Muscle Glycogen Dominates Females Fuel Usage in Moderately Intensive Endurance Exercise Even in the Cold

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Abstract
Carbohydrate metabolism has been observed to be altered in males exercising in the cold. Whether metabolism is affected in females in the cold is unknown, therefore, the present study was conducted. Female cyclists (n=11) conducted two trials of 75 min cycling (74±4% VO2max) each followed by a 4-km time-trial, at 5 ºC (Cold) and 15 ºC (Temperate), in random order. U-13Cglucose (0.12 g) was fed. Indirect calorimetry (with urea measurements) and 13CO2 (breath) and 13Cglucose (blood) enrichment were used to calculate substrate metabolism. Total energy, carbohydrate, fat, protein, muscle glycogen and liver-derived, blood glucose utilization were similar (P> 0.05) over 75 min of exercise in Cold and Temperate with muscle glycogen being the predominant energy source (3664±374, 3605±420 kJ; 2.05± 0.40, 2.13±0.25 g/min; 0.31±0.14 g/min, 0.27±0.13, 0.14± 0.05, 0.13±0.05 g/min; 1.61±0.43, 1.71 ± 0.25 g/min; 0.44 ± 0.14, 0.43 ± 0.08 g/min, resp.). Rectal temperature increased similarly (~1.5 °C). Time-trial performance wasn’t different between Cold and Temperate (419±25, 425±21 s, resp, P=0.23, 95% CI: 1.4 ± 2.4%). Similar amounts of muscle glycogen (the major fuel source) and other substrates were metabolized by women during moderately intensive (74% VO2max) exercise in cold (5 ºC) and temperate (15 ºC) environments.

ABBREVIATIONS
RER: Respiratory Exchange Ratio; VO2 max: Maximal Aerobic Capacity; Bpm: Beats Per Minute; Tc: Core (Rectal) Temperature; Tsk: Skin Temperature; RES: Respectively; W: Watt; LBM: Lean Body Mass

INTRODUCTION
Environmental temperature can affect substrate metabolism during exercise. In particular, high temperature has been consistently shown to elevate carbohydrate utilization [1], in males and females. Cold temperature has received less research attention and the limited data are conflicting and are solely from male participants. In the studies conducted on exercise in the cold an increased utilization of fat [2-5], or carbohydrate [6,7] or no effect [8] has been observed. This lack of consistency in effect on metabolism may be due, at last in part, to the fact that in these studies various exercise types, intensities, and environmental conditions were utilized. Furthermore, the presence or absence of shivering is often unreported. Although it is acknowledged that it is difficult to detect at low levels [9], shivering can have a dramatic effect on metabolism, increasing metabolic rate [9], muscle glycogenolysis and carbohydrate oxidation [10,11].

Although the paucity of research on physiological responses to cold in women has been highlighted periodically [12,13], women remain underrepresented and it cannot be assumed that females will respond similarly to males. Females generally use fat oxidation to greater extent than males during exercise at the same relative intensity [14,15], at least partly due to 17β-estradiol increasing mobilization and oxidation of fat [14-16]. However, most studies have been at intensities below the ‘crossover point’ (< 65% VO2max) where fat oxidation should predominate regardless of sex [17]. Females’ body composition...
may further make their metabolism unique in the cold. Compared with males, females have more subcutaneous fat, less lean and total mass, and a larger surface area-to-volume ratio; these factors may passively and actively affect substrate metabolism. Females may also have more brown adipose tissue (BAT), and BAT activation [18], although there is an inverse relationship of BAT activation with adiposity in response to cold exposure [19], so a net effect-if any- is difficult to predict.

Therefore, the present study was designed to provide female-specific data on the metabolic demands of moderately-intensive exercise, including muscle glycogen and liver-derived blood glucose utilization, and evaluate any effect cold may have.

**MATERIALS AND METHODS**

**Participants**

Eleven trained ($\dot{V}O_{2max}$ > 40 ml $\dot{V}O_2$/kg/min and training >1.0 h > 3x/wk) female cyclists volunteered. All were taking monophonic contraceptive pills for ≥ three months. Ethical approval and informed consent were obtained. Body fat and lean mass were estimated using multi-frequency bioimpedance (In Body 230, Biospace Co Ltd, Seoul, Korea) and skin folds [20].

