Group effect; P < 0.001; ES = 1.85). No Condition·Group interaction was observed (P = 0.878). $\dot{V}O_{2peak}$ measured during PRE and TEMP was not different for TR (PRE: 63.1 ± 5.1 vs. TEMP: 62.1 ± 7 mL·kg^{-1}·min^{-1}; P = 1.0; ES = 0.15) or IA (40.7 ± 5.1 vs. 38.5 ± 4.9 mL·kg^{-1}·min^{-1}; P = 0.200; ES = 0.44). $\dot{V}O_{2peak}$ was lower in HOT compared with TEMP for TR (63.1 ± 5.1 vs. 52.5 ± 5.8 mL·kg^{-1}·min^{-1}; P < 0.001; ES = 1.43) and for IA (38.5 ± 4.9 vs. 33.3 ± 4.5 mL·kg^{-1}·min^{-1}; P < 0.001; ES = 1.06). $\dot{V}O_{2peak}$ reduction between TEMP and HOT represented −15.2 ± 7% for TR and −13.2 ± 4.5% for IA and was not different between groups (P = 0.571; ES = 0.25). HR was greater in HOT compared with TEMP (P < 0.001; ES = 0.64) with no Group·Condition interaction observed (P = 0.506).

Substrate Utilization and Metabolic Flexibility

Maximal lipid oxidation (i.e., highest value of lipid oxidation measured during experimental trials; MLO) was higher

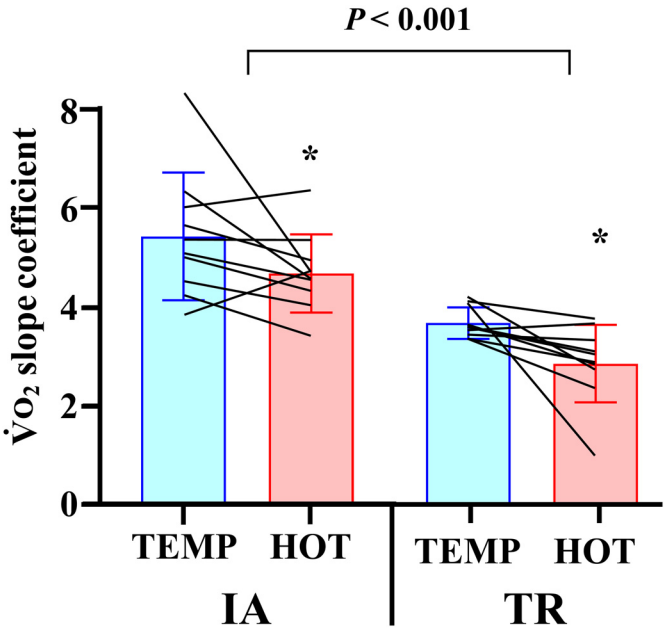

Figure 1. $\dot{V}O_2$ slope coefficient relative to speed during an incremental running exercise performed by IA and TR, in TEMP (blue) and HOT (red). *Main condition effect. HOT, hot conditions; IA, inactive individuals; TEMP, temperate condition; TR, trained runners; $\dot{V}O_2$, oxygen consumption.

in TR than in IA regardless of the environmental condition (P < 0.001; ES = 1.66) (Fig. 2). HOT led to significantly reduced MLO for TR (0.54 ± 0.21 vs. 0.45 ± 0.15 g·min^{-1}; P = 0.003; ES = 0.60) but not for IA (0.25 ± 0.11 vs. 0.23 ± 0.08 g·min^{-1}; P = 0.495; ES = 0.23). Carbohydrate oxidation at submaximal intensities (i.e., 30%–70% $\dot{V}O_{2peak}$; Fig. 2) was greater in HOT compared with TEMP in TR (P = 0.013; ES = 0.45) but not different between conditions for IA (P = 0.276; ES = 0.21). For both groups, lipid oxidation was lower in HOT compared with TEMP (TR: P < 0.001; ES = 0.72; IA: P = 0.044; ES = 0.43). RER was higher in IA than TR at any intensity (P = 0.036; ES = 0.69), and higher in HOT compared with TEMP (P < 0.001; ES = 0.40) with no Group·Condition interaction observed (P = 0.109) (Table 2).

