Fueling shivering thermogenesis during passive hypothermic recovery

François Haman,1 Chris G. Scott,2 and Glen P. Kenny2
1Faculty of Health Sciences and 2School of Human Kinetics, University of Ottawa, Ottawa, Ontario, Canada

Submitted 22 August 2006; accepted in final form 6 July 2007

Haman F, Scott CG, Kenny GP. Fueling shivering thermogenesis during passive hypothermic recovery. J Appl Physiol 103: 1346–1351, 2007. First published July 19, 2007; doi:10.1152/japplphysiol.00931.2006.—In humans, the relative importance of oxidative fuels for sustaining shivering during passive hypothermic recovery or rewarming is still unclear. The main goals of this study were 1) to quantify the respective contributions of lipids and carbohydrates (CHO) during passive rewarming and 2) to determine the effects of precooling exercise on the pattern of fuel utilization. With indirect calorimetry methodologies, changes in fuel metabolism were quantified in nonacclimatized adult men shivering to rewarm from moderate hypothermia (core temperature ∼34.5°C) not following (Con) or following a precooling exercise at 75% \( V\overset{\text{O2max}}{\text{m}} \), for 15 min (Pre-CE). As hypothermic individuals shiver to normothermia, results showed that CHO dominate at all shivering intensities above 50% \( \text{Shiv}_{\text{peak}} \), while lipids were preferred at lower intensities. This change in the relative importance of CHO and lipids to total heat production was dictated entirely by modulating CHO oxidation rate, which decreased by as much as 10-fold from the beginning to the end of rewarming (from 1,611 ± 396 to 141 ± 361 mg/min for Con and 1,555 ± 230 to 207 ± 261 mg/min for Pre-CE). In contrast, lipid oxidation rate remained constant and low (relatively to maximal rates at exercise) throughout rewarming, averaging 183 ± 141 for Con and 207 ± 118 mg lipids/min for Pre-CE. In addition, this pattern of fuel selection remained the same between treatments. We concluded that fuel selection is regulated entirely by changes in CHO oxidation rate. Further research should focus on establishing the exact regulatory processes involved in achieving this large upregulation of CHO utilization rate following hypothermia.

IN COLD EXPOSED HUMANS, significant decreases in core temperature or hypothermia occur when increases in heat production are not sufficient to compensate for increases in heat loss. Following a hypothermic episode, shivering thermogenesis (ST) remains the only physiological process available for reestablishing core temperature in nonexercising, nonassisted individuals (15). Most studies during passive hypothermic recovery or passive rewarming focused on the efficiency of external warming methods (for example, warm water immersion, warm air breathing; Ref. 6) and, therefore, little is known about the metabolic requirements of ST following hypothermia. In the only study focusing on the energy demand of ST during rewarming, Neuffer et al. (15) showed that core warming rate in nonacclimatized hypothermic men remained unaffected by glycogen depletion (0.61 vs. 0.71°C/h for control vs. glycogen depleted). However, the respective importance of CHO and lipids to total heat production (\( \text{H}_{\text{prod}} \)) still remains unknown because rates of oxidation have never been quantified. During sustained low and moderate shivering intensities, recent work has shown that the relative contribution of CHO to total \( \text{H}_{\text{prod}} \) increases progressively as shivering intensifies, while that of lipids and proteins decreases progressively (11). Most importantly, these changes in fuel selection occur entirely by upregulating rates of CHO utilization because lipid and protein oxidation rates remain unchanged. Whether this same pattern of fuel selection is found during passive rewarming remains to be established.

Therefore, the main goals of this study were 1) to determine the pattern of fuel selection during rewarming and 2) to investigate the potential effects of high-intensity, short-duration precooling exercise on changes in fuel utilization rates. More specifically, changes in CHO and lipid oxidation rates were quantified in nonacclimatized adult men following moderate hypothermia (core temperature ∼34.5°C). These experiments were conducted either without performing precooling exercise (Con) or following a precooling exercise bout at 75% \( V\overset{\text{O2max}}{\text{m}} \) for 15 min on a cycle ergometer (Pre-CE). Based on the previously reported fuel selection pattern observed during low- and moderate-intensity shivering (11), we predict that the contribution of CHO to total heat production will be dominant at high shivering intensities, while lipids will be the preferred fuel at lower intensities. Second, assuming that the combination of cycling at 75% \( V\overset{\text{O2max}}{\text{m}} \) for 15 min is sufficient to induce neuromuscular fatigue and a reduction in CHO availability, we anticipate that CHO utilization rate will be decreased during rewarming following precooling exercise due to an overall reduction in the recruitment of type II, fatigue-inducible fibers, and/or CHO availability (9, 14, 22).

