CORRESPONDENCE

The Importance of Brown Adipose Tissue

TO THE EDITOR: In the April 9 issue, van Marken Lichtenbelt et al.,[1] Cypess et al.,[2] and Virtanen et al.[3] report on functioning supraclavicular brown adipose tissue in adult humans. Van Marken Lichtenbelt et al. conclude that brown-adipose-tissue activity induced by exposure to cold is impaired in overweight healthy subjects, and Cypess et al., using epidemiologic-association techniques, conclude that older subjects and those with a higher body-mass index (BMI) have less brown adipose tissue. It is suggested that obesity is associated with reduced brown-adipose-tissue function or activity. However, disseminated brown adipocytes[4] within the large subcutaneous adipose-tissue mass may cumulatively represent substantial brown-adipocyte activity that may not be detected with the use of integrated positron-emission tomography and computed tomography (PET–CT) or crude biochemical studies.

Using gene profiling, we found an indication that the brown-adipose-tissue marker uncoupling protein 1 (UCP1) is positively associated with BMI in human subcutaneous adipose tissue (from 33 subjects) (Fig. 1A), as is angiopoietin 2 (ANGP2), a brown-adipose-tissue–enriched[5] angiogenic gene that is a surrogate for ^{18}F-fluorodeoxyglucose (^{18}F-FDG) uptake.[6] Furthermore, cell death–inducing DNA-fragmentation factor (CIDEA), a negative regulator of lipolysis,[7] which is inversely associated with the basal metabolic rate in humans (potentially through indirect UCP1 activation[8]), is reduced in obese subjects matched for glycemic control (Fig. 1B). Such BMI associations are markedly influenced by the presence of diabetes (Fig. 1C and 1D), tempering conclusions about disease drawn from these studies. We have found brown-adipose-tissue markers in obese subjects without diabetes, suggesting that anatomically dispersed brown adipocytes may promote metabolic homeostasis.

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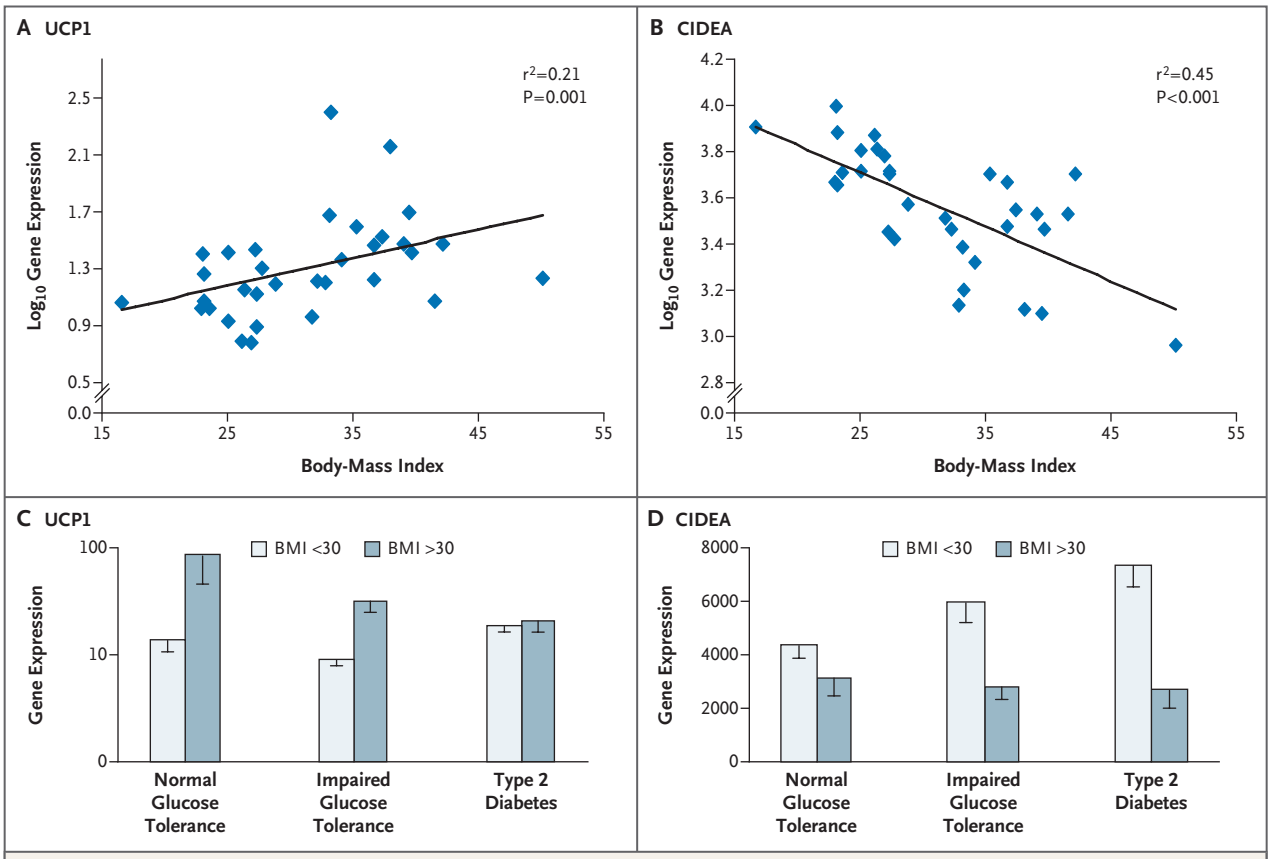