Figure 3 presents the relative contribution of lipid and carbohydrate oxidation across the exercise intensity spectrum

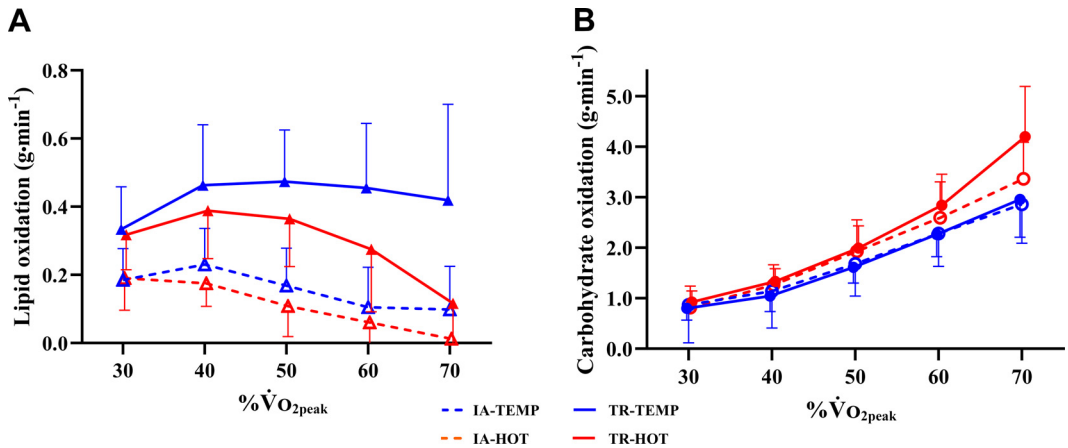

Figure 2. Lipid (*A*; triangle) and carbohydrate (*B*; circle) oxidation against %$\dot{V}O_{2peak}$ in IA (dashed line) and TR (solid line), in TEMP (blue) and HOT (red). HOT, hot conditions; IA, inactive individuals; TEMP, temperate condition; TR, trained runners; $\dot{V}O_{2peak}$, peak oxygen consumption.

Table 2. *Heart rate and respiratory exchange ratio over %$\dot{V}O_{2peak}$ during an incremental running exercise performed in TEMP and HOT*

	Group	Condition	%$\dot{V}O_{2peak}$ 30	40	50	60	70	Group Effect	Condition Effect	Group·Condition	Exhaustion
HR, beats·min^{-1}	IA	TEMP	101 ± 7	112 ± 6	126 ± 8	141 ± 8	157 ± 9	F (1,16) = 3; P = 0.109; ES = 0.28	F (1,164) = 385; **P < 0.001**; ES = 0.64	F (1,164)=0; P = 0.506	189 ± 9
		HOT	112 ± 8	127 ± 9	144 ± 10	159 ± 11	177 ± 13				190 ± 9
	TR	TEMP	91 ± 10	103 ± 12	117 ± 15	134 ± 15	152 ± 15				187 ± 11
		HOT	101 ± 9	113 ± 11	138 ± 11	157 ± 9	174 ± 10				189 ± 9
RER	IA	TEMP	0.90 ± 0.15	0.90 ± 0.10	0.93 ± 0.06	0.96 ± 0.04	0.98 ± 0.05	F (1,16) = 5; **P = 0.036**; ES = 0.69	F (1,164) = 29; **P < 0.001**; ES = 0.40	F (1,164) = 3; P = 0.109	1.06 ± 0.05
		HOT	0.89 ± 0.08	0.91 ± 0.04	0.96 ± 0.05	0.99 ± 0.04	1.03 ± 0.03				1.06 ± 0.04
	TR	TEMP	0.85 ± 0.04	0.84 ± 0.04	0.87 ± 0.03	0.90 ± 0.03	0.92 ± 0.05				1.03 ± 0.06
		HOT	0.86 ± 0.03	0.87 ± 0.03	0.90 ± 0.03	0.94 ± 0.04	1.00 ± 0.06				1.06 ± 0.06

Data are means ± standard deviation. Values in bold indicate a significant effect with $P < 0.05$. %$\dot{V}O_{2peak}$, relative intensity compared with the maximal value of peak oxygen consumption measured among the three sessions; HOT, hot conditions; HR, heart rate; IA, inactive individuals; RER, respiratory exchange ratio; TEMP, temperate condition; TR, trained runners.

during incremental running exercise, for IA and TR. Reduced lipid oxidation and increased carbohydrate oxidation were observed in HOT compared with TEMP (Condition effect; $P < 0.001$; ES = 0.48). A Group effect was observed with reduced lipid oxidation and increased carbohydrate oxidation in IA compared with TR (Group effect; $P = 0.025$; ES = 0.70) but no Group·Condition interaction was reported ($P = 0.083$).

Blood lactate concentrations were higher in IA than TR at any intensity ($P = 0.003$; ES = 0.56), and higher in HOT compared with TEMP ($P < 0.001$; ES = 0.47) with no Group·Condition interaction observed ($P = 0.909$) (Table 3). Blood glucose concentrations were higher in HOT compared with TEMP ($P < 0.001$; ES = 0.61) with no Group·Condition interaction observed ($P = 0.082$) (Table 3).

Blood lactate concentrations were greater in HOT than in TEMP regardless of the group ($P < 0.001$; ES = 0.56). TR demonstrated reduced blood lactate concentrations compared with IA ($P < 0.001$; ES = 0.47).

Core and Skin Temperature

T_{core} was greater in HOT compared with TEMP for TR ($P < 0.001$; ES = 0.66) but not for IA ($P = 0.283$; ES = 0.25) (Table 4). At exhaustion, no differences between HOT and TEMP were reported in T_{core} for TR. T_{skin} was greater in HOT than in TEMP for TR ($P < 0.001$; ES = 7.31) and for IA ($P < 0.001$; ES = 7.56). At exhaustion, RTS was greater in HOT than in TEMP for both groups (6 ± 1 vs. 1 ± 2 in HOT and TEMP, respectively, for TR and vs. 5 ± 2 vs. 1 ± 3 for IA in HOT and TEMP, respectively; $P < 0.001$).

DISCUSSION

The aim of the study was to compare substrate oxidation while exercising in hot and temperate conditions in individuals with different physical activity and training statuses (i.e., inactive individuals vs. trained runners). Despite differences in physiological capacities between inactive individuals and trained runners, both groups exhibited important alterations in oxygen consumption and metabolic flexibility in a hot environment. However, differences observed in trained runners for carbohydrate oxidation in a hot environment were not observed in inactive individuals.

As expected, running speed at exhaustion and peak oxygen consumption were lower in inactive compared with trained runners. In addition, trained runners demonstrated enhanced lipid oxidation capacities (e.g., an MLO twice as high as that of inactive individuals) and delayed lactate accumulation compared with inactive individuals, regardless of environmental conditions. Greater oxidative and enzymatic capacities (mainly linked to greater mitochondrial abundance and function (1); with endurance training likely explain improved capacities to oxidize lipids (4), lower reliability on carbohydrate metabolism (i.e., reduced glucose and glycogen utilization) and a delayed lactate accumulation at low intensity (10). Consequently, and as previously reported (29), in inactive individuals, there is a greater reliance on carbohydrate-derived fuels compared with lipids.

Exercise in hot conditions (34°C and 40% of RH) led to a significant decrease in peak oxygen consumption regardless of the training status of participants despite similar blood

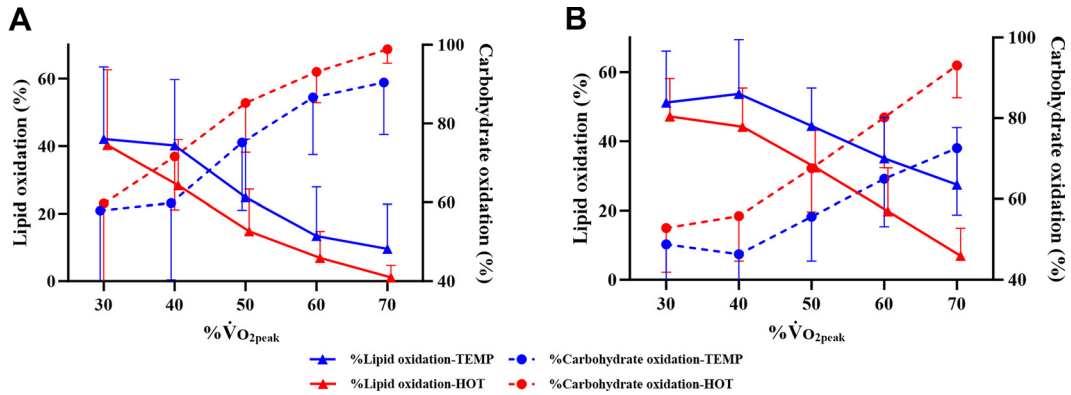

Figure 3. Relative contribution of lipids (Lipid oxidation; solid line and triangle) and carbohydrates (Carbohydrate oxidation; dashed line and circle) to total energy expenditure over %$\dot{V}O_{2peak}$ in IA (*A*) and TR (*B*), in TEMP (blue) and HOT (red). HOT, hot conditions; IA, inactive individuals; TEMP, temperate condition; TR, trained runners; $\dot{V}O_{2peak}$, peak oxygen consumption.

lactate concentration, heart rate, and perceptual responses (i.e., RPE) at exhaustion. Interestingly, the extent of the reduction was similar for trained runners (−15.2 ± 7.0%) and inactive individuals (−13.2 ± 4.5%) despite significant differences in peak oxygen consumption in temperate conditions (40.7 ± 5.1 $mL \cdot kg^{-1} \cdot min^{-1}$ for inactive individuals and 63.1 ± 5.1 $mL \cdot kg^{-1} \cdot min^{-1}$ for trained runners). These reductions corroborate previous studies in healthy and trained individuals (30–33) in hot environments (40°C, 46°C, 49°C, and 35°C, respectively). However, many studies reported smaller reductions (~3%–8% (34–36); 37–48°C). The variation in results observed may be attributed to several factors, such as the severity of the heat stress, the duration of the exposure (including passive or active preheating), the specificities of the test or premature cessation of the exercise due to thermal discomfort. In the present study, extended heat exposure, facilitated by a combination of whole body preheating (i.e., 10 min of passive exposure followed by 10 min of active exposure) and the prolonged test duration (~25–30 min), likely contributed to a shift in physiological mechanisms (i.e., earlier substrate oxidation crossover point and lactate accumulation; described in the same section below) and consequently the significant reductions observed in maximal capacities.

Contrary to previous studies, which attributed changes greater than 10% in maximal oxygen consumption to elevated core temperature (37), no significant difference in gastro-intestinal temperature was observed between environmental conditions for inactive individuals in this study. Although significant condition effects were noted for skin temperature for both groups, these findings do not support the theory that a decrease in maximal oxygen consumption occurs only when both core and skin temperature are elevated (33). Consequently, it appears that gastro-intestinal temperature is not the primary driver for oxygen consumption and substrate use. The thermoregulatory network should be considered as a central integrative circuit that incorporates information from both peripheral (e.g., skin) and central (e.g., viscera and brain) thermoreceptors (38). As thermal strain develops (shown here by increased skin temperature), detection by the central nervous system leads to increased sympathetic neural activity (38), which is supported by increased blood glucose concentrations in hot conditions. This is triggered by the redistribution of blood flow, necessary to meet the demands of both active muscles and thermoregulation processes [i.e., to dissipate heat by increasing skin blood flow; (39)]. Despite the increased heart rate in hot conditions, at either relative or absolute intensity, which confirms the heightened sympathetic outflow [(40); in addition to the increased blood glucose in hot conditions], these regulatory mechanisms can result in compromised muscle perfusion, leading to impaired oxygen delivery/consumption in exercising muscles, especially at high intensity. As a result, the maximal heart rate, the rise in blood lactate concentration, and earlier use of carbohydrate/crossover between lipids and carbohydrates occur at lower intensities in hot conditions, as was observed in the present study in both groups, leading to decreased exercise performance/capacity. Therefore, sustained peripheral signaling may be sufficient to impact oxygen consumption and lead to metabolic shifts (i.e., increased use of carbohydrates and increased reliance on anaerobic metabolism as evidenced by increased lactate) in the absence of central temperature changes. Interestingly, the main condition effect reported for oxygen consumption at submaximal intensities (with the absence of core temperature changes) seems to support the importance of peripheral signals. Due to the short duration of the exercise, it is unlikely that exercise-related dehydration would have significantly amplified the effects of heat stress (41).

As reported in previous studies involving healthy and trained individuals (5), a significant decrease in lipid oxidation occurred during exercise in hot conditions among trained runners. Notably, as observed by Ruíz-Moreno et al. (8), these effects appear to be more pronounced at higher exercise intensities, particularly beyond 60% of $\dot{V}O_{2peak}$. This downward shift in lipid oxidation, coupled with the concurrent increase in carbohydrate oxidation at the same relative intensity, as demonstrated during constant-paced exercise (7), indicates earlier reliability (i.e., at lower intensity) on carbohydrate-derived fuels. Interestingly, significant reductions in lipid oxidation were also observed in inactive individuals, despite their inherently lower lipid oxidation capacities due to their poor training status. However, no changes in carbohydrate oxidation were

Table 3. *Blood lactate and glucose concentrations over %$\dot{V}O_{2peak}$ during an incremental running exercise performed in TEMP and HOT*

			%$\dot{V}O_{2peak}$								
	Group	Condition	30	40	50	60	70	Group Effect	Condition Effect	Group· Condition	Exhaustion
Lactate, mmol·L^{-1}	IA	TEMP	1.4 ± 0.5	1.4 ± 0.5	1.8 ± 0.5	2.5 ± 0.6	3.4 ± 0.9	F (1,16) = 12;	F (1,164) = 65;	F (1,164) = 0;	8.9 ± 2.8
		HOT	1.3 ± 0.2	1.6 ± 0.4	2.3 ± 0.6	3.3 ± 0.7	5.0 ± 1.0	**P = 0.003**;	**P < 0.001**;	P = 0.909	8.2 ± 2.8
	TR	TEMP	1.3 ± 0.3	1.3 ± 0.3	1.3 ± 0.4	1.3 ± 0.5	1.8 ± 0.6	ES = 0.56	ES = 0.47		9.4 ± 2.2
		HOT	1.2 ± 0.4	1.2 ± 0.4	1.4 ± 0.3	2.1 ± 0.7	4.2 ± 1.4				9.0 ± 2.2
Glucose, mmol·L^{-1}	IA	TEMP	4.1 ± 0.6	4.0 ± 0.5	4.0 ± 0.3	4.0 ± 0.3	3.9 ± 0.4	F (1,16) = 2;	F (1,164) = 47;	F(1,164) = 3;	4.5 ± 1.0
		HOT	4.3 ± 0.6	4.2 ± 0.6	4.2 ± 0.4	4.1 ± 0.4	4.2 ± 0.6	P = 0.136;	**P < 0.001**;	P = 0.082	4.5 ± 1.0
	TR	TEMP	4.2 ± 0.3	4.2 ± 0.2	4.1 ± 0.3	4.2 ± 0.4	4.2 ± 0.6	ES = 0.57	ES = 0.61		5.7 ± 0.9
		HOT	4.5 ± 0.3	4.4 ± 0.2	4.4 ± 0.3	4.6 ± 0.3	4.8 ± 0.4				6.0 ± 1.4

Data are means ± standard deviation. Values in bold indicate a significant effect with $P < 0.05$. %$\dot{V}O_{2peak,}$ relative intensity compared with the maximal value of peak oxygen consumption measured among the three sessions; HOT, hot conditions; IA, inactive individuals; TEMP, temperate condition; TR, trained runners.

Table 4. *Core and skin temperature over %$\dot{V}O_{2peak}$ during an incremental running exercise performed in TEMP and HOT*

			%$\dot{V}O_{2peak}$								
	Group	Condition	30	40	50	60	70	Group Effect	Condition Effect	Group·Condition	Exhaustion
T_{core}, °C	IA	TEMP	37.3 ± 0.4	37.5 ± 0.2	37.5 ± 0.2	37.5 ± 0.2	37.6 ± 0.1	F (1,16) = 3;	F (1,164) = 31;	F (1,164) = 11;	38.1 ± 0.2
		HOT	37.4 ± 0.2	37.5 ± 0.3	37.5 ± 0.2	37.6 ± 0.2	37.8 ± 0.2	P = 0.123;	**P < 0.001**;	**P < 0.001**†	38.1 ± 0.3
	TR	TEMP	37.1 ± 0.4	37.1 ± 0.4	37.2 ± 0.4	37.4 ± 0.3	37.6 ± 0.3	ES = 0.32	ES = 0.48		38.5 ± 0.3
		HOT	37.2 ± 0.2	37.2 ± 0.2	37.5 ± 0.1	37.7 ± 0.2	38.1 ± 0.2				38.6 ± 0.3
ΔT_{core}, °C	IA	TEMP	-0.1 ± 0.5	0.0 ± 0.1	0.1 ± 0.2	0.1 ± 0.2	0.2 ± 0.2	F (1,16) = 11;	F (1,164) = 47;	F (1,164) = 12;	0.7 ± 0.3
		HOT	0.0 ± 0.2	0.1 ± 0.2	0.2 ± 0.3	0.2 ± 0.3	0.4 ± 0.4	**P = 0.005**;	**P < 0.001**;	**P = 0.021**†	0.7 ± 0.5
	TR	TEMP	0.0 ± 0.1	0.0 ± 0.1	0.1 ± 0.2	0.3 ± 0.2	0.6 ± 0.2	ES = 0.67	ES = 0.56		1.4 ± 0.4
		HOT	0.1 ± 0.2	0.2 ± 0.2	0.4 ± 0.3	0.7 ± 0.3	1.1 ± 0.3				1.6 ± 0.5
T_{skin}, °C	IA	TEMP	28.5 ± 1.1	28.3 ± 1.0	28.3 ± 1.0	28.2 ± 1.0	28.2 ± 1.0	F (1,16) = 11;	F (1,164)=6,227;	F (1,164) = 14;	28.5 ± 1.0
		HOT	34.7 ± 0.8	34.8 ± 0.7	34.9 ± 0.7	34.8 ± 0.6	34.9 ± 0.5	**P = 0.002**;	**P < 0.001**;	**P < 0.001**$†	35.0 ± 0.5$
	TR	TEMP	29.3 ± 0.9	29.2 ± 0.9	29.4 ± 1.0	29.8 ± 1.1	30.0 ± 1.2	ES = 0.29	ES = 6.47		30.3 ± 1.3
		HOT	35.2 ± 0.4	35.3 ± 0.4	35.6 ± 0.3	35.7 ± 0.3	35.8 ± 0.2				35.8 ± 0.5†

Data are means ± standard deviation. Values in bold indicate a significant effect with $P < 0.05$. %$\dot{V}O_{2peak}$, relative intensity compared with the maximal value of peak oxygen consumption measured among the three sessions; HOT, hot conditions; IA, inactive individuals; T_{core}, core temperature; TEMP, temperate condition; TR, trained runners; T_{skin}, skin temperature; ΔT_{core}, difference between measured core temperature and core temperature at rest. $HOT different from TEMP for IA. †HOT different from TEMP for TR.

observed in inactive individuals compared with trained runners. These differences, in addition to the absence of differences in maximal lipid oxidation between conditions for inactive individuals, could be explained by a lack of change in T_{core} in inactive individuals. As an increase in core temperature has been suggested as a driver for cardiovascular and metabolic changes in hot environments (33), differences in substrate use between groups could be explained by this factor. Moreover, an increase in T_{core} is often accompanied by a rise in muscle temperature, which can directly stimulate muscle glycogenolysis (2), possibly explaining the absence of differences in carbohydrate oxidation in inactive individuals. When comparing equivalent relative intensities between inactive individuals and trained runners, the difference in speed (i.e., reduced in inactive individuals) resulted in a lower production of metabolic heat (Supplemental File S1; Fig. S1), combined with a greater test duration for trained runners, thus limiting the increase in gastrointestinal temperature and effects on substrate utilization. Despite these differences, relative contributions of lipid and carbohydrate oxidation did not appear to differ between groups.

For both populations, the decline in lipid oxidation could be attributed to several (interlinked) factors. The increase in skin temperature and gastro-intestinal temperature, although not observed for inactive individuals, is likely to affect thermoregulatory control by the central nervous system and, as a consequence, increase sympathetic activation (38) and consequently circulating glucose. Although the greater sympathetic response mainly impacts redistribution of blood flow, necessitated by the demands of both active muscle and thermoregulation (39), it also leads to the increased hepatic glucose production and the reduction in the use of lipids (42). Moreover, earlier lactate accumulation was observed in hot conditions, probably linked to reduced oxygen delivery (39), and suggests a downregulation in lipid use. Indeed, elevated lactate concentrations inhibit lipolysis (43) and function of carnitine palmitoyl transferase 1, the transporter of free fatty acids into mitochondria (44). Early lactate accumulation, and related effects, could reinforce the hypothesis of a greater effect of heat stress on substrate oxidation at high intensity (8).

Limitations

The limitations of our study mainly reside in the indirect nature of the assessment of substrate utilization. An ideal study would involve a direct examination of muscle glycogen utilization, as well as free fatty acids and lactate oxidation, through muscle biopsies and tracer methodologies. Furthermore, carbohydrate and lipid oxidation are presented until exhaustion, but this was at high speed, and consequently high intensity. The increased production of H^+ ions (confirmed by lactate accumulation) would have been buffered by bicarbonate, producing non-metabolic CO_2, and possibly overestimating carbohydrate oxidation (9). In the present study, blood variables such as triglyceride and catecholamine concentrations were not measured. The inclusion of these measurements could have offered valuable insight into the underlying mechanisms influencing the outcomes of this investigation, such as carbohydrate oxidation (2).

Applications

Inactive individuals exhibit poorer metabolic flexibility compared with trained runners, and this disparity is observed regardless of environmental conditions. These findings underscore the potential implications of global warming-induced temperature increases, as they may hinder individuals' ability to efficiently oxidize lipids. Investigations are warranted to explore whether these findings extend to individuals with impaired metabolic flexibility stemming from metabolic or genetic diseases [e.g., type 2 diabetes, obesity (3), and sickle cell disease (45)].

Conclusions

The present study reveals significant alterations in lipid utilization and metabolic flexibility in both inactive individuals and trained runners when exposed to hot environments, although these are greater for trained runners. Moreover, peak oxygen consumption was consistently reduced in hot conditions compared with temperate conditions across both groups. These results underscore the profound influence of environmental factors on metabolic responses during exercise, regardless of the population, highlighting the importance of further investigation in this area.

DATA AVAILABILITY

Data will be made available upon reasonable request.

SUPPLEMENTAL MATERIAL

Supplemental Files S1–S3: https://doi.org/10.6084/m9.figshare.27630087.v2.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

L.M. and S.A.M. conceived and designed research; L.M., H.Z.M., A.H., and T.G.C. performed experiments; L.M. analyzed data; L.M. interpreted results of experiments; L.M. prepared figures; L.M. drafted manuscript; L.M., H.Z.M., A.H., T.G.C., L.T., L.J.J., and S.A.M. edited and revised manuscript; L.M., H.Z.M., A.H., T.G.C., L.T., L.J.J., and S.A.M. approved final version of manuscript.

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