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Adaptive Thermogenesis Is Intact in B6 and A/J Mice Studied at Thermoneutrality

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ADAPTIVE THERMOGENESIS IS INTACT IN B6 AND
A/J MICE STUDIED AT THERMONEUTRALITY

By

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A Thesis submitted to the
Department of Nutrition, Food and Exercise Sciences
in partial fulfillment of the
requirements for the degree of
Master of Science

Degree Awarded:
Fall Semester, 2003

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ABSTRACT

We determined the influence of thermoneutrality on the oxygen consumption (VO_2) and heart rate (HR) responses to short-term moderate fat feeding in male C57BL/6J (B6; obesity-prone) mice and A/J (obesity-resistant) mice. Mice were adapted to either $T_a=23^\circ\text{C}$ or $T_a=30^\circ\text{C}$ for at least 5 weeks, implanted with telemetry devices, and housed in metabolic chambers for measurement of food intake, VO_2 , HR, mean arterial pressure (MAP), and locomotor activity. Access to moderate fat diet resulted in similar increases in normalized VO_2 at both $T_a=23^\circ\text{C}$ (B6 Δ : 2.7 ± 0.4 ml/min/kg^{0.75}; A/J Δ : 3.8 ± 0.5 ml/min/kg^{0.75}) and $T_a=30^\circ\text{C}$ (B6 Δ : 2.4 ± 0.4 ml/min/kg^{0.75}; A/J Δ : 4.4 ± 0.6 ml/min/kg^{0.75}) within each respective strain. However, the A/J mice (Δ : 4.1 ± 0.4 ml/min/kg^{0.75}) had an enhanced metabolic response compared to the B6 mice (Δ : 2.5 ± 0.3 ml/min/kg^{0.75}). Tachycardia following the nutritional intervention occurred at $T_a=30^\circ\text{C}$, and was similar in magnitude in B6 mice (Δ : 55 ± 10 beats/min at $T_a=30^\circ\text{C}$ vs. 12 ± 8 beats/min at $T_a=23^\circ\text{C}$) and A/J mice (Δ : 61 ± 15 beats/min at $T_a=30^\circ\text{C}$ vs. 25 ± 18 beats/min at $T_a=23^\circ\text{C}$). By the third day of access to moderate fat diet, caloric intake for A/J mice returned to baseline chow levels. However, the B6 mice remained hyperphagic throughout the 7 days of moderate fat feeding. The results demonstrate that an enhanced ability to engage adaptive thermogenesis is one mechanism utilized by the A/J mice to prevent obesity. Because the thermogenic response was observed in mice chronically adapted to thermoneutrality, we conclude it is not likely mediated by brown adipose tissue. Finally, the observation that the cardiovascular responses to short term moderate fat feeding in mice were more evident at thermoneutrality suggests that rodent studies examining the role of obesity on cardiovascular disease should be performed at thermoneutral temperatures.

INTRODUCTION

Several lines of evidence support the hypothesis that adaptive thermogenesis, mediated by increases in sympathetic activity, is a critical mechanism of resistance to obesity (1-10). Support for the critical role of the sympathetic nervous system mediating adaptive thermogenesis comes from studies utilizing β -adrenergic receptor (AR) blockade prior to overfeeding, demonstrating that the elevation in oxygen consumption (VO_2) is prevented in rats and mice (5-7). In addition, the lack of resistance to obesity in the $\beta_{1,2,3}$ -AR knockout mouse demonstrates the importance of the sympathetic nervous system mediating obesity resistance (8). Both rodents (6,9) and humans (10) possess various abilities to engage adaptive thermogenesis in order to defend body weight. Thus, the ability to engage adaptive thermogenesis is likely to be genetic (11) and mediates the degree of obesity resistance in certain individuals.

The C57BL/6J (B6) and A/J mouse strains are a valuable tool for studying natural genetic differences in obesity resistance. The B6 mouse, an established model for the study of diet-induced obesity, has been shown to develop obesity, hyperglycemia, and hyperinsulinemia following long-term access to higher fat diets (12-17). In contrast, the A/J mouse strain is resistant to obesity over the same period of time although it has similar food intake as the B6 mice (12-16). These differences arise although both the B6 and A/J mouse strains are consuming similar amounts of calories. This observation suggests potent mechanisms for obesity resistance in A/J mice. Given that sympathetic activation appears to mediate obesity-resistance, one purpose of this study was to examine the cardiovascular response to short-term moderate-fat feeding in the A/J and B6 mouse strains.