Figure 1. Gene-Expression Profiles in Subcutaneous Adipose Tissue.

Gene-expression profiles from 33 samples of human subcutaneous adipose tissue were generated with the use of the Affymetrix U133 Plus 2.0 platform followed by the selection of brown versus white mature murine adipocyte markers.[5] Gene-chip normalization avoids the use of unstable, single housekeeping genes and thus can be technically superior for clinical biomarker studies. Significant correlations for markers of brown adipocytes were subsequently calculated according to World Health Organization guidelines for type 2 diabetes, impaired glucose tolerance, and normal glucose tolerance. Panel A shows uncoupling protein 1 (UCP1) expression, and Panel B shows cell death–inducing DNA-fragmentation factor (CIDEA) expression. UCP1 and CIDEA expression are shown in Panels C and D, respectively, according to the presence or absence of metabolic disease and the body-mass index (BMI, the weight in kilograms divided by the square of the height in meters). No subject had a BMI of exactly 30. T bars indicate the standard error.

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TO THE EDITOR: Cypess et al. identified brown adipose tissue with the use of ^{18}F-FDG PET–CT scans in adults and found that the probability of detecting brown adipose tissue decreased with increasing age and BMI. No significant relationship of brown adipose tissue with fasting glycemia was found.

We analyzed data from 3604 subjects who underwent ^{18}F-FDG PET–CT scanning under thermoneutral conditions; in 110 of these subjects, brown adipose tissue was documented by the radiologist. When 198 age-matched and study-date–matched subjects without brown adipose tissue were included, the prevalence of brown adipose tissue decreased with increasing age, BMI, and fasting glycemia (Table 1). In a multivariate model, brown adipose tissue remained significantly associated with age and BMI ($P<0.001$ for both comparisons) but not with glycemia ($P=0.76$). Our data extend the findings of Cypess et al., showing an independent relationship between brown adipose tissue and BMI over a broad range of ages, and indicate that, unlike visceral fat and liver fat,[1,2] brown adipose tissue is not an independent regulator of glycemia. This hypothesis needs to be studied prospectively with the

Table 1. Relationship of Age, Body-Mass Index, and Fasting Glycemia to the Presence of Brown Adipose Tissue in 308 Selected Subjects.*