This study will also address another important issue. Previous shivering work indicates that maximal lipid oxidation rate seems to be already reached at low metabolic rates (∼135 mg·lipids·kg⁻¹·h⁻¹; Ref. 11); a value more than three times lower than the one reported for sustained exercise by Achten et al. (1). Physiological reasons for this limitation are still unknown because subsequent measurements during shivering were only performed at low [∼2.5 × resting metabolic rate (RMR)] or [∼15% \( V\overset{\text{O2max}}{\text{m}} \)] and moderate intensities (∼3.5 × RMR or ∼20% \( V\overset{\text{O2max}}{\text{m}} \)). In the present study, it is expected that shivering intensities during rewarming will exceed 3.5 × RMR and may even reach maximal shivering intensity at ∼5 × RMR (5) due to the severity of the cold stress. Consequently, this study will allow us to determine if higher rates of lipid oxidation can be achieved during shivering.

METHODS

Subjects. Six healthy, physically active men with no history of cardiorespiratory disease or cold injuries volunteered for this study. The subject’s characteristics were as follows (mean ± SD): age:

Address for reprint requests and other correspondence: F. Haman, Faculty of Health Sciences, Univ. of Ottawa, 125 Univ. St, Ottawa, Ontario, Canada K1N 6N5 (e-mail: fhaman@uottawa.ca).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
values measured in the cold by the highest metabolic rate recorded postcooling for Con and Pre-CE. This maximal shivering rate (Shivpeak in ml·kg⁻¹·min⁻¹) was not different from those calculated using the shivering peak estimated for each subject using the equation proposed by Eyolfson et al. (5):

\[
\text{Shivpeak} = 30.5 + (0.348 \times \text{VO}_{2\text{max}}) - (0.909 \times \text{BMI}) - (0.233 \times \text{age})
\]  

where, \(\text{VO}_{2\text{max}}\) is the maximal oxygen consumption (ml·kg⁻¹·min⁻¹), BMI is the body mass index (kg·m⁻²), and the age is in years.

Isopahgeal temperature (\(T_e\)) was monitored continuously using a pediatric probe (Mon-a-therm general purpose, Mallinckrodt Medical, St. Louis, MO). Rewarming rate (\('\text{O}2\text{min}\)'/(min)) was calculated using \(T_e\) values measured at 15-s intervals following cold water immersion. Mean skin temperature (\(T_{\text{skin}}\)) was averaged from 12 sites (i.e., finger tip plus 11 heat transducer sites mentioned above for heat flux measurements) using an area-weighted equation (3).

**Metabolic rate and fuel utilization.** Pulmonary ventilation (\(V_e\)), oxygen consumption (\(V_O2\)), and carbon dioxide production (\(V\text{CO}_2\)) were determined using a mouthpiece and a calibrated automated metabolic analyzer (Med-Graphics CPX-D, St. Paul, MN). \(V\text{O}_2\) and \(V\text{CO}_2\) were averaged every 5 min and carbohydrate (CHOox) and lipid (FATox) oxidation rates (g/min) were calculated using the following equations (2, 13):

\[
\begin{align*}
\text{CHOox}(g/min) &= 4.59V\text{CO}_2/l/min - 3.23V\text{O}_2/l/min \quad (4) \\
\text{FATox}(g/min) &= -1.70V\text{CO}_2/l/min + 1.70V\text{O}_2/l/min \quad (5)
\end{align*}
\]

where \(V\text{CO}_2/l/min) and \(V\text{O}_2/l/min) were corrected for the volumes of \(O_2\) and \(CO_2\) corresponding to protein oxidation (1.010 and 0.843 l/g, respectively). Protein oxidation rate was estimated at 66 mg/min based on previously published urinary urea excretion measurements made on 12-h postabsorptive men with normal CHO reserves (8, 10). Energy potentials of 16.3, 40.8, and 19.7 kJ/g were used to calculate the amount of heat produced from glucose, lipid (fatty acids), and protein (amino acids) oxidation, respectively (4, 17).

