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Resting energy expenditure in type 2 diabetes subjects: is obesity important?

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Background: Type 2 diabetes is associated with a higher energy expenditure, however, the influence of obesity on resting metabolic rate (RMR) within this subject group is uncertain. The aim of this study was to analyze the differences in RMR in type 2 diabetes subjects with (O / DM) and without (DM) obesity.

Materials and methods: Body composition and the distribution of adipose tissue was estimated using the body mass index (BMI), waist and hip circumferences and deuterium oxide (D_2O) dilution; RMR was measured by indirect calorimetry in 66 obese with type 2 diabetes and 28 non-obese type 2 diabetes subjects.

Results: The measured RMR was by 2327 kJ/d (26.8%, $p < 0.001$) higher in the O / DM than in the DM group. This difference remained even after adjustment for age and gender and correction for fat-free mass (FFM) with a 9.1% higher RMR kJ/FFM kg/d in O / DM than in DM (143.3 ± 2.2 vs. 129.9 ± 3.6, $p < 0.001$). Fat mass (FM), FFM, waist-to-hip ratio, plasma glucose and triglyceride concentration were significant determinants of RMR with an adjusted R^2 value of 0.71 ($p < 0.001$) in O / DM, whereas FFM and plasma glucose with the adjusted R^2 value of 0.73 ($p < 0.001$) in the DM group. RMR correlated significantly with body weight, height, FFM, waist circumference ($p < 0.001$) and BMI ($p < 0.01$) in both groups.

Conclusions: Obese type 2 diabetes patients have a higher RMR compared to non-obese type 2 diabetes subjects. Body composition, especially abdominal distribution of adipose tissue, in parallel with metabolic changes have an influence on RMR, thus the impact of obesity on energy metabolism in type 2 diabetes patients cannot be ignored.

Key words: type 2 diabetes, resting metabolic rate, obesity

INTRODUCTION

The growing incidence of obesity resulted in more than one billion overweight and 315 million obese people worldwide by the end of 2000 and became the major global health problem (1). In March 2005 the International Obesity Task Force, a global coalition of obesity scientists and research centers advising the European Union, estimated that some European countries have exceeded the United States' figure of 67% for overweight and obesity (2). The incidence of type 2 diabetes mellitus (type 2 DM) during the same period has been mirrored and is presumed to be a direct result of the obesity epidemic (3). The international Diabetes Federation has predicted the prevalence of type 2 diabetes for the Euro-pean region to be 10.1% in 2025 (4).

Type 2 diabetes and obesity are closely related conditions known to influence energy expenditure. Resting metabolic rate (RMR) is the main component of daily energy expenditure, and a minor change in RMR could lead to a significant energy imbalance and huge change of body weight with the development of obesity and, later on, type 2 diabetes (5). The results of studies comparing RMR of formerly obese subjects with controls are discordant: some studies found no significant difference (6, 7), whereas others reported a significantly lower RMR in formerly obese subjects (8). Cross-sectional studies in Pima Indians with type 2 diabetes indicated an ~5% increased RMR compared with non-diabetic individuals after adjustment for age and body composition (9–11). This was supported by a longitudinal study in a group of 17 Pima Indians in whom glucose tolerance deteriorated from normal to impaired and to type

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2 diabetes in parallel with an increase of RMR by 6.8% (12). Thus, diabetes status leads to an increase in RMR, irrespective of body composition, age and gender. Fat-free mass (FFM), fat mass (FM), basal endogenous glucose output, fasting insulin and free fatty acid concentrations, age, gender and even race were estimated to be significant determinants of RMR in obese type 2 diabetes subjects (13).

Thought about 80% to 85% of type 2 diabetes subjects are obese, the remaining 20% have a normal body weight. It could be speculated that the pathogenesis of the disease in normal body weight type 2 diabetes patients is slightly different, with the predominance of insulin deficiency rather than insulin resistance, however, the possible variations in energy metabolism, responsible for the maintenance of body weight, could not be excluded. Previous studies have shown a higher resting energy and total energy expenditure in obese type 2 diabetes subjects compared to lean controls, but the difference disappeared after correction for differences in fat-free mass (14). However, considering the influence of obesity on energy metabolism, it remains unclear whether the paradoxical elevation of RMR in type 2 diabetes subjects is related to metabolic disturbances or also to the differences in body weight and composition.