We have previously demonstrated that ambient temperature (T_a) plays a critical role for food intake, VO_2 , heart rate (HR), and mean arterial pressure (MAP), as they are elevated in rodents at standard lab temperatures ($T_a=23^\circ\text{C}$) compared to thermoneutrality

($T_a \approx 30^\circ\text{C}$) (18-20). Thermoneutrality, typically $T_a = 28^\circ\text{C}$ - 31°C for rodents, is the temperature where energy expenditure to maintain body temperature is at a minimum (21). Primarily, the decrease in metabolic rate at thermoneutrality is mediated by decreased sympathetic nervous system activity (SNA) to brown adipose tissue (BAT) (22-24); BAT is a thermogenic organ that is activated by sympathoexcitation in response to overfeeding in rodents (3,4,7,25). Therefore, it is hypothesized that the sympathetic response to overfeeding in mice will be attenuated at thermoneutrality. Thus, the overall purpose of this study is to elucidate the thermogenic and cardiovascular responses to short-term overfeeding at both $T_a = 23^\circ\text{C}$ and $T_a = 30^\circ\text{C}$ in the B6 and A/J mouse strains as determined by VO_2 and HR. We hypothesize that obese-resistant A/J mice will exhibit a greater increase in VO_2 and HR compared to the B6 mice following access to moderate fat diet. It is hypothesized that at thermoneutrality there will be a blunted thermogenic response following overfeeding in both strains, with the responses of A/J mice more closely resembling B6 mice.

METHODS

Male C57BL/6J (n=18) and A/J (n=14) mice were obtained from Jackson Labs (Bar Harbor, ME). Mice from each strain were housed at either standard lab temperature ($T_a=23^{\circ}\text{C}$), or at thermoneutrality ($T_a=30^{\circ}\text{C}$) for at least five weeks prior to data collection. At 16-24 weeks of age, mice were anesthetized with halothane (1-2% in 95% oxygen-5% nitrogen mixture) and surgically instrumented with telemetry devices (TA11PA-C20, Data Sciences International, St. Paul, MN) in the right common carotid artery for measurement of cardiovascular function (26-28) as described previously (29). A/J mice were injected subcutaneously with atropine (1.0 ml/100 g body weight of 0.3 mg atropine/mL saline) following induction with halothane prior to any surgical manipulations, and immediately post-surgery. Atropine was used to minimize excessive airway secretions only present in the A/J mice. Mice were housed individually in standard polycarbonate cages to allow recovery after surgery. Body weight, food and fluid intake, and general behavioral appearance were monitored daily to assess the effects of surgery. In addition to standard chow and water *ad libitum*, a liquid diet of chocolate Ensure™ was placed in the cage for 3 days following surgery. We have found this liquid diet to improve recovery rates in mice. Only animals that appeared completely healthy and had regained body weight were studied. Following recovery from surgery (≈ 7 days), mice were transferred to larger cages (18 X 9.5 X 5 in) and provided *ad libitum* access to deionized water and powdered chow (Purina 5001; 3.3 kcal/g) contained within a stainless steel feeder that minimized spillage. The cages were placed within previously described environmental chambers (30) that provided computer control of T_a (23°C or 30°C) and the 12-h light/dark schedule. Body weight (corrected for transmitter weight of 3.3 g) and food and water consumption were measured daily during the 12th hour of the light phase.

The cages were made with a near air-tight seal for continuous measurement of VO_2 (ml/min) and carbon dioxide production (VCO_2 ; ml/min) using an approach previously described (31). The cages were set with a fixed constant flow rate of 0.5 l/min. Air was dried and compressed prior to sampling every 4 min for 30s. Open circuit respirometry was used to determine VO_2 and VCO_2 by modifying the approach of Bartholomew et al. (32).

A custom designed force platform with stiff strain-gauge load-beam transducers attached under two adjacent corners was used to measure locomotor activity. The cage was positioned on the platform with a pivot under its center to determine locomotor activity as previously described (31). Total locomotor activity, with a 1-mm resolution, was accumulated in 30-s bins and measured in meters.

A telemetry receiver (model RPC-1, Data Sciences) was placed under the mouse platform within the metabolic chambers used for metabolic data collection. As described previously (31), MAP and HR were calculated from the direct measurement of systolic blood pressure (SBP), diastolic blood pressure (DBP) and interbeat interval (IBI) for each 30-s bin and stored for off-line analysis.

Following adequate acclimation to the testing chambers ($T_a=23^\circ\text{C}$ or $T_a=30^\circ\text{C}$), a 3-day baseline period was implemented for all experiments. During acclimation and baseline, mice were given *ad libitum* access to powdered chow (4.5 % fat, 3.3 kcal/g, Purina) and deionized water. Following a baseline period mice were given a moderate fat diet (32% fat, 4.41 kcal/g, Research Diets) for 7 days.

Metabolic data were collected and stored in 4-min bins. Cardiovascular and locomotor activity were collected and stored in 30-s bins. Average light-phase data were based on 11-h means (daily maintenance procedures were performed during the last hour of the light cycle), while dark-phase data were based on 12-h means.