**Experimental protocol**

Participants performed two, 75-min exercise trials at ~70% $\dot{V}O_{2max}$, followed by a 4-km time-trial, in 5.2 ± 0.3 °C, 92 ± 5% relative humidity (Cold) and 14.9 ± 0.2 °C, 77 ± 6% (Temperate) conditions, in randomized order. Trials were within days 3-11 of the menstrual cycle, >one month apart, in winter-autumn (mean temperature 5–13 °C). Plasma progesterone and estrogen were measured to confirm menstrual phase.

Participants arrived by 0700 h after fasting for ≥10 h, voided and fitted a heart rate monitor (Polar S810i, Polar Electro Inc., Port Washington, NY) before seated at rest for ≥10 min. Baseline body mass (shorts and bra only) and respiratory measures were collected before participants ate a standardized breakfast (time = -120); 45 g Sustagen™ Sport powder, (Nestlè, Auckland, New Zealand) and fitted a heart rate monitor (Polar S810i, Polar Electro Inc., Rotkreuz, Switzerland), plasma glucose with the hexokinase reaction (GLUC2, Roche Diagnostics GmbH, Indianapolis, USA), and respiratory water loss. Temperatures were logged every 15s through all exercise. Mean skin temperature ($T_{sk}$) was calculated from area weightings (ISO 9886, 2004). Body surface area was calculated using Du Bois and Du Bois [21]:

\[ BSA = 0.007184 \times \text{mass (kg)}^{0.425} \times \text{height (cm)}^{0.725} \]

Participants rated perceived exertion (6-20; very light to very hard), thermal sensation (1: Unbearably Cold – 13: Unbearably Hot), and thermal discomfort (1: Comfortable – 10.0: Extremely Uncomfortable) (both adapted from, [22]) before each blood sampling.

**Standardization and isotope ingestion**

Participants were required to minimize exercise to <2 h within 2 d before each trial, then avoid exercise completely within 1 d. Dietary intake was recorded for two days beforehand with instruction to avoid foods from plants high in $^{13}$C. In each trial, participants consumed 0.12 g uniformly labeled $^{13}$Cglucose (99 atom%, Isotech Inc., Miamisburg, Ohio, USA) in seven aliquots totaling 800 mL. Specifically, participants bolus-ingested 200-mL $^{13}$C-labeled water (0.04 g $^{13}$Cglucose) before the warm up ($t$ = -30), then 100-mL (0.013 g $^{13}$Cglucose) every 15 min until 60 min of exercise.

**Sampling**

Venous forearm blood samples were taken at rest ($t$ = -120) and during exercise ($t$ = 15, 30, 45, 60, 75, 83). Plasma and serum were stored at -80 °C until analyses for estrogen and progesterone, insulin and glucose (from plasma) and $^{13}$Cglucose (from serum).

Respiratory measures, heart rate, and perceptual responses were recorded for 3 min every 15 min. Rates of oxygen consumed ($\dot{V}O_2$), and carbon dioxide produced ($\dot{V}CO_2$) were measured using open-circuit spirometry (CPET COSMED srl, Rome, Italy). During the last 30 s of every 15 min, expire was collected into a latex Douglas bag $\dot{V}$ and 1-mL samples transferred in duplicate into tubes for analysis of $^{13}$C. Urea production was estimated from urine and sweat concentrations (from a plastic pouch adhered to the lower back), the volume of urine produced and estimated sweat volume. Sweat volume was estimated from body mass change, corrected for fluid intake, mass loss through gas exchange, and respiratory water loss. Temperatures were logged every 15s (Grant 1200, Squirrel, Grant Instruments Cambridge, England).

**Temperature and perceptual responses**

Core temperate ($T_c$) was measured using a rectal thermistor. Temperatures were logged (Grant 1200 series Squirrel Data Logger, Grant Instruments Cambridge, England) at 15-s intervals throughout all exercise. Mean skin temperature ($T_{sk}$) was calculated from area weightings (ISO 9886, 2004). Body surface area was calculated using Du Bois and Du Bois [21]:

Body surface area = 0.007184 x mass (kg)$^{0.425}$ x height (cm)$^{0.725}$

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**Metabolite and hormone assays**

Plasma glucose, sweat, and urine urea were analyzed spectrophotometrically (Cobas c 111, Roche Instrument Centre, Rotkreuz, Switzerland), plasma glucose with the hexokinase reaction (GLUC2, Roche Diagnostics GmbH, Indianapolis, USA), and urea with the glutamate dehydrogenase reaction (UREAL Urea/BUN, Roche Diagnostics, Basel, Switzerland). Plasma insulin was determined with electrochemiluminescence immunoassay (ECLI A Insulin, Elecsys, Roche Diagnostics, Basel, Switzerland). Coefficients of variation (CV) were <2.7%.

