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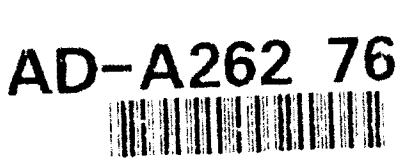

DECEMBER 1992

DCIEM No. 92-60

INFLUENCE OF THE COLD BUSTER™ SPORTS BAR ON HEAT DEBT, MOBILIZATION AND OXIDATION OF ENERGY SUBSTRATES

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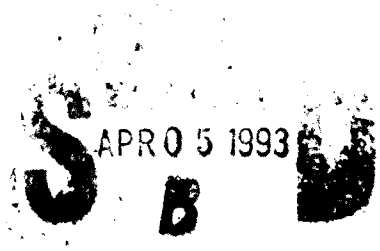

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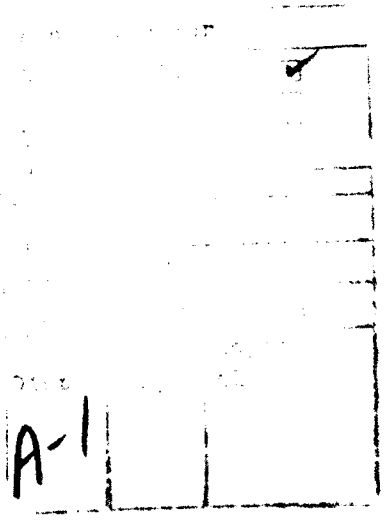


EXECUTIVE SUMMARY

A D Food S tasking was carried out to determine whether the commercially available Cold Buster™ Sports bar, purported to improve resistance to cold, should be considered for use as a CF Cold Weather Supplement Ration. We reported that the bar did not improve metabolic heat production ($\dot{M}$) or heat debt (S) in our subjects exposed to a relatively severe cold test (Vallerand, Tikuisis, Ducharme & Jacobs, In Review). One possible explanation for these conflicting results is that our $\dot{M}$ was high enough to prevent the detection of the small amount of heat associated with the bar ingestion. The goal of this study was therefore to re-evaluate, under milder conditions, the influence of the Cold Buster™ on heat balance ($\dot{M}$ - heat losses = S) and body temperatures (rectal and skin temperatures). Eight semi-nude fasted subjects were exposed to the cold (3h at rest, 10°C) on two occasions following two ingestions of either a placebo (100 ml water) or a Cold Buster™ (all feedings at min 0 and 90). There were no differences between treatments with respect to $\dot{M}$, heat losses, S and body temperatures. Compared to the placebo, ingestion of the Cold Buster™ significantly increased the mobilization and oxidation of carbohydrates entirely at the expense of the mobilization and oxidation of lipids. The results confirm previous data where the ingestion of the Cold Buster™ Sports bar did not change heat production, heat losses, heat debt or body temperatures, and extend these observations to a mild cold stress, even though the bar altered the mobilization and oxidation of energy substrates. Taken together, these studies do not provide any evidence to support the contention that the ingestion of the Cold Buster™ Sports bar is advantageous during cold stress.

RESUME EXECUTIF

Nous avons récemment montré que le complément énergétique alimentaire Cold Buster™, commercialisé dans le but d'améliorer la résistance au froid, ne la modifiait pas chez 8 sujets exposés à un test au froid relativement sévère (Vallerand, Tikuisis, Ducharme et Jacobs, En Révision). Une explication possible de ces résultats est que ce test a provoqué une augmentation très importante du métabolisme, masquant l'effet thermogénique espéré du Cold Buster™. Nous avons voulu ré-évaluer l'effet du Cold Buster™ sur le bilan thermique (production - pertes de chaleur = dette thermique) et les températures corporelles (rectale et cutanée moyenne) sous des conditions expérimentales moins intenses. Pour cela, 8 sujets ont été exposés semi-nus à un test au froid (10°C, 3h, au repos et à jeun) après ingestion de placebo (100 mL d'eau) ou du Cold Buster™ (une aux temps 0 et min 90). Dans ces conditions, aucune différence n'a été constatée entre les deux tests quant à l'ensemble des paramètres thermiques mesurés et calculés. L'ingestion du Cold Buster™ a significativement augmenté la mobilisation plasmatique ainsi que l'oxydation des hydrates de carbone entièrement au dépends des lipides. Ces résultats confirment l'expérimentation antérieure. Cold Buster™ ne modifie ni le bilan thermique ni les températures corporelles lors d'un test au froid relativement intense ou modéré. Ceci est observé bien que Cold Buster™ modifie la mobilisation et l'oxidation des substrats énergétiques. Ensemble, ces études n'apportent aucun argument pour recommender l'utilisation de Cold Buster™ au froid.

ABSTRACT

In a recent study, we have shown that the commercially available Cold Buster™ Sports bar, purported to improve cold resistance, did not do so in our subjects exposed to a relatively severe cold test. (Vallerand, Tikuisis, Ducharme & Jacobs, In Review). One possible explanation for these conflicting results is that our metabolic rate ($\dot{M}$) was so high that it may have masked any thermogenic effect of the bar. The goal of this study was therefore to re-evaluate, under milder conditions, the influence of the Cold Buster™ on heat balance (heat debt = heat production - heat losses) and body temperatures. Eight semi-nude fasted subjects were exposed to the cold (3h at rest, 10°C, <0.4 $m.s^{-1}$ wind) on two occasions following two ingestions of either a placebo (100 ml water) or a Cold Buster™ (all feedings at min 0 and 90). As a result of the cold, $\dot{M}$, dry heat losses and heat debt (S) increased whereas mean skin temperature decreased ($P<0.05$). Rectal temperature profile remained unchanged due to the mild cold. For all of the above variables, there were no differences between treatments. Ingestion of the Cold Buster™ significantly increased carbohydrate oxidation at min 150 compared to the placebo ($P<0.05$). However, this was without impact on $\dot{M}$, since it occurred entirely at the expense of fat oxidation (n.s.). Interestingly, the Cold Buster™ increased plasma glucose and insulin levels 2h into the cold ($P<0.05$). This secretion of insulin seems to have blunted lipid mobilization since it significantly reduced plasma free fatty acids levels ($P<0.05$). The results confirm previous data where the ingestion of the Cold Buster™ Sports bar did not alter heat production, heat losses, heat debt or even body temperatures, and extend these observations to a mild cold stress. Although the Cold Buster™ enhanced CHO mobilization and oxidation, this phenomenon occurred entirely at the expense of mobilization and oxidation of lipids. Taken together, these studies do not provide any evidence to support the contention that the ingestion of the Cold Buster™ Sports bar is advantageous during cold stress.

BACKGROUND

Humans will often expose themselves to extreme cold temperatures, but they must do so without losing sight of the delicate balance that they must maintain between metabolic heat production and heat loss, to ensure the proper regulation of body temperatures (Burton & Edholm, 1969). Failure to properly regulate body temperatures can be tragic in the case of a local failure (frostbite) or it can be fatal in the case of a whole body failure of temperature regulation (hypothermia).