In this study, we have tested the hypothesis that resting metabolic rate is higher in obese type 2 diabetes subjects (O / DM) compared to those without obesity (DM) and is related to the differences in fat mass, fat-free mass, regional body fat distribution and metabolic parameters.

MATERIALS AND METHODS

Ninety-four adults (66 O / DM and 28 DM) aged between 21 and 75 years were recruited from Woolmanhill Obesity and Diabetic Clinic in Aberdeen (Scotland, UK) or by newspaper advertisement. Written informed consent was obtained from all subjects. The study was approved by the Grampian Research Ethics Committee.

Inclusion criteria were: male or female aged 18 to 75 years, body mass index (BMI) >30 kg/m^2 for O / DM and BMI <25 kg/m^2 for DM subjects. Patients with any severe cardiac problems, malignant tumours, psychiatric disorders, abnormal thyroid function or taking L-thyroxin, beta-blockers and / or diuretics were excluded from the study.

All tests were undertaken in the morning at the Clinical Research Unit at Foresterhill Hospital, under standardized conditions. Subjects arrived after an overnight rest and fast of at least 12 hours. They were asked to avoid vigorous physical activity and smoking prior to attending the Unit.

Measurement of body composition

Body weight (BW) was measured with digital weighing scales to the nearest 0.1 kg (TANITA Corporation, Tokyo, Japan). The percentage of fat mass (%FM) in arms, legs and the trunk (upper body) area was measured using an eight-electrode bioelectrical impedance analyzer (BC-418 MA, TANITA Corporation, Tokyo, Japan). All measurements were taken with empty bladder, barefoot, without clothing and wearing a hospital gown of known weight, which then was subtracted from the measured body weight. BMI was calculated as body weight in kilograms divided by the square of the height in meters (15). Waist and gluteal (hip) circumferences were measured as described in the Anthropometric Standardization Reference Manual (16).

Measurement of total body water (TBW)

Total body water (TBW-kg) was measured by deuterium oxide dilution (D_2O) as described by Speakman (17). A two-point plateau method was used to obtain pre-dose and equilibration samples. Baseline urine analyses were used to determine the background deuterium concentration. A dose of ~0.1mg deuterium / kg body weight (99.9% deuterium oxide), adjusted to about 100 ml with Aberdeen tap water, was given to drink through a straw. Another 100 ml of tap water was used as a rinse and then consumed to ensure complete ingestion of the tracer. Subjects were asked to void 3.5 hours after the deuterium dose and then again, at 4 hours after deuterium dose equilibrium samples of urine were obtained. Urine samples were collected in glass airtight containers and stored at -80 °C until analysis. Deuterium concentrations in the urine samples were determined on an isotope-ratio mass spectrometer (IsoPrime, Micromass UK Ltd) which was calibrated against Vienna Standard Mean Ocean Water. Each sample and dose aliquot was analysed in duplicate to a precision of 0.3–0.4 ppm.

TBW was calculated as described elsewhere (18), using a 4% correction factor for the exchange of deuterium with labile H of protein and other body constituents. FFM was calculated on the assumption that water occupies 73.2% of FFM mass (19). FM was determined as follows:

$$FM\ (kg) = BW\ (kg) - FFM\ (kg).$$

The estimated FM in kg was used to calculate %FM:

$$\%FM = [(BW\ (kg) - FFM\ (kg))/BW] \times 100.$$

Measurement of resting metabolic rate (RMR)

RMR was measured by indirect calorimetry (ventilated hood) using DELTATRAC™ MBM-200 Metabolic Monitor (Datex-Engstrom Division, Instrumentarium Corporation, Finland). The monitor was calibrated before each measurement and the flow range was adjusted according to the patient's body weight.

