Adiposity and human regional body temperature\textsuperscript{1–3}

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ABSTRACT

Background: Human obesity is associated with increased heat production; however, subcutaneous adipose tissue provides an insulating layer that impedes heat loss. To maintain normothermia, therefore, obese individuals must increase their heat dissipation.

Objective: The objective was to test the hypothesis that temperature in a heat-dissipating region of the hand is elevated in obese adults.

Design: Obese [body mass index (in kg/m\(^2\)) \(\geq 30\)] and normal-weight (NW; body mass index = 18–25) adults were studied under thermoneutral conditions at rest. Core body temperature was measured by using ingested telemetric capsules. The temperatures of the third fingernail bed of the right hand and of abdominal skin from an area 1.5 cm inferior to the umbilicus were determined by using infrared thermography. Abdominal skin temperatures were also measured via adhesive thermistors that were placed over a prominent skin-surface blood vessel and over an adjacent nonvessel location.

Results: Core temperature did not differ significantly between the sex, race, and room temperature as covariates.

Core temperature did not differ significantly between the sex, race, and room temperature as covariates.

Conclusions: Greater subcutaneous abdominal adipose tissue in obese adults may provide a significant insulating layer that blunts abdominal heat transfer. Augmented heat release from the hands may offset heat retention in areas of the body with greater adiposity, thereby helping to maintain normothermia in obesity. This trial was registered at clinicaltrials.gov as NCT00266500. Am J Clin Nutr 2009;90:1124–31.

INTRODUCTION

Obesity is a condition associated with high body heat content (1–5). Several physiologic changes that accompany the development of obesity tend to increase heat production or impede heat loss. First, resting metabolic heat production is significantly greater in obese than in lean individuals (6). This greater heat production is primarily due to the larger fat-free mass (FFM), ie, muscle, that accompanies excessive adiposity (6). Second, adipose tissue, because of its reduced thermal conductivity and increased insulatory capacity (7), provides an insulating barrier to conductive heat flow and reduces the body’s ability to respond to changes in core temperature (8). The degree of thermal insulation from adipose tissue has been shown to be positively related to the degree of obesity (8, 9). Third, obese individuals may lose a smaller fraction of their metabolic heat because the core temperature triggering vasoconstriction is greater in obesity (10). Finally, with obesity, weight increases without a proportional increase in height, resulting in a lower ratio of surface area to body mass (11), and, because cutaneous heat loss is relatively proportional to skin surface area (12), obese individuals may lose their metabolic heat more slowly than do those with normal body weight (13). Thus, obesity itself reduces the ratio of heat loss to heat production and should lead to retention of body heat. However, because the core temperature in obese individuals is homeostatically regulated, thermoregulatory reflexes must compensate and be biased toward heat dissipation in those with excessive adiposity.

In addition to the well-known action of insensible perspiration and sweating as mechanisms for heat loss (14), peripheral glabrous skin regions, including the skin found on the palmar surface of the hands and the soles of the feet, play an important role in body heat management. Such sites are rich in arteriovenous anastomoses (AVAs), which are capable of regulating heat loss to the ambient environment by modulating distal blood flow (15, 16). With increases in body temperature, open AVAs provide a low-resistance route that allows large quantities of blood to flow through superficial venous plexi and away from peripheral capillary beds (15, 16). This allows for more efficient heat loss to the environment and the return of cooled blood to the core (15, 17). Peripheral sites, such as the hands, would be anticipated to

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remain effective for heat dissipation in obese individuals, because such sites have relatively less adipose accumulation than more central locations such as the abdomen or hips (18). We therefore hypothesized that, under thermoneutral conditions, heat dissipation from distal extremities (in particular, the hands) would be augmented in obesity, whereas heat dissipation from central sites (eg, the abdomen) would be reduced.

The aim of this study was to evaluate the relations between adiposity and the temperature of the hand and abdomen in both obese and normal-weight adults. We therefore obtained thermal data at rest under thermoneutral conditions, as well as during local cooling of the palmar surface of the hand, from obese and normal-weight individuals.

