Hypermetabolism and progression of HIV infection

Dear Sir:

Several studies evaluating resting energy expenditure (REE) in HIV-infected patients have been published. This component of energy expenditure, when adjusted for differences in body composition, has been reported as being increased (1–4), decreased (5), or even normal (6, 7) in these patients. The reason for this lack of consensus remains unclear.

Given the evidence that viral load is an important predictor of the progression of HIV infection, it is of interest to assess the relation between this variable and the degree of hypermetabolism. A significant relation ($r = 0.404$, $P = 0.011$) between plasma viral load and REE was described in 1997 by Mulligan et al (8) in 36 clinically stable HIV-positive men, suggesting that energy expenditure would increase as a part of the host response to viral replication.

Recently, Grinspoon et al (9) evaluated the probable determinants of energy expenditure in 33 ambulatory HIV-infected female patients. In the accompanying editorial, Kotler and Heymsfield (10) found it surprising that there was not a significant relation between REE and viral burden in the study by Grinspoon et al. They hypothesized that such a relation would have been statistically significant had the study sample size been increased or had the viral load variable been log-transformed in the statistical analyses. Our experience suggests that such a relation does not exist.

We studied 85 HIV-infected patients (20 women, 65 men) ranging in age from 24 to 65 y. They were recruited from 2 different hospitals [Hospital Universitari de Sant Joan de Reus ($n = 50$) and Hospital Virgen del Rocio de Sevilla ($n = 35$)] at different stages of HIV infection. Thirty-three patients were free of any acute opportunistic infection and the rest ($n = 52$) had clinical evidence of an active secondary infection. After an overnight fast, body composition was estimated by bioelectrical impedance analysis (Human-Im Scan; Dietosystem, Milano, Italy) and REE was estimated by 30-min indirect calorimetry (Deltatrac; Datex, Instrumentarium, Finland). Subjects rested for 30 min before the testing began. Blood samples were taken the same day of the study to determine CD4 cell counts and viral load (Amplicor HIV-1 Monitor; Roche Molecular Systems, Inc, Branchburg, NJ). The malabsorption and nutritional status of some of the patients included in this letter were reported previously (7).

Nutritional status varied a great deal between patients. The body mass index (in kg/m^2) of our patients ranged from 14.03 to 30.78 and mean changes in body weight over the previous 3- and 1-mo periods were 5.4 ± 5.1 and 3.5 ± 3.3, respectively. REE ranged from 84.7% to 143.0% of the value predicted by the Harris-Benedict equation in patients free of opportunistic infection and from 84.8% to 152.4% in patients with active opportunistic infection.

As did Grinspoon et al (9), we found no significant relation between REE and CD4 cell counts ($r = -0.07$) or plasma concentrations of HIV RNA ($r = 0.02$) even when the viral RNA load in the total study population was log-transformed. These relations were not significant in the group of subjects free of opportunistic infections or in the rest of patients. The relation between REE and plasma viral load was consistent when patients from either hospital were analyzed separately.

The lack of a significant relation between REE and progression markers of HIV infection is, perhaps, not so surprising. After all, a considerable number of factors known to be present in HIV-infected patients are able to modulate energy expenditure and although the effects of energy intake, malabsorption, weight loss, and physical activity on energy expenditure have been studied in these patients, the considerable variability in the degree of hypermetabolism between subjects remains unexplained. Furthermore, the effects of proinflammatory cytokines, which affect intermediary metabolism, as well as those of antiretroviral therapies and other factors need to be explored.

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Reply to PP García Luna et al

Dear Sir:

In our recent article, "Determinants of Increased Energy Expenditure in HIV-Infected Women," we showed significantly increased resting energy expenditure (REE) in ambulatory, HIV-infected women (1). REE was, in fact, 119% of that predicted by the Harris-Benedict equation, which agrees with the results of many previous studies showing increased energy expenditure in this population (2–4). In the patients studied, we found no correlation between REE and viral burden. In their accompanying editorial, Kotler and Heymsfield (5) noted that the results from our study are in contrast with those of Mulligan et al (6) obtained in 36 HIV-positive men, in whom REE was also significantly increased to 112% of that predicted by the Harris-Benedict equation. In the patients reported on by Mulligan et al, a significant positive correlation between REE, expressed per kilogram lean body mass, and viral load was noted. Furthermore, Kotler and Heymsfield suggested that a potential relation between REE and viral load might have been noted if our data had been log-transformed or if the patient population had been larger. However, no such positive relation was found even when the data were log-transformed (**Figure 1**). In contrast, our data suggested an inverse correlation between REE and viral load.