**Statistical analyses.** Changes in \(T_e\), \(T_{\text{skin}}\), \(H_{\text{loss}}\), and \(H_{\text{prod}}\) were assessed by two-way ANOVA for repeated measures. Differences in \(H_{\text{prod}}\), fuel utilization for CHO (CHOox) and lipids (FATox) at baseline (23°C) as well as during exercise (only for the precooling exercise experiment), cooling and rewarming were determined using one-way ANOVA to verify the main effect of the treatment (Con vs. Pre-CE). Statistical differences were considered significant when \(P \leq 0.05\). The statistical power of the two-way ANOVA for repeated measures was calculated for CHOox and FATox and it reached 0.54 and 0.74, respectively. All values presented are means ± SD (n = 6) unless indicated otherwise.

**RESULTS**

**Thermal responses.** Changes in \(T_{\text{skin}}\), \(T_e\), \(H_{\text{prod}}\), and \(H_{\text{loss}}\) measured prior to cooling, during precooling exercise (Pre-CE only), during cooling and postcooling for Con and Pre-CE are presented in Fig. 1. While postexercise values were significantly higher for Pre-CE, no difference between Con and Pre-CE were observed by the end of cooling and throughout rewarming. By the end of immersion, \(T_{\text{skin}}\) decreased from 31.9 ± 0.6°C at baseline to 12.2 ± 4.7°C prior to rewarming for Con and from 31.7 ± 0.9 to 11.2 ± 3.5°C for Pre-CE (Fig. 1). \(T_e\) decreased from 37.1 ± 0.3 to 31.4 ± 0.3°C for Con and from 37.1 ± 0.2°C to 34.5 ± 0.03°C for Pre-CE (Fig. 1). \(H_{\text{prod}}\) increased 3.7-fold for Con from 7.2 ± 1.1 to 26.5 ± 7.5 kJ/min and 3.9-fold for Pre-CE from 6.7 ± 1.2 to 25.1 ± 7.7 kJ/min (Fig. 1). For both Con and Pre-CE, metabolic rates reached by the beginning of rewarming was equivalent to maximal shiv-
ering intensity (5). H˙loss increased fourfold for Con from 7.4/110060.9 to 29.8/1100610.3 kJ/min and for Pre-CE from 7.4/110061.4 to 29.7/1100613.8 kJ/min (Fig. 1). By the end of rewarming, Tskin and Tes increased to 27.5/110061.3 and 36.2/110060.3°C for Con (27.6/110061.0 and 36.2/110060.2°C for Pre-CE). Average values measured at baseline (B), at the end of exercise (PE), end of cold-water immersion (PC), and at the after-drop level (AD, the lowest post-cooling Tes) are also shown. Values are presented for the longest rewarming period common to all subjects for each condition. *Immersion time averaged 56 ± 21 min for Con and 53 ± 23 min for Pre-CE.

Changes in rewarming rates and heat balance during rewarming are shown in Fig. 2. Rewarming rate and heat balance were not different between Pre-CE and Con. Maximal rewarming rate was measured at 15 min and averaged 0.09 ± 0.05°C/min (Con) and 0.08 ± 0.05°C/min (Pre-CE). In addition, Fig. 3 illustrates changes in shivering intensity (%Shivpeak) as a function of changes in Tes and Tskin for Con and Pre-CE. Again, no difference was found between Con and Pre-CE.

Fuel selection. Changes in the absolute rates and relative contributions of CHO and lipids to total heat production as a function of percent shivering intensity (%Shivpeak) during rewarming are presented in Fig. 4. From the beginning to the end of rewarming, shivering intensity ranged from 97 ± 4 to 26 ± 5.1%Shivpeak for Con and from 95 ± 3 to 27 ± 7%Shivpeak for Pre-CE. Along this range of shivering intensities, no significant difference in absolutes rates of oxidation and relative contribution to total H˙prod were observed between Con and Pre-CE. Absolute rates of CHO oxidation varied from 1,611 ± 396 to 141 ± 360 mg/min for Con and 1,555 ± 237 to 207 ± 261 mg/min for Pre-Ex. In contrast, absolute rates of

Fig. 1. Changes in mean skin (Tskin) and esophageal temperatures (Tes) as well as rates of heat production (H˙prod) and heat loss (H˙loss) measured in men recovering from moderate hypothermia not following (Con) and following precoooling exercise at 75% VO2max for 15 min (Pre-CE). Average values measured at baseline (B), at the end of exercise (PE), end of cold-water immersion (PC), and at the after-drop level (AD, the lowest post-cooling Tes) are also shown. Values are presented for the longest rewarming period common to all subjects for each condition. *Immersion time averaged 56 ± 21 min for Con and 53 ± 23 min for Pre-CE.