SUBJECTS AND METHODS

Participants

Adults (age: 18–70 y) were recruited through flyers posted on public bulletin boards at the National Institutes of Health (NIH), at local libraries, and at supermarkets in the Washington, DC, greater metropolitan area. The flyers explained that the study was investigating the relation between body weight and body heat management in healthy adults. Nonsmoking adults were eligible for participation if their body mass index (BMI; in kg/m²) was calculated within the range for normal weight (NW; BMI = 18–25) or obesity (BMI ≥30), and if they had been weight-stable (±3%) for the past 2 mo. None of the participants had medical conditions that would preclude accurate completion of study assessments, nor were any participants taking medication to treat obesity-related medical conditions or drugs that affected cardiovascular function or heat balance. Female participants reported having a normal menstrual cycle or were postmenopausal. The Eunice Kennedy Shriver National Institute of Child Health and Human Development institutional review board approved the clinical protocol. Each volunteer gave written consent for his or her participation and was financially compensated in accordance with established NIH Human Research Protection Program policy.

Experimental design

Each participant visited the Hatfield Clinical Research Center (CRC) at the NIH on 2 separate occasions. At the first visit, a brief history and physical examination were conducted by a physician or a nurse practitioner, eligibility was assessed, and body-composition measures were collected. Weight and height were measured in the postabsorptive state with the use of calibrated instruments. Body fat mass (FM) and FFM were determined by dual-energy X-ray absorptiometry with an instrument from Hologic (Bedford, MA), or, in the case of 3 individuals whose weight exceeded 136 kg, with an instrument from GE Health Care (Waukesha, WI). Half-body values were doubled to calculate total body results for 2 individuals whose supine body width exceeded the dimensions of the scan window (19).

At the second visit, all participants were admitted to the CRC allowing for acclimatization to the experimental environment. Participants were provided with a calorie-controlled diet based on their predicted resting energy expenditure (20) and an activity multiplier of 1.5 to maintain weight stability (±3%). Female participants with a normal menstrual cycle were tested in the midfollicular phase (calculated from self-stated cycle lengths), and physical activity was constrained by the study protocol and was comparable across all participants. Regional body temperatures were assessed 3 h after a light meal under thermoneutral ambient conditions in a temperature-controlled room (23.1±0.2 °C), and all participants wore their usual light indoor clothing. Room temperature was continuously recorded with an accuracy of ±0.1 °C in 1-min intervals via a temperature data-logging monitor (Extech Instruments, Waltham, MA). Resting metabolic rate (RMR) was assessed by open-circuit indirect calorimetry performed with the use of a respiratory metabolic cart (Parvomedics, Sandy, UT) in the morning after regional body-temperature measurements, after an overnight fast.

Regional body temperature measurements

Infrared imaging

Thermographic images of the right hand and the abdomen were acquired by using a Santa Barbara Focalplane ImageIR camera system (Lockheed Martin Inc, Goleta, CA). The infrared thermography camera used a 640 × 512 array of sensors operating in the 3–5-μm wavelength range (mid-infrared) and was sensitive to 0.02–°C temperature changes. Two-point linear image calibration via a black-body infrared radiation source and nonuniformity correction of the entire array allowed reading of absolute temperatures in each of the 327,680 pixels of an infrared image. Sequential thermographic images were acquired at a rate of 2 images/s and stored on a computer for off-line analysis of body surface temperature. The plane of the infrared camera’s lens was positioned parallel to the plane of the region of interest at a distance of 40–60 cm. To reduce motion artifacts, participants were asked to remain as still as possible during the periods of infrared imaging.

During fingernail-bed temperature imaging, participants were seated on a hospital bed with the upper torso angle adjusted to 45°, and a vertical hospital curtain was drawn to minimize the effects of drafts. Each participant placed his or her right hand, palm down, on a grid (1 cm²) made of thin black nylon line strung on a solid polycarbonate ring, which was suspended over a basin. The purpose of the grid was to support the hand, while minimizing skin contact with the surface providing support, during temperature measurements of the dorsal surface of the hand. Sequential infrared thermographic images were collected for 20 min after a 20-min acclimatization period with the participant at rest.

