# Postabsorptive Resting Metabolic Rate and Thermic Effect of Food in Relation to Body Composition and Adipose Tissue Distribution

F. Armellini, M. Zamboni, A. Mino, L. Bissoli, R. Micciolo, and O. Bosello

**One hundred thirty subjects were studied to investigate relationships between the body composition and fat distribution as evaluated by computed tomography and the resting metabolic rate (RMR) as evaluated by indirect calorimetry: 82 premenopausal women (age, 18 to 52 years; body mass index [BMI], 27 to 52 kg/m$^2$), 27 postmenopausal women (46 to 71 years; 28 to 49 kg/m$^2$), and 21 men (18 to 70 years; 31 to 43 kg/m$^2$). The thermic effect of food (TEF) was evaluated in all men and in 2 subgroups of 55 and 19 women. The best-fitting equations for predicting RMR, obtained by multiple regression, included the following as covariates: fat-free mass and both subcutaneous and visceral adipose tissue in premenopausal women ($R^2$ = .55, $P$ = .0001), fat-free mass and visceral adipose tissue in postmenopausal women ($R^2$ = .58, $P$ = .001), and age, with minus sign, and visceral adipose tissue in men ($R^2$ = .44, $P$ = .0051). Fasting insulin and fat-free mass, with minus sign, and both visceral and subcutaneous adipose tissue were the predictors of the TEF ($R^2$ = .25, $P$ = .0055) in premenopausal women. This study demonstrates that visceral fat distribution is important in determining the RMR in postmenopausal women and men. In premenopausal women, total adipose tissue is a main determinant of both the RMR and TEF. This last effect could be counterbalanced by insulin resistance.**


THE RESTING METABOLIC RATE (RMR) and thermic effect of food (TEF) are 2 main components of total daily energy expenditure in each individual. These rates are determined by factors such as sex, age, body weight, fat-free mass, body fat, and body fat distribution.[1-8]

Studies in obese and non-obese individuals showed no difference in the RMR between the 2 groups after matching for fat-free mass.[9] Results for the relationship between the RMR and fat distribution measured by reliable methods are controversial.[10-17] For postprandial thermogenesis, some studies have shown that the TEF is significantly smaller in obese versus lean subjects,[18-23] while other studies fail to demonstrate any difference.[24-27] Significant associations have been reported between the TEF and visceral fat location in women.[12,28,29]

The aim of the present study was to investigate in a group of obese men and women the relationships between body composition and fat distribution as evaluated by computed tomography and the RMR and TEF as evaluated by indirect calorimetry.

## SUBJECTS AND METHODS

One hundred thirty subjects were studied: 82 premenopausal women aged 18 to 52 years with a body mass index (BMI) between 27 and 54 kg/m$^2$, 27 postmenopausal women aged 46 to 71 years with a BMI between 28 and 49 kg/m$^2$, and 21 men aged 18 to 70 years with a BMI between 31 and 43 kg/m$^2$. Females were tested for pregnancy and gonadotropin levels were measured. No pregnancies were found. Amenorrheic but not menopausal women were excluded from the study. All subjects had normal circulating levels of thyroid hormones and thyrotropin. Characteristics of the subjects are shown in Table 1. Thirty-five women did not complete the study with the test meal, for different reasons. As a consequence, the TEF was assessed in all men and in 2 subgroups of women. Their characteristics are listed in Table 2. Patients were asked not to change their dietary habits in order to avoid changing their weight. After recovery, they were placed on a weight-maintaining diet containing 55% of energy as carbohydrates. All were inpatients in a metabolic unit. The subjects were familiarized with the experimental procedures and provided informed consent. The study was approved by the Ethics Committee of Verona Hospital.

Patients were weighed in the morning in their nightclothes. The following measurements were taken: age, weight, height, BMI, body fat, fat-free mass, total adipose tissue area, visceral adipose tissue area, subcutaneous adipose tissue area (total-visceral), RMR, TEF, and blood glucose and insulin both fasting and after a glucose load.

Total and visceral adipose tissue areas were determined by computed tomography according to the method of Sjöström et al.[30,31] The usual radiographic parameters for abdominal investigations were used: 125 kV, 350 mA, scanning time 4 seconds, and slice thickness 8 mm. A lateral topogram (scout view) was performed to identify the standard level, the fourth lumbar vertebra, with precision. Regions of interest were outlined using a light-pen cursor (joystick). By assessing the number of pixels within the fat density range (−150 and −50 Hounsfield units), the cross-sectional areas of both visceral (including retroperitoneal, mesenteric, and omental fat) and total fat were calculated. Subcutaneous adipose tissue area was calculated by difference. The visceral to subcutaneous adipose tissue area ratio was calculated. The total body fat volume was calculated from the total adipose tissue area according to the following formulas[32]: for males, body fat volume = 0.0356 (total adipose tissue area) +0.704 (weight/height) −22.9; for females, body fat volume = 0.0385 (total adipose tissue area) +0.841 (weight/height) −20.7. Body fat in kilograms was obtained by multiplying body fat volume by the mean density of human fat, 0.923.[33] Fat-free mass was obtained from the difference between body weight and body fat. The coefficient of variation of the method was 1.2%.

The RMR and TEF were assessed by indirect calorimetry[34,35] using a MMC Horizon System 6 (Beckman Sensormedics, Milan, Italy) that measures resting oxygen uptake and resting carbon dioxide production. Subjects were familiarized with the canopy of the calorimeter so that they did not feel suffocated during the measurement period. Gas analysis was performed in the morning after 12 hours of fasting. The RMR was measured continuously for 60 minutes. Mean energy expenditure for the last 45 minutes was used as the RMR after a steady-state condition was achieved. The TEF was evaluated after RMR measurement. Measurements were performed for 6 hours, starting 1

*From the Division of Geriatrics, San Lorenzo Hospital, Valdagno; Institute of Geriatrics, University of Verona, Verona; and Institute of Statistics, University of Trento, Trento, Italy.*

*Submitted March 2, 1998; accepted July 21, 1999.*

*Supported by grants from the Ministero dell'Università e della Ricerca Scientifica e Tecnologica and the National Research Council targeted Project "Disease Prevention and Control Factors," subproject "Nutrition" (94.00771.PF41).*

*Address reprint requests to F. Armellini, MD, Division of Geriatrics, San Lorenzo Hospital, 36078 Valdagno, Italy.*



*0026-0495/00/4901-0013$10.00/0*

**Table 1. Characteristics of All Subjects**

| Characteristic | Women | | Men |
|---|---|---|---|
| | Premenopausal | Postmenopausal | |
| Subjects (n) | 82 | 27 | 21 |
| Age (yr) | 34 ± 9.8[a] | 58 ± 6.3[b] | 41 ± 13.2[a] |
| Weight (kg) | 100 ± 18.8[a] | 96 ± 17.8[a] | 116 ± 13.8[b] |
| BMI (kg/m²) | 38 ± 6.5 | 38 ± 5.5 | 37 ± 3.3 |
| Body fat (kg) | 54 ± 14.9[a] | 52 ± 12.6[ab] | 44 ± 8.4[b] |
| Fat-free mass (kg) | 46 ± 6.8[a] | 44 ± 7.8[a] | 72 ± 8.3[b] |
| Visceral AT area (cm²) | 137 ± 85[a] | 191 ± 76[b] | 224 ± 103.1[b] |
| Subcutaneous AT area (cm²) | 563 ± 178.7[a] | 481 ± 141.7[b] | 470 ± 114.9[b] |
| Visceral/subcutaneous AT | 0.31 ± 0.597 | 0.43 ± 0.204 | 0.51 ± 0.276 |
| Fasting insulin (pmol/L) | 97 ± 36.8 | 97 ± 43.8 | 111 ± 62.5 |
| Insulin area (fmol/min)* | 362 ± 516.6[ab] | 254 ± 225.1[a] | 665 ± 792.1[b] |
| Respiratory quotient | 0.79 ± 0.094 | 0.81 ± 0.081 | 0.80 ± 0.073 |
| RMR (kJ/min) | 5.1 ± 0.76[a] | 4.6 ± 0.66[b] | 6.4 ± 0.99[c] |

NOTE. Results are the mean ± SD. Values not sharing a common superscript are significantly different, $P < .05$ (Scheffé test after ANOVA).

Abbreviation: AT, adipose tissue.

*Incremental area after glucose load.

half-hour after the meal and alternating 1 half-hour of measurement with 1 half-hour of rest under an open canopy to permit patients to go to the bathroom. Values were considered acceptable when a steady-state condition was achieved. Mean values for oxygen intake and carbon dioxide production were calculated for each hour for 6 hours. Meal energy was calculated on the basis of fat-free mass, using a liquid formula (1 mL = 4.2 kJ) containing 53% carbohydrates, 30% lipids, and 17% proteins. Ten milliliters were given for every 1 kg of fat-free mass up to a maximum of 500 mL. This value was reached by 8 premenopausal women, 2 postmenopausal women, and all men.

**Table 2. Characteristics of the Subjects Who Completed the Study**

| Characteristic | Women | | Men |
|---|---|---|---|
| | Premenopausal | Postmenopausal | |
| Subjects (n) | 55 | 19 | 21 |
| Age (yr) | 34 ± 9.7[a] | 58 ± 5.7[b] | 41 ± 13.2[c] |
| Weight (kg) | 99 ± 17.1[a] | 92 ± 16.7[a] | 116 ± 13.8[b] |
| BMI (kg/m²) | 38 ± 5.6 | 37 ± 5.6 | 37 ± 3.3 |
| Body fat (kg) | 53 ± 13.2[a] | 49 ± 12.9[ab] | 44 ± 8.4[b] |
| Fat-free mass (kg) | 46 ± 7.2[a] | 43 ± 6.8[a] | 72 ± 8.3[b] |
| Visceral AT area (cm²) | 136 ± 89.8[a] | 167 ± 53.8[ab] | 224 ± 103.1[b] |
| Subcutaneous AT area (cm²) | 554 ± 173 | 466 ± 144.5 | 470 ± 114.9 |
| Visceral/subcutaneous AT | 0.34 ± 0.723 | 0.39 ± 0.155 | 0.51 ± 0.276 |
| Fasting insulin (pmol/L) | 97 ± 35.4 | 90 ± 45.1 | 111 ± 62.5 |
| Insulin area (fmol/min)* | 314 ± 313.1[a] | 194 ± 196.1[a] | 665 ± 792.1[b] |
| Respiratory quotient | 0.80 ± 0.105 | 0.82 ± 0.085 | 0.80 ± 0.073 |
| RMR (kJ/min) | 5.1 ± 0.78[a] | 4.5 ± 0.6[b] | 6.4 ± 0.99[c] |
| TEF (kJ/6 h)† | 260 ± 144[a] | 221 ± 143.5[a] | 396 ± 156.6[b] |
| TEF (%)‡ | 14 ± 8 | 12 ± 7.8 | 13 ± 4.7 |

NOTE. Results are the mean ± SD. Values not sharing a common superscript are significantly different, $P < .05$ (Scheffé test after ANOVA).

*Incremental area after glucose load.

†Incremental area above the RMR value.

‡Incremental area above the RMR value as a percent of the energy content of the meal.

Postprandial energy expenditure was considered as the area under the curve of the energy expenditure measurements during 6 hours. The TEF during the 6-hour period after ingestion of the test meal was calculated in 2 ways: (1) TEF expressed as the difference of areas (the absolute increment in energy expenditure above the postabsorptive status due to ingestion of the test meal), TEF (kJ/6 h) = postprandial EE area − RMR × 360 min; and (2) TEF expressed as a percent of the energy content of the test meal, TEF (%) = (kJ/6 h) × 100/meal energy. The within-person day-to-day coefficients of variation by double determinations in 17 subjects for the RMR and 11 subjects for the TEF, were 5.57% for the RMR and 27.4% for the TEF.

Specimens for fasting blood analysis were obtained in the morning from an antecubital vein. A 75-g glucose solution was then administered orally and specimens were drawn at 30, 60, 90, 120, and 180 minutes after the glucose load. Eighteen premenopausal women (11 in the TEF group), 10 postmenopausal women, and 6 men showed impaired glucose tolerance according to World Health Organization criteria.[36] Among subjects with impaired glucose tolerance, 2 premenopausal women, 3 postmenopausal women, and 2 men showed a mild diabetic pattern (blood glucose at 120 minutes, 248, 251, 227, 239, 241, 204, and 207 mg/dL, respectively). The plasma glucose level was measured by a glucose analyzer (Beckman Instruments, Palo Alto, CA). Plasma immunoreactive insulin was measured by radioimmunoassay using a commercial kit (Medgenis, Brussels, Belgium); the intraassay and interassay coefficient of variation was 3% and 6%, respectively.

Differences between groups were analyzed by 1-way ANOVA. When ANOVA was significant, multiple comparisons were performed by Scheffé tests. Simple linear correlation was calculated to quantify the degree of association between variables. Multiple regression analysis was performed to determine the predictive power of several measurements for energy expenditure both fasting and after a mixed meal. Independent variables were age, fat-free mass, visceral and subcutaneous adipose tissue area, and insulin both fasting and after a glucose load.[37]

## RESULTS

The women in our study (Table 1) had the same BMI as the men. Postmenopausal women were older than premenopausal women and men. Men had higher fat-free mass than women. Postmenopausal women and men had a larger visceral and smaller subcutaneous adipose tissue area than premenopausal women. Postmenopausal women showed the lowest values for insulin area. No differences were observed in fasting insulin and the respiratory quotient. Men had higher RMR than premenopausal women, who in turn had higher RMR than postmenopausal women.

Characteristics of the subjects who also performed the meal test (Table 2) were similar. TEF values in absolute terms were higher in men but similar in the 3 groups of patients when expressed as a percent of the energy content of the meal.

Table 3 shows correlations between calorimetric measures and age, anthropometric measures, and both fasting insulin and the insulin area after a glucose load. Correlations between the RMR and anthropometric measures were always significant in premenopausal women. No significant correlations were observed with the TEF. Visceral adipose tissue area was correlated (not shown) with fat-free mass ($r = .28$, $P < .05$) and fasting insulin ($r = .35$, $P < .01$). Significant correlations were observed between the RMR and body fat, fat-free mass, and fasting insulin in postmenopausal women. They also showed a significant negative correlation between the TEF and visceral

**Table 3. Correlations Between Calorimetric Variables and Age, Anthropometric, and Metabolic Variables in 82 Premenopausal Women, 27 Postmenopausal Women, and 21 Men**

| | Women | | | | | |
|---|---|---|---|---|---|---|
| | Premenopausal | | Postmenopausal | | Men | |
| Variable | RMR | TEF* | RMR | TEF† | RMR | TEF |
| Age | −.076 | −.099 | −.026 | −.423 | −.538§ | −.066 |
| Weight | .703‖ | .094 | .666‖ | −.026 | .374 | −.026 |
| Body fat | .659‖ | .227 | .548§ | −.064 | .203 | −.071 |
| Fat-free mass | .504‖ | −.193 | .631‖ | .051 | .417 | .029 |
| Visceral AT area | .407‖ | .148 | .318 | −.569§ | −.131 | .036 |
| Subcutaneous AT area | .455‖ | .176 | .269 | .151 | .261 | −.211 |
| Fasting insulin | .289§ | −.192 | .477‡ | .018 | .336 | −.314 |
| Insulin area | −.055 | .026 | .177 | −.239 | .045 | −.427‡ |

*In 55 subjects.
†In 19 subjects.
‡$P < .05$.
§$P < .01$.
‖$P < .001$.

adipose tissue area. No significant correlations were observed between anthropometric and calorimetric measures in men. They showed a significant negative correlation between age and RMR and a strong significant correlation (not shown) between age and visceral adipose tissue area ($r = .739$, $P < .001$). A significant negative correlation was observed in men between the TEF as a percent of meal energy and the insulin area.

Results of multiple regression analysis are shown in Table 4. Dependent variables were the RMR and TEF as a percent of meal energy. Independent variables were age, fasting insulin, insulin area, visceral adipose tissue area, subcutaneous adipose tissue area, and fat-free mass. The predictive equation for the RMR in premenopausal women ($R^2 = .55$) included age, with minus sign, fat-free mass, and visceral and subcutaneous adipose tissue area as covariates (standardized coefficients, 14%, 25%, 29%, and 32%, respectively). The predictive equation for the RMR in postmenopausal women ($R^2 = .58$) included visceral adipose tissue area and fat-free mass as covariates (standardized coefficient, 38% and 62%, respectively). The predictive equation for the RMR in men ($R^2 = .44$) included age, with minus sign, and visceral adipose tissue area as covariates (standardized coefficient, 62% and 38%, respectively). As already stated, the TEF was inversely related to insulin area in men and to visceral adipose tissue area in postmenopausal women. These correlations disappeared when the joint effect of the other covariates was considered. The predictive equation for the TEF in premenopausal women ($R^2 = .25$) included fasting insulin and fat-free mass as negative predictors and visceral and subcutaneous adipose tissue area as positive predictors (standardized coefficient, 26%, 20%, 31%, and 23%, respectively).