Results are reported as mean $\pm$ S.E.M. Differences in means between groups (baseline, days 1 and 7 of moderate fat diet) were assessed by 1-way ANOVA. The Scheffé post hoc test was used for comparison between groups when significant F ratios were observed. The effect of moderate fat diet over time within each respective group was assessed by repeated measures ANOVA. Tukey's post hoc tests were performed to determine significant differences between means at specific time points following the

intervention of moderate fat diet. The Student's t-test was used to analyze deltas between groups with the Bonferroni correction method applied for multiple comparisons. $P < 0.05$ was accepted as significant.

RESULTS

Body weight, caloric, and water intakes

B6 mice were heavier than A/J mice, with no effect of T_a on body weight (Figure 1A, Table 1). B6 mice at $T_a=23^\circ\text{C}$ consumed more calories at baseline than B6 mice housed at $T_a=30^\circ\text{C}$ (Figure 1B, Table 1). However, there were no strain differences in food intake at baseline (Table 1). All groups decreased fluid intake transiently throughout the intervention (data not shown), although B6 mice at $T_a=30^\circ\text{C}$ continued to have decreased fluid intake for all days following moderate fat diet (Table 1). Following access to moderate fat diet, there was no effect of ambient temperature on caloric intake, although marked strain differences arose by day 3 (Figure 1B). The A/J mice only transiently increased food intake, but the B6 mice increased caloric intake throughout the 7 days.

VO_2 , RQ and locomotor activity

During baseline conditions, dark period VO_2 was elevated at $T_a=23^\circ$ in B6 vs. A/J mice (Figure 2A, Table 2A). However, when normalized for metabolic body mass, we observed no baseline strain differences in the dark or light phase VO_2 (Figure 2C and 2D, Table 2A and 2B). Housing mice at $T_a=30^\circ\text{C}$ reduced baseline VO_2 (Table 2A and 2B) in both strains compared to $T_a=23^\circ\text{C}$. Although baseline VO_2 was reduced at $T_a=30^\circ\text{C}$, access to moderate fat diet elevated VO_2 across time (Figure 2A-D) for all groups, independent of temperature. The magnitude of increase for normalized VO_2 was analyzed by the increase from the 3-day baseline on day 1 and day 7 of moderate fat diet during the light phase. This was to prevent any effect of locomotor activity on VO_2 , since locomotor activity was similar in all groups during the light phase (Table 3A and 3B). There were no temperature differences in the magnitude of increase within strain, so data from both temperatures were pooled for the B6 and A/J mouse strains. The

magnitude of increase for normalized VO_2 (Figure 3) was higher in the A/J mice compared to the B6 mice on day 1, but not day 7 of moderate fat diet.

Baseline RQ was similar for all groups in both the light and dark cycles (Table 2A and 2B). Light phase RQ increased in all groups at day 1, although by day 7 all groups had returned to basal levels (Figure 2F). However, the B6 mice at $T_a=30^\circ\text{C}$ increased light-phase RQ from days 1-5, while all other groups increased RQ for only one day. Dark phase RQ increased at day 1 at both temperatures for B6 mice, although only B6 at $T_a=23^\circ\text{C}$ remained different from baseline by day 7 (Figure 2E).

Light phase locomotor activity was not different between groups or across time during the experiment (Table 3B). However, at both temperatures, B6 mice had elevated dark phase baseline locomotor activity compared to A/J mice (Table 3A). All groups except B6 mice at $T_a=23^\circ$ demonstrated increases in dark phase locomotor activity at day 1, although activity returned to baseline levels by day 7 for all groups.

Heart rate and blood pressure

Baseline dark phase MAP was elevated in both B6 and A/J mice at $T_a=23^\circ$ compared to B6 and A/J at $T_a=30^\circ\text{C}$ (Table 3A). No blood pressure changes occurred across time during the dark phase. Dark phase baseline HR was elevated in the A/J vs. B6 mice at $T_a=30^\circ\text{C}$ and the B6 mice at $T_a=23^\circ$ had increased baseline HR compared to B6 mice at $T_a=30^\circ\text{C}$ (Figure 4A, Table 3A). Light phase HR was increased in both B6 and A/J mice housed at $T_a=23^\circ\text{C}$ compared to $T_a=30^\circ\text{C}$ (Figure 4B, Table 3B). B6 mice at $T_a=23^\circ\text{C}$ increased HR across time only during the dark phase on days 3 and 5, while B6 mice at $T_a=30^\circ\text{C}$ increased HR for all days on moderate fat diet during both the dark (Figure 4A) and light phases (Figure 4B and Figure 5). A/J mice at either temperature did not increase HR across time, although A/J mice housed at $T_a=30^\circ\text{C}$ had a similar magnitude of increase in HR compared to the B6 mice.