Several techniques have been used to enhance metabolism or to improve resistance to cold, and a dietary and/or pharmacological approach has received significant attention (Vallerand, Jacobs & Kavanagh, 1989a; Vallerand & Jacobs, 1992; Vallerand, In Press; Wang, Man & Belcastro, 1987; Wang, 1990). To that effect, a commercially available high-carbohydrate (CHO) "Recreation and Sports bar", the Cold Buster™, is purported to delay the onset of hypothermia in cold-exposed individuals. The theory behind the bar's effectiveness appears to be the thermogenic combination of theobromine (a caffeine-like substance) and the timely supply of energy substrates (Wang, 1990). It is well understood that ingesting substrates will obligatorily increase exogenous substrate mobilization. More than ten years ago, it was suggested that the main physiological mechanism which limits maximum cold-induced thermogenesis is the timely supply of metabolic fuels (Wang, 1980) and that substances like methylxanthines, which appear to enhance energy substrate mobilization, could further improve cold-induced thermogenesis and consequently cold tolerance (Wang, 1981).

We were not able to confirm this theory in a recent study in which the Cold Buster™ had no influence on any parameter of the heat balance (heat production, heat losses, heat debt) or body temperatures in cold-exposed subjects (Vallerand, Tikuisis, Ducharme & Jacobs, In Review). One possible explanation for our conflicting results was that, due to the relative severity of our cold exposure, metabolic rate was too high to enable the detection of the thermogenic effect of the bar.

The goal of this study was therefore to determine, under milder conditions, whether the Cold Buster™ can enhance cold resistance in humans and whether it does so via alterations in heat production ($\dot{M}$) or heat losses. This was accomplished by determining not only core and mean skin temperatures, but also by performing a full heat balance analysis, by calculating rates of energy substrate oxidation and by analyzing levels of various plasma substrates and hormones, as indices of energy substrate mobilization.

METHODS

SUBJECTS

Eight healthy male volunteers participated in the present study. Each subject was examined by a physician who approved his participation. The nature, purpose and possible risks of the study were explained in detail to each individual before he gave his consent to participate. All subjects could withdraw from the study at any time without bias. Their standard physical characteristics were normal, and they are summarized in the Results section. A 1.5 h familiarization run was used to familiarize the subjects with the cold air and the protocol.

EXPERIMENTAL PROTOCOL

Two cold exposure tests, each for 3 h at 10°C, <0.4 $m.s^{-1}$ wind speed, sitting at rest, were performed about 1 wk apart on each fasted subject (wearing jogging shorts and foam slippers only). Each subject served as his own control. The subjects could not be blind to the conditions since a suitable placebo chocolate bar could not be located. However, we could ensure that the experimenters were kept blind, by keeping both the subject i.d. and the treatment number secret until the end of data analysis. Subjects were also asked to avoid alcohol at least 48 h prior to the test, to avoid exercise 24 h before, and to report in a fasting state (only water for 12-14h). Treatments involved two ingestions of either a placebo (100 ml water) or a Cold Buster™ Sports bar (L&R Wang Enterprises Inc., Edmonton Alberta Canada) each containing 170 kcal, of which

approximately 68, 21, 11% of the energy was derived from CHO, fat and protein, respectively. One bar was eaten immediately before and the other after 1.5 h, as recommended on the label, and as performed elsewhere (Vallerand et al., In Review). Our lot of Cold Buster™ bars weighed ~42 g (170 kcal) instead of 38 g (154 kcal) printed on the wrapper. Only the 42±2 g bars were used. The order of treatments was balanced (4 subjects had the Cold Buster™ test first and 4 others had the placebo first; there was no order effect).

Subjects were instrumented with a rectal probe (Sherigan, Argyle, NY), 12 re-calibrated heat flux transducers (Concept Engineering, Old Saybrook NJ), an intravenous catheter (inserted into an antecubital vein; Insyte, Deseret Medicals, Sandy UT) and a heart rate monitor (Polar Vantage XL, Polar USA, Stamford CT) early in the morning. Thereafter, they were allowed to rest quietly in the subject preparation room (sitting in the experimental chair at 22°C) for approximately 0.5 h before the cold test. The first feeding occurred about 2 min before entering the cold room. The cold test consisted of exposing semi-nude subjects to cold air for 3 h at rest (seated on a modified lawn chair, which was used to wheel the subjects in and out of the chamber). Individuals were asked to provide subjective ratings of discomfort, thermal preference, and sense of shivering a few min before entering the chamber and at the end of each hour of the 3 h test, using previously established questionnaires (Gwosdow & Berglund, 1989). The experiment was terminated if: (a) rectal temperature dropped below 35.0°C; (b) the subject wished to withdraw from the test; or (c) after 3 h of exposure.

MEASUREMENTS

During the tests, rectal temperature (T_{re}) was used as an index of core temperature and it was monitored using a thin thermistor probe inserted 10 cm beyond the anus. It was also secured into place using a modified loincloth. Skin temperatures and dry heat losses were measured with 12 re-calibrated heat flux transducers taped to the skin. Using a 12 point area-weighted system, mean skin temperature ($\bar{T}_{sk}$) and mean dry heat loss were calculated as described elsewhere (Vallerand et al., 1989b). All thermal data were

continuously recorded with a computerized Hewlett-Packard 236 data acquisition system (Vallerand et al., 1989b).

O_2 consumption and CO_2 production (in $L.min^{-1}$ STPD) were measured using a computerised metabolic cart comprising of an IBM PC, a polarographic O_2 analyzer (S-3A/I) and a infrared CO_2 (CD-3A) analyzer (Applied Electrochemistry, Pittsburgh PA), a ventilation module (Interface Associates, Irvine CA) and a Turbofit A/D computer interface (Vacumetrics, Ventura CA). The use of a Hans Rudolf face mask (Hans Rudolf Inc., Kansas City MO) permitted the continuous measurement of respiratory gas exchanges for the entire 3 h period, except for about 5 min at min 90. Analyzers were recalibrated before and during the tests (at min 90 if required). The recalibration/feeding period at min 90 provided the subjects with a 5 min break with respect to the wearing of the face mask.

THERMAL AND METABOLIC CALCULATIONS

Metabolic rate ($\dot{M}$ in watts per square meter of body surface area) was calculated according to the table of Lusk (1928) and the non-protein respiratory exchange ratio (NPRER), exactly as previously reported (Acheson, Zahorska-Markiewicz, Pittet, Anantharaman & Jéquier, 1980; Ravussin, Schutz & Acheson, 1985; Vallerand et al, 1989a; Vallerand & Jacobs, 1989). After having determined protein oxidation via the analysis of the urinary urea nitrogen excretion (Sigma Chemicals, Kit 640, St Louis MO), rates of carbohydrate and lipid utilization (in $g \cdot min^{-1}$) were calculated using the non-protein oxygen consumption and the NPRER, as before (Acheson et al., 1980; Ravussin et al., 1985; Vallerand et al., 1989a; Vallerand & Jacobs, 1989).

The heat balance equation summarizes whole body heat exchange in terms of not only heat production but also in terms of the various avenues of heat loss (Tikuisis, McCracken & Radomski, 1991b; Vallerand, Savourey & Bittel, 1992a, Vallerand, Savourey, Hanniquet & Bittel, 1992b) and it is described below (all terms in $W.m^{-2}$):

$$\dot{M} - \dot{W} - (\dot{R}+\dot{C}) - \dot{E}_{persp} - \dot{C}_{resp} - \dot{E}_{resp} - \dot{S} - \dot{K} = 0 \qquad \text{(eq. 1)}$$

or

$$\dot{S} = \dot{M} - (\dot{R}+\dot{C}) - \dot{E}_{persp} - \dot{C}_{resp} - \dot{E}_{resp} \qquad \text{(eq. 2).}$$

where $\dot{M}$ is the average metabolic rate, $\dot{W}$ is the external work rate (zero in the present study), $\dot{R}+\dot{C}$ is the measured dry heat exchange, $\dot{E}_{persp}$ is the calculated evaporative heat loss from the skin via cutaneous perspiration, $\dot{C}_{resp}$ and $\dot{E}_{resp}$ are respectively the calculated convective and evaporative heat loss by the respiratory tract, $\dot{K}$ represents conductive heat loss (assumed negligible) and $\dot{S}$ is the rate of heat debt (determined as the balance of heat gains and heat losses). A negative $\dot{S}$ signifies a positive heat debt.