Catecholamines, progesterone and estradiol were analyzed using HPLC-EP, enzyme-linked immunosorbent assay (ELISA) and radioimmunoassay (Spectria E2 sensitive, Orion Diagnostics, Finland), respectively. CV for noradrenaline, adrenaline,
progesterone and estradiol (averaged across concentrations) were < 3.8%, < 8.0%, < 6.4%, and < 18%, respectively.

Glucose was isolated from serum using double-bed ion exchange chromatography [23]. The resulting supernatant was freeze-dried for analysis using isotope ratio mass spectrometry (IRMS) (Thermo Finnigan Trace GC Ultrace with GC combustion III interface, Bremen, Germany). Breath samples were analyzed using gas chromatography continuous flow isotope ratio mass spectrometry (Thermo Finnigan Delta Plus Advantage, Bremen, Germany).

Substrate oxidation and glucose kinetics calculations

\( \dot{V}_O_2 \) and \( \dot{V}CO_2 \) were averaged over 2 min each 15-min in constant-load exercise, and over the entire time trial. Carbohydrate and fat oxidation were computed from \( \dot{V}_O_2 \) and \( \dot{V}CO_2 \) corrected for exchanges from protein oxidation (1.010 L·g\(^{-1}\) and 0.843 L·g\(^{-1}\), respectively) [24, 25], calculated from urea production[23, 26]. An RER>1.0 precluded calculation of substrate oxidation, which occurred for one participant in the last 15 min in both conditions.

Calculations of muscle glycogen and liver-derived, blood glucose oxidation are described in Harvey et al. [27]. To account for delay equilibrating\(^{13}C_2\) in expired breath and tissues, computations were made during the last 45 min of exercise, allowing for 60 min of equilibration, similar to other studies [23, 27, 28].

Statistics

SPSS (Version 16.0, SPSSInc, Chicago, IL) was used to conduct Linear Mixed Model analyses for each measure, with factors of subject (between), time and condition (repeated). Work rate and \( \sum \) Skin folds were used as covariates. Post-hoc analyses used Sidak tests. Paired t-tests with unequal variances were used for singular measures across conditions for resting and time trial measures. Data are presented as means (± SD), with 95% confidence limits for comparison effects. Significance was accepted as \( P < 0.05 \).

Results and Discussion

Participant characteristics and compliance

Participant characteristics are presented in (Table 1). All 11 participants completed both trials, each within three months. One participant was removed from \( \dot{V}_O_2 \) and \( \dot{V}CO_2 \)\(^{13}C_2\) analyses, and thus carbohydrate oxidation partitioning, due to consuming maize during the night before testing. One participant was removed from core temperature (T\( _c \)) analyses due to insufficient valid T\( _c \) recordings. Participants completed 7±3 h cycling training per week before their first trial and 7±4 h before their last trial. Estradiol concentration was similar in Cold and Temperate (38±17, 46±15 pmol/L, resp., \( P=0.16 \)) as was progesterone (3.6±1.8, 3.5±0.13 pmol/L, resp., \( P=0.23 \)).

Work load, heart rate and perceptual responses

The workload was 158±19 W. Absolute \( \dot{V}_O_2 \) (2.16±0.25, 2.12±0.26 L/min, \( P<0.43 \)) and relative intensity (75 ± 5%, 73 ± 2%, \( P=0.18 \)) were similar between conditions.

Core and skin temperatures

Mean resting T\( _c \) was similar in Cold and Temperate (\( P=0.74 \)). Mean T\( _c \) increased (\( P<0.001 \)) similarly throughout exercise in both conditions (\( P=0.92 \), Figure 1). Work rate was positively correlated with the extent of change in T\( _c \). Mean T\( _c \) declined during exercise (\( P<0.01 \)), more so in Cold (\( P<0.001 \)), becoming ~4°C cooler (95% CI: 3.4 - 4.4°C) by 75 min (Figure 1). The extent of reduction in T\( _c \) between Cold and Temperate was related to subcutaneous adiposity (\( \sum \) Skin folds) (\( P<0.001 \)).