Variable	Lowest Third	Middle Third	Highest Third	P Value
	percent of subjects with brown adipose tissue			
Age	61	28	18	<0.001
BMI	56	32	18	<0.001
Fasting glycemia	38	42	26	0.049

* For these analyses, 110 subjects with brown adipose tissue were matched according to age and study date with 198 subjects without brown adipose tissue. Subjects were divided into thirds according to age and the values for body-mass index (BMI) and fasting glycemia. A univariate model was used. P values were calculated with the use of the likelihood-ratio chi-square test.

use of ^{18}F-FDG PET–CT measurements under cold exposure, as van Marken Lichtenbelt et al. and Virtanen et al. did in their studies, and precise measurements of glucose metabolism.

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TO THE EDITOR: Virtanen et al., Cypess et al., and van Marken Lichtenbelt et al. report that brown adipose tissue is present and active in adult humans and is positively associated with energy expenditure. Patients with human immunodeficiency virus (HIV)–associated lipodystrophy, in addition to having altered distribution of adipose depots, have abnormally increased energy expenditure and increased postprandial thermogenesis.[1] The mechanisms that could explain these findings remain unclear. We recently identified a high level of induction of UCP1 expression, the specific marker of brown as opposed to white adipocytes, in the enlarged dorsocervical fat depots from patients with HIV lipodystrophy.[2] In a follow-up of this study, we further identified enhanced UCP1 expression in fat-biopsy specimens from the neck and supraclavicular areas. All these regions are reported to contain potentially active brown fat. We propose that abnormal induction of brown adipose tissue in patients with HIV lipodystrophy may underlie dysregulated energy expenditure. In pharmacologic or nutritional attempts to ameliorate the metabolic status and wasting-related processes in patients with HIV infection, overactivated brown adipose tissue should be considered as a likely target.

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TO THE EDITOR: Cypess et al. suggest a potential role of brown adipose tissue in adult human metabolism but refer to "inconclusive" prior literature regarding its metabolic state. ^{18}F-FDG uptake in apparent brown adipose tissue was previously reported in 2.5 to 6.8% of patients undergoing PET–CT.[1,2] Cohade et al. reported an increased prevalence of brown adipose tissue in cold weather.[2] We reported that serum glucose levels were lower in patients with than in those without ^{18}F-FDG uptake in brown adipose tissue and suggested a possible role of low brown-adipose-tissue activity in diabetes causation.[3] Alternatively, patients with diabetes may be unable to sustain brown-adipose-tissue stores because reduced insulin signaling and UCP1 expression promote brown-adipocyte death in vitro.[4] Lean adults may require increased metabolism in brown adipose tissue for "non-shivering" thermogenesis to maintain body temperature. Whether thinner patients have

high brown adipose tissue to meet these thermogenic needs or are thin because they have active brown adipose tissue requires further study. ^{18}F-FDG PET–CT may prove vital for determining causality between increased brown-adipose-tissue metabolism and a lean phenotype, a nondiabetic phenotype, or a lean and nondiabetic phenotype. This literature and the reports in the *Journal* appear to be "conclusive" regarding the presence and metabolic activity of brown adipose tissue in many adults.

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TO THE EDITOR: Cypess et al. report the detection of brown adipose tissue by PET–CT in 5.4% of 1972 patients, a prevalence lower than that in earlier studies. They do not report on scan–rescan reproducibility, which can be highly variable,[1] and brown-adipose-tissue detection based on a single scan may underestimate the prevalence when the scan is negative. We analyzed 4834 consecutive PET–CT scans in 2934 patients between 2003 and 2008 for oncologic diagnosis and staging. Brown adipose tissue was detected in 250 patients, yielding a prevalence of 8.5%. Among 747 patients who underwent scanning more than once, 145 patients had at least one positive scan, yielding a much higher prevalence, 19.4%. We also observed a lower blood glucose level in patients with brown adipose tissue. This association was particularly evident within the same patient. In patients with multiple scans, the mean (±SD) fasting glucose level was significantly lower when brown adipose tissue was detected (4.9±0.3 mmol per liter, vs. 5.5±0.3 mmol per liter in the absence of brown adipose tissue; P=0.03). Our results indicate that brown adipose tissue is present in up to one in five patients in our cohort, and its detection by PET–CT is associated with fasting glycemia.

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TO THE EDITOR: In their article, van Marken Lichtenbelt et al. report apparently substantial uptake of ^{18}F-FDG by the heart in five of six lean young men after 2 hours of exposure to cold air (16°C), which was blunted in obese young men and absent on exposure to 22°C (Fig. 1A of their article). Cypess et al. also show one PET-CT scan that suggests uptake of ^{18}F-FDG by the heart (Fig. 1C of their article). Neither group of authors mentions or discusses the tracer uptake by the heart, although van Marken Lichtenbelt et al. cited Heaton's autopsy study,[1] performed in the 1970s, in which she demonstrated brown adipocytes in several human adult tissues, including pericardial (epicardial) fat.

It is important to know whether cold-activated ^{18}F-FDG uptake by the heart is mediated by the myocardium or the epicardial fat layer. The latter would be evidence that human adult epicardial fat contains biologically active brown adipose tissue, which, like predominant brown adipose tissue in the supraclavicular and anterior neck regions, shows less activity in obese subjects.

Effects on putative epicardial brown adipose tissue might be a relevant consideration in strategies targeting brown adipose tissue for the treatment of obesity.

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1. Heaton JM. The distribution of brown adipose tissue in the human. J Anat 1972;112:35-9.

DR. VAN MARKEN LICHTENBELT AND COLLEAGUES REPLY: The point raised by Sacks about a possible role of epicardial brown adipose tissue is interesting. At least three reasons for ^{18}F-FDG uptake in or close to the heart are possible in healthy

subjects: myocardial uptake due to glucose as a substrate for basal heart metabolism, including contraction; myocardial adaptive thermogenesis; and epicardial brown fat activity.

Indeed, the presence of epicardial brown adipocytes was demonstrated earlier.[1,2] From the data published, it is not clear how much epicardial brown fat can actually be present.

In a study by Crisan et al.,[3] one of us found that UCP1 messenger RNA (mRNA) could be detected in the skeletal muscle in adult humans, indicating UCP1 activity. It is possible that this is also true for myocardial fibers. If so, this could result in additional cold-induced ^{18}F-FDG uptake.

However, in our brown-fat study, the cardiac ^{18}F-FDG uptake during cold exposure was not present in several subjects, whereas brown adipose tissue was present in 23 of 24 subjects.[4] Of the three subjects in whom measurements were performed under thermoneutral conditions, two had ^{18}F-FDG uptake in the heart, whereas no brown-fat ^{18}F-FDG uptake could be detected. It is well known that patients have spatially and temporally variable uptake in the heart,[5] and this nonuniform uptake is likely to be related to the nonuniform transition from glucose to fatty acid metabolism. Thus, our results do not provide an indication of the alternative routes.

Timmons and Pedersen provide their findings with respect to subcutaneous UCP1, ANGP2, and CIDEA expression. They suggest that subcutaneous brown-fat activity might be increased in subjects with a high BMI, contrary to the results for brown-fat activity in our study. However, mRNA expression cannot be translated into the amount of brown-fat activity. Further studies would be needed to expand observations on UCP1 mRNA into hypotheses about functional brown-adipose-tissue activity.

Moreover, the function of such superfluous subcutaneous brown fat in overweight subjects is not clear. Subcutaneous fat appears to be a significant insulator in overweight subjects, causing reduced dissipation of body heat. Extra heat production by brown adipocytes does not seem likely. Nevertheless, the potential occurrence of subcutaneous brown fat and a possible relation to diabetes deserve further study.

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DR. CYPESS AND A COLLEAGUE REPLY: Like Lee et al., we saw scan–rescan variability. Therefore, we classified patients as being positive for brown adipose tissue if they had at least one positive scan during the study period. The prevalence we reported was per patient, not per scan.