$\dot{R}+\dot{C}$ was measured by individually recalibrated heat flux transducers. To take into account the thermal resistance of the transducer itself, measured dry heat losses and skin temperatures were corrected as previously described (Ducharme & Frim, 1990). Determination of the last three variables $\dot{E}_{persp}$, $\dot{C}_{resp}$ and $\dot{E}_{resp}$ of Eq. 2 follow *method 2* in Tikuisis et al. (1991b). Due to the importance of an accurate heat balance analysis, the following is a brief review of these three estimated variables.

$$\dot{E}_{persp} = 0.06 \cdot 16.5 \cdot h_c (P_{sk} - P_a) \qquad \text{(eq. 3)}$$

where 0.06 (or 6%) is the minimum cold value of skin wettedness, 16.5 is the reciprocal of the psychrometer constant at sea level ($^{\circ}C.kPa^{-1}$), h_c is the convective heat transfer coefficient ($W.m^{-2}.^{\circ}C^{-1}$) deduced from the measured skin heat flux (Tikuisis, Bell & Jacobs, 1991a), P_{sk} is the saturated water vapour pressure at the skin surface (in kPa) and P_a is the ambient water vapour pressure (in kPa).

$$\dot{C}_{resp} = 0.001293 \cdot 0.28 \cdot \dot{V}E \cdot (T_{exp} - T_{db}) \cdot bsa^{-1} \qquad \text{(eq. 4)}$$

where 0.001293 is the air density in $kg.L^{-1}$ STPD, 0.28 is the specific heat of air in $W.h.kg^{-1}.^{\circ}C^{-1}$, $\dot{V}E$ is the ventilation in $L.h^{-1}$, T_{exp} is the expired air temperature and bsa is the body surface area in m^2.

$$\dot{E}_{resp} = 0.001293 \cdot 673 \cdot \dot{V}E \cdot (W_e - W_a) \cdot bsa^{-1} \qquad \text{(eq. 5)}$$

where 673 is the latent heat of vaporization in $W.h.kg^{-1}$ and W_e and W_a are the humidity ratios of expired air and ambient air given by:

$$W_e - W_a = 0.622 \cdot [P_{satexp}/(101 - P_{satexp}) - P_{satdb}/(101 - P_{satdb})] \quad \text{(eq. 6)}$$

where P_{satexp} and P_{satdb} are the saturated water vapour pressure of the expired air and ambient air respectively.

The heat debt in $kJ.kg^{-1}$ (S) was then obtained by the integration of the rate of heat debt ($\dot{S}$ in $W.m^{-2}$, as in eq. 2):

$$S = \int \{ \dot{M} - (\dot{R}+\dot{C}) - \dot{E}_{persp} - \dot{C}_{resp} - \dot{E}_{resp} \} \, dt \, / \text{mass} \; (kJ.kg^{-1}) \quad \text{(eq. 7)}.$$

PLASMA HORMONES AND SUBSTRATES

Blood was drawn from an indwelling catheter at about 5 min prior to the cold test, while subjects were sitting at a comfortable ambient temperature (22°C), and in the cold at min 60, 120 and 180 of tne cold exposure test. To ensure catheter patency after blood sampling, without using a heparin lock, we have used a slow infusion of warm isotonic saline (~1 $ml.min^{-1}$) coupled with regular flushes of warm saline. A temperature controller was employed to maintain the insulated saline bag and insulated i.v. line at about 37°C. Venous blood samples (12 ml) were collected into chilled EDTA tubes (5 ml) and heparinized tubes with glutathione and EGTA (7 ml). Thereafter, the plasma was promptly separated by centrifugation, and kept frozen at -70°C. Plasma was assayed for glycerol (Boobis & Maughan, 1973), FFA (Wako Chemicals NEFA kit, Dallas TX), glucose (YSI Instruments Glucose Analyzer, Yellow Springs OH), and insulin (Pharmacia Diagnostics, Insulin kit, Uppsala Sweden). Plasma values were corrected for changes in plasma volume according to changes in haemoglobin (Sigma Chemicals kit #545, St Louis MO) and hematocrit (Dill & Costill, 1974)

STATISTICS

The main effects of time and treatments as well as time vs treatment interactions were tested by repeated measures ANOVA on all data (Biomedical Computer Programs, BMDP-90, Los Angeles, CA).

ANOVA were corrected by the Huynh-Feldt epsilon adjusted degrees of freedom when the sphericity test was significant (BMDP-90). When interactions of effects were shown significant, paired t-tests (adjusted for multiple comparisons) were used to locate significant differences (Glantz, 1981). Results are expressed as mean ± standard error of the mean (SEM).

RESULTS

Table 1. Subject Characteristics

	$\bar{x}$ ± SEM	Range
Age (Yr)	33.5 ± 1.3	[26.0 - 37.0]
Height (m)	1.79 ± 0.01	[1.76 - 1.84]
Body Mass (kg)	86.3 ± 3.6	[72.0 - 98.0]
VO_2 max ($ml \cdot min^{-1} \cdot kg^{-1}$)	43.0 ± 2.4	[32.4 - 49.6]
Body fat (%)	18 ± 1	[11 - 22]
N = 8		

Subjects were eight healthy male volunteers whose standard physical characteristics are described in Table 1. All subjects were able to complete the 3 h test in both the placebo and Cold Buster™ conditions. Even though the cold was too mild to cause a significant drop in rectal temperature (T_{re}), it was sufficient to reduce $\bar{T}_{sk}$ by about 8.5°C (Fig. 1) and increase oxygen consumption in both conditions (Fig. 2A; $P<0.05$). However, there were no differences between tests.