Metabolic responses and substrate partitioning

Energy utilization was similar in Cold and Temperate (3664 ± 374, 3605 ± 420 kJ, resp., \( P=0.28 \)). RER was not different between conditions at rest (\( P=0.43 \)) or during exercise (\( P=0.58 \)) (Figure 2). Protein oxidation was similar between Cold and Temperate (0.14 ±0.05, 0.13±0.05 g·min\(^{-1} \), resp., \( P=0.65 \)), as was carbohydrate (\( P=0.40 \)), fat (\( P=0.52 \)) and protein (\( P=0.76 \)); carbohydrate providing the most energy (Figure 3). No differences in carbohydrate oxidation in the last 45 min were observed (Figure 3, \( P=0.84 \)), and the majority was muscle glycogen. Total carbohydrate oxidation over 75 min of exercise was also similar in Cold and Temperate (154±30, 160±19 g, resp., \( P=0.49 \)), as was muscle glycogen utilization (Figure 4, \( P=0.75 \)). Total muscle glycogen oxidation was 121± 32 and 128± 18 g in Cold and Temperate, respectively (\( P=0.92 \)). Liver-derived, blood glucose oxidation increased across time (Figure 4, \( P=0.003 \)) to a similar extent (\( P=0.94 \)) in both conditions and the total amount oxidized over 75 min of exercise was similar in Cold and Temperate (33± 11, 32 ± 6 g, resp., \( P=0.95 \)). Total fat oxidation was also similar in Cold and Temperate over 75 min (23± 10 g, 20 ± 10 g, resp., \( P=0.26 \)) as was that utilized in the last 45 min of exercise (Figure 4, \( P=0.94 \)).

Circulating metabolite and hormone concentrations

Plasma glucose concentration was similar in both conditions (across steady state exercise: 4.6±0.4, 4.6 ± 0.3 mmo/L, resp., \( P=0.56 \)). Insulin concentration at baseline was also similar between Cold and Temperate (9.9 ± 4.8, 9.4 ± 3.9 µIU/ml, resp., \( P=0.69 \)). Changes in insulin from baseline to 15 min were not different (-0.0 ± 4.0 µIU/mL, -4.8 ± 2.9 µIU/mL, \( P=0.83 \)) nor those to 75 min (-7.2 ± 5.5 µIU/mL, -7.4 ± 3.6 µIU/mL, \( P=0.91 \)).

Adrenaline concentration was similar in Cold and Temperate at rest (136 ± 126, 67±42 pmol/L, resp., \( P=0.09 \)), but increased

### Table 1: Physical characteristics of participants (n=11, Mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>Body mass (kg)</th>
<th>Height (cm)</th>
<th>Body fat (%)</th>
<th>LBM† (kg)</th>
<th>Sum of skin folds‡ (mm)</th>
<th>( \dot{V}_O_2 ) max (L/min)</th>
<th>( \dot{V}_O_2 ) max (mL/kg/min)</th>
<th>( \dot{V}_O_2 ) max (mL/kg LBM/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>26 ± 7</td>
<td>63.9 ± 7.2</td>
<td>169 ± 8</td>
<td>20 ± 3</td>
<td>50.4 ± 6.0</td>
<td>1116 ± 22.5</td>
<td>3.09 ± 0.39</td>
<td>48 ± 8</td>
<td>61 ± 7</td>
</tr>
</tbody>
</table>

Abbreviations: † from Bioimpedence; LBM: Lean Body Mass; ‡8 sites

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**Figure 1** Mean (±SD) (A) change in core temperature (rectal) and (B) mean skin temperature (T_{sk}, nine sites) from baseline (rest) during a 30-min warm up at ~30%VO\textsubscript{2} max in 19°C, 75 min cycling at ~74% VO\textsubscript{2} max in 5°C (▼ Cold) and 15°C (ο Temperate), before a 4-km time-trial (4KTT) in the same environment.

**Figure 2** Mean (±SD) Respiratory Exchange Ratio (RER) measured at rest in 19°C, then during 75 min cycling at ~74% VO\textsubscript{2} max in 5°C (▼ Cold) and 15°C (ο Temperate), immediately followed by a 4-km time-trial (4KTT) in the same environment.