During abdominal skin temperature imaging, the participants lay supine on a hospital bed with the upper torso adjusted to 15° above horizontal, with a hospital curtain drawn to minimize the effects of drafts. Participants positioned their clothing to allow for full abdominal exposure extending approximately from the level of the xiphoid process to 1 in (2.54 cm) above the symphysis pubis. A thin plastic barrier was positioned vertically across the participant at the level of the chest to restrict interference of respiratory airflow with infrared thermography measures. After a 20-min acclimatization period with the participant at rest, and his or her abdomen exposed, sequential infrared thermographic images were collected for 20 min.

To assess the response to a mild cold challenge of the hand, the participants were seated and acclimatized as described above for
The right.

Temperatures are represented by colors, as indicated by the gradient on location 1.5 cm inferior to the umbilicus selected for image analysis. Obese female (A) and a normal-weight female (B). Arrows indicate the baseline imaging, water cooled to 20°C. Extraneous cooling due to evaporation. After 4 min of sequential thermography, the ROIs were specified as the third fingertip bed in all participants. The nail bed was selected because it is rich in AVAs, one of the fundamental elements of body thermoregulation (21). Furthermore, preliminary infrared thermography testing in 2 healthy, NW participants showed that the temperatures measured at the third fingertip bed were strongly correlated with skin temperatures measured on the palmar surface during baseline (n = 2378, r² = 0.93) and after 2 min of palmar hand cooling (n = 3466, r² = 0.86). For abdominal infrared temperature measurements, a rectangular ROI (100 pixels total) 1.5 cm inferior to the umbilicus was analyzed. This abdominal subcutaneous fat depot was selected because it was the most homogeneous across all participants with regard to the absence of prominent surface vessels, as determined by infrared thermography (Figure 1).

FIGURE 1. Abdominal (left) and right-hand (right) thermograms of an obese female (A) and a normal-weight female (B). Arrows indicate the location 1.5 cm inferior to the umbilicus selected for image analysis. Temperatures are represented by colors, as indicated by the gradient on the right.

Core and abdominal skin temperature measurements with thermistors

Gastrointestinal tract ("core") and abdominal skin temperatures were acquired via the VitalSense Integrated Physiologic Monitoring System (Philips Respironics Inc, Bend, OR) during all infrared imaging studies. This system consists of biocompatible ingestible capsules and adhesive dermal patches, each with integrated thermistors and telemetric circuitry that transmit temperatures with an accuracy of ±0.1°C, in 1-min intervals to a data-logging monitor.

Temperature measurements by the core capsule and dermal patches were initiated ≥1 h before infrared imaging studies. Dermal patches were placed on the abdomen of each participant. Given that variations in torso skin temperatures occur between adjacent fixed sites (24), infrared imaging was used in real-time to guide patch placement such that the thermistor of one patch was placed directly over the warmest location on the abdomen (warm patch), and the thermistor of a second patch (cool patch) was placed directly over the coolest location in proximity (<10 cm) to the warm patch. Warm sites suggested the presence of cutaneous perforator-type vessels, which are easily detected by infrared thermography (23, 25).

Statistical analyses

Differences in anthropometric and demographic characteristics between the study participants were evaluated by Student’s t test and chi-square analyses for continuous and categorical variables, respectively. RMR corrected for FFM was analyzed by analysis of covariance (ANCOVA), with FFM included as a covariate. Basal hand and abdominal data were averaged over the 20 min of each measurement for all individuals and were analyzed by ANCOVA followed by least-significant-difference pairwise multiple comparison tests to compare results between the NW and obese groups. Age, sex, race (coded as African American or white/other), and room temperature were included as covariates. “Other” races/ethnicities (ie, nonblack Hispanic and self-declared “other”) were combined with whites because of their small numbers (n = 2). Race was considered an important potential covariate because of the known difference in RMR between African Americans and whites that remains even after adjustment for total lean body mass (26). Temperatures measured by core capsule and dermal patches during the 20-min abdominal infrared imaging are reported. Temperatures measured during the cold challenge of the hand were averaged within bins for each stage (2-min bins for both baseline and recovery, and a 75-s bin for cooling) for all individuals and were assessed by ANCOVA with repeated measures.