Fig. 2. Changes in rewarming rate (top) and heat balance (bottom) in men recovering from moderate hypothermia measured in men recovering from moderate hypothermia not following (Con) and following precoooling exercise (Pre-CE). Values are presented for the longest rewarming period common to all subjects for each condition.

Fig. 3. Changes in shivering intensity (%Shivpeak) as a function of esophageal temperature (Tes) and mean skin temperature (Tskin) measured in men recovering from moderate hypothermia not following (Con; • and solid line) and following precoooling exercise (Pre-CE; ○ and dotted line). %Shivpeak is presented in reverse order to better illustrate the rewarming process.
lipid oxidation remained constant averaging 183 ± 141 mg/min for Con and 207 ± 118 mg/min for Pre-Ex. Relative contributions of CHO to total \( H_{\text{prod}} \) decreased continuously from beginning to the end of rewarming from 79.3 ± 15.6 to 25.5 ± 13.2% \( H_{\text{prod}} \) for Con and from 69.9 ± 9.9 to 37.7 ± 11.8% \( H_{\text{prod}} \) for Pre-CE), whereas that of lipids increased from 16.7 ± 15.7 to 60.9 ± 13.2% \( H_{\text{prod}} \) for Con and from 26.4 ± 10.3 to 48.4 ± 12.4% \( H_{\text{prod}} \) for Pre-CE. The crossover point or point at which CHO and lipid provide equally to total heat production occurred at 50% \( \text{Shiv}_{\text{peak}} \) (Con) and 60%\( \text{Shiv}_{\text{peak}} \) (Pre-CE) and was not different between treatments. Table 1 summarizes total amounts of CHO and lipids used during rewarming in Con and Pre-CE, as well as the relative contributions of these fuels to total \( H_{\text{prod}} \) averaged over the entire rewarming period. Again, no overall differences between Con and Pre-CE were found in the total amount of CHO or of lipid used to sustain shivering and in the relative contributions of these fuels to total \( H_{\text{prod}} \) production when averaged over the entire rewarming period.

**DISCUSSION**

This study quantifies the contributions of CHO and lipids to total heat production during passive rewarming. It shows that as individuals shiver to normothermia, CHO dominate at all shivering intensities above 50%\( \text{Shiv}_{\text{peak}} \), while lipids are pre-
ferred at lower intensities (Fig. 4). This change in CHO and lipid utilization rate is achieved entirely by a modulation of CHO oxidation rate, which varies by as much as 10-fold from the beginning to the end of rewarming (Fig. 4). In contrast, over the entire range of shivering intensities, lipid oxidation rate remains stable and low (relative to maximal rates during exercise, see *Oxidizing lipids*). The pattern of fuel selection found here during rewarming is consistent with the one observed previously in men during sustained low- and moderate-intensity shivering (11; Fig. 5). Finally, pre-cooling exercise at 75% \( V_{\text{O2max}} \) for 15 min is insufficient to cause the anticipated decrease in CHO utilization rate during rewarming (\( P = 0.20; \) Fig. 4 and Table 1).

**Fuel selection during passive hypothermic recovery.** The 7°C water immersion used in this study to induce hypothermia in nonacclimatized men resulted in decreases of \( \sim 3°C \) for \( T_{es} \) and \( \sim 20°C \) for \( T_{\text{skin}} \) by the end of cold exposure. Together these changes in deep and peripheral temperatures were sufficient to elicit maximal shivering by the beginning of warming [4–5 times resting metabolic rate or \( \sim 50% \ V_{\text{O2max}} \); Eyolfson et al. (5)]. As individuals shiver to normothermia, results show that absolute rates of CHO oxidation decrease by as much as 10-fold (from 1,611 ± 396 to 141 ± 361 mg/min for Con and 1,555 ± 230 to 207 ± 261 mg/min for Pre-CE), whereas lipid utilization rate remains constant (discussed later, see *Oxidizing lipids*; Fig. 4, A and B). In effect, the relative contribution of CHO decreases progressively (from \( \sim 80 \) to 25% \( H_{\text{prod}} \) for Con and from \( \sim 70 \) to 38% \( H_{\text{prod}} \) for Pre-CE), whereas that of lipids increases from \( \sim 17 \) to 61% \( H_{\text{prod}} \) for Con (from \( \sim 26 \) to 48% \( H_{\text{prod}} \) for Pre-CE) as shivering lessens in intensity and individuals approach normothermia (Fig. 4, C and D). The crossover point or the shivering intensity at which CHO and lipids contribute equally to total heat production occurs at \( \sim 50\% \text{Shiv}_{\text{peak}} \) when precooling exercise is not performed. This pattern of fuel oxidation rate is consistent with the one described recently during sustained shivering in nonhypothermic adult men exposed for 90 min to either 10°C (low-intensity shivering (L); or 5°C (moderate-intensity shivering (M); Ref. 11). We compared patterns of fuel selection found previously