![Figure 1](image1.png)  
![Figure 2](image2.png)

thereafter at different rates \((P=0.02)\). Adrenaline was greater in Cold by 15min of exercise \((593±222, 476±121 \text{ pmol/L, resp., } P=0.046)\), but tended to be lower in Cold at 75 min \((P=0.057, 95\% \text{ CI: -1215 – 20 mmol/L})\). Noradrenaline concentration was similar between Cold and Temperate at rest \((1819 ± 936, 1744 ± 705 \text{ pmol/L, resp., } P=0.69)\), and increased in exercise \((P<0.001)\), to similar extents \((P=0.19)\).

**Time-trial**

Time-trial performance was similar in Cold and Temperate when measured as time \((419 ± 25, 425 ± 21 \text{ s, resp., } P=0.23)\), work rate \((219 ± 31, 213 ± 29 \text{ W, resp., } P=0.37)\) or work rate relative to VO\textsubscript{2} max \((91 ± 6\%, 88 ± 6\%, P=0.053)\). Mean heart rate (Cold: 180±11 bpm, Temperate: 179±11 bpm,\(P=0.45)\)and changes over time (Figure 5) were not different between conditions. RER was similar in Cold and Temperate \((P=0.11)\) and as mean values were >1.0 substrate partitioning was not possible. Finally, perceived exertion \((18±1 \text{ in both conditions})\) and thermal discomfort (Cold: 4 ± 2, Temperate: 3 ± 1) were similar at the end of the time trial across conditions \((P>0.05)\), whilst thermal sensation was higher in Temperate than Cold \((9±2 \text{ (i.e. warm), 7±2 (i.e. neutral), resp., } P=0.01, 95\% \text{CI: 1 – 3 points)})\).

This is the first study to report the metabolic response to exercise in the cold in females and describe the source and rate of carbohydrate oxidized. Reduced ambient temperature (5 vs. 15°C) affects neither metabolic rate nor substrate use in fed females cycling at a moderate intensity with realistic airflow. Despite T\text{sk} being ~4°C lower in Cold (5°C), T\text{c} was similar to Temperate (15°C), which may account for the lack of difference in metabolic response. In both conditions carbohydrate provided the main energy source (~70%), the majority (~80%) of which came from muscle glycogen. Thus, muscle glycogen contributed ~56% to total energy, consistent with observations in males [29] and females [30, 31].

Absolute and relative rates of substrate oxidation were stable throughout exercise in this study, regardless of environmental temperature. Typically RER declines during moderately intensive exercise lasting more than 1.5-2 h, as muscle glycogen becomes depleted, shifting metabolism towards fat [5,6,32]. The women in this study were trained and, having had two days of reduced exercise, probably had replete muscle glycogen reserves. Furthermore they had had breakfast, reducing liver glycogenolysis; hence, 75 min of exercise was probably not enough to deplete reserves. However, the extent of substrate oxidation is in line with current literature on females exercising at moderate intensities for > 1 h. Campbell at al. [31] observed

**Figure 3** Mean relative contribution of substrates to total energy expenditure during the last 45min of 75 min cycling at ~74% VO\textsubscript{2} max in 5°C (Cold) and 15°C (Temperate).

![Figure 3](image3.png)
Figure 4 Mean (±SD) oxidation rates of A carbohydrate (CHO), B fat, C muscle glycogen, and D liver-derived (plasma) glucose measured at rest in 19°C, then during 75 min cycling at ~74% VO_{2\text{max}} in 5 °C (▼ Cold) and 15°C (ο Temperate), immediately followed by a 4-km time-trial (4KTT) in the same environment.

Figure 5 Mean (±SD) heart rate during 75 min cycling at ~74% VO_{2\text{max}} in 5°C (▼ Cold) and 15°C (ο Temperate) immediately followed by a 4 km time-trial (4KTT) in the same environment.

a mean rate of carbohydrate oxidation of 0.46 g/min in women cycling at 70% VO_{2\text{peak}} for 2 h which is in line with our observed rate of 0.47 g/min over 75 min at 75% VO_{2\text{max}}.