DISCUSSION

This study tested the hypothesis that adaptive thermogenesis would be blunted in mice after long-term (5 weeks) housing at $T_a=30^{\circ}\text{C}$. However, thermoneutrality did not attenuate the increase in VO_2 following access to a moderate fat diet in the two strains of mice studied. In agreement with our other hypothesis, the obesity resistant A/J mice had an augmented VO_2 response to overfeeding compared to the obesity-prone B6 mice. A third major finding arose at thermoneutrality, as tachycardia following the moderate fat diet was only revealed at this temperature, although the magnitude of increase was not greater in the A/J mice as hypothesized.

Although the magnitude of increase in VO_2 was not different between $T_a=23^{\circ}\text{C}$ and $T_a=30^{\circ}\text{C}$, baseline VO_2 was decreased at $T_a=30^{\circ}\text{C}$ in both strains. This decrease in metabolic rate at thermoneutrality is mediated by decreased SNA to BAT (23,24). The mice were housed at $T_a=30^{\circ}\text{C}$ for 5 weeks prior to the nutritional intervention to minimize the potential contribution of BAT to thermogenesis. Therefore, it was an unexpected finding that long-term housing of mice in a thermal neutral environment did not attenuate the magnitude of increase in VO_2 following access to moderate fat diet. The light phase RQ of the B6 mice was elevated at $T_a=30^{\circ}\text{C}$ from days 1-5 of moderate fat diet, which suggests these mice were burning more carbohydrate as fuel and less fat compared to the other three groups. This suggests that a warmer T_a may increase fat deposition in B6 mice with access to moderate fat diet. Although previous reports have demonstrated that adaptive thermogenesis is impaired at warmer temperatures in *rats* (3,33,34), data of this sort are lacking in mice. However, our data agree with previous reports that mice are able to engage adaptive thermogenesis at standard lab temperatures ($T_a=23^{\circ}\text{C}$) (6,35).

The augmented VO_2 response in the A/J mouse at day 1 supported our hypothesis that the lean phenotype was in part due to augmented adaptive thermogenesis following

overfeeding. These data support the role of a heightened sympathetic nervous system response mediating obesity resistance. Support for this hypothesis is drawn from studies utilizing β -AR blockade prior to overfeeding, demonstrating that the VO_2 response is prevented in rats and mice (5-7). Further evidence for a sympathetic component to the prevention of obesity is the lack of resistance to obesity in the $\beta_{1,2,3}$ -AR knockout mouse (8). However, there are other additional mechanisms that differ in the two strains of mice, but these differential mechanisms arise after long-term overfeeding. β_3 -AR (15), uncoupling protein-1 (UCP-1) mRNA in BAT (13) and UCP-1 increases in response to exogenous leptin (14) all remain intact in the A/J mouse after long-term overfeeding with higher fat diets compared to the B6 mouse.

The finding that B6 and A/J mice differentially regulate food intake over one week of overfeeding is interesting, considering previous reports that both strains consume kcal levels similar to a low-fat chow diet (12-15). However, these studies monitored food intake over many weeks, not days. One mechanism responsible for the divergent food intake behavior in the mice may be altered satiety signaling as demonstrated by an increase in pro-opiomelanocortin mRNA in A/J mice, but not B6 mice, although this data is after long-term overfeeding (16). A previous report found that both the B6 and the A/J mice preferred a higher fat diet compared to high carbohydrate or low-fat chow diet (36). Thus, it is unexpected that taste preference played a role in the divergent food intake responses in the B6 and A/J mice. Although it has been well documented that mice are not hyperphagic after long-term access to higher fat diets (37-39), B6 mice have been found to take over two weeks to regulate caloric intake back to low-fat (4.5% fat) chow levels with *ad libitum* access to a higher fat diet (17). It is possible that after only a few weeks on the moderate fat diet the B6 mice in our study would have reduced their caloric intake. However, we investigated the initial response to overfeeding which revealed another difference in the two strains of mice.

An activity component has been suggested to play a role in obesity resistance as demonstrated by various levels of non-exercise activity thermogenesis and spontaneous physical activity in humans overfed equal amounts (10,40). Increased activity in response to overfeeding may also contribute to obesity resistance in A/J mice. Compared to baseline, the A/J mice had heightened dark-phase locomotor activity on day 1 at both

temperatures that corresponded with elevated caloric intake. In comparison, the B6 mice increased dark phase locomotor activity only on day 1 at $T_a=30^{\circ}\text{C}$, although caloric intake was elevated days 1-7 at both temperatures. Our data are in agreement with previous reports demonstrating that at baseline and following long-term overfeeding, B6 mice are more active compared to A/J mice (12,41). However, the initial response following overfeeding suggests locomotor activity may be a factor contributing to the obesity resistance in A/J mice.

Although temperature did not contribute to the magnitude of increase in metabolic, food intake or locomotor parameters, there was a marked tachycardia evident only at $T_a=30^{\circ}\text{C}$ following access to moderate fat diet in both strains. The tachycardia of the A/J mice failed to reach significance at $T_a=30^{\circ}\text{C}$, but the magnitude of the increase in HR was similar to that of the B6 mice. The poor recovery rate of the A/J mice to surgery prevented further experiments with this strain. During surgery, A/J mice had excessive airway secretions and respiration was more adversely affected by the anesthesia. Following surgery, many of the A/J mice did not consume adequate calories for recovery. The HR at $T_a=30^{\circ}\text{C}$ for the A/J mice was not decreased compared to $T_a=23^{\circ}\text{C}$ at any of the time points except baseline in the light phase. It is possible that a lower baseline HR would have been necessary in order to attain an elevated HR that would have reached significance in the A/J mice. It is speculated that the A/J mice, because of their smaller body size, may have required a warmer ambient temperature in order to elicit a decrease in HR compared to the HR at $T_a=23^{\circ}\text{C}$. Recently, it has been reported that body size is a primary indicator for thermal conductance in rodents (42). However, the magnitude of tachycardia in both the B6 ($\Delta: 55 \pm 10$ beats/min) and A/J ($\Delta: 61 \pm 15$ beats/min) mice at thermoneutrality was similar, demonstrating that the A/J mice are still capable of increasing HR at $T_a=30^{\circ}\text{C}$.

Because adaptive thermogenesis is clearly mediated by sympathetic activation of beta-receptors, we predicted that acute overfeeding would produce tachycardia. Interestingly, this HR response was most clearly observed in mice studied at thermoneutrality. This suggests both sympathoexcitation and vagal inhibition to the heart following overfeeding. This conclusion is based upon pharmacological and spectral analysis data demonstrating that indeed, there is very little parasympathetic tone in mice

at standard lab temperatures and sympathetic tone is predominant (43,44). However, spectral analysis data from our lab suggest that vagal tone is present in mice at $T_a=30^\circ\text{C}$, but not $T_a=23^\circ\text{C}$ (45). Additional data from our lab demonstrate that following β_1 -AR blockade with atenolol, HR still drops over 100 beats/min when B6 mice are warmed from $T_a=23^\circ\text{C}$ to $T_a=30^\circ\text{C}$, suggesting a vagal component (18). Although intrinsic HR is unknown in mice at $T_a=30^\circ\text{C}$, unpublished data from our lab show that in rats intrinsic HR is similar at $T_a=23^\circ\text{C}$ and $T_a=30^\circ\text{C}$. Thus, it is hypothesized that if intrinsic HR is similar at $T_a=23^\circ\text{C}$ and $T_a=30^\circ\text{C}$ in mice, then mice have vagal tone present at $T_a=30^\circ\text{C}$ demonstrated by the 100 beats/min bradycardia still present after atenolol, which is able to be withdrawn with overfeeding. In addition, with a lower background of sympathetic activation to the heart at thermoneutrality, there is a greater ability for increased sympathetic activation to alter HR.

Further evidence for altered autonomic regulation following overfeeding comes from studies demonstrating that increased cardiac sympathetic tone accounts for some, but not all of the increase in HR with overfeeding in rabbits (46) and humans (47). In addition, spectral analysis (48) and pharmacological blockade of the SNS and PNS (49) suggest a decrease in parasympathetic tone with overfeeding in humans. Obese dogs also have also been shown to have decreased cardiac vagal tone (50), suggesting that the autonomic nervous system is altered in a state of obesity.

In conclusion, this study demonstrates the potent thermogenic effect of overfeeding in mice. During access to moderate fat diet, both the obesity-prone B6 and obesity-resistant A/J mice increase VO_2 regardless of T_a . However, B6 mice displayed hyperphagia throughout the 7-day experiment (compared to only 2 days for the A/J mice), reduced locomotor activity response to the overfeeding, and a blunted increase in VO_2 compared to the A/J mice. These behavioral and physiological differences suggest a genetic component to obesity resistance. In addition to the strain differences, this study demonstrates that thermogenesis, measured by elevation in VO_2 , is not impaired at $T_a=30^\circ\text{C}$ in mice. In addition, the cardiovascular response to overfeeding is dependent on T_a . These data demonstrate the importance of T_a on mouse physiology, and the need for further investigation of the autonomic regulation at warmer ambient temperatures in mice.

1A. Body Weight

1B. Caloric Intake

Figure 1. Body weight adjusted for the 3.3 g telemetry device (A) and caloric intake (B) in B6 (circles) and A/J (triangles) mice during 3 baseline days and 7 days of moderate fat diet at $T_a=23^\circ\text{C}$ (filled symbol) and $T_a=30^\circ\text{C}$ (open symbol). Powdered chow (3.3 kcal/g) was provided *ad libitum* during baseline, while powdered moderate fat diet (4.41 kcal/g) was provided *ad libitum* during the 7-day nutritional intervention. * $P < 0.05$ vs. baseline for B6 mice, † $P < 0.05$ vs. baseline for A/J mice. Mice were removed from the food intake analysis (Fig. 1B) if there was consistent placement of food outside the feeder. This is reflected in the n value.

Figure 2. Dark phase and light phase absolute VO_2 (A-dark, B-light), normalized VO_2 (C-dark, D-light), and RQ (E-dark, F-light) in B6 (circles) and A/J (triangles) mice at $T_a=23^\circ\text{C}$ (filled symbols) and $T_a=30^\circ\text{C}$ (open symbols) during 3 baseline days and 7 days of moderate fat diet. Powdered chow (3.3 kcal/g) was provided *ad libitum* during baseline, while powdered moderate fat diet (4.41 kcal/g) was provided *ad libitum* during the 7-day nutritional intervention. * $P<0.05$ vs. baseline for B6 mice, † $P<0.05$ vs. baseline for A/J mice.

2A. Dark Absolute VO_2 2B. Light Absolute VO_2 2C. Dark Normalized VO_2 2D. Light Normalized VO_2

2E. Dark RQ

2F. Light RQ

● B6 (23°C) n=10 ○ B6 (30°C) n=8
 ▲ AJ (23°C) n=6 △ AJ (30°C) n=8

3. Delta VO₂

Figure 3. Increase in light phase normalized VO₂ in B6 (solid bars) and A/J (hashed bars) mice on day 1 and day 7 of moderate fat diet. Data from both T_a=23°C and T_a=30°C were combined in the B6 and A/J mice because the magnitude of increase was not different between temperature within strains. Each bar represents the increase from the 3-day baseline mean on powdered chow (3.3 kcal/g) for day 1 or day 7 of moderate fat diet (4.41 kcal/g) in B6 or A/J mice. † P<0.05 vs. B6 mice on the same day.

4A. Dark HR

4B. Light HR

Figure 4. Dark phase (A) and light phase (B) HR in B6 (circles) and A/J (triangles) mice at $T_a=23^\circ\text{C}$ (filled symbols) and $T_a=30^\circ\text{C}$ (open symbols) during 3 baseline days and 7 days of moderate fat diet. Powdered chow (3.3 kcal/g) was provided ad libitum during baseline, while powdered moderate fat diet (4.41 kcal/g) was provided ad libitum during the 7-day nutritional intervention. * $P<0.05$ vs. baseline for B6 mice, † $P<0.05$ vs. baseline for A/J mice.

5. Delta HR

Figure 5. Diet induced changes in HR in B6 (solid bars, n=8 at $T_a=23^\circ\text{C}$ and n=6 at $T_a=30^\circ\text{C}$) and A/J (hashed bars, n=4 at $T_a=23^\circ\text{C}$ and $T_a=30^\circ\text{C}$) mice at $T_a=23^\circ\text{C}$ (filled bars) and $T_a=30^\circ\text{C}$ (open bars) during the light phase. Each bar represents the change in HR at either day 1 or day 7 of moderate fat diet (4.41 kcal/g) from the 3-day baseline mean on powdered chow (3.3 kcal/g). * $P<0.05$ temperature effect within strain on the same day, † $P<0.05$ A/J different from B6 at the same temperature on the same day.

Table 1. Adjusted body weight, food intake and fluid intake for 3-day baseline mean, day 1 and day 7 moderate fat diet for B6 and A/J mice housed at 23°C and 30°C.

	Adjusted Body Weight (g±SEM)		Food Intake § (kcal/d±SEM)		Fluid Intake (g/d±SEM)	
	B6	A/J	B6	A/J	B6	A/J
23°C	n=10	n=6	n=10	n=5	n=10	n=6
30°C	n=8	n=8	n=6	n=7	n=8	n=8
3-day Baseline Mean						
23°C	28.41±0.72	23.60±0.94*	14.42±0.27	13.35±0.82	5.68±0.11	4.37±0.24*
30°C	26.10±0.59	21.73±1.02*	9.40±1.31†	8.95±1.07	4.79±0.22	3.65±0.36*
Moderate Fat Day 1						
23°C	29.55±0.78‡	24.97±0.93*	21.37±1.72‡	21.34±2.00‡	4.67±0.16‡	3.38±0.24*‡
30°C	26.69±0.66	23.38±0.95*‡	14.61±0.97†‡	16.91±0.85‡	5.00±0.14	3.83±0.13*
Moderate Fat Day 7						
23°C	29.84±0.87‡	25.02±0.48*	20.79±1.80‡	15.79±1.82	3.58±0.41‡	3.40±0.39
30°C	28.78±0.94‡	24.09±0.81*‡	18.01±2.08‡	8.60±0.84*	4.30±0.29	2.70±0.14*‡

* P<0.05 vs. B6 mice, † P<0.05 vs. T_a=23°C, and ‡ P<0.05 vs. baseline.

§ Mice were removed from the analysis if there was consistent placement of food outside the feeder. This is reflected in the n value.

Table 2A. Dark phase absolute VO₂, normalized VO₂ and RQ for 3-day baseline, day 1 and day 7 of moderate fat diet for B6 and A/J mice housed at 23°C and 30°C. See Figure 2 for n values.

	Absolute VO ₂ (ml/min±SEM)		Normalized VO ₂ (ml/min/kg ^{0.75} ±SEM)		Respiratory Quotient (CO ₂ /O ₂ ±SEM)	
	B6	A/J	B6	A/J	B6	A/J
3-day Baseline Mean						
23°C	2.24±0.04	1.91±0.10*	32.39±0.63	32.58±1.12	0.934±0.005	0.952±0.006
30°C	1.43±0.07†	1.19±0.09†	22.77±0.47†	21.78±1.11†	0.925±0.001	0.923±0.024
Moderate Fat Day 1						
23°C	2.41±0.05‡	2.17±0.13‡	33.63±0.53	36.26±1.62‡	0.955±0.003‡	0.953±0.011
30°C	1.59±0.05†‡	1.47±0.08†‡	24.72±0.25†‡	25.48±0.96†‡	0.947±0.006‡	0.971±0.008
Moderate Fat Day 7						
23°C	2.46±0.05‡	2.14±0.15‡	34.32±0.54‡	35.76±2.36‡	0.884±0.008‡	0.909±0.006
30°C	1.62±0.07†‡	1.32±0.08†‡	24.25±0.53†	22.45±1.14†	0.013±0.004	0.933±0.014

* P<0.05 vs. B6 mice, † P<0.05 vs. T_a=23°C, and ‡ P<0.05 vs. baseline.

Table 2B. Light phase absolute VO₂, normalize VO₂ and RQ for 3-day baseline, day 1 and day 7 of moderate fat diet for B6 and A/J mice housed at 23°C and 30°C. See Figure 2 for n values.

	Absolute VO ₂ (ml/min±SEM)		Normalized VO ₂ (ml/min/kg ^{0.75} ±SEM)		Respiratory Quotient (CO ₂ /O ₂ ±SEM)	
	B6	A/J	B6	A/J	B6	A/J
3-day Baseline Mean						
23°C	1.68±0.09	1.52±0.08	24.33±0.63	26.03±1.14	0.849±0.004	0.891±0.018
30°C	0.89±0.05†	0.85±0.05†	14.34±0.43†	16.66±1.30†	0.886±0.008	0.872±0.026
Moderate Fat Day 1						
23°C	1.93±0.10‡	1.82±0.06‡	27.00±0.43‡	30.48±0.92*‡	0.945±0.006‡	0.936±0.021‡
30°C	1.05±0.05†‡	1.14±0.05†‡	16.74±0.50†‡	21.03±1.10†*‡	0.960±0.013‡	0.988±0.011‡
Moderate Fat Day 7						
23°C	1.84±0.11‡	1.69±0.08‡	25.71±0.49‡	28.23±1.30‡	0.846±0.006	0.808±0.013
30°C	1.09±0.07†‡	0.95±0.05†	16.05±0.59†‡	17.12±0.94†	0.924±0.008†	0.837±0.010*

* P<0.05 vs. B6 mice, † P<0.05 vs. T_a=23°C, and ‡ P<0.05 vs. baseline.

Table 3A. Dark phase HR, MAP, and locomotor activity for 3-day baseline, day 1 and day 7 of moderate fat diet for B6 and A/J mice housed at 23°C and 30°C.

	Heart Rate (beats/min±SEM)		Mean Arterial Pressure (mmHg±SEM)		Locomotor Activity (m/12 hr±SEM)	
	B6	A/J	B6	A/J	B6	A/J
23°C	n=8	n=4	n=8	n=4	n=10	n=6
30°C	n=6	n=4	n=6	n=4	n=8	n=8
3-day baseline Mean						
23°C	636±7	657±5	119±2	117±22	196±18	87±12*
30°C	486±15†	571±25*	104±3†	96±5†	244±17	91±14*
Moderate Fat Day 1						
23°C	661±10	673±14	117±2	114±5	211±26	152±30‡
30°C	540±13†‡	615±21*	105±3	94±6†	325±41‡	172±26*‡
Moderate Fat Day 7						
23°C	659±15	637±8	119±1	104±3*	200±25	145±33
30°C	549±15†‡	570±4	107±1†	95±5*	280±35	132±21*

* P<0.05 vs. B6 mice, † P<0.05 vs. T_a=23°C, and ‡ P<0.05 vs. baseline.