Subjects were always measured in the morning after overnight sleep at home. Patients rested on the bed for about 10 min in a quiet and thermoneutral (20–22 °C) room until the procedure was explained prior to the

measurement. A ventilated hood was then placed over the patients' head and they were instructed to lie still but not to sleep. Data points were collected every minute for 40 minutes. The first 5 to 10 min were excluded from calculations and the mean RMR was calculated from the remaining data as 15 min of steady state, when respiratory quotient (RQ), oxygen consumption (VO_2) and carbon dioxide production (VCO_2) volumes did not vary >10%. RMR was calculated as follows:

RMR (kJ/24 hours) = [(15.818 × VO_2) + (5.176 × VCO_2)] × 1440 where VCO_2 and VO_2 in l/min; 15.818 and 5.176 are the energy equivalents of oxygen and carbon dioxide (20).

Blood sampling and analytical methods

Whole blood was sampled from a large antecubital vein, using an 18G butterfly needle (Sarstedt, Nuernbrecht, Germany) and adaptor prior to deuterium dosing. Biochemical analyses were done immediately by the Clinical Biochemistry Laboratory of National Health Service Grampian Aberdeen Royal Infirmary. Blood samples for nonesterified fatty acids (NEFA), insulin and leptin were collected into EDTA tubes and then spun (30,000 rpm at 4 °C for 10 min) immediately in a chilled centrifuge to obtain plasma which was stored at –80 °C for analysis. Plasma glucose was measured by the glucose oxidase technique on an AVDIA 1650/2400 autoanalyzer (Bayer PLC Diagnostic Division, UK). Glycosylated hemoglobin (HbA_{1c}) was analyzed by the high performance liquid chromatography method on an automated analyzer HLP-723G7 (Tosoh Corporation, Scientific Instruments Division, Japan). Insulin and leptin were each measured in duplicate on a Luminex 100 system employing the multiplexed biomarker immunoassay X-map technology (Luminex Corporation Technology Blvd Austin, USA). Total cholesterol (Chol), high density lipoprotein (HDL) cholesterol and triglycerides (TG) were measured with the AVDIA 1650/2400 analyzer (Bayer PLC Diagnostic Division, UK). Low density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula (21):

LDL cholesterol = Cholesterol – HDL – (Triglyceride × 0.45). NEFA were measured in duplicate each sample on a KONE clinical analyzer (Labmedics, Manchester, UK) using the *in vitro* enzymatic colorimetric method. The homeostasis model assessment (HOMA) insulin resistance (IR) index was calculated according to the following formula of Matthews et al. (22):

HOMA – IR = [fasting insulin (μU/ml) × fasting glucose (mmol/l)]/22.5.

Statistical analysis

Normal probability plot tests were used to determine whether the variables followed a normal distribution. The two-sample paired Student test was used to compare the means from two samples. One-way analysis of variance (ANOVA) was then used to compare the means from three samples. Analysis of covariance (ANCOVA) was used to adjust different variables to age and gender within the groups. Correlation analysis was used to assess Pearson's correlation. Several regression models were performed by multivariate regression analysis using RMR as the dependent variable and age, gender, FFM, BW, waist-to-hip (W / H) ratio, leptin, insulin, glucose and HOMA-IR as independent variables. Statistical analysis was performed using the GenStat statistical program (8th edition for Windows, Rothamstead Experimental Station, Harpenden, UK).

RESULTS

The groups shown in Table 1 were unequal with respect to subject number, gender and age ($p < 0.001$), thus all other variables were adjusted to age and gender. O / DM had a significantly higher BW, BMI, FM kg, FFM kg, %FM distributed in arms, legs and trunk area, waist and hip circumferences ($p < 0.001$) and W / H ratio ($p = 0.004$) compared with the DM group. Plasma glucose, HbA_{1c}, Chol, LDL cholesterol, NEFA and insulin concentration did not differ between the groups. However, the other parameters of lipid profile were different, with a significantly higher concentration of TG and a lower concentration of HDL cholesterol in O / DM subjects compared to DM ($p < 0.001$). Obese type 2 diabetes patients had also higher amounts of leptin ($p < 0.001$) and tended to display a higher insulin resistance ($p = 0.054$) compared to lean controls. Thus, O / DM patients had an overall higher body weight with the abdominal distribution of adipose tissue combined with a high plasma glucose and a poor long-time glucose control, dyslipidemia, hyperleptinemia and insulin resistance, whereas DM subjects only displayed a disturbed glucose metabolism.