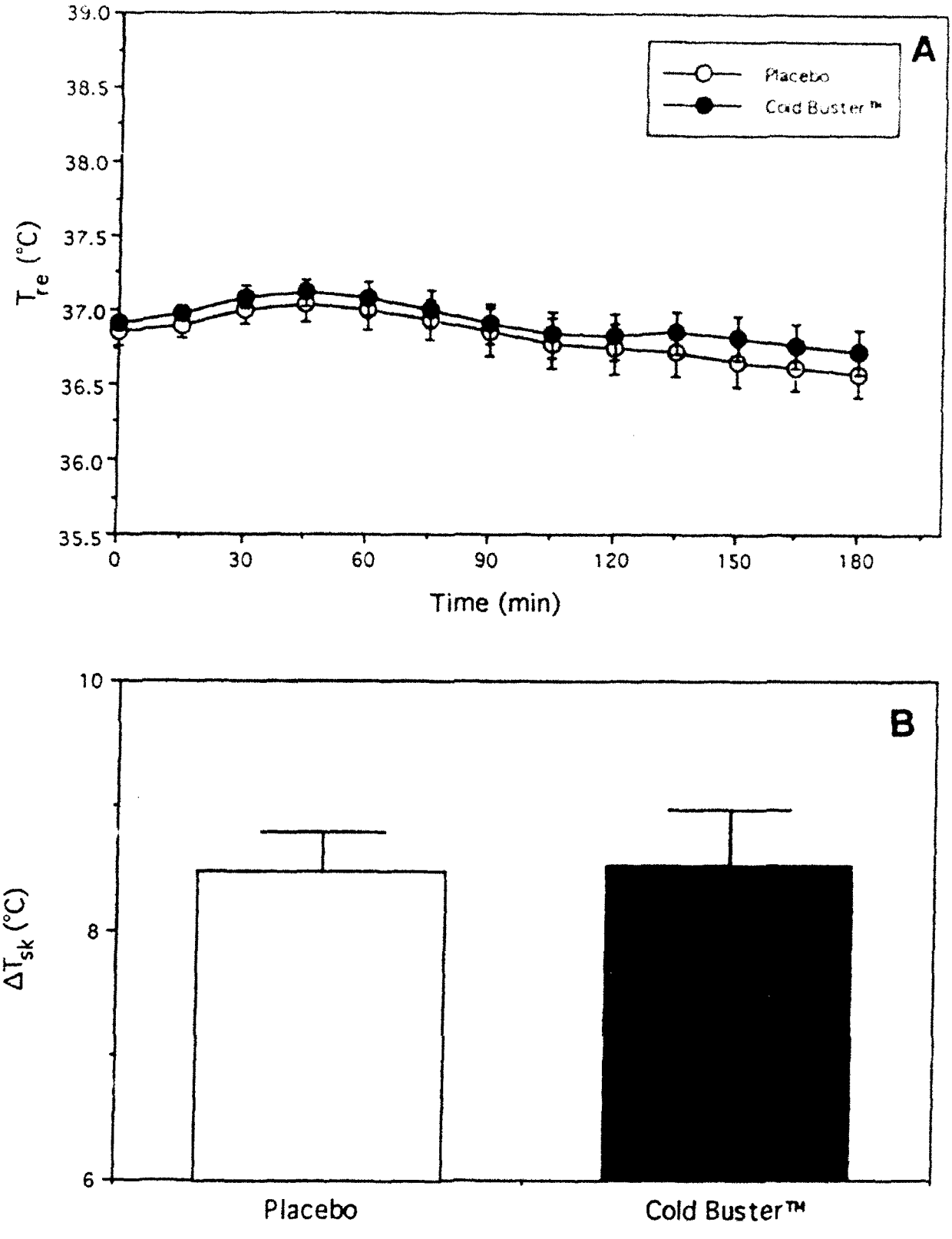


Figure 1: Rectal temperature (T_{re}; Fig. 1A) profile and drop in mean skin temperature ($\overline{T}_{sk}$; Fig. 1B), in the cold (3h at 10°C; 0.8-1.0 $m.s^{-1}$ wind speed, 50%r.h.) following the ingestion of a placebo or a high-CHO Cold Buster™ Sports bar. Results are expressed as mean±SEM (n=8). There were no differences between treatments.

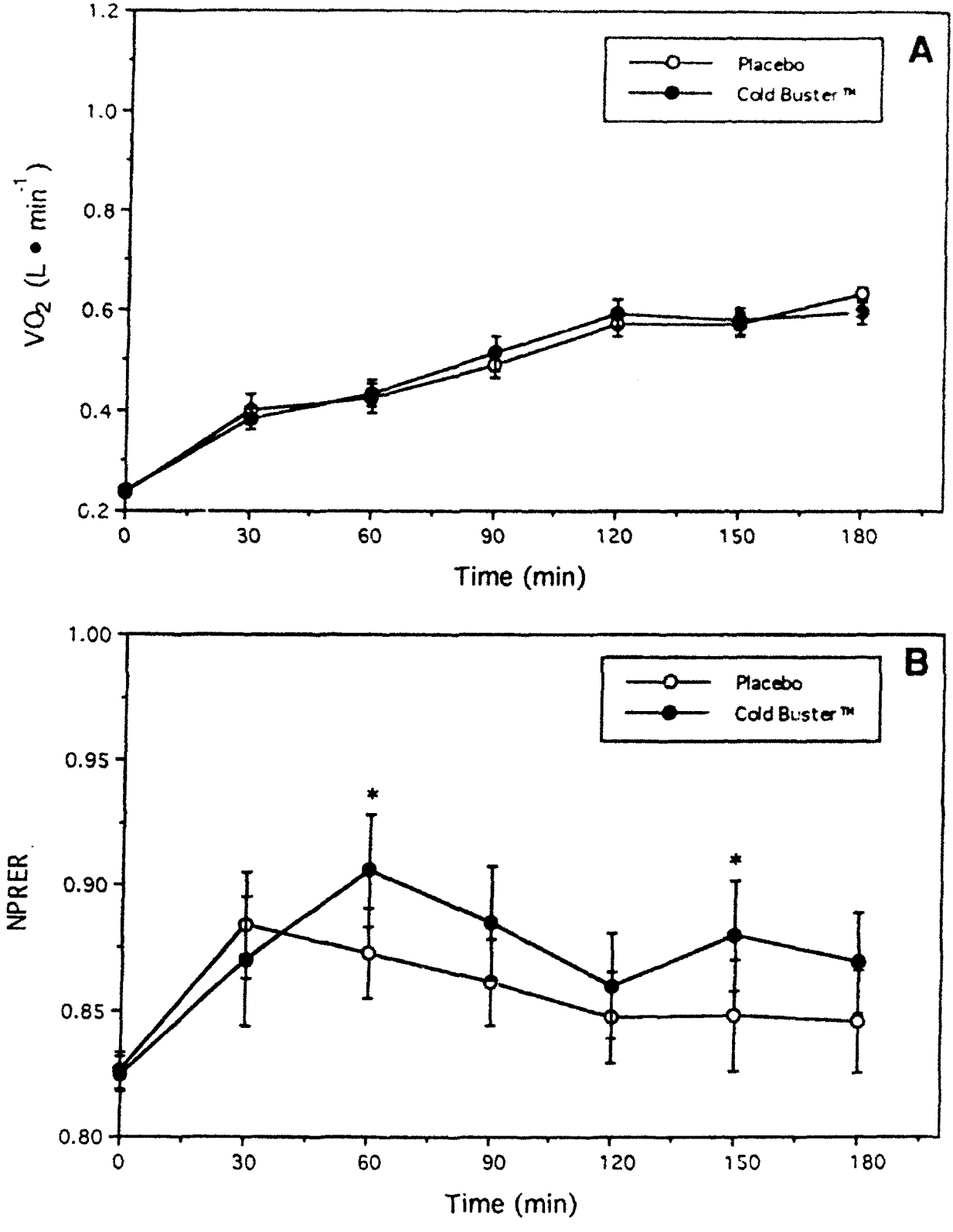


Figure 2: Oxygen consumption (in $L.min^{-1}$; STPD; Fig. 2A), and non protein respiratory exchange ratio (NPRER; Fig. 2B) during the 3h cold test. The symbol "*" indicates a significant difference from the placebo condition ($P<0.05$).