Similarly, Suttmann et al (7) did not find a consistent relation between REE and immune function as measured by CD4 cell count in 60 HIV-infected patients (primarily men). In the accompanying letter, García Luna et al (8) also reported on the relation between REE and viral load. In 85 HIV-infected patients, no significant relation was found between REE and viral load and the overall *r* value for the comparison was negative. However, the data from García Luna et al are not broken down by sex, which would be useful, considering that the data from Mulligan et al were collected in men in contrast with our data in women.

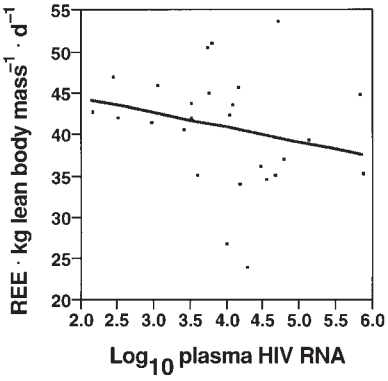

FIGURE 1. Relation between resting energy expenditure (REE) and viral load.

The mechanism of increased REE in HIV-infected patients remains unknown and may relate to cytokine abnormalities or other factors (2). If REE were positively associated with viral load, it might be hypothesized, as suggested by Mulligan et al, that increased energy expenditure is a direct result of progressive HIV infection (6). As a corollary, control of HIV replication might result in reduced energy expenditure. Although this hypothesis remains an interesting one, we still must determine whether there is a relation between REE and viral load, and if so, whether there are sex-specific differences between HIV-infected men and women with respect to this relation. Further research is needed to better define the relation, if any, between REE and viral load. Useful data might be obtained from longitudinal studies in patients receiving potent antiviral agents, in whom REE could be assessed before and after therapy.

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Reply to PP García Luna et al

Dear Sir:

Resting energy expenditure (REE) in humans is closely linked with body mass (1). A rise of ≈10% or more above a predicted REE for body mass, age, and other predictor variables represents a hypermetabolic state (2). A corresponding decrease of 10% or more in resting metabolic rate represents a hypometabolic state.

Both absolute and relative REE elevations are well documented with hyperthyroidism, catabolic injury, overfeeding, fever, and many other conditions (3). Similarly, absolute and relative reductions in REE are recognized in hypothyroidism, semistarvation, and hypothermia. Periodic fluctuations in REE are also observed with variation in menstrual cycle activity (3). REE is thus a dynamic physiologic measure that varies in magnitude either up or down with many common physiologic states and pathologic conditions.

Although early investigators used body surface area to adjust REE for between-individual comparisons, the modern approach is to adjust REE for metabolically active tissue (1, 4). The usual compartment selected is fat-free body mass (FFM), although other approaches are recognized. Measured REE is first regressed against FFM for a group of fasting subjects and this establishes the basic relation between resting thermogenesis and body mass (4). Additional potential predictors of REE are then entered into the model, and usually others such as sex, fat mass, and age are found significant (4). However, the contribution of the variables other than FFM to REE prediction is very small and large subject samples (eg, several hundred subjects) are often required to achieve statistical significance. Hyper- or hypometabolism would presumably be established after accounting for REE variance secondary to these recognized physiologic predictors.

García Luna et al (5) in their letter examine the relation between REE and body mass in a heterogeneous group of 85 HIV-positive patients. The patients ranged from severely underweight to obese [body mass index (in kg/m^2) range: 14–31] and hypometabolic to hypermetabolic (REE ranged from 85% to 143% of predicted based on height, weight, and age). The authors explore the possibility that REE is significantly correlated with plasma viral load, a finding that would suggest that energy expenditure is increased as part of the host response to viral infection. Studies examining this question have produced mixed results and García Luna et al did not detect a significant relation between REE and CD4 cell counts or plasma HIV RNA concentrations. However, the authors did not provide information on how REE prediction models were developed. Was FFM used as an independent variable representing metabolically active tissue? Were other covariates, such as sex and age, statistically significant in developed REE models? Was the magnitude of observed associations between REE and other predictor variables (eg, R^2) similar to that reported by other investigators? Was the sample size adequate to detect the hypothesized difference?

We agree with García Luna et al that our understanding of the underlying mechanisms of widely observed hyper- and hypometabolism in HIV-positive patients remains an incompletely understood and fascinating problem. Because HIV is a prevalent infection, the opportunity remains to unravel the many and complex REE determinants in large scale, carefully executed, prospective studies. The information they could provide will give new insights into the many complex and interacting factors that determine REE and human energy requirements.

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Lactose maldigestion and calcium from dairy products

Dear Sir:

The recent publication of a paper in this Journal suggesting that lactose maldigestion should not be considered an impediment to consuming dairy products to obtain dietary calcium (1), which was sponsored by the National Dairy Council, may mislead the readers. First, dairy products may not be a good source of calcium for reasons other than lactose intolerance. A recent report from the Nurses' Health Study, which included 121 701 women aged 30–55 y at enrollment in 1976, concluded that the cohort study data do not support the hypothesis that a high consumption of milk or other food sources of calcium by adult women protects against hip or forearm fractures (2).

To examine the link between milk and osteoporosis further, an ecologic approach was used to study hip fracture incidence rates for the white and total populations from 9 countries (3). The data for Finnish women were omitted because they were both an outlier with respect to women from other countries and inconsistent with rates of hip fracture for the Finnish men. As shown in **Table 1**, dietary milk and its components, especially milk protein, have a much higher statistical association with hip fracture incidence

than do other likely factors such as fat, protein, and sweeteners (4). When linear regressions were run for milk protein, the *r* value for women was 0.800 (P = 0.005) and for men was 0.593 (P = 0.054). What the statistical results show is that living in countries with a high dairy consumption is a risk factor for osteoporosis. They do not necessarily imply that consumption of dairy products causes osteoporosis; however, they do suggest that further investigations be conducted to determine why the associations are so high.

In addition, the annual hip-fracture rate of black females in California was 43% that of white females (219 compared with 559 cases/100 000 persons) (3), whereas the hip-fracture rate of black females in Washington, DC, was 51% that of white females (118.8 compared with 231.8 cases/100 000 persons) (5, 6). African Americans are generally lactose intolerant and have lower milk consumption rates than do white Americans. Perhaps their diet, genetic makeup, or both lead to strong bones and therefore dairy products or large amounts of dietary calcium are not as important as they are for whites.

Other common chronic diseases are now linked to calcium and milk consumption. Lactose from unfermented dairy products such as milk and yogurt has the highest association with ischemic heart disease of any dietary macronutrient for men of all ages and postmenopausal women (7–9). A possible mechanism is the metabolism of lactose into triacylglycerol and its incorporation into VLDL cholesterol. In addition, milk and calcium intakes have been found in cohort studies in 5 countries to be the highest risk factors for prostate cancer (10). The proposed mechanism is a reduction in circulating vitamin D by calcium because vitamin D is involved in the incorporation of calcium into bone (10). Vitamin D has been shown to kill prostate cancer cells in vitro (11).

Thus, there are many good reasons not to consume dairy products. Those concerned about osteoporosis, which has a complex etiology, should review the report by Brown (5), which delves far beyond the relation between osteoporosis and calcium intakes into such other factors as the dietary acid-alkaline balance, trace minerals, exercise, and exposure to sunlight.

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Reply to WB Grant

Dear Sir:

We emphasize that the sole purpose of our study was to determine whether lactose maldigesters are able to tolerate lactose in dairy products that provide ≈1500 mg Ca/d. The results of our study clearly suggested that this is the case (1).

The complex issue of the potential benefit or harm of a dairy-rich diet was not the topic of our study. However, the results of many studies have supported the benefits of a high-calcium diet in the prevention of osteoporosis (2–9) and the evidence in this regard was sufficient to convince a National Institutes of Health consensus conference to recommend that postmenopausal women ingest 1500 mg Ca/d (9).

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Protein intake and the risk of hip fracture in postmenopausal women

Dear Sir:

I read with interest the study by Munger et al (1), who reported a negative association between protein intake and the risk of hip fracture in postmenopausal women. They also reported a negative association between total carbohydrate intake, which was higher in the women with hip fractures than in those without hip fractures, and animal protein intake. A high consumption of sugars increases the urinary excretion of calcium and magnesium (2–4) and may well be a risk factor for osteoporosis (5). It would have been useful, therefore, to have controlled for simple carbohydrate intake in the multivariate analyses. Similarly, a high sodium intake increases the urinary excretion of calcium and represents a risk factor for osteoporosis (6, 7). Controlling for dietary sodium would also have been of value.