Linear regression analysis was also performed to examine the relation between FM as a percentage of total body mass (%FM) and temperature measures. In addition to %FM, the aforementioned covariates [age, sex (coded as F = 0, M = 1), race (coded as white/other = 0 and African American = 1), and room temperature] were included simultaneously as independent variables in all regression analyses. All analyses were conducted by using SPSS for Windows (version 16.0; SPSS Inc, Chicago, IL) software, and statistical significance was set at an α level of 0.05. Adjusted mean (±SE) values are shown, unless otherwise indicated.
RESULTS
The demographic and physical characteristics of the NW and obese participants are shown in Table 1. There were no significant differences between the NW and obese groups in sex, age, or blood pressure. There was, however, a greater proportion of African American participants in the obese group \((P = 0.04)\). By design, the obese group had significantly higher BMIs and measures of body composition \((all \ P < 0.001)\). As expected, the obese group exhibited a significantly greater mean RMR \((obese: 1980 \pm 57 \text{ kcal/d}; \ NW: 1469 \pm 78 \text{ kcal/d}; \ P < 0.001)\), indicative of greater total heat generation during rest. Mean RMR, corrected for FFM, was not significantly different between the obese and NW groups \((obese: 1845 \pm 43 \text{ kcal/d}; \ NW: 1729 \pm 66 \text{ kcal/d}; \ P = 0.2)\).

Room temperature during imaging experiments did not significantly differ between the NW and obese participants \((P = 0.95)\) and averaged \(23.1 \pm 0.2 \degree C\) for both groups. Mean core body temperatures were also comparable for both groups \((NW: 37.1 \pm 0.1 \degree C; \ obese: 37.0 \pm 0.1 \degree C; \ P = 0.74)\).

Mean fingernail-bed temperature, adjusted for covariates as described in Subjects and Methods, was significantly greater in obese than in NW participants \((33.9 \pm 0.7 \degree C; \ P < 0.001; \ Figure 1 \ and \ Figure 2A)\). The difference between fingernail bed temperature and core temperature was significantly smaller in the obese than in the NW participants \((-3.1 \pm 0.7 \degree C; \ P < 0.001; \ Figure 2B)\). The size of the fingernail bed, determined by the number of pixels analyzed, did not differ significantly between obese and NW participants \((360 \pm 37 \text{ compared with } 331 \pm 37 \text{ pixels}, \ respectively; \ P = 0.61)\).

The adjusted mean abdominal skin temperature, measured by infrared thermography from the monitored site \(1.5 \text{ cm} \) inferior to the umbilicus, was significantly lower in the obese participants than in the NW participants \((31.8 \pm 0.2 \degree C; \ P = 0.02; \ Figure 1 \ and \ Figure 2C)\). The difference between the infrared abdominal skin temperature and the core temperature was significantly greater in the obese than in the NW participants \((-5.3 \pm 0.2 \degree C; \ P = 0.001)\). As expected, abdominal skin temperature did not differ between the obese and NW groups from thermistors placed directly over a cool location on the abdomen showed a difference between the obese and NW participants \((31.7 \pm 0.3 \degree C; \ P = 0.04; \ Figure 3)\) that was similar to the difference for these groups as determined by infrared measurements of abdominal skin temperature \((Figure 2C)\). As expected, abdominal skin temperature did not differ between the obese and NW groups from thermistors placed directly over the warm surface vessel located on the abdomen \((32.6 \pm 0.2 \degree C; \ P = 0.27; \ Figure 3)\).