An ANOVA for repeated measures revealed that NPRER was affected by a significant main effect of time and time by treatment interaction. Paired t-tests indicated that the Cold Buster™ NPRER were higher than those of the placebo at min 60 and 150 (Fig. 2B; $P<0.05$). Consequently, it affected the rates of substrate oxidation. Similar to the NPRER above, rates of CHO oxidation were influenced by a significant main effect of time and a time by treatment interaction. On average, the Cold Buster™ treatment produced rates of CHO oxidation that were slightly higher than those of the placebo at several points in time, but it reached significance only at min 150 ($P<0.05$; Fig. 3A). The opposite was observed with fat oxidation, which was correspondingly reduced with the ingestion of the bar (Fig. 3B, n.s.). Protein oxidation was not affected by the ingestion of the bar (0.052 ± 0.004 $g.min^{-1}$) compared to the placebo (0.059 ± 0.003 $g.min^{-1}$).

Metabolic rate was about 38 $W.m^{-2}$ in our fasted resting subjects under comfortable ambient temperatures (prior to entering the chamber; Fig. 4A). By min 120 of the cold exposure, $\dot{M}$ had increased to about 95 $W.m^{-2}$ where it remained relatively stable for the last 60 min (Fig. 4A), representing an increase of about 2.5 times resting values. Again, there were no differences between treatments. Further, since there were no differences between treatments in the rates of dry ($\dot{R}+\dot{C}$), wet ($\dot{E}_{persp}$) and respiratory heat loss ($\dot{C}_{resp}+\dot{E}_{resp}$) (values at min 180 depicted in Table 2), there were no main effect of treatment and no interaction of effects with respect to the heat debt profiles shown in Fig. 4B.

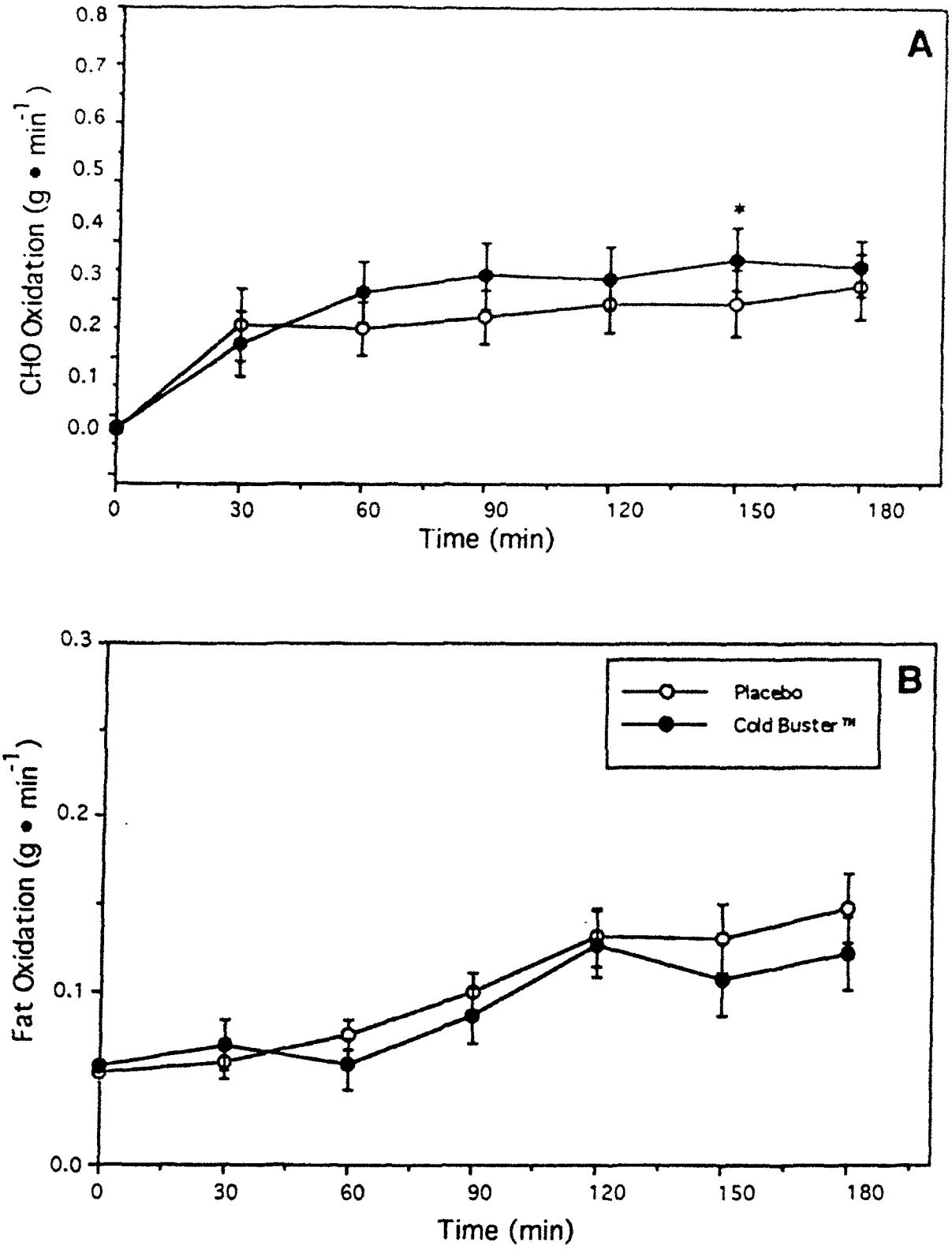


Figure 3: Rates of carbohydrate (CHO; Fig. 3A) and lipid oxidation in the cold (Fig. 3B). Symbol for statistical differences is as in Fig. 2.

