Obese Japanese Adults with Type 2 Diabetes Have Higher Basal Metabolic Rates than Non-Diabetic Adults

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Summary Several cross-sectional studies in Pima Indians and Caucasians have indicated that obese individuals with type 2 diabetes have a higher basal metabolic rate (BMR) than healthy, obese individuals. However, no study has investigated this comparison in Japanese subjects, who are known to be susceptible to type 2 diabetes due to genetic characteristics. Thirty obese Japanese adults with pre-type 2 diabetes (n=7) or type 2 diabetes (n=13) or without diabetes (n=10) participated in this study. BMR was measured using indirect calorimetry. The relationships between residual BMR (calculated as measured BMR minus BMR adjusted for fat-free mass, fat mass, age, and sex) and biomarkers including fasting glucose, glycosylated hemoglobin (HbA1c), fasting insulin, homeostasis model assessment of insulin resistance (HOMA-R), triglycerides, and free fatty acids were examined using Pearson’s correlation. BMR in diabetic subjects adjusted for fat-free mass, fat mass, age, and sex was 7.1% higher than in non-diabetic subjects. BMR in diabetic subjects was also significantly (p<0.05) higher than in non-diabetic subjects. There was a significant correlation between residual BMR and fasting glucose (r=0.391, p=0.032). These results indicate that in the Japanese population, obese subjects with type 2 diabetes have higher BMR compared with obese non-diabetic subjects. The fasting glucose level may contribute to these differences.

Key Words basal metabolic rate, Japanese, obesity, diabetes, predictive equation

As type 2 diabetes and obesity are closely related, the number of patients with type 2 diabetes in Japan has increased as a result of the rise in prevalence of obesity (1). In general, the fundamental treatment for type 2 diabetes is improvement in lifestyle such as diet and physical activity, associated with pharmacotherapy (2). Control of daily energy balance remains one of the most important treatment principles. Management of daily energy balance is usually conducted by diet control and maintenance of higher levels of physical activity. Accurate assessments of energy intake and energy expenditure are therefore required during treatment of diabetes.

Several cross-sectional studies have examined whether or not individuals with type 2 diabetes have a higher basal metabolic rate (BMR). Previous studies in Pima Indians (3) and Caucasians (4) using calorimetry showed obese subjects with type 2 diabetes had 5.2% and 6.9% higher BMR, adjusted for body composition, compared with their respective non-diabetic counterparts. Although the physiological mechanisms responsible for the increased BMR in individuals with type 2 diabetes are poorly understood, several mechanisms have been proposed to explain this change in BMR. These include increases in protein turnover (5), futile substrate cycling (6), gluconeogenesis (7), plasma glucose (8), and sympathetic nervous system activity (9). As Japanese people are susceptible to type 2 diabetes (9), mainly due to a lower ability to secrete insulin than Caucasians (10), this genetic characteristic may provide different results in BