

Effects of reduced ambient temperature on fat utilization during submaximal exercise

JOSEPH D. LAYDEN, MARK J. PATTERSON, and MYRA A. NIMMO

Strathclyde Institute for Biomedical Sciences, University of Strathclyde, Glasgow, UNITED KINGDOM

ABSTRACT

LAYDEN, J. D., M. J. PATTERSON, and M. A. NIMMO. Effects of reduced ambient temperature on fat utilization during submaximal exercise. *Med. Sci. Sports Exerc.*, Vol. 34, No. 5, pp. 774–779, 2002. **Purpose:** The influence of cold air exposure on fuel utilization during prolonged cycle exercise was investigated. **Methods:** Nine male subjects cycled for 90 min in ambient temperatures of -10°C , 0°C , 10°C , and 20°C . External work performed between conditions was constant. Mean oxygen consumption ($\dot{V}\text{O}_2$) over the 90 min in the 20°C trial corresponded to $64 \pm 5.8\%$ $\dot{V}\text{O}_{2\text{peak}}$. **Results:** Although mean skin temperature was different between trials ($P < 0.05$), rectal temperatures were not different. At -10°C and 0°C , the respiratory exchange ratio was higher compared with 10°C and 20°C (0.98 ± 0.01 and 0.97 ± 0.01 vs 0.92 ± 0.01 and 0.91 ± 0.01 ; $P < 0.05$). The associated rates of fat oxidation were lower at -10°C and 0°C compared with 10°C and 20°C (0.15 ± 0.06 and 0.17 ± 0.06 vs 0.35 ± 0.06 and 0.40 ± 0.04 $\text{g}\cdot\text{min}^{-1}$; $P < 0.05$). Blood glycerol was lower at -10°C and 0°C compared with 20°C ($P < 0.05$); mean values were 0.13 ± 0.0 , 0.13 ± 0.0 , and 0.18 ± 0.0 $\text{mmol}\cdot\text{L}^{-1}$ for the -10°C , 0°C , and 20°C trials, respectively. Mean $\dot{V}\text{O}_2$ was lower in the -10°C trial than the 20°C trial (2.53 ± 0.06 vs 2.77 ± 0.09 $\text{L}\cdot\text{min}^{-1}$; $P < 0.05$). Mean blood glucose concentrations were lower at -10°C than 20°C (4.9 ± 0.2 vs 5.3 ± 0.1 $\text{mmol}\cdot\text{L}^{-1}$; $P < 0.05$). Although plasma epinephrine concentrations were greater during the 20°C trial compared with all other trials ($P < 0.05$), plasma norepinephrine did not differ between trials. **Conclusion:** The diminished fat oxidation at colder temperatures potentially reflects a reduction in lipolysis and/or mobilization of FFA or impairment in the oxidative capacity of the muscle. **Key Words:** COLD, FAT OXIDATION, CATECHOLAMINES, OXYGEN CONSUMPTION, SKIN TEMPERATURE

During cold exposure at rest, despite an overall increase in lipid and carbohydrate (CHO) oxidation resulting from increased thermogenesis and shivering, the percentage contribution from lipids decrease (34). However, during submaximal exercise in cold air, whole body fat utilization has been reported to be elevated (7,20,33), unchanged (15,27,32), or reduced (11). Because the response to cold exposure is different at rest than during exercise and is dependent on the intensity and duration of the exercise and on the nature and severity of the exposure (17,24), the metabolic responses observed cannot be generalized. Hurley and Haymes (20) suggest the differing fuel utilization patterns are related to a decrease in both the skin and rectal temperatures, whereas a reduction in skin temperature alone causes no effect. Although studies have been conducted at $\leq 0^{\circ}\text{C}$ (32,33), they have combined whole body precooling before the exercise, and although not reported, this potentially resulted in a diminished core temperature and possibly initiated the shivering response. Thus, it is not known what influence exposure to temperatures of $\leq 0^{\circ}\text{C}$ will have on whole body fat utilization during submaximal exercise when shivering is inhibited via maintenance of the core temperature elevation associated with exercise.

The aim of this study was to investigate the influence of ambient temperatures of $\leq 0^{\circ}\text{C}$ on whole body fat utilization

during submaximal exercise where the intensity is sufficient to maintain core temperature elevation and to alleviate shivering. The metabolic responses will be compared with exercise at 10°C , which has been reported to be the optimal temperature at which to perform exercise (15), and 20°C , which is often regarded as a control environment.

MATERIALS AND METHODS

Nine recreationally active men volunteered as subjects in the present study. Their physical characteristics were (mean \pm SD): body mass, 81.1 ± 9.5 kg; age, 26.1 ± 2.8 yr; body fat (8), $13.6 \pm 3.5\%$ fat; and $\dot{V}\text{O}_{2\text{peak}}$ 51.4 ± 5.6 $\text{mL}\cdot\text{kg}\cdot\text{min}^{-1}$. The University of Strathclyde Ethics Committee approved all procedures and subjects provided written informed consent.

Experimental protocol. Before the main experimental trials, subjects performed a continuous incremental cycling test to volitional exhaustion on a mechanically braked cycle ergometer (Monark 864, Varberg, Sweden), at a room temperature of $20.8 \pm 1.5^{\circ}\text{C}$, to determine peak oxygen uptake ($\dot{V}\text{O}_{2\text{peak}}$). Expired air was collected and analyzed via an on-line system (Oxycon Gamma, Mijnhardt, The Netherlands).

Each subject completed five bouts of 90-min submaximal cycling. During the first visit, subjects cycled in a 20°C environment at a workload corresponding to $64 \pm 5.8\%$ $\dot{V}\text{O}_{2\text{peak}}$. This trial served to familiarize subjects with the experimental procedures and confirmed that they could complete the 90-min exercise bout. In the remaining four trials, subjects cycled at the workload set in the familiarization trial at an ambient temperature (T_a) of $-9.9 \pm 0.1^{\circ}\text{C}$,

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0.0 ± 0.1°C, 10.0 ± 0.1°C, or 20.0 ± 0.1°C, presented in a balanced order 5–7 d apart. Relative humidity was 50.0 ± 0.0%, 49.9 ± 0.3%, 38.4 ± 2.1%, and 42.7 ± 4.8% for the 20°C, 10°C, 0°C, and –10°C trials respectively. Air velocity was constant at 0.35 ± 0.1 m·s⁻¹ between trials.

Subjects reported to the laboratory, between 9 a.m. and 10 a.m., in a fasted state. In the 24-h period before each trial, subjects abstained from alcohol, caffeine, and vigorous physical activity. To minimize differences in muscle metabolite concentration, individuals recorded their dietary intake, fluid consumption, and physical activity during the 48-h period preceding the first experimental trial and were asked to repeat this for each subsequent trial. To ensure euhydration, subjects consumed 1 L of water the night before and 0.5 L 2 h before each experiment.

On arrival at the laboratory, subjects were weighed nude and inserted a rectal thermistor 12 cm beyond the anal sphincter (Grant Instruments Ltd., Cambridge, UK). An indwelling cannula (Venflon 18G, BOC Ohmeda, Helsingborg, Sweden) was inserted into an antecubital forearm vein and was kept patent by flushing with saline after each sample collection. Subjects rested in a seated position for 30 min during which they were instrumented, and a resting blood sample was drawn to assess metabolic homogeneity before exercise between conditions. After the resting blood sample, subjects entered the environmental chamber (SANYO Gallenkamp PLC, Loughborough, UK) and commenced cycling immediately. Every 15 min during exercise, a blood sample was drawn, expired gas was collected, and heart rate (HR; Polar Vantage NV, Kempele, Finland), skin temperature, and rectal temperature (T_{re}) were recorded.

Skin temperatures were measured at eight sites by using surface thermistors (Grant Instruments Ltd., UK) attached with a single layer of waterproof tape. Mean skin temperature (\bar{T}_{sk}) was calculated as (after ISO-9886; International Standards Organization, 1992); $\bar{T}_{sk} = 0.07 \cdot \text{forehead} + 0.175 \cdot \text{right scapula} + 0.175 \cdot \text{left upper chest} + 0.07 \cdot \text{right upper arm} + 0.07 \cdot \text{left forearm} + 0.05 \cdot \text{left hand} + 0.19 \cdot \text{right anterior thigh} + 0.2 \cdot \text{left calf}$. All thermistors were calibrated (± 0.1°C) against a certified reference mercury thermometer (Zeal, London, UK). Temperatures were recorded using a portable data logger (1206 Series Squirrel, Grant Instruments Ltd.).

The mouthpiece was inserted 4 min before expired gas being collected into a Douglas bag over a 1-min period. The gas was immediately analyzed for O₂ and CO₂ concentrations (Servomex 1440, Crowborough, UK). The expired volume was determined using a dry gas meter (Harvard Apparatus Ltd., Kent, UK). The respiratory exchange ratio (RER) and $\dot{V}O_2$ was then used to estimate CHO and fat oxidation and total energy expenditure (13).

Subjects wore the same shorts and shoes in all trials. Mittens and earmuffs were worn to protect the extremities from cold injury. An additional skin thermistor was placed on the subject's fingertip to monitor peripheral skin temperature. Cold exposure was discontinued if skin temperature decreased below 8°C for more than 15 min or below 6°C at any time.

Preliminary trials indicated that temperatures below 0°C affected the workload of the bicycle. To overcome this, an

insulating hood was built around the flywheel and drive chain to maintain the temperature above 0°C. During the –10°C and 0°C trials, the hood maintained the ambient temperature around the flywheel at 10.7 ± 1.9 and 19.9 ± 2.0°C, respectively.

Analytical techniques. From an 8-mL blood sample, 5 mL was dispensed into a chilled tube containing lithium heparin, EGTA, and reduced glutathione and centrifuged (Eppendorf 1440, Hamburg, Germany) at 4000 rpm at 4°C for 10 min. The plasma was then frozen at –80°C for subsequent determination of norepinephrine (NE) and epinephrine (Epi) (Waters HPLC with electrochemical detection, Waters HPLC Ltd., Elstree, UK) by using the method described by Goldstein et al. (16). The remaining blood was dispensed into a chilled tube containing K-EDTA. From this, duplicate 100- μ L aliquots were deproteinized in 0.4 mM perchloric acid (PCA), centrifuged, and later analyzed for lactate (25), glycerol (2), and glucose (glucose oxidase method, Boehringer Mannheim GmbH Diagnostica, Lewes, UK). Whole blood was used to determine hematocrit (microcapillary technique) and hemoglobin (cyanmethemoglobin method), with changes in plasma volume being estimated using the method of Dill and Costill (6). The remaining blood was centrifuged and the resultant plasma used for the determination of free fatty acid (FFA, enzymatic colorimetric method, Boehringer Mannheim GmbH Diagnostica) concentrations. The coefficient of variation, calculated according to the formula of Essen (10), was 1.7, 2.8, 1.8, 2.8, 2.4, and 1.7% for the lactate, glycerol, glucose, FFA, Epi, and NE assays, respectively.

On occasions where it was difficult to obtain a blood sample due to cold-induced vasoconstriction, an occlusion cuff was applied to the arm for 1–2 min. To determine the effect of the cuff on blood constituents, a small substudy was conducted on five subjects. At rest, hematocrit was elevated with the application of the cuff (44.0 ± 0.7 vs 45.2 ± 1.0%; $P = 0.06$; $N = 5$). However, during exercise, the reduction in plasma volume was not significantly altered by the application of the cuff. With and without stasis, values at 15 or 30 min of exercise were –4.57 ± 2.78% vs –2.54 ± 3.15% and –1.6 ± 1.32% vs –0.22 ± 0.95%, respectively. Subsequently, blood metabolite concentrations were also unaffected by application of the cuff during exercise.

Statistical analysis. A two-way (trial by time) repeated measures ANOVA was used to compare data collected during the exercise period. When analysis revealed a significant main effect, one-way ANOVA tests were performed to determine at which time points an effect was observed between trials. Where appropriate, a Tukey *post hoc* test was used to locate the differences. In all cases, significance was taken at $P < 0.05$. Comparative data are presented as mean ± SEM unless otherwise stated.

RESULTS

The mean oxygen uptake over the 90-min exercise period was 2.77 ± 0.17, 2.67 ± 0.12, 2.61 ± 0.12, and 2.53 ± 0.12 L·min⁻¹ for the 20°C, 10°C, 0°C, and –10°C

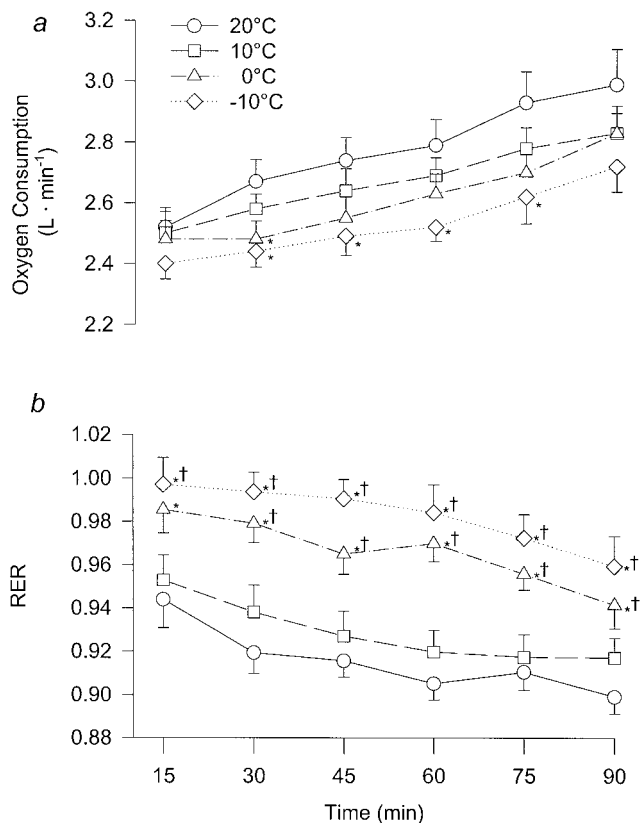


FIGURE 1—Oxygen consumption (a) and respiratory exchange ratio (RER; b) during 90 min of cycle exercise in ambient temperatures of 20°C, 10°C, 0°C, and -10°C. Values are mean \pm SEM ($N = 9$). * and † indicate significantly different ($P < 0.05$) from 20°C and 10°C, respectively.

trials, respectively, with the -10°C trial being significantly less than 20°C ($P < 0.05$; Fig. 1a). As ambient temperature decreased, RER increased with 0°C and -10°C being significantly higher than both the 20°C and 10°C trials ($P < 0.05$; Fig. 1b). RER decreased over the 90 min in all trials. HR was significantly higher at all time points in the 20°C trial compared with all other trials ($P < 0.05$). Mean HR values are presented in Table 1. Fat oxidation was significantly lower in the 0°C and -10°C trial than the 10°C and 20°C trials ($P < 0.05$), whereas CHO oxidation exhibited the converse response (Fig. 2). Total energy expenditure was not different among trials. Values were 298 ± 8 , 288 ± 5 , 286 ± 6 , and 285 ± 11 kJ for the 20°C, 10°C, 0°C, and -10°C trials, respectively.

Rectal temperature increased from resting values in all trials with no significant differences being observed among trials nor at any time point ($P < 0.05$). \bar{T}_{sk} was significantly different between all trials and at all time points ($P < 0.05$). Mean values for T_{re} and \bar{T}_{sk} are presented in Table 1. Mean sweat loss over the 90 min was not significantly different

between 0°C and 10°C. All other comparisons of trials showed significant differences ($P < 0.05$; Table 1).

The resting levels of blood glucose, lactate, glycerol, and plasma FFA were not significantly different between trials. The mean change in plasma volume during 90 min of exercise did not significantly differ between trials. Plasma FFA progressively increased in all trials with no significant differences being noted between trials (Fig. 3a). Blood glycerol was significantly greater in the 20°C trial than both 0°C and -10°C by 60 min of exercise ($P < 0.05$; Fig. 3b). Blood glucose concentrations were significantly lower at all time points in the -10°C trial compared with the 20°C trial ($P < 0.05$; Fig. 3c). Blood lactate was not significantly different among trials at any time (Table 2).

Plasma Epi concentrations were significantly greater at all time points in the 20°C trial compared with 0°C and -10°C ($P < 0.05$). Plasma NE concentrations were not significantly different between trials at any time point. Mean plasma Epi and NE concentrations are presented in Table 2.

DISCUSSION

This study was unique in that it was the first to investigate the influence of ambient temperature on whole body fuel utilization at temperatures $\leq 0^\circ\text{C}$, while exercising at an intensity sufficient to maintain rectal temperature. The main finding is that whole body fat oxidation is reduced when exercising at 0°C and -10°C compared with both 10°C and 20°C.

The reduced fat oxidation in the colder environments, as indicated by the increased RER values, may be due to a number of factors. These include diminished lipolysis, a reduction in FFA mobilization and/or transport, decreased FFA uptake into the muscles, or a reduction in the oxidative capacity of the muscle. Although it is recognized that changes in circulating glycerol levels do not solely reflect the release of FFA and glycerol from the triacylglycerol stores (9,23), the two parameters are highly correlated (14,22) and have been shown to be an accurate reflection of whole body lipolysis (28). Thus, the reduction of blood glycerol noted during the 0°C and -10°C trials in the current study is interpreted as reflecting reduced lipolysis. Equally so, the lower skin temperature at colder ambient temperatures may have caused a reduced subcutaneous adipose tissue blood flow (ATBF; 1), thus inhibiting the supply of albumin, which would consequently inhibit both lipolysis and removal of FFA from adipose tissue (3,4,30). Any increase in the FFA/albumin ratio and subsequent increase in FFA concentration will favor reesterification at the expense of mobilization into the blood (31). An accumula-

TABLE 1. Mean values for rectal (T_{re}) and skin temperatures (\bar{T}_{sk}), heart rate (HR) and mean sweat loss during 90-min cycle exercise at a range of ambient temperatures.

	20°C	10°C	0°C	-10°C
T_{re} (°C)	37.94 ± 0.18	37.86 ± 0.24	37.93 ± 0.26	37.85 ± 0.57
\bar{T}_{sk} (°C)	31.54 ± 0.35	$26.74 \pm 0.38^*$	$22.7 \pm 0.57^{*\dagger}$	$18.27 \pm 0.76^{*\ddagger}$
HR (beats \cdot min $^{-1}$)	147.3 ± 3.8	$136.9 \pm 2.6^*$	$134.5 \pm 2.3^*$	$132 \pm 2.2^*$
Sweat loss (% body mass)	1.7 ± 0.5	$1.1 \pm 0.4^*$	$0.8 \pm 0.2^*$	$0.6 \pm 0.1^{*\ddagger}$

* , † , ‡, indicate significantly different ($P < 0.05$) when compared with 20°C, 10°C, and 0°C trials, respectively; values are mean \pm SEM.

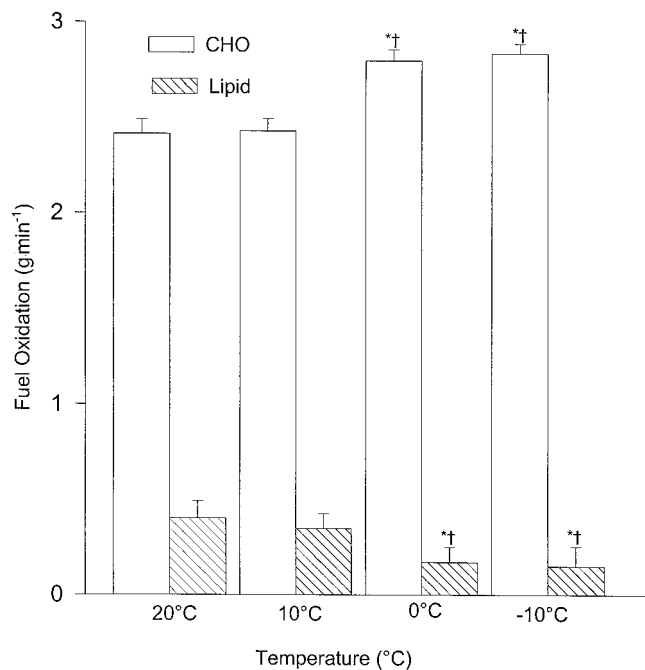


FIGURE 2—Mean rate of lipid and carbohydrate (CHO) oxidation during 90 min of cycle exercise in ambient temperatures of 20°C, 10°C, 0°C, and -10°C. Values are mean \pm SEM ($N = 9$). * and † indicate significantly different ($P < 0.05$) from 20°C and 10°C trials, respectively.

tion of FFA in the peripheral adipose tissue has also been proposed in studies where blood flow to the adipose tissue is reduced because of diversion of the blood to active muscle (28,35). A reduced lipolysis, as evidenced by the reduced glycerol concentrations, together with the evidence of a reduced fat utilization (RER data) and no change in circulating FFA concentrations suggest a possible reduction in the uptake of FFA into the muscle (21). Alternatively, it may be that the oxidative capacity of the muscle has been impaired in the cold, with a consequent reduction in oxidation of both circulating and intramuscular fat. This latter hypothesis is supported by the finding of Febbraio et al. (11), that despite exercising for 40 min at an intensity corresponding to 65% $\dot{V}O_{2max}$, the exercising muscle temperature is significantly lower at 3°C than 20°C, although in that study T_{re} was also decreased.

Thus, despite rectal temperature being maintained in this study, it is possible that low skin temperatures and potentially low muscle temperatures (11) resulted in a reduction in the rate of lipolysis, mobilization/transport, uptake, and oxidation of fat. The contribution that each of these make requires further investigation.

In the current study, significant reductions in fat oxidation were only observed at $\leq 0^\circ\text{C}$ and below. Thus, our data would support those studies reporting no differences in fuel utilization when comparisons are made between 20°C and temperatures $>0^\circ\text{C}$ (15,27). It is unclear why Febbraio et al. (11) found a reduction in fat utilization when 3°C was compared with 20°C, but it may be related to variables such as high fitness status and body composition of the subjects involved. Although Sink et al. (32) utilized 0°C, subjects

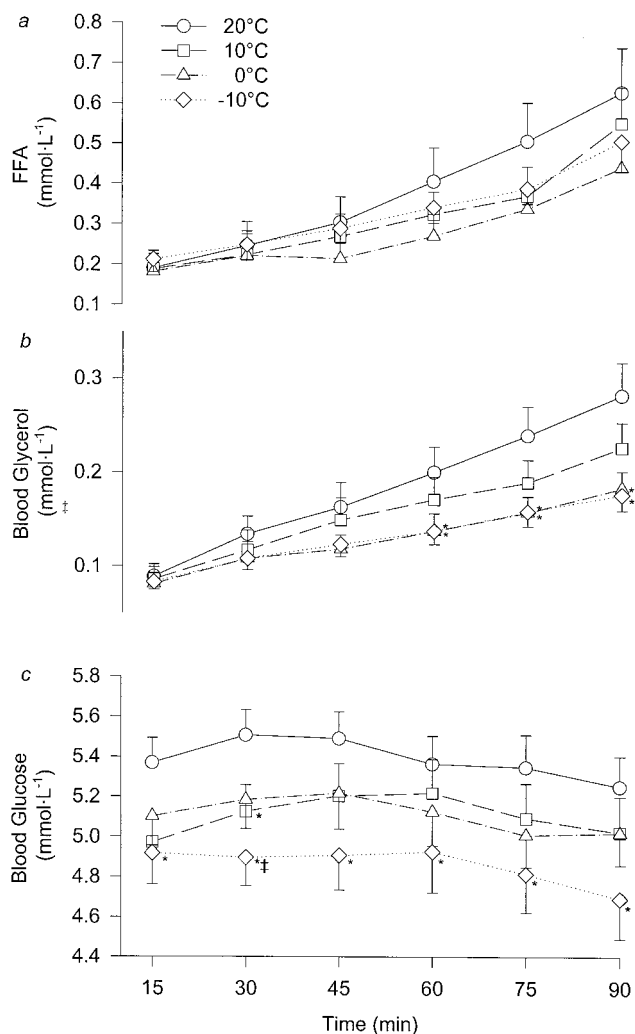


FIGURE 3—Plasma free fatty acids (FFA; a), blood glycerol (b), and blood glucose (c) concentrations during 90 min of cycle exercise in ambient temperatures of 20°C, 10°C, 0°C, and -10°C. Values are mean \pm SEM ($N = 9$). * and † indicate significantly different ($P < 0.05$) from 20°C and 0°C trials, respectively.

wore t-shirts throughout the exercise period and consequently the \bar{T}_{sk} in the 0°C trial ($26.2 \pm 0.4^\circ\text{C}$) was similar to that observed during the 10°C trial ($26.7 \pm 0.38^\circ\text{C}$) in the current investigation. Therefore, if reductions in lipolysis and ATBF, due to the reduced skin temperature, determine the availability of fats it is not surprising that Sink et al. (32) reported no difference in substrate utilization between 22°C and 0°C. Timmons et al. (33) reported greater fat utilization when subjects exercised at -10°C compared with 22°C. The enhanced fat utilization may have been related to changes in core temperature as the subjects experienced a combination of 15-min seated rest and a low exercise intensity (100 W) in the -10°C environment. However, core temperature was not reported in that study. The current study was the first to exercise subjects at T_a above and below 0°C without the confounding effect of decreased core temperature.

Lower rates of fat utilization have been attributed to lower plasma Epi concentrations as the hydrolysis of triglycerides

TABLE 2. Mean values for blood lactate, plasma epinephrine, and plasma norepinephrine concentrations during 90-min cycle exercise at a range of ambient temperatures.

	20°C	10°C	0°C	-10°C
Lactate (mmol·L ⁻¹)	1.8 ± 0.2	1.5 ± 0.2	1.5 ± 0.2	1.8 ± 0.2
Epinephrine (nmol·L ⁻¹)	1.0 ± 0.3	0.7 ± 0.1*	0.6 ± 0.2*	0.6 ± 0.2*
Norepinephrine (nmol·L ⁻¹)	9.2 ± 1.4	6.2 ± 0.7	6.1 ± 0.7	8.6 ± 1.6

* indicates significantly ($P < 0.05$) when compared with 20°C trial; values are mean ± SEM.

involves catecholamine-mediated lipolysis (5) and Epi increases ATBF and the efflux of FFA from adipose tissue (30). However, plasma Epi concentrations were similar in the 10°C, 0°C, and -10°C environments, yet fat utilization was significantly diminished at 0°C and -10°C when compared with 10°C. Therefore, it is more likely that the severity of cold exposure, and the subsequent vasoconstriction, was responsible for the reduction in fat utilization. The elevated Epi concentrations in the 20°C trial is potentially due to an indirect effect of the greater heat stress (18) and dehydration (19) relative to the colder trials. Elevated skin temperature in the 20°C trial reflects a redistribution of blood from the central to the peripheral circulation with a resulting increase in sympathetic nervous activity to maintain cardiac output and mean arterial pressure (MAP) (29). This is supported by the elevated HR observed during the 20°C trial

Considering that external work and total energy expenditure were unaltered between trials, it is likely that oxidation of other fuel sources compensated for the decreased fat oxidation. The derived CHO oxidation was greater in the 0°C and -10°C trials, as evidenced by the respiratory data. It is unlikely that the greater CHO oxidation reflected an elevated muscle glycogen utilization because others have reported a diminished glycolytic rate at low ambient temperatures (11,12,27). The lower blood glucose concentrations in the -10°C trial, compared with 20°C trial, may imply that blood glucose utilization was increased in the cold. However, blood glucose was not different between the 10°C and 0°C despite CHO oxidation being different between trials and therefore cannot fully explain the differ-

ences. The indirect method of measuring substrate utilization in this study does not take account of the protein sources of fuel and therefore an increased protein utilization at the colder temperatures remains a possibility.

The reduced $\dot{V}O_2$ in the -10°C trial supports the diminished fat oxidation, because more oxygen is required to oxidize fat than CHO (26). Galloway and Maughan (15) reported an inverse relationship between oxygen consumption and ambient temperature with the greatest uptake being observed in the 4°C trial, although CHO oxidation was greater in the 4°C trial when compared with the 21°C trial. However, these authors reported an unaltered HR response between trials, which is unusual. It would be expected that the peripheral vasoconstriction, associated with the lower \bar{T}_{sk} in the cold environments, would shunt more blood to the central volume (29), consequently reducing HR, as observed in the current and previous investigations (7,20,32). Therefore, it is possible that the combination of a greater $\dot{V}O_2$ and unaltered HR imply that subjects were exercising at a higher intensity in the 4°C trial reported by Galloway and Maughan (15).

In summary, our data demonstrate that when exercising in cold ambient temperatures the contribution of fats to total fuel utilization is reduced. It is suggested that potential causes are a reduced adipose tissue lipolysis and/or subcutaneous ATBF, which decreases the ability to mobilize and utilize fats.

Address for correspondence: Joseph Layden, School of Sport, Exercise, and Health Sciences, University College of Chichester, College Lane, Chichester, P019 4PE, United Kingdom; E-mail: j.layden@ucc.ac.uk.

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