Given the slight mean age difference between NW and obese study participants and the potential effect of age on temperature measures, temperature differences were also examined in a subpopulation analysis of NW and obese subjects \(\text{mean } \pm \text{SD}: \ BMI = 22.5 \pm 1.5 \text{ compared with } 40.8 \pm 8.1; \ P < 0.001)\) restricted to those aged \(<50 \text{ y}\). Age \((32.5 \pm 10.9 \degree C; \ P = 0.30)\) and sex distribution were not significantly different between these subgroups. The fingernail bed and abdominal temperature differences observed between the NW \((n = 11)\) and obese \((n = 14)\) participants in this subgroup analysis confirmed those found for the entire study cohort \(\text{(Table 2)}\).

Repeated-measures ANCOVA of the temperature response, measured by infrared in the fingernail bed after a mild cold challenge of the hand, showed that there was a significant effect of group \(\text{NW compared with obese; } P = 0.005)\), such that the obese group had greater fingernail-bed temperatures throughout the cooling experiment, but no significant group \(\times \text{ time interaction; } P = 0.47; \ Figure 4)\).

In a regression model relating temperatures measured by infrared to \%FM, fingernail-bed temperature was positively related to \%FM \((\beta = 0.56, \ P = 0.001)\). Race \((\beta = 0.28, \ P = 0.04)\) and room temperature \((\beta = 0.30, \ P = 0.03)\) also served as significant contributors to fingernail-bed temperature \(\text{model-adjusted } R^2 = 0.45, \ P < 0.001; \ Figure 5A)\). Analysis of the relation between \%FM and abdominal skin temperature measured by infrared found that there was a negative association \((\beta = -0.54, \ P = 0.005)\); sex \((\beta = -0.45, \ P = 0.01)\) and room temperature \((\beta = 0.34, \ P = 0.03)\) were also significant predictors of abdominal skin temperature \(\text{model-adjusted } R^2 = 0.30, \ P = 0.006; \ Figure 5B)\).

TABLE 1

<table>
<thead>
<tr>
<th>Sex (% female)</th>
<th>Normal weight ((n = 13))</th>
<th>Obese ((n = 23))</th>
<th>(P^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Race (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>23 (8, 51)</td>
<td>61 (41, 78)</td>
<td>0.3</td>
</tr>
<tr>
<td>White or other</td>
<td>77 (49, 93)</td>
<td>39 (22, 59)</td>
<td>0.04</td>
</tr>
<tr>
<td>Age (y)</td>
<td>36.3 ± 13.7(^1)</td>
<td>44.7 ± 12.5</td>
<td>0.07</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>84.5 ± 11.3</td>
<td>88.1 ± 6.8</td>
<td>0.2</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>22.9 ± 1.7</td>
<td>39.0 ± 7.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>17.5 ± 5.7</td>
<td>46.3 ± 13.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>44.7 ± 8.0</td>
<td>66.1 ± 12.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fat mass (% of total mass)</td>
<td>28.3 ± 8.5</td>
<td>41.0 ± 8.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RMR (kcal/d)</td>
<td>1469 ± 238</td>
<td>1980 ± 286</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

\(^1\) 95% CIs in parentheses. MAP, mean arterial pressure; RMR, resting metabolic rate (uncorrected).

\(^2\) Differences between groups were determined by chi-square tests for categorical variables and by ANOVA for continuous variables.

\(^3\) Mean ± SD (all such values).