| Table 1. \( H_{\text{prod}} \) as well as absolute and relative contributions of CHO and lipids to \( H_{\text{prod}} \) measured in men recovering from moderate hypothermia not following or following pre-CE |
|---------------------------------|-----------------|-----------------|
|                                 | Con             | Pre-CE          |
| \( H_{\text{prod}} \), kJ       | 806±213         | 797±289         |
| CHO, g                         | 28.9±7.4        | 25.6±10.5       |
| %\( H_{\text{prod}} \)          | 61.5±19.1       | 52.6±11.9       |
| Lipids, g                      | 7.0±5.5         | 8.1±3.8         |
| %\( H_{\text{prod}} \)          | 32.5±19.0       | 41.0±11.8       |

Values are means ± SD. \( H_{\text{peak}} \), total heat production; CHO, carbohydrates, Con, control [not following precooling exercise (Pre-CE)].
same crossover point found at tance of CHO and lipids to total heat production also reveal the that of lipids decreases. These changes in the relative impor-
sifies during sustained shivering in nonhypothermic men while that of CHO increases progressively as shivering intensity declines and individuals approach normothermia. However, this change is not achieved by upregulating lipid oxidation rate as it remains constant, averaging 183 ± 141 for Con and 207 ± 118 mg lipids/min for Pre-CE throughout rewarming (Fig. 4). Similarly, during sustained shivering in the cold, previous work has shown that lipid oxidation rate never exceeds ~165 mg lipids/min even when shivering intensity decreases from low to moderate (Fig. 5; Ref. 11). Together these findings indicate that maximal rates of lipid oxidation are already achieved at low shivering intensities. Clearly, however, we anticipated that much higher rates could be reached as shivering intensified from 3.5 to 5× RMR. To date, the highest RFox values measured during shivering are still more than three times lower than reported for sustained exercise (1). What limits lipid utilization rate during shivering? Although the exact physiological reasons are unclear at best, we can speculate on a number of possible mechanisms that may limit RFox during shivering. For example, cold exposure is associated with reduced peripheral blood flow, which may lead to impaired fatty acid supply to shivering muscles via the circulation. In addition, as shivering intensity increases, the progressive recruitment of proportionally more type II glycolytic fibers may occur, whereas the recruitment of type I lipolitic fibers remains constant. Finally, one may also consider the influence of cold exposure on other factors responsible for controlling fuel oxidation such as circulating hormones (e.g., catecholamines), trans-membrane transporters (fatty acid transporters), and a series of intracellular metabolites (acetyl-CoA, malonyl-CoA, Ca^{2+}, ADP, AMP, P_{i}, and AMPK l). Future work in this field of research should attempt to better understand the relative importance of these factors on energy substrate regulation in the cold.

**Effect of precooling exercise.** The second objective of this study was to examine whether precooling exercise at 75% VO_{2max} for 15 min is sufficient to reduce CHO utilization rate by inducing neuromuscular fatigue and, thus, the recruitment of type II, fatigable muscle fibers. Recent work had shown that the recruitment of type II fatigable fibers was key in dictating fuel selection in the cold by modulating CHO oxidation rate (8). Based on previous observations during rewarming, we also recruiting muscles varying in fiber composition (20). In the cold, the first two mechanisms of fuel selection have been identified (7, 8). During low-intensity shivering (~2.5 times resting metabolic rate (RMR)), CHO-depleted and CHO-loaded individuals were able to sustain the same rate of heat production by recruiting different metabolic pathways within the same fibers (7). In contrast, during moderate shivering (~3.5 times RMR), alterations in fuel selection are achieved by recruiting subpopulations of fuel specific fibers within the same muscle (8). In view of the large range of shivering intensities found here during hypothermic recovery (up to 5 times RMR), we can speculate that the 8- to 10-fold increase in CHO utilization rate is achieved by proportionally increasing the specific recruitment of “CHO specific” fibers (i.e., type II glycolytic fibers) within shivering muscles; assuming that the same muscles were recruited throughout warming. Clearly, however, additional work is needed to identify the exact mechanisms responsible for this regulation in CHO oxidation rate.

**Oxidizing lipids.** During hypothermic recovery, the relative importance of lipids increases progressively as shivering intensity declines and individuals approach normothermia. However, this change is not achieved by upregulating lipid oxidation rate as it remains constant, averaging 183 ± 141 for Con and 207 ± 118 mg lipids/min for Pre-CE throughout rewarming (Fig. 4). Similarly, during sustained shivering in the cold, previous work has shown that lipid oxidation rate never exceeds ~165 mg lipids/min even when shivering intensity decreases from low to moderate (Fig. 5; Ref. 11). Together these findings indicate that maximal rates of lipid oxidation are already achieved at low shivering intensities. Clearly, however, we anticipated that much higher rates could be reached as shivering intensified from 3.5 to 5× RMR. To date, the highest RFox values measured during shivering are still more than three times lower than reported for sustained exercise (1). What limits lipid utilization rate during shivering? Although the exact physiological reasons are unclear at best, we can speculate on a number of possible mechanisms that may limit RFox during shivering. For example, cold exposure is associated with reduced peripheral blood flow, which may lead to impaired fatty acid supply to shivering muscles via the circulation. In addition, as shivering intensity increases, the progressive recruitment of proportionally more type II glycolytic fibers may occur, whereas the recruitment of type I lipolitic fibers remains constant. Finally, one may also consider the influence of cold exposure on other factors responsible for controlling fuel oxidation such as circulating hormones (e.g., catecholamines), trans-membrane transporters (fatty acid transporters), and a series of intracellular metabolites (acetyl-CoA, malonyl-CoA, Ca^{2+}, ADP, AMP, P_{i}, and AMPK l). Future work in this field of research should attempt to better understand the relative importance of these factors on energy substrate regulation in the cold.
expected that such changes in fuel use could occur without significant alterations in rates of heat production and/or rewarming. For example, during sustained shivering in the cold, a growing body of evidence indicates that heat production rates can be sustained independently of 1) changes in the size of glycogen reserves (or fuel mixtures being used; Refs. 9, 14, 22) or 2) whether exhaustive exercise is performed prior to cold exposure (21). In addition, during passive rewarming in hypothermic men, Neufer et al. (15) showed that rewarming rates remain unimpaired even when the size of muscle glycogen reserves are reduced prior to cooling. However, to date, the effects of precocing exercise on thermal responses and on oxidative fuel selection during rewarming had never been quantified. In the present study, results show that exercising at 75% V\textsubscript{O}\textsuperscript{max} for 15 min prior to cooling has no significant effect on H\textsubscript{prod} and rewarming rate (Figs. 1 and 2, Table 1). In addition, contrary to what was expected, absolute rates and relative contributions of CHO to total H\textsubscript{prod} remained unchanged between Con and Pre-CE (Fig. 4, Table 1). It is likely that the precocing exercise intensity and duration selected in this study were insufficient to influence the expected neuromuscular fatigue and associated reduction in type II fiber recruitment and/or CHO availability. It still remains unclear whether more intense and longer duration exercise could modify thermal responses and the pattern of fuel selection during passive rewarming.

Conclusion. This study shows that both CHO and lipids play substantial roles in sustaining heat production during passive hypothermic rewarming (CHO above and lipids below 50% Shiv\textsubscript{peak}); a pattern unaffected by precocing exercise (75% V\textsubscript{O}\textsubscript{max} for 15 min). More importantly, it indicates that this regulation in fuel selection is modulated entirely by changes in CHO oxidation rate, which is downregulated by as much as 10-fold from the beginning to the end of rewarming. In contrast, lipid oxidation rate remains unchanged, reaching a maximum rate at very low shivering intensity. Further research should focus on establishing the exact regulatory processes involved in achieving this large upregulation of CHO utilization rate following hypothermia.

REFERENCES