Figure 1 is a scatterplot of fasting insulin and body fat. Points were arbitrarily divided into 4 groups and the mean values for the TEF were calculated. Subjects with lower body fat and higher fasting insulin showed the lowest TEF values.

## DISCUSSION

Body composition, and adipose tissue distribution in particular, were studied as determinants of the RMR and TEF in 130 subjects, 82 premenopausal women, 27 postmenopausal women, and 21 men. Body fat and fat-free mass are considered 2 important determinants of the RMR.[38] Our study results confirm this and point to independent contributions by the 2 components of body fat: visceral and subcutaneous. Visceral adipose tissue was always a significant predictor of the RMR in the 3 groups of patients when the joint effect of several parameters was considered. Age, fat-free mass, and both subcutaneous and visceral adipose tissue were the determinants of the RMR in premenopausal women. Fat-free mass and visceral adipose tissue were the determinants of the RMR in postmenopausal women. Predictors of the RMR in men were age and visceral adipose tissue.

Age, taking the joint effect of covariates into consideration, was a significant negative predictor of the RMR in men and to a lesser extent in premenopausal women, but not in postmenopausal women. The following findings can help to explain such a result. It is known[38] that the RMR shows a curvilinear decline with age beginning earlier in men (~40 years) than in women (~50 years). In addition, this decline is greater in men than in

**Table 4. Predictive Equations for RMR and TEF**

| Parameter | Value | Standard Error | Standardized Coefficient (%) | $t$ | $P$ |
|---|---|---|---|---|---|
| Premenopausal women | | | | | |
| Dependent variable: RMR | | | | | |
| $R^2 = .547$, $P = .0001$ | | | | | |
| Intercept | 2.259 | | | | |
| Age (yr) | −0.015 | 0.0006 | 14 | 2.3 | .0222 |
| Fat-free mass (kg) | 0.038 | 0.009 | 25 | 4.2 | .0001 |
| Visceral AT area ($cm^2$) | 0.004 | 0.001 | 29 | 4.8 | .0001 |
| Subcutaneous AT area ($cm^2$) | 0.002 | 0.0003 | 32 | 5.6 | .0001 |
| Dependent variable: TEF (%) | | | | | |
| $R^2 = .25$; $P = .0055$ | | | | | |
| Intercept | 23.2 | | | | |
| Fat-free mass (kg) | −0.32 | 0.14 | 20 | 2.9 | .0265 |
| Fasting insulin (pmol/L) | −0.59 | 0.21 | 26 | 2.8 | .0078 |
| Subcutaneous AT area ($cm^2$) | 0.015 | 0.006 | 23 | 2.5 | .0169 |
| Visceral AT area ($cm^2$) | 0.039 | 0.013 | 31 | 3.1 | .0035 |
| Postmenopausal women | | | | | |
| Dependent variable: RMR (kJ/min) | | | | | |
| $R^2 = .578$, $P = .001$ | | | | | |
| Intercept | 1.305 | | | | |
| Visceral AT area ($cm^2$) | 0.004 | 0.001 | 38 | 3.2 | .0039 |
| Fat-free mass (kg) | 0.059 | 0.011 | 62 | 5.2 | .0001 |
| Men | | | | | |
| Dependent variable: RMR (kJ/min) | | | | | |
| $R^2 = .444$, $P = .0051$ | | | | | |
| Intercept | 8.112 | | | | |
| Visceral AT area ($cm^2$) | 0.006 | 0.003 | 38 | 2.2 | .0377 |
| Age (yr) | −0.073 | 0.02 | 62 | 3.7 | .0016 |

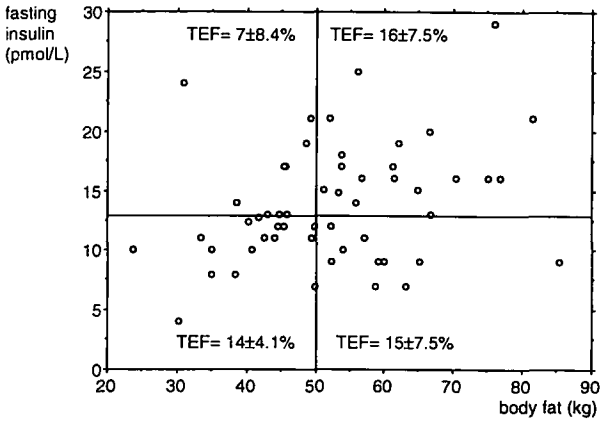


**Fig 1. Scatterplot of fasting insulin versus body fat in 55 premenopausal women. Subjects were arbitrarily subdivided into 4 groups according to a cutoff value of 12.8 pmol/L for fasting insulin and 50 kg for body fat. High insulin–low fat, n = 9; high insulin–high fat, n = 19; low insulin–low fat, n = 16; low insulin–high fat, n = 11. TEF values for the high insulin–low fat group were significantly different ($P < .05$) from those of the low insulin–high fat group.**

women. It is also known that growth hormone is a regulator of the RMR,[39] and in fact, RMR values are increased in acromegaly[40] and in growth hormone replacement therapy for growth hormone deficiency.[39] On the other hand, age is negatively related to 24-hour growth hormone release, and this phenomenon is considerably stronger in men than in women.[41]

Fat-free mass was not a significant predictor of the RMR in men, a finding that is in contrast with the literature.[4] A possible explanation for this could be an unusual selection of men with a narrow range of BMIs in contrast to a large range of age and visceral adipose tissue.

Only the visceral part of body fat predicted the RMR in men and menopausal women. This could depend on the large quantity of visceral adipose tissue in these subjects. A short comment is necessary to clarify how the visceral adipose tissue area, which in men was not significantly correlated with the RMR ($r = -.131$), became significant when connected with age. The correlation between visceral adipose tissue and the RMR is hidden by the negative correlation between age and RMR ($r = -.538$) and the positive correlation between age and visceral adipose tissue ($r = .739$). When the joint effects of age and visceral adipose tissue are considered, the latter becomes a significant predictor.

Another possible interesting joint effect of covariates concerned the TEF in premenopausal women. In this equation, body fat (taking visceral and subcutaneous fat together) is a positive predictor and fat-free mass and fasting insulin are negative predictors of the TEF. Fasting insulin significantly correlates with the degree of insulin resistance[42] in both normal and impaired glucose tolerance. Insulin-resistant subjects could have a low TEF as a consequence of insulin resistance with reduced glucose uptake and storage.[4,43] A relatively large inactive fat-free mass could contribute to hyperinsulinemia with insulin resistance. This phenomenon is illustrated in Fig 1, where insulin-resistant subjects with lower body fat show the lowest TEF values. The other groups show similar TEF values, probably as a consequence of different degrees of insulin resistance and different proportions among fat-free, visceral fat, and subcutaneous fat compartments. Fasting insulin is actually only a marker and not a direct measurement of insulin resistance. In addition, the weakness of associations and the wide coefficient of variation for the TEF advise caution in formulating hypotheses.

An important bias in our study concerns the nutrient load. The nutrient load was given per kilogram of fat-free mass until this reached 50 kg. Beyond this limit, energy intake was the same. As a consequence, 2 postmenopausal women, 8 premenopausal women, and all men had the same 2,100-kJ load. This could have influenced the negative results in men. For the women, we repeated our analysis without these subjects and the results did not change. This is probably because the fat-free mass of the above-50-kg women was only slightly over this limit.

Only 4 reports in the literature[5,6,16,17] have examined visceral adipose tissue in relation to the RMR, and only 2[5,16] in relation to the TEF. Our results partially agree with these.

In a previous study[17] that examined 27 fertile women, we were not able to show a significant association between visceral fat and the RMR. The statistical analysis was different and the joint effect of covariates was not studied. In the present study, both subcutaneous and visceral adipose tissue entered the final regression equation. This suggests that total adipose tissue is more important than visceral or subcutaneous adipose tissue in determining the RMR in fertile women.

Busetto et al[6] studied fat distribution as measured by single-slice computed tomography in 12 morbidly obese premenopausal women who underwent bariatric surgery. By multiple regression, they found that visceral fat and fat-free mass changes after a 24-kg weight loss were independently related to RMR changes.

Leenen et al[5] studied 78 healthy obese subjects, 40 premenopausal women and 38 men. Fat distribution was evaluated by magnetic resonance imaging between the lower rib margin and the iliac crest. They reported a significant correlation between the visceral adipose tissue area and RMR in women but not in men. They also found a significant correlation between the visceral adipose tissue area and TEF in absolute values in women but not in men.