The RMR kJ/d was by 2327 kJ/ (26.8%, $p < 0.001$) higher in the O / DM than in the DM group. This difference remained even after adjustment for age and gender and correction for FFM with a 9.1% higher RMR kJ / FFM kg/d in O / DM compared to DM (143.3 ± 2.2 vs. 129.9 ± 3.6, $p < 0.001$) (Figure).

Adjusted to age and gender, RMR correlated positively with FFM kg, BW, height, BMI and waist circumference (WC) in both groups, but other relations were different within each group (Table 2). We performed a multivariate regression analysis to determine which variables entered as significant predictors of RMR within each group. As anticipated, FFM kg was the most significant factor accounting for 49.5% and 65.1% of RMR in the O / DM and DM groups, respectively ($p < 0.001$). The significance of other potentially explanatory variables was different within each group:

– in the O / DM group (n = 66), FM kg, plasma glucose, TG and W / H ratio explained a further 6.4%, 9.3%, $p < 0.001$, 2.8%, $p = 0.007$, 3%, $p = 0.016$, respectively and were thus all significant variables in the final model: **RMR kJ/d = (78 × FFM kg) + (36 × FM kg) + (109 × Glu mmol/l) + (344 × TG mmol/**

Table 1. **Clinical characteristic of obese patients with type 2 diabetes (O / DM) and non-obese with type 2 diabetes (DM) patients**

	O / DM (n = 66)	DM (n = 28)	P value
N (M/F)	66 (35/31)	28 (19/9)	
Age (years)	55.9 ± 1.4	61.7 ± 1.8	0.016
BW (kg)	106.8 ± 2.6	65.9 ± 1.6	<0.001
Height (cm)	168.4 ± 1.2	166.8 ± 1.5	0.452
BMI	37.6 ± 0.7	23.6 ± 0.4	<0.001
FM%*	44.3 ± 1.1	32.3 ± 1.9	<0.001
FM kg*	47.9 ± 1.7	22.3 ± 2.8	<0.001
FFM%*	56.2 ± 0.8	71.5 ± 1.4	<0.001
FFM kg*	59.9 ± 1.0	47.3 ± 1.7	<0.001
%fat legs*	40.1 ± 0.6	27.7 ± 1.0	<0.001
%fat arms*	42.3 ± 0.9	23.8 ± 1.5	<0.001
% fat trunk*	39.4 ± 0.7	24.3 ± 1.0	<0.001
Waist (cm)*	120.4 ± 1.5	88.3 ± 2.5	<0.001
Hips (cm)*	120.2 ± 1.6	94.0 ± 2.6	<0.001
W/H ratio*	1.00 ± 0.01	0.94 ± 0.02	0.004
RMR kJ/d*	8464 ± 143	6137 ± 231	<0.001
RMR/FFM (kJ/d/kg) *	143.3. ± 2.2	129.9 ± 3.6	<0.001
Glu mmol/l	9.7 ± 0.5	8.4 ± 0.6	0.116
HbA_{1c} %	8.1 ± 0.2	7.6 ± 0.3	0.211
Chol mmol/l	4.76 ± 0.13	4.81 ± 0.19	0.829
HDL mmol/l	1.23 ± 0.03	1.61 ± 0.07	<0.001
LDL mmol/l	2.64 ± 0.11	2.64 ± 0.18	0.971
TG mmol/l	2.01 ± 0.12	1.26 ± 0.11	<0.001
NEFA pmol/l	0.46 ± 0.03	0.44 ± 0.04	0.635
Leptin pmol/l	1665.0 ± 152.9	502.0 ± 120.7	<0.001
Insulin pmol/l	294.0 ± 79.4	156.0 ± 37.5	0.281
HOMA-IR	16.0 ± 2.68	7.7 ± 1.47	0.054

* Data are adjusted to age and gender.