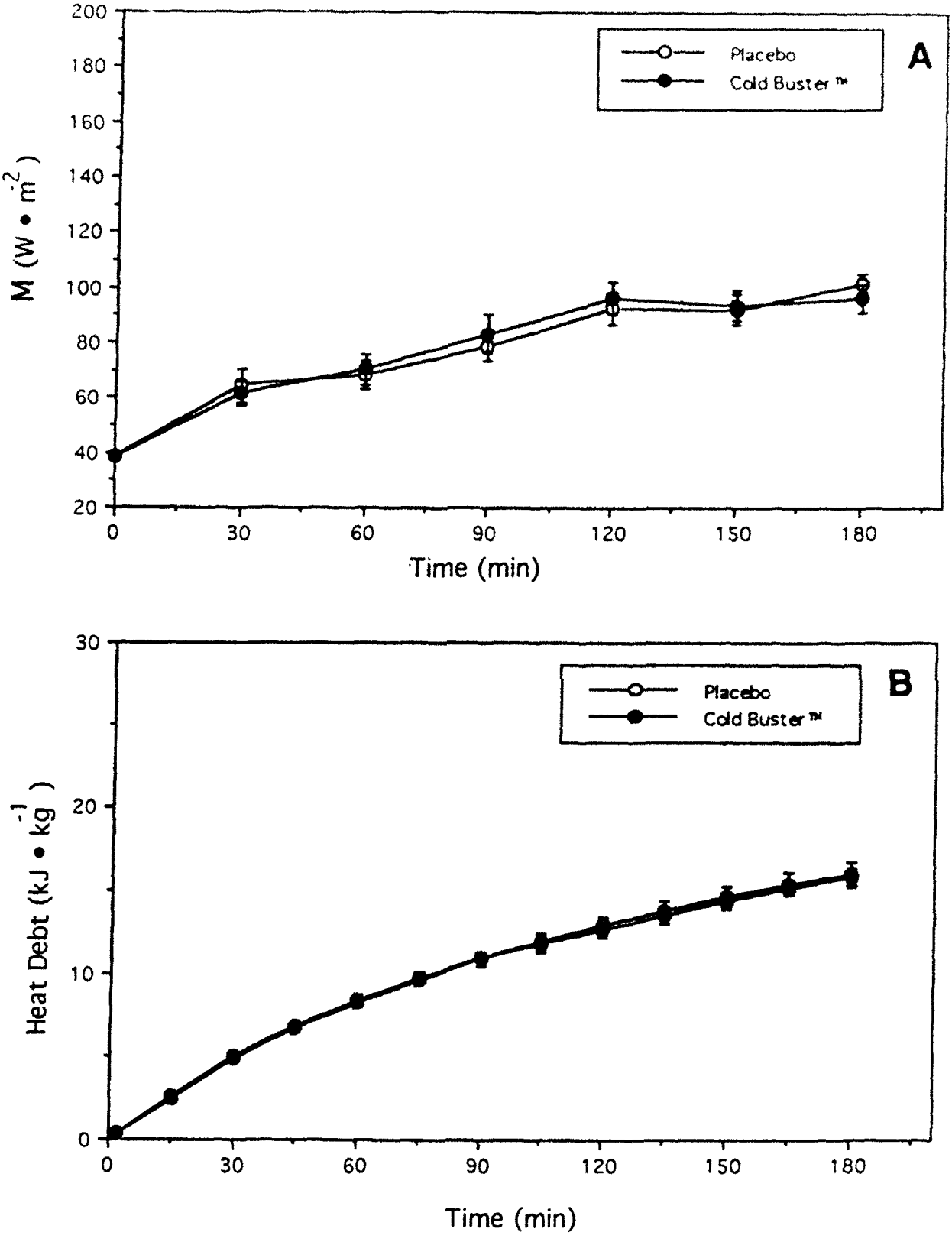


Figure 4: Metabolic rate (M; Fig. 4A) and heat debt (S; Fig. 4B) during the 3h cold test.

Table 2. Heat Balance analysis at min 180 of cold exposure test.

		$\dot{M}$	$\dot{R}+\dot{C}$	$\dot{E}_{persp}$	$\dot{C}_{resp}$	$\dot{E}_{resp}$	$\dot{S}$
				($W \cdot m^2$)			
min 180	Placebo	+101.5 ±3.3	-115.7 ±3.5	-5.6 ±0.3	-3.2 ±0.3	-7.3 ±0.6	-30.3 ±4.5
	Cold Buster™	+96.0 ±4.9	-113.8 ±4.9	-5.3 ±0.5	-3.0 ±0.2	-6.7 ±0.5	-32.8 ±3.2

Plasma insulin levels were affected by significant main effects of time, treatments and by a time by treatments interaction. Basal plasma insulin levels were similar between treatments prior to the cold (Fig. 5A). Whereas the placebo treatment slightly reduced basal levels, the Cold Buster™ triggered a significant increase of insulin levels during the test, which peaked at min 120 with an almost 4-fold rise ($P<0.05$). This is likely the result of the corresponding increment of plasma glucose levels that occurred at the same time ($P<0.05$; Fig. 5B). Plasma glucose were indeed affected by a significant time by treatment interaction. Although plasma FFA levels were greatly reduced by the Cold Buster™ throughout the 3 h cold test (significant main effect of time and treatments, $P<0.05$), plasma glycerol levels were reduced to a much lesser extent (n.s.; Fig. 6). Finally, there were no differences in either heart rate or in the subjective ratings of discomfort, sense of shivering and thermal preference (data not shown).

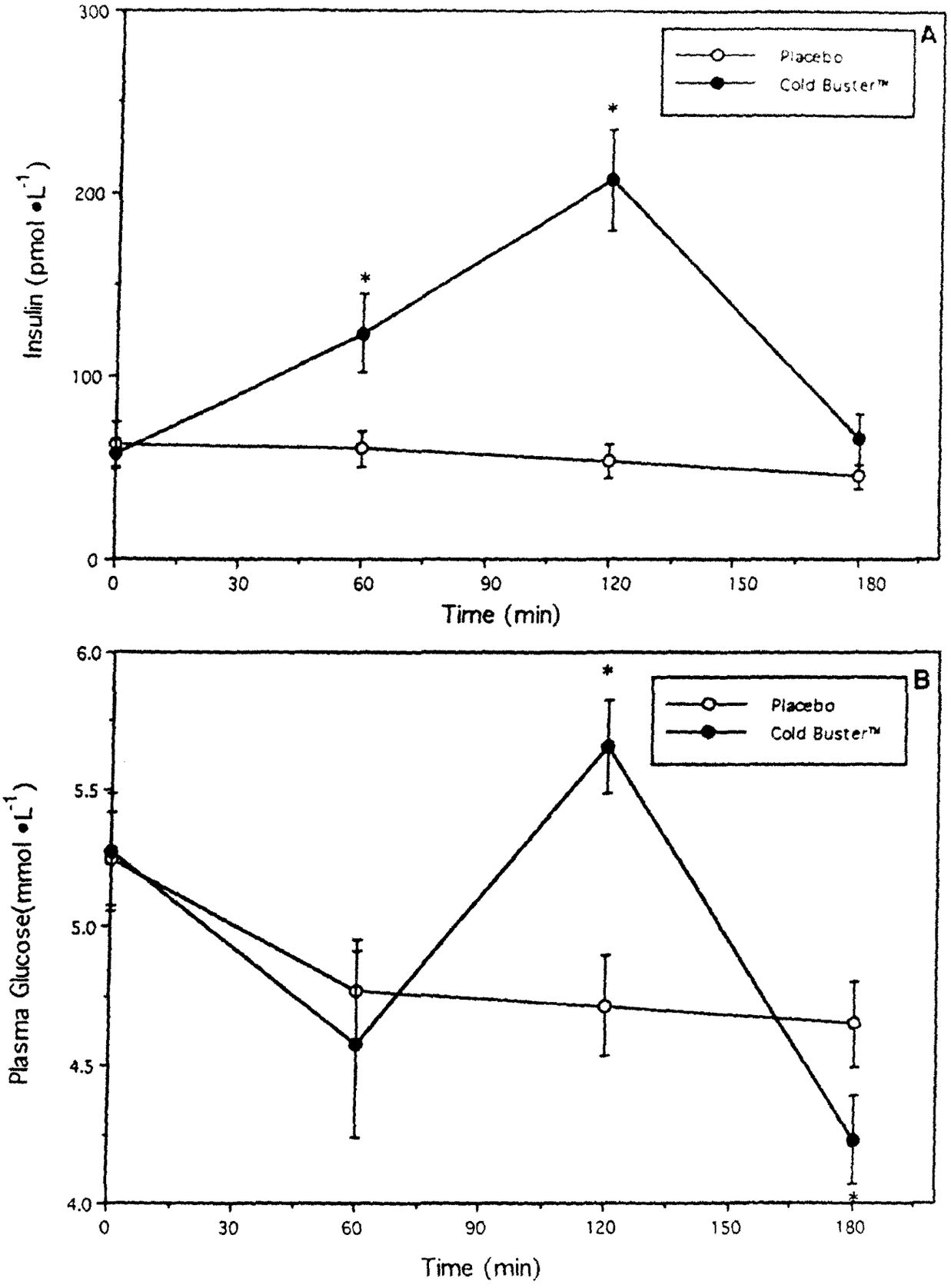
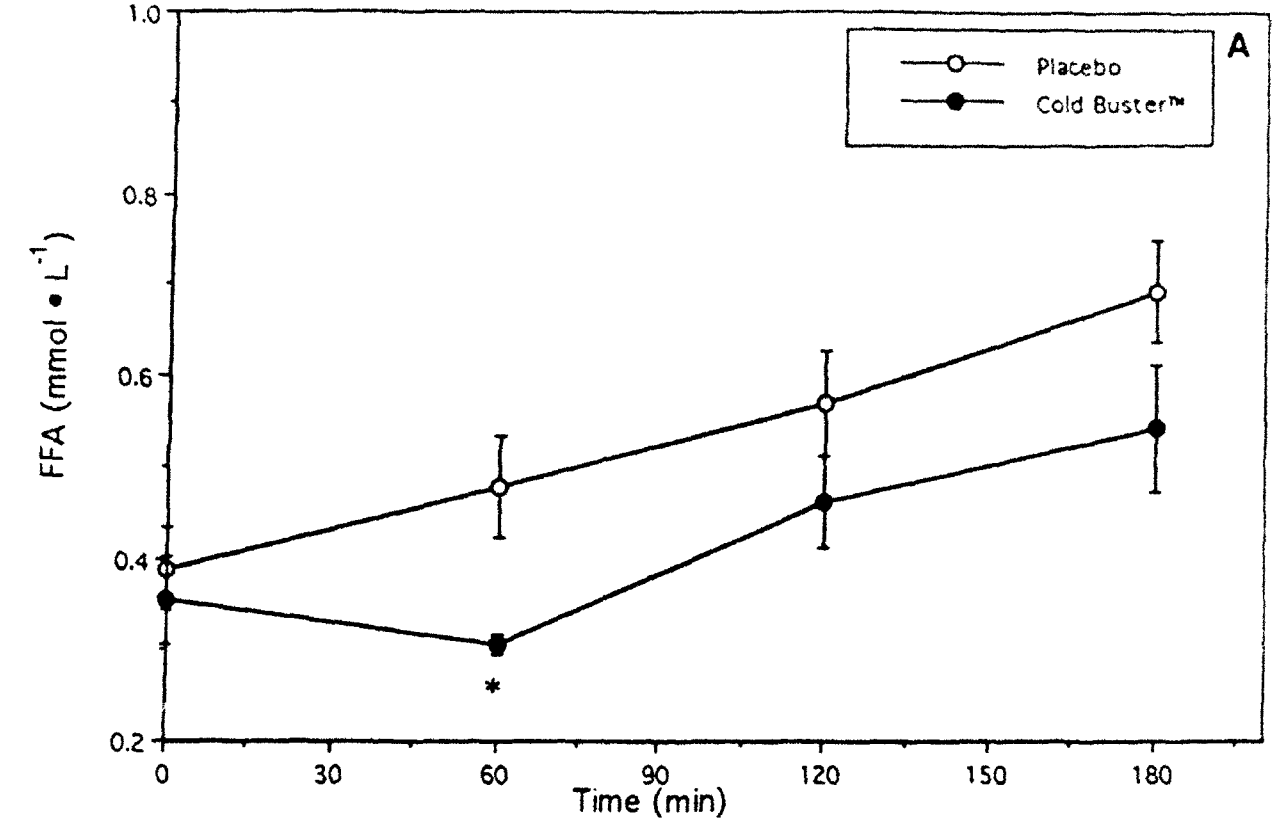