discussion

The aim of this study was to achieve a better understanding of body heat management in obesity by evaluating the relations between adiposity and temperature profiles of the hands and abdomen under thermoneutral conditions. Even though obese individuals had greater resting metabolic heat production, due to increased FFM, there were no differences in core temperatures between the NW and obese groups were detected. These results suggest that there are physiologic differences that occur in obesity that diminish body heat storage by increasing heat dissipation to compensate for the additional thermal load. Given the important role of peripheral sites for body heat dissipation and retention \(\text{(17, 27)}\), we hypothesized that, compared with the NW subjects, obese subjects would have increased heat dissipation through the hands. Indeed, we found that obese participants had warmer fingernail-bed temperatures than did the NW participants. Fingernail-bed temperatures were measured because we found that they are
strongly correlated with skin temperatures measured on the palmar surface of the hand under ambient conditions as well as after palmar hand cooling. We also observed, using both surface thermistors and infrared thermography, significantly lower core-to-skin temperature differences in the obese than in the NW subjects in abdominal regions that were selected for study because they did not contain surface vessels observable by infrared. This decreased abdominal core-to-skin temperature gradient is consistent with the concept that the trunk retains body heat in obesity (8). Our findings suggest that, in obesity, reduced heat loss related to high abdominal fat is accompanied, under thermoneutral conditions, by augmented heat dissipation from the hands.

These data support the concept that, because of adipose tissue’s low thermal conductivity (7), body heat management is related to regional adiposity (8, 9, 28–30). Other convincing data for differences between obese and NW humans relevant for the insulating properties of adipose tissue have been collected from subjects studied with the use of direct calorimetry, in which whole-body heat losses (radiant, convective, conductive, and evaporative heat losses) were measured. For example, Jequier et al (8) reported that at an environmental temperature of 20 °C, thermal insulation was directly proportional to subcutaneous fat thickness, such that total heat losses in overweight and obese women were less than and internal temperature fell more slowly than in normal-weight women. We observed that, under thermoneutral conditions, obese participants had lower abdominal

**TABLE 2**

<table>
<thead>
<tr>
<th>Body temperature measures of participants younger than 50 y†</th>
<th>Normal weight (n = 11)</th>
<th>Obese (n = 14)</th>
<th>P²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fingernail bed (°C)</td>
<td>29.8 ± 1.1</td>
<td>33.9 ± 0.9</td>
<td>0.02</td>
</tr>
<tr>
<td>Abdomen (°C)</td>
<td>33.0 ± 0.3</td>
<td>31.8 ± 0.3</td>
<td>0.02</td>
</tr>
<tr>
<td>Abdomen warm patch (°C)</td>
<td>33.1 ± 0.2</td>
<td>32.7 ± 0.3</td>
<td>0.2</td>
</tr>
<tr>
<td>Abdomen cool patch (°C)</td>
<td>32.9 ± 0.3</td>
<td>31.8 ± 0.3</td>
<td>0.02</td>
</tr>
</tbody>
</table>

† All values are means ± SEs.

² Differences between groups were determined by ANCOVA with age, race, sex, and room temperature as covariates.
skin temperatures measured by infrared thermography and dermal thermistors. These results agree with other reports that local skin temperatures are influenced by subcutaneous adiposity (9, 18, 24, 30–32) and suggest that core-to-skin heat dissipation in sites with significant subcutaneous fat depots is blunted in obese individuals.

Infrared thermography showed local heterogeneities in abdominal skin temperatures and enabled placement of dermal thermistors on warm or cool areas of the skin more reliably than by depending on standard torso sites (24). In addition to the thickness and distribution of adipose tissue, another source of variability in measurement of skin temperature is due to the presence or absence of cutaneous perforator-type vessels, which can easily be detected by infrared thermography (23, 25). We did not detect significant skin temperature differences between NW and obese participants when using patches placed over perforator-type vessels, which suggested that the ability of individual abdominal perforator vessels to dissipate heat does not appear to be augmented in obesity. Our participants were assessed at rest; at present, it is unknown whether these abdominal vessels would exhibit differential responses between the NW and obese groups during exercise or under conditions outside of thermoneutrality.