Table 2. **Correlation coefficients of measured RMR and different variables among obese with type 2 diabetes (O / DM) and non-obese with type 2 diabetes (DM)**

	O/DM (n = 66)	DM (n = 28)
FFM kg	0.721	0.816
FM kg	0.414*	
BW kg	0.697	0.819
Height cm	0.598	0.689
BMI	0.465*	0.482*
Waist cm	0.636	0.499*
Hips cm		0.604
%fat legs		-0.686
%fat arms		-0.499*
Glu mmol/l		0.526*

Data indicate statistical significance ($p < 0.001$) unless noted otherwise.

* Data indicate statistical significance $p < 0.01$.

l) + (3333 × W / H ratio) – 2991 (R^2 adjusted = 0.71, $p < 0.001$);

– in the DM group (n = 28), plasma glucose explained a further 8.5% (P=0.007) of variance: **RMR kJ/d = 1294 + (86 × FFM kg) + (90.3 × Glu mmol/l) (R^2 adjusted = 0.73, $p < 0.001$).**

As gender did not enter any regression model as a significant variable, we compared RMR between men and women to assess the differences between these groups (Table 3). There were no difference in age, BW, BMI, FM kg, glucose, HbA_{1c} and lipid profile between the groups, but men had a larger FFM ($p < 0.001$) and a higher W / H ratio ($p = 0.001$), whereas women displayed a peripheral body fat distribution with more

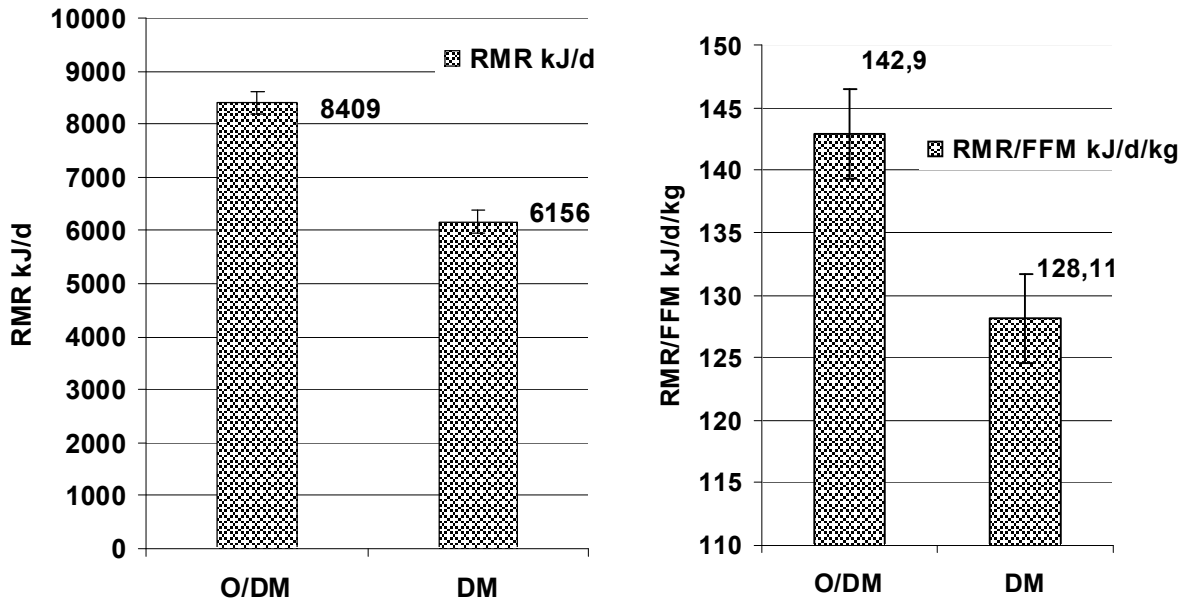

Figure. Comparison of measured resting metabolic rate (A) and RMR adjusted to age and gender and corrected for fat-free mass (B) in type 2 diabetes subjects with (O / DM) and without (DM) obesity

Table 3. **Some clinical characteristics of type 2 diabetes men and women groups**

	Men (n = 54)	Women (n = 40)	P value
FM kg	36.5 ± 2.6	41.1 ± 2.6	0.233
FFM kg	62.5 ± 1.5	48.6 ± 1.3	<0.001
% fat legs	26.5 ± 1.1	46.1 ± 1.0	<0.001
% fat arms	27.1 ± 1.4	45.0 ± 1.4	<0.001
% fat trunk	31.3 ± 1.3	36.3 ± 1.5	<0.001
W / H ratio	1.02 ± 0.01	0.96 ± 0.01	0.001
RMR kJ/d	8285 ± 241	6963 ± 234	<0.001
RMR / FFM (kJ/d/kg)	134.5 ± 2.9	143.7 ± 2.9	0.03
Leptin pmol/l	781.0 ± 91	2003 ± 213	<0.001

adipose tissue in legs, arms and the trunk area ($p < 0.001$). There was also a gender-specific evidence of a higher leptin concentration in women ($p < 0.001$).

The measured resting metabolic rate was by 1322 kJ/d higher in men than in women ($p < 0.001$), but when RMR was corrected for FFM, women displayed a 6.3% higher RMR / FFM ratio ($p = 0.03$). The relation analysis in women revealed a strong positive correlation between RMR and FM, FFM, % of fat in arms, legs, trunk area ($r = 0.81$, 0.79, 0.82, 0.75, 0.72 respectively, $p < 0.001$) and a negative correlation with age ($r = -0.60$, $p < 0.001$). Neither leptin concentration nor W / H ratio correlated with RMR in women. Data analysis in men revealed the same tendency, showing a significant positive correlation between RMR and FM, FFM, % fat in arms, legs, trunk area ($r = 0.70$, 0.72, 0.66, 0.58, 0.70 respectively, $p < 0.001$). Age was not significant, whereas the W / H ratio also correlated positively with RMR ($r = 0.51$, $p < 0.001$) in men group.

DISCUSSION

The main finding of this study that O / DM patients have 2327 kJ/ (26.8%) higher measured RMR and 9.1% higher RMR corrected for fat-free mass (RMR kJ / FFM kg/d) compared with the DM group is not in agreement with the previous studies that have shown a higher total RMR in O / DM group, but this difference disappeared after correction for lean body mass (14), implying that the influence of obesity on RMR is rather limited in diabetic patients.

In accordance with the estimated difference in RMR kJ / FFM kg/d within type 2 diabetes group, we suggest that abdominal obesity in association with metabolic alterations are significant factors influencing RMR in type 2 diabetes patients. Since the W / H ratio was shown to be associated with the amount of visceral adipose tissue, this index has been widely used to investigate the relations between regional adipose tissue distribution and metabolic profile (23). Multivariate regression analysis within O / DM and DM groups revealed that together with FFM kg and plasma glucose, TG and W / H ratio are also important predictors of RMR for the O / DM group. Thus, the influence of obesity status and associated factors on RMR cannot be ignored in type 2 diabetes patients. Gougeon et al. in 2002 also estimated fasting glucose to be a significant factor increasing the prediction of RMR by more than 3% (24), however, we estimated this influence to be 9.3% in obese and 8.5% in non-obese type 2 diabetes patients. This indicates that glucose control is essential with respect to energy metabolism in type 2 diabetes. In the current study, we failed to find an association between RMR and NEFA or insulin concentration, unlike the study of Weyer et al. (12). The absence of a relation in our study may reflect the relatively small sample size combined with a reduced frequency of sampling.

Gender effect on body composition, estimated in our study, was similar to previous results showing a greater amount of lean tissue and a higher W / H ratio in men as compared with women (25). Measured RMR was higher in men, possibly because of a higher amount of lean body tissue. However, when RMR was corrected to FFM kg, women displayed a 9.2 kJ/d/kg higher RMR / FFM than men. Previous studies have shown no difference in RMR between men and women (26) or a higher RMR in men both before and after adjustment for the differences in body composition (27, 28). However, these data were collected in healthy or in older subjects (29), but not in obese type 2 diabetes patients. Evaluating the results of the current study, we cannot ignore the diabetic status of our patients, which has already been proven to influence energy metabolism. On the other hand, there are known sex differences in fuel metabolism, basal and exercise substrate oxidation, post-absorption lipolysis, storage of dietary fatty acids, post-meal glucose flux and whole-body insulin sensitivity (30, 31). Perhaps the combination of these factors could result in a tendency of a greater fat deposition, sparing of endogenous body fat stores and energy metabolism in women. These perturbations may be even more expressed in type 2 diabetes subjects.