Macor et al[16] studied 26 obese subjects, 8 males and 18 females. They used the same instruments we used to measure energy expenditure and visceral adipose tissue, and could not find any correlation between the visceral adipose tissue area and RMR nor glucose-induced thermogenesis. They found a significant correlation between insulin sensitivity, evaluated by an intravenous insulin tolerance test and glucose-induced thermogenesis. In conclusion, the results of our study demonstrate that adipose tissue distribution is an important determinant of the RMR. This is especially true for visceral adipose tissue in men and menopausal women and for the entire body fat in fertile women. The results also suggest that the TEF could be the result of the positive effects of body fat compartments counterbalanced by the negative effects of insulin resistance.

## ACKNOWLEDGMENT

We wish to thank R. Mandragona and L. Frigo, dieticians, for their cooperation in making the anthropometric measurements.

## REFERENCES


1. Ravussin E, Burnard B, Schutz Y, et al: Twenty-four hour energy expenditure and resting metabolic rate in obese, moderately obese, and control subjects. Am J Clin Nutr 35:566-573, 1982
2. Bessard T, Schutz Y, Jéquier E: Energy expenditure and postprandial thermogenesis in obese women before and after weight loss. Am J Clin Nutr 38:680-693, 1983
3. Weinsier RL, Schutz Y, Bracco D: Reexamination of the relationship of resting metabolic rate to fat-free mass and to the metabolically active components of fat-free mass in humans. Am J Clin Nutr 55:790-794, 1992
4. Ravussin E, Swinburn BA: Energy metabolism, in Stunkard AJ, Wadden TA (eds): Obesity: Theory and Therapy. New York, NY, Raven, 1993, pp 97-123
5. Leenen R, van der Kooy K, Deurenberg P, et al: Visceral fat accumulation in obese subjects: Relation to energy expenditure and response to weight loss. Am J Physiol 263:E913-E919, 1992
6. Busetto L, Perini P, Giantin V, et al: Relationship between energy expenditure and visceral fat accumulation in obese women submitted to adjustable silicone gastric banding. Int J Obes 19:227-233, 1995
7. Golay A, Schutz Y, Broquet C, et al: Decreased thermogenic response to an oral glucose load in older subjects. J Am Geriatr Soc 31:144-148, 1983
8. Thorne A, Wahren J: Diminished meal-induced thermogenesis in elderly man. Clin Physiol 10:427-437, 1990
9. De Boer JO, van Es AJH, van Raaij JMA, et al: Energy requirements and energy expenditure of lean and overweight women, measured by indirect calorimetry. Am J Clin Nutr 46:13-21, 1987
10. Den Besten C, Vansant G, Weststrate JA, et al: Resting metabolic rate and diet-induced thermogenesis in abdominal and gluteal-femoral obese women before and after weight reduction. Am J Clin Nutr 47:840-847, 1988
11. Weststrate JA, Dekker J, Stoel M, et al: Resting energy expenditure in women: Impact of obesity and body-fat distribution. Metabolism 39:11-17, 1990
12. Vansant G, Van Gaal L, De Leeuw I: Decreased diet induced thermogenesis in gluteal-femoral obesity. J Am Coll Nutr 8:597-601, 1989
13. Vansant G, Van Gaal L, Van Acker K, et al: Importance of glucagon as a determinant of resting metabolic rate and glucose-induced thermogenesis in obese women. Metabolism 40:672-675, 1991
14. Weck M, Fischer S, Karst H, et al: Fat distribution in obese men and women is not related to energy expenditure. Int J Obes 15:71, 1991 (suppl 1, abstr)
15. Vaughan L, Zurlo F, Ravussin E: Aging and energy expenditure. Am J Clin Nutr 53:821-825, 1991
16. Macor C, Ruggeri A, Mazzonetto P, et al: Visceral adipose tissue impairs insulin secretion and insulin sensitivity but not energy expenditure in obesity. Metabolism 46:123-129, 1997
17. Armellini F, Robbi R, Zamboni M, et al: Resting metabolic rate, body-fat distribution, and visceral fat in obese women. Am J Clin Nutr 56:981-987, 1992
18. Pittet P, Chappuis P, Acheson K, et al: Thermic effect of glucose in obese subjects studied by direct and indirect calorimetry. Br J Nutr 35:281-292, 1976