Lee et al., Jacene and Wahl, and Stefan et al. all discuss the relationship between brown adipose tissue and glycemia. Our multivariate analysis showed that age confounded the effect of glucose to the point that it no longer achieved significance. We agree with Stefan et al., whose multivariate analysis supports our finding that the probability of detecting brown adipose tissue decreases with age and BMI but not with glucose.

We agree with Sacks that it will be important to distinguish the ^{18}F-FDG uptake seen in the myocardium from that in epicardial fat, which could be important for the treatment of obesity.

Both Villarroya et al. and Timmons and Pedersen report finding UCP1 in subcutaneous white-adipose-tissue depots, which we have also observed (unpublished data). The observation by Villarroya et al. of increased UCP1 expression in patients with HIV lipodystrophy connects brown adipose tissue to dysregulated energy expenditure and suggests a path to the identification of mediators that could safely increase metabolism in patients with obesity.

Timmons and Pedersen observed that in white adipose tissue, BMI correlated positively with UCP1 expression but negatively with CIDEA expression. This is surprising, since both are considered markers of brown adipose tissue.[1] We agree that conclusions about disease must be made carefully. As shown in Table 1 of our article, we found that the effects of BMI and glucose were confounded in part by sex and age, respectively, highlighting the complexity of the metabolic control associated with human brown adi-

pose tissue. Timmons and Pedersen also note that detection of brown adipose tissue by PET–CT is not as sensitive as biopsies followed by quantitative polymerase-chain-reaction (PCR) assay. This is almost certainly true, but the latter approach is not practical for clinical assessment. Indeed, there may be more brown adipose tissue distributed in white adipose tissue or in muscle[2] than that which was measured by us or by van Marken Lichtenbelt et al. However, the limitation of gene profiling is that the presence of UCP1 is insufficient for determining whether a particular brown-adipose-tissue depot is functional.[3] By measuring activity, PET–CT provides essential information about the potential effect of brown adipose tissue on metabolism. There may be smaller collections of functional brown adipose tissue that cannot be detected currently, and it remains to be determined how much additional metabolic importance these smaller collections will have.

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DR. ENERBÄCK AND COLLEAGUES REPLY: Villarroya et al. raise the interesting question of whether activation of brown adipose tissue can explain the dysregulated energy expenditure seen in patients with HIV lipodystrophy. In support of this possibility, they cite their study of gene expression in the so-called Buffalo hump (enlarged dorsocervical adipose tissue) in a subgroup of patients who had lipodystrophy associated with HIV type 1 (HIV-1) infection and highly active antiretroviral therapy (HAART). The gene-expression pattern in adipose-tissue samples from the Buffalo hump depots was analyzed and compared with that in tissue samples from other depots. Villarroya et al. report decreased levels of mitochondrial markers for both mRNA and protein; also,

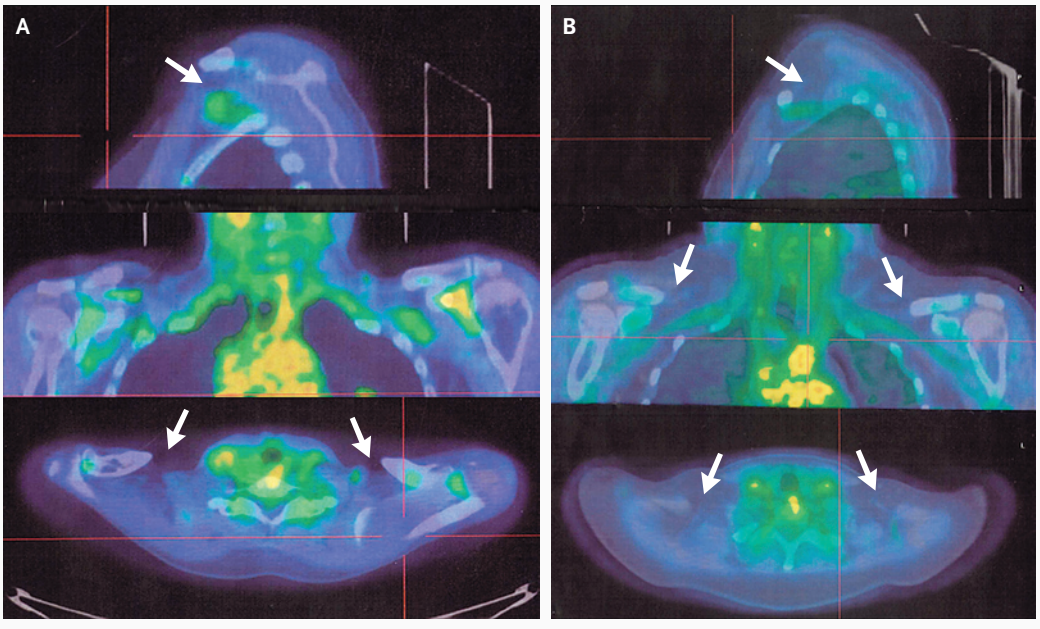

Figure 1. PET–CT Images Obtained during Cold Exposure in Two Women with Lipodystrophy Associated with Human Immunodeficiency Virus Type 1 Infection and Highly Active Antiretroviral Therapy.
No enhanced glucose uptake is evident in the supraclavicular paracervical brown-adipose-tissue depot (arrows) or in the dorsocervical region in either Patient 1 (Panel A) or Patient 2 (Panel B).

UCP1 mRNA was detected with the use of quantitative real-time PCR.[1] Unfortunately, no data regarding UCP1 protein levels or histologic studies are presented. In our view, this information is essential before any conclusions can be made regarding the potential involvement of Buffalo hump–derived brown adipose tissue in the regulation of energy expenditure in patients who have lipodystrophy associated with HIV-1 infection and HAART. To determine whether such patients have signs of enhanced cold-induced glucose uptake in the supraclavicular paracervical region, which our study showed is linked to the presence of bona fide brown adipose tissue, we performed PET–CT in two such patients (both women). No signs of cold-induced glucose uptake could be identified (Fig. 1).

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Electronic Health Records in Hospitals

TO THE EDITOR: In their study of the use of electronic health records, Jha et al. (April 16 issue)[1] report that only 1.5% of U.S. hospitals have comprehensive electronic-records systems, and 7.6% have a basic system that includes a capacity to store physicians' notes and nursing assessments in at least one clinical unit. Interestingly, doctors overwhelmingly say that electronic-records systems improve care,[2] a view that is borne out by the Veterans Health Administration (VHA) experience.[3] Nevertheless, very few U.S. physicians use electronic health records. The reasons range from cost to the lack of a national standard.[2]

The VHA already has an excellent system, the Computerized Patient Record System (CPRS), which has been successfully used for the past 10 years to manage the care of approximately 8 million veterans.[4] This system is highly reliable, and because it is government shareware, it is available free of charge (www1.va.gov/cprsdemo). The only criticism I have heard about CPRS is that it does not allow for billing, which is something that could be added. CPRS should be made the nationwide standard tomorrow or, better yet, today.

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TO THE EDITOR: Jha et al. have provided an important incremental advance in our knowledge regarding the use of electronic health records in U.S. hospitals. The authors did not include an anesthesia information management system (AIMS) in their definition of either a comprehensive inpatient electronic health record or a basic electronic record. This is an important omission. An AIMS is the anesthesia component of an inpatient electronic health record.[1] As such, it is the electronic record for a high-risk and expensive episode of inpatient clinical care. Approximately 30% of U.S. hospital admissions involve surgical care.[2]

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TO THE EDITOR: Jha et al. do not address the question of the preservation of patients' confidential information. As Gillon wrote, "The prin-