Figure 5: Plasma levels of insulin (Fig. 5A) and glucose (Fig. 5B) during the 3h cold test. Symbol for statistical differences is as in Fig. 2.



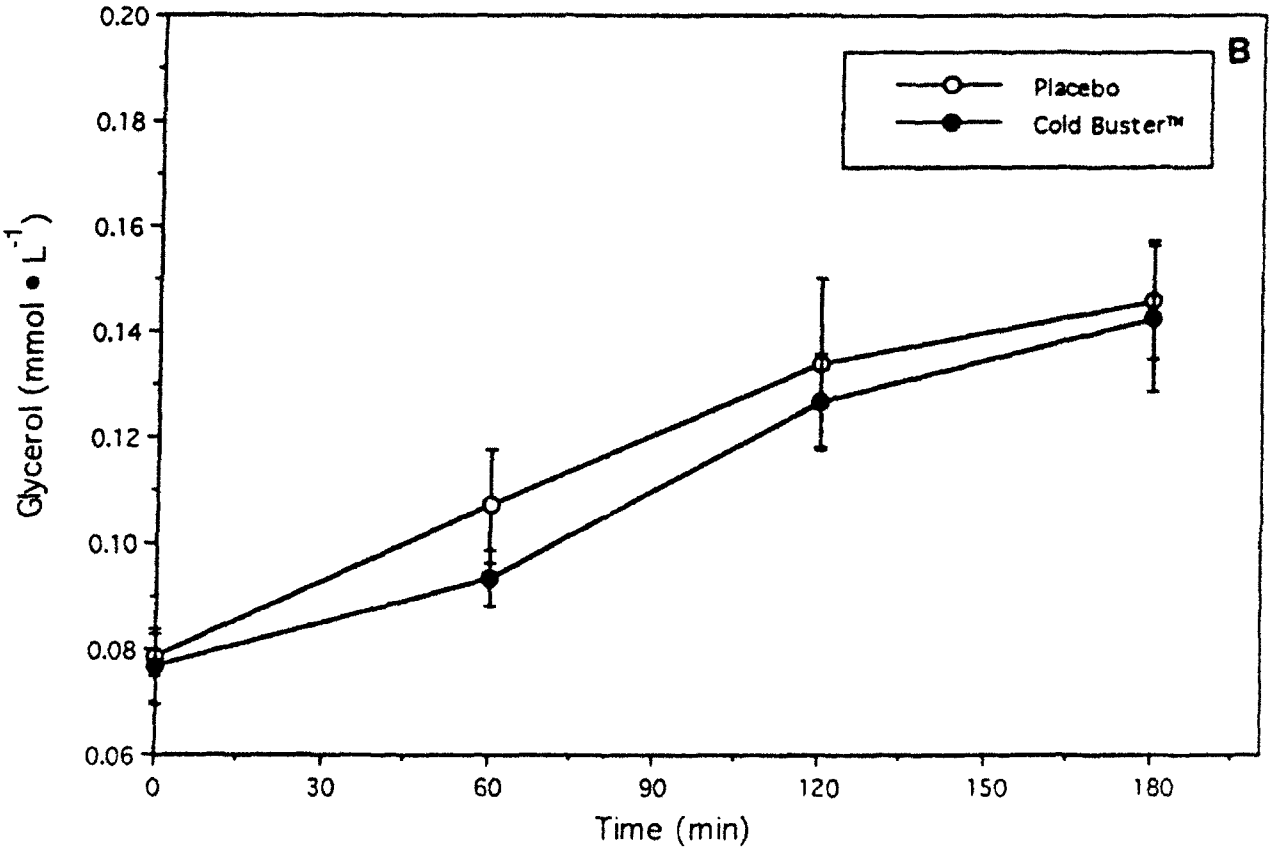


Figure 6: Plasma levels of free fatty acids (FFA; Fig. 6A) and glycerol (Fig. 6B) during the 3h cold test. Symbol for statistical differences is as in Fig. 2.