Whereas basal metabolic heat production, adjusted for body mass, is generally comparable between NW and obese subjects, cold-induced metabolic heat production is attenuated in overweight men (30) and in overweight and obese women (8). It is thought that thermal insulation attributable to adipose tissue attenuates core cooling and that this results in a smaller metabolic response to cold environments in obese individuals (8). Cutaneous thermoregulatory responses during cold exposure, such as reductions in vascular conductance through AVAs, also provide substantial protection against core hypothermia (16, 33). In the current study, we observed that both groups exhibited a similar local thermoregulatory response pattern to hand cooling, although recovery to baseline temperatures in the obese group appeared to be slightly, albeit nonsignificantly, attenuated (Figure 4). Mechanisms that could account for a delay in postcooling recovery in obesity have not been established, but could involve delayed withdrawal of sympathetic constrictor tone (34). Finger temperature depends largely on finger blood flow (35), and skin vascular resistance plays a role in systemic vascular resistance and blood pressure regulation (36). Indeed, defects in sympathetic tone in obesity have been reported by others (37, 38).

Some limitations of our study must be noted. First, temperature differences were not assessed in a homogenous sample. A somewhat greater percentage of obese participants were African American, which potentially influenced peripheral blood flow

FIGURE 4. Mean (±SE) fingernail-bed temperatures, adjusted as indicated in Subjects and Methods, measured by infrared thermography in response to a mild cold (20°C) challenge of the hand. Differences in temperature between the normal-weight (NW; n = 13) and obese (OB; n = 23) participants were determined by using repeated-measures ANCOVA with age, sex, race, and room temperature as covariates. No significant group × time interaction was observed (P = 0.47).

FIGURE 5. Fingernail-bed temperature (A) and abdominal skin temperature 1.5 cm inferior to the umbilicus (B) relative to the percentage of total body mass from fat in normal-weight (n = 13) and obese (n = 23) participants. Measured temperatures were adjusted for relevant covariates (age, sex, race, and room temperature) in the regression model.
and, thus, hand temperature measures because of racial differences in vascular resistance and blood pressure regulation (39). Although our analyses controlled for factors such as sex, age, and race, which might affect body heat management and peripheral vascular tone or vasomotion, it is possible that adjusting for these factors did not fully account for their potential to influence our measures. However, our subgroup analyses of participants aged <50 y showed that a reduction in age-related variability had no appreciable affect on the obesity-related temperature differences that we found for the entire cohort. Second, we did not measure skin-surface temperature of the forearm. Forearm-minus-finger temperature gradients are accurate indexes of thermoregulatory peripheral vasoconstriction (35). Temperature gradient data might have allowed for further characterization of hand-temperature differences between the NW and obese participants during the hand cooling study and at baseline. Future studies should measure temperatures in more body regions of interest. Third, whereas we did not detect significant skin temperature differences between the NW and obese participants when patches were placed over perforator-type vessels, we did not measure whether obese subjects might have more or fewer such perforator vessels than NW subjects. Finally, we did not assess differences in other factors that promote heat dissipation, such as evaporative cooling via sweating (14). Obesity-associated changes in skin physiology may be related to increased sweat gland activity (40); however, there do not appear to be any specific alterations in the structure and function of apocrine and eccrine sweat glands in obesity (41).

In summary, we observed that abdominal skin temperature was cooler and fingernail-bed temperature was warmer in the obese than in the NW participants measured under thermoneutral conditions. This weight-related divergence in abdominal skin temperatures and fingernail-bed temperatures was detectable both based on classification by BMI and when measured temperatures were related to %FM in regression models, where abdominal skin temperature was negatively related and fingernail bed temperature was positively related to %FM. This study thus provides additional evidence that body regions of increased adiposity have blunted core-to-skin heat loss and that heat dissipation from peripheral regions, such as the hands, is augmented in obesity. It remains to be determined whether heat extraction from peripheral sites such as the hand is feasible or beneficial for the clinical management of obesity.

We thank the participating volunteers for their help in completing these studies. The authors’ responsibilities were as follows—DMS, AMG, HSE, and JAY: conception and design of the study; and DMS, AMG, and JAY: analysis and interpretation of the data and drafting of the article. All authors contributed to the collection and assembly of data, provided critical revision of the article for content, and approved the final version of the manuscript. JAY is a Commissioned Officer in the US Public Health Service, Department of Health and Human Services. All authors reported that they had no disclosures relevant to this publication.

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