19. Schutz Y, Golay A, Felber JP, et al: Decreased glucose-induced thermogenesis after weight loss in obese subjects: A predisposing factor for relapse of obesity? Am J Clin Nutr 39:380-387, 1984
20. Schwartz RS, Halter JB, Bierman EL: Reduced thermic effect of feeding in obesity: Role of norepinephrine. Metabolism 32:114-117, 1983
21. Golay A, Schutz Y, Meyer HU, et al: Glucose-induced thermogenesis in nondiabetic and diabetic obese subjects. Diabetes 31:1023-1028, 1982
22. Kaplan ML, Leveille GA: Calorigenic response in obese and nonobese women. Am J Clin Nutr 29:1108-1113, 1976
23. Shetty PS, Jung RT, James WPT, et al: Postprandial thermogenesis in obesity. Clin Sci (Colch) 60:519-525, 1981
24. Welle SL, Campbell RG: Normal thermic effect of glucose in obese women. Am J Clin Nutr 37:87-92, 1983
25. Cunningham J, Levitt M, Hendler R, et al: Energy expenditure in obesity: Increased metabolic rate in the fasting and postprandial state. Clin Res 29:681A, 1981 (abstr)
26. Sharief NN, Macdonald I: Differences in dietary-induced thermogenesis with various carbohydrates in normal and overweight men. Am J Clin Nutr 35:267-272, 1982
27. Ravussin E, Acheson KJ, Vernet O, et al: Evidence that insulin resistance is responsible for the decreased thermogenic effect of glucose in human obesity. J Clin Invest 76:1268-1273, 1985
28. Van Gaal LF, Vansant GA, De Leeuw IH: Factors determining energy expenditure during very–low-calorie diets. Am J Clin Nutr 56:224S-229S, 1992 (suppl)
29. Van Gaal LF, Vanuytsel JL, Vansant GA, et al: Sex hormones, body fat distribution, resting metabolic rate and glucose-induced thermogenesis in premenopausal obese women. Int J Obes 18:333-338, 1994
30. Sjöström L: New aspects of weight-for-height indices and adipose tissue distribution in relation to cardiovascular risk and total adipose tissue volume, in Berry EM, Blondheim SH, Elihau HE, Shafrir E (eds): Recent Advances in Obesity Research, vol 5. London, UK, Libbey, 1987, pp 66-76
31. Sjöström L, Kvist H, Tylén U: Methodological aspects of measurements of adipose tissue distribution, in Vague J, Björntorp P, Guy-Grand B (eds): The Metabolic Complications of Human Obesity. Amsterdam, The Netherlands, Elsevier, 1985, pp 13-19
32. Kvist H, Chowdhury B, Grangard U, et al: Total and visceral adipose-tissue volumes derived from measurements with computed tomography in adult men and women: Predictive equations. Am J Clin Nutr 48:1351-1361, 1988
33. Sjöström L: Methods for measurements of the total and visceral adipose tissue volume and relationships between visceral fat and disease in 1006 severely obese subjects, in Oomura Y, Tarui S, Inoue S, Shimazu T (eds): Progress in Obesity Research 1990. London, UK, Libbey, 1991, pp 277-284
34. Jequiér E: Long-term measurement of energy expenditure in man: Direct or indirect calorimetry? in Björntörp P, Cairella M, Howard AN (eds): Recent Advances in Obesity Research, vol 3. London, UK, Libbey, 1981, pp 130-135
35. Weir JBV: New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol (Paris) 109:1-9, 1949
36. World Health Organization: Diabetes mellitus: Report of a WHO study group. World Health Organ Tech Rep Ser 727:7-113, 1985
37. Armitage P: Statistical Methods in Medical Research. New York, NY, Wiley, 1971
38. Poehlman ET: Regulation of energy expenditure in aging humans. J Am Geriatr Soc 41:552-559, 1993
39. Salomon F, Cuneo RC, Hesp R, et al: The effects of treatment with recombinant human growth hormone on body composition and metabolism in adults with growth hormone deficiency. N Engl J Med 321:1797-1809, 1989
40. OSullivan AJ, Kelly JJ, Hoffman DM, et al: Body composition and energy expenditure in acromegaly. J Clin Endocrinol Metab 78:381-386, 1994
41. Weltman A, Weltman JY, Hartman ML, et al: Relationship between age, percentage body fat, fitness, and 24-hour growth hormone release in healthy young adults: Effects of gender. J Clin Endocrinol Metab 78:543-548, 1994
42. Laasko M: How good a marker is insulin level for insulin resistance? Am J Epidemiol 137:959-965, 1993
43. Ravussin E, Zawadzki JK: Thermic effect of glucose in obese subjects with non–insulin-dependent diabetes mellitus. Diabetes 36:1441-1447, 1987