The higher RMR in obese type 2 diabetes patients could in theory promote weight loss, however, it is known that for type 2 diabetes subjects it is more difficult to lose or / and to maintain a stable body weight. The paradoxical elevation in resting metabolic rate cannot explain persistent obesity in type 2 diabetes subjects. Thus, further longitudinal studies are needed to analyse the peculiarities of energy balance regulation, energy intake and even the genetic background of energy metabolism to understand the factors predisposing type 2 diabetes patients to obesity.

CONCLUSIONS

Obese type 2 diabetes patients have a higher resting metabolic rate compared to non-obese type 2 diabetes subjects. Body composition, especially abdominal distribution of adipose tissue, in parallel with metabolic changes have an influence on resting metabolic rate, thus obesity affects

energy metabolism in type 2 diabetes patients. Men have a higher measured resting energy expenditure than women, but it is lower when corrected for fat-free mass. Gender effect on resting metabolic rate is not significant when differences in body composition are considered.

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DIABETU SERGANČIŲ ASMENŲ RAMYBĖS ENERGIJOS APYKAITA: AR NUTUKIMAS YRA SVARBUS?

Santrauka

Įvadas. Yra nustatyta, jog antro tipo cukriniu diabetu (II tipo CD) sergantys asmenys turi didesnę ramybės energijos apykaitą (REA), lyginant su sveikais. Apie 80–85% diabetu sergančiųjų turi per didelį kūno svorį, tačiau nutukimo poveikis jų energijos apykaitai yra neaiškus. Šio tyrimo tikslas buvo palyginti REA tarp nutukusių (N/CD) ir nenutukusių (neN/CD) II tipo diabetu sergančiųjų asmenų.

Metodai. Kūno kompozija bei riebalų pasiskirstymas buvo vertintas naudojant kūno masės indeksą (KMI), liemens (L) ir klubų (K) apimčių matmenis ir deuterio oksido (D_2O) praskiedimo metodiką. Ramybės energijos apykaita matuota netiesioginės kalorimetrijos būdu. Tyrimai atlikti 66 nutukusiems ir 28 nenutukusiems II tipo diabetu sergantiems asmenims.

Rezultatai. Išmatuota REA buvo 2327 kJ/ (26,8%, $p < 0.001$) didesnė N/CD grupėje, lyginant su neN/CD grupe. REA išliko 9,1% didesnė rezultatus pakoregavus pagal amžių ir lytį ir išreiškus kilogramui liesosios kūno masės (LKM) N/CD asmenims lyginant su neN/CD asmenimis ($143,3 \pm 2,2$ vs. $129,9 \pm 3,6$, $p < 0.001$). Kūno riebalų masė (KRM), LKM, L/K santykis, gliukozės bei trigliceridų kiekis kraujo plazmoje buvo reikšmingi veiksniai, turintys įtakos REA N/CD asmenims, (koreguotas R^2 0,71, $p < 0,001$), o LKM ir gliukozės kiekis kraujo plazmoje buvo reikšmingi veiksniai neN/CD asmenims (koreguotas R^2 0,73, $p < 0,001$). Abiejose grupėse REA reikšmingai koreliavo su svoriu, ūgiu, LKM, L apimtimi ($p < 0,001$) ir KMI ($p < 0,01$).

Išvados. Nutukę II tipo diabetu sergantys asmenys turi didesnę ramybės energijos apykaitą, lyginant su nenutukusiais II tipo diabetu sergančiaisiais. Kūno kompozicija, ypač centrinio tipo riebalų pasiskirstymas, kartu su metaboliniais pakitimais yra ramybės energijos apykaitos pokyčius lemiantys veiksniai, todėl negalima ignoruoti diabetu sergančių asmenų nutukimo poveikio energijos metabolizmui.