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Reply to JB Lavine

Dear Sir:

We agree with Lavine that dietary intake of carbohydrate and other nutrients may be involved in osteoporosis. We emphasized the possible role of protein intake in our report (1) because it was the nutrient with the strongest dose-response relation to risk of hip fracture and because a good deal of other evidence has accumulated suggesting that protein intake is important in bone health.

In our report we stated that "analyses based on single nutrients derived from dietary questionnaires must be interpreted with caution because of the collinearity of nutrient intakes." The relation between carbohydrate and protein intakes is a good example of this problem. In our study, intake of animal protein was negatively correlated with carbohydrate intake ($r = -0.56$, $P < 0.0001$). Carbohydrate intake was positively associated with risk of hip fracture, but this finding was diminished in multivariate analyses with nonnutrient variables and fell below the threshold of statistical significance. Because of our relatively small number of hip fracture cases and the collinearity of protein and carbohydrate intakes, multivariate models with simultaneous inclusion of these 2 nutrient variables were not interpretable. A clearer picture may emerge in subsequent analyses with larger sample sizes. A better understanding of the possible role of carbohydrate intake in the risk of osteoporotic fractures is important because of current dietary trends in which fat and protein are replaced with carbohydrates.

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REFERENCE

1. Munger RG, Cerhan JR, Chiu BC-H. Prospective study of dietary protein intake and risk of hip fracture in postmenopausal women. Am J Clin Nutr 1999;69:147–52.

Erratum

Thureen PJ, Anderson AH, Baron KA, Melara DL, Hay WW Jr, Fennessey PV. Protein balance in the first week of life in ventilated neonates receiving parenteral nutrition. Am J Clin Nutr 1998;68:1128–35.

On page 1131, Table 2, the energy values under the column heading "Nonprotein energy intake" are inaccurate. The correct values, from top to bottom, are as follows: 334, 240, 202, 208, 207, 215, 188, 256, 180, 196, 167, 233, 132, 139, 80, 103, 194, 177, and 187 $kJ \cdot kg^{-1} \cdot d^{-1}$.

Erratum

Willett WC. Is dietary fat a major determinant of body fat? Am J Clin Nutr 1998;67(suppl):556S–62S.

Figures 3 and 4 were inadvertently switched in the publication process and the weight scale for Figure 3 was incorrectly converted to kilograms. The corrected figures appear below.

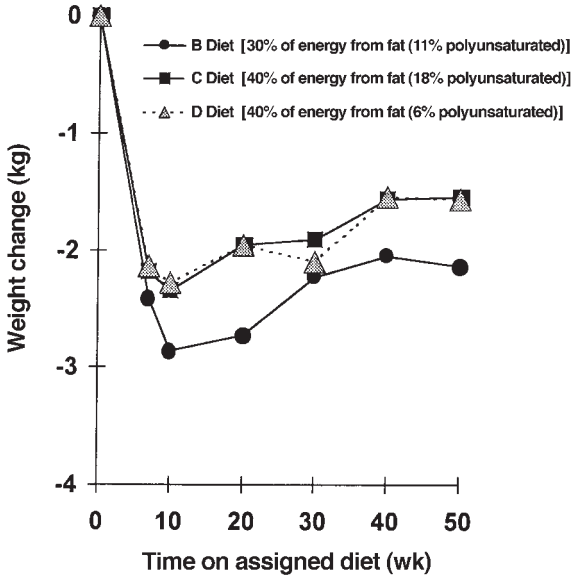

FIGURE 3. Changes in fat and energy intake and weight change over 1 y (37).

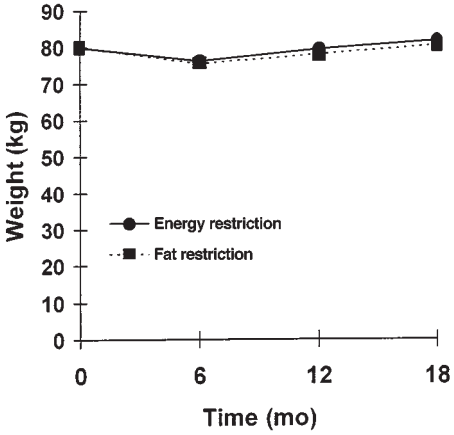

FIGURE 4. Fat and energy restriction and weight change over 18 mo (41).