Table 3B. Light phase heart rate, mean arterial pressure, and locomotor activity (baseline, day 1 and day 7 of moderate fat diet) of B6 and A/J mice housed at 23°C and 30°C.

	Heart Rate (beats/min±SEM)		Mean Arterial Pressure (mmHg±SEM)		Locomotor Activity (m/12 hr±SEM)	
	B6	A/J	B6	A/J	B6	A/J
23°C	n=8	n=4	n=8	n=4	n=10	n=6
30°C	n=6	n=4	n=6	n=4	n=8	n=8
3-day baseline Mean						
23°C	587±16	597±22	105±2	101±6	50±6	32±8
30°C	409±12 [†]	480±17 [†]	95±6	85±4	44±3	30±4
Moderate Fat Day 1						
23°C	599±16	614±27	103±2	102±9	36±4 [‡]	32±8
30°C	464±14 ^{†‡}	541±26	101±8	85±4	36±6	37±5
Moderate Fat Day 7						
23°C	593±23	546±12 [‡]	106±3	86±4 ^{*‡}	34±4 [‡]	23±8
30°C	478±21 ^{†‡}	477±19	100±5	83±3	32±9	22±4

* P<0.05 vs. B6 mice, [†] P<0.05 vs. T_a=23°C, and [‡] P<0.05 vs. baseline.

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BIOGRAPHICAL SKETCH

Lara M. Roberts

EDUCATION

2003	M.S.	Nutritional Neuroscience, Florida State University Advisor: Dr. J. Michael Overton
2001	B.S.	Exercise Science, Florida State University Advisor: Dr. John E.A. Bertram

ACADEMIC EMPLOYMENT

2003-present	Alumni Fellow and Graduate Research Assistant Lab Director: Dr. Linda F. Hayward
2002-2003	Graduate Research Assistant Lab Director: Dr. J. Mike Overton
2000-2001	Laboratory Technician Lab Director: Dr. John Bertram

TEACHING EXPERIENCE

2002	Anatomy and Physiology Lab	2 sections
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HONORS AND AWARDS

2003-present	University of Florida, Alumni Fellowship
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1999-2001	State of Florida, Florida Bright Futures Tuition and Book Scholarship
2001	Florida State University, Minority Association of Pre-Health Students, Summer Book Stipend
1997-1998	University of Utah, Full Out-of-State Tuition Scholarship

EXTRACURRICULAR INVOLVEMENT

2003	Florida State University, Member, Congress of Graduate Students
2001-present	Boggy Creek Gang Camp: Volunteer to aid with chronically ill children and their families 3 family weekends and 1 week of summer camp

2000-2001	Florida State University, Member, Minority Association of Pre-Health Students
2000	Medical mission to Honduras: Health, dental and optical care to children and adults
2000	Florida State University, Treasurer of Kappa Omicron Nu

PROFESSIONAL AFFILIATIONS

2002-present	American Physiological Society; student member
2002-present	Society for Neuroscience; student member

PEER-REVIEWED PUBLICATIONS

Todd D. Williams, J.B. Chambers, S.P. Gagnon, **L.M. Roberts**, R.P. Henderson, and J.M. Overton. Cardiovascular and metabolic responses to fasting and thermoneutrality in Ay mice. *Physiology and Behavior* April 2003; 78 (4-5): 615-23.

Todd D. Williams, J.B. Chambers, **L.M. Roberts**, R.P. Henderson and J.M. Overton. Diet-induced obesity and cardiovascular regulation in B6 mice. In Press: *Clinical and Experimental Pharmacology and Physiology*.

Keith C. DeRuisseau, **L.M. Roberts**, M.R. Kushnick, A.M. Evans, K. Austin, and E.M. Haymes. Iron status of young males and females performing weight training exercise. Submitted to *Medicine and Science in Sports and Exercise*: 11/2002.

ABSTRACTS

Lara M. Roberts, T.D. Williams, and J.M. Overton. Acute high fat feeding produces concurrent diet-induced thermogenesis and tachycardia in B6 mice. Submitted for: *Experimental Biology*. San Diego, CA. 04/11/2003-04/15/2003.

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Keith C. DeRuisseau, C.S. Katsanos, **L.M. Roberts**, and E.M. Haymes. The Influence of Iron Status on Exercise Induced Lipid Peroxidation in Male Runners. *American College of Sports Medicine*. St. Louis, MO. 05/22/2002-05/25/2002

CONFERENCES ATTENDED

Experimental Biology: San Diego, CA; 4/2003
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