Most of the previous research in which carbohydrate metabolism was observed to be increased in the “cold” used male participants and a lower relative exercise intensity. Studies in which increased fat oxidation is observed typically had a much warmer temperature to compare with cold than the present study [2-5] and “cold” in one of these study could be considered temperate (13°C) [4]. Carbohydrate metabolism is increased with exercise in both hot and cold conditions- if the perturbation of homeothermy is sufficient to elicit either cold- or heat-defensive thermoregulatory responses. As such, exercise itself is heat stressful and endurance exercise is optimized in temperatures below thermo neutrality at rest [32]. Thus, previous studies may have been comparing a heat stressed with a cooler (or less heat stressed) condition.

Exercise-cold stress includes scenarios such as low ambient temperature combined with wind, water (rain or immersion), insufficient clothing, or low exercise intensity, which can cause an increase in metabolic cost to maintain thermal balance [33]. Exercise in cool water typically hastens T_{sk} cooling despite an increased metabolic response, and exercise in the rain can similarly increase the metabolic cost markedly despite a rise in T_{c} above resting values; raising the question as to what is an appropriate T_{c} response to exercise [34].

When exercising in cold air, T_{sk} and T_{c} have been reported to both decrease [35], or just T_{sk} to decrease with no change in T_{c} [6]. In our study, T_{c} increased across time, independent of ambient conditions, and the lower T_{sk} did not stimulate a higher metabolic cost.

In contrast to many lab-based studies, which are more heat stressful by virtue of low or no air velocity, semi-realistic, forced
The convection provides more valid thermoregulatory responses [36]. The convection in the present study (~14 km·h⁻¹), making the wind chill temperatures closer to ~2 °C and ~14 °C [33,37], may have made Temperate less heat stressful than in ‘temperate’ conditions in previous studies [6,35] and the higher intensity and heat created reducing effects of Cold. Accordingly, the 17 °C [35] and 20°C [6] difference in temperature in previous studies may have had a greater effect on metabolism than that of the effective ~12°C difference in the present study. Considering these observations, the women in our study may have been physically and physiologically more heat stressed than cold stressed due to their high exercise intensity and favorable adiposity, while also being psychophysically mildly cold stressed (i.e. rated themselves less thermally comfortable, but nevertheless found the exercise less strenuous).

The similarity in insulin concentration in both conditions further supports the lack of difference in substrate usage. The transient difference in adrenaline concentrations was not large enough to have a measurable effect on carbohydrate metabolism between the two conditions, or was overshadowed by other regulatory mechanisms. Febbraio et al. [35] found reduced adrenaline and nor adrenaline and decreased muscle glycogenolysis, core and muscle temperatures at 3 than 20°C (which would have been more heat stressful than our 15°C convection-cooled condition). Catecholamine profiles in the present study (~14 km·h⁻¹), making the wind chill temperatures closer to ~2 °C and ~14 °C [33,37], may have made Temperate less heat stressful than in ‘temperate’ conditions in previous studies [6,35] and the higher intensity and heat created reducing effects of Cold. Accordingly, the 17 °C [35] and 20°C [6] difference in temperature in previous studies may have had a greater effect on metabolism than that of the effective ~12°C difference in the present study. Considering these observations, the women in our study may have been physically and physiologically more heat stressed than cold stressed due to their high exercise intensity and favorable adiposity, while also being psychophysically mildly cold stressed (i.e. rated themselves less thermally comfortable, but nevertheless found the exercise less strenuous).

The lack of effect of ambient temperature on time-trial performance was probably related to there being no difference in thermoregulation and substrate metabolism during the preceding 75 min of exercise, and muscle glycogen thus being similarly taxed. The absence of any extra cardiac or thermal reserve during 75 min of exercise in the cooler condition, would also contribute to the lack of effect on subsequent performance.

CONCLUSION

The results of the present study indicate that, in females, in the fed state, moderately intensive cycling (with realistic airflow) elicits the same metabolic strain when conducted in 5°C as in 15°C. Core temperature is unaffected despite differences in skin temperature and perceptions of cold and discomfort in 5 °C. The lack of difference in core temperature (and perhaps muscle temperature) and, in turn, the minimal difference in sympathetic response, could explain the similarity in metabolism in these disparate environments. Methodological differences might contribute to the discrepant findings, when comparing with previous work conducted in men, particularly the fact that we used realistic airflow and a less warm (more thermo neutral) temperature to compare cold with than in many studies. Nonetheless, it appears that women may be more able to exercise in colder environments before metabolism is altered, most likely due to body composition differences, thus making nutritional adjustments unnecessary.

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Conflict of Interest

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