DISCUSSION

The results of the present study confirm our previous results that the ingestion of the Cold Buster™ does not alter cold resistance (Vallerand et al., In Review) and extend these results to a milder cold stress. Further, the conclusion remains unchanged whether the results were analyzed by a thermometric analysis (via body temperatures) or by a calorimetric analysis (via heat balance). In thermophysiological studies, we firmly believe that it is now essential to report both types of analyses, particularly the latter, because a change in body temperatures originates *from* an imbalance between heat gains and heat losses, known as heat debt or heat storage (Tikuisis et al., 1991b; Vallerand et al., 1992a, 1992b). With respect to the Cold Buster™ and these two types of analyses, we were not able to document any differences in the rates of heat production, heat loss, heat debt or even in the drop of body temperatures, in comparison with the placebo (Figs. 1, 2, 4).

We have reviewed unpublished data by Dr. L.C.H. Wang, the inventor of the Cold Buster™ (personal communication). His data (core temperature only) conflict with ours and suggest that ingestion of the bar (same feeding protocol as in the present study) reduces significantly the rate at which rectal temperature decreases during cold exposure by about 50% (ΔT_{re} of about -0.7 vs -1.4°C; Wang LCH, personal communication). It must be pointed out that these are the only data available so far to support this claim and that a heat balance analysis was not performed. As stated above, we consider such an analysis essential to explain whether this particular improvement in the drop of T_{re} is due to either a greater $\dot{M}$ and/or reduced heat loss. A heat balance analysis is much more informative than a simple core temperature profile, which has been known to vary out of proportion to the calorimetric heat debt (or heat storage) (Tikuisis et al., 1991b; Vallerand et al., 1992a, 1992b). The use of core temperature itself as an index of cold resistance becomes problematic for instance, whenever two (or more) sites of core are measured and they differ, as reported (Bittel, Livecci-Gonnot, Hanniquet, Poulain & Etienne, 1989; Livingstone, Grayson, Frim, Allen & Limmer, 1983; Vallerand et al., 1992a, 1992b).

Without the information provided by the heat balance, it is very difficult to try to explain the different findings between Wang's and our studies. However, one possible explanation is that the experimental conditions differed appreciably between studies. Our subjects rested during the entire cold exposure at 10°C. Wang's subjects interspersed 10 minutes of light exercise after every 20 minutes of rest during their cold exposure (3 h with gradual cooling to below freezing temperatures). Therefore Wang's protocol produced a higher $\dot{M}$ and required a greater mobilization of endogenous energy substrates, two conditions which could be important. However, we feel that this is unlikely since we have already used an identical intermittent exercise protocol in the cold (3h at 0°C; Vallerand & Jacobs, unpublished) with results similar to the present study. When these subjects were fed a high-CHO xanthine-containing cookie (300 kcal) in the cold (Vallerand & Jacobs, unpublished), the data on $\dot{M}$, S, T_{re} and substrate oxidation were almost a carbon copy of the present Figs. 1-4, and our previous Cold Buster™ trial (Vallerand et al., In Review).

In summary, the present results demonstrate that ingesting the Cold Buster™ does not significantly alter thermoregulatory thermogenesis, heat losses, heat debt or body temperatures in resting subjects during a 3 h exposure at 10°C. In addition, our data do not support the theory that energy mobilization is a limiting factor for thermoregulatory thermogenesis, since oxidation and mobilization (in plasma) of one substrate were significantly increased entirely at the expense of the mobilization and oxidation of another.

RECOMMENDATIONS

In conclusion, our recent studies (Vallerand et al., In Review; Vallerand & Jacobs, unpublished) as well as the present one, do not provide any evidence that the procurement of the Cold Buster™. would be more beneficial to Canadian Forces operations in the cold than would the procurement of any other foodstuff with a similar energy content or composition.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the assistance of Ms. Debbie Kerrigan-Brown, Mr. Jerry Laufer and Mr. Kevin McDonald. Medical coverage was kindly provided by Lt Cdr Bill Bateman. Our thanks are also extended to Mr. Jan Pope for performing maximal aerobic power tests and body fat determinations. We would like to acknowledge Dr. L.C.H. Wang for kindly sharing some of his data with us.

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(Security classification of title, body of abstract and indexing annotation must be entered when the overall document is classified)

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Influence of the Cold Buster Sports Bar on Heat Debt, Mobilization and Oxidation of Energy Substrates

4 DESCRIPTIVE NOTES (the category of the document, e.g. technical report, technical note or memorandum. If appropriate, enter the type of report, e.g. interim, progress, summary, annual or final. Give the inclusive dates when a specific reporting period is covered.)

DCIEM Report No. 92-60

5 AUTHOR(S) (Last name, first name, middle initial. If military, show rank, e.g. Doe, Maj. John E.)

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6 DOCUMENT DATE (month and year of publication of document)	7a NO. OF PAGES (total containing information. Include Annexes, Appendices, etc.)	7b NO. OF REFS (total cited in document)
November 1992	32	24

8a PROJECT OR GRANT NO. (if appropriate, the applicable research and development project or grant number under which the document was written. Please specify whether project or grant)	8b CONTRACT NO. (if appropriate, the applicable number under which the document was written)
51CA-17	

9a ORIGINATOR'S DOCUMENT NUMBER (the official document number by which the document is identified by the originating activity. This number must be unique to this document.)	9b OTHER DOCUMENT NO.(S) (Any other numbers which may be assigned this document either by the originator or by the sponsor)

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In a recent study, we have shown that the commercially available Cold Buster™ Sports bar, purported to improve cold resistance, did not do so in our subjects exposed to a relatively severe cold test. (Vallerand, Tikuisis, Ducharme & Jacobs, In Review). One possible explanation for our conflicting results is that our metabolic rate (M) was too high for the possibly small thermogenic effect of the bar to be measurable. The goal of this study was therefore to re-evaluate, under milder conditions, the influence of the Cold Buster™ on heat balance (heat debt = heat production - heat losses) and body temperatures. Eight semi-nude fasted subjects were exposed to the cold (3h at rest, 10°C, <0.4 m.s-1 wind) on two occasions following the ingestion of either a placebo (100 ml water) or a Cold Buster™ (all feedings at min 0 and 90). As a result of the cold, M, dry heat losses and heat debt (S) increased whereas mean skin temperature decreased ($P<0.05$). Rectal temperature remained unchanged due to the mild cold. In all of the above parameters, there were no differences between treatments. Ingestion of the Cold Buster™ significantly increased carbohydrate oxidation at min 150 compared to the placebo ($P<0.05$). However, this was without impact on M, since it occurred entirely at the expense of fat oxidation (n.s.). Interestingly, the Cold Buster™ increased plasma glucose levels after 2h into the cold ($P<0.05$), a phenomenon which appears to have triggered a large increase in insulinemia ($P<0.05$) This secretion of insulin seems to have blunted lipid mobilization since it significantly reduced plasma free fatty acids levels ($P<0.05$). The results confirm previous data where the ingestion of the Cold Buster™ Sports bar did not alter heat production, heat losses, heat debt or even body temperatures, and extend these observations to a mild cold stress. Although the Cold Buster™ enhanced CHO mobilization and oxidation, this phenomenon occurred entirely at the expense of mobilization and oxidation of lipids. Taken together, these studies do not provide any evidence to support a recommendation for the use of the Cold Buster™ Sports bar.

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Body temperatures, carbohydrate metabolism, heat balance, heat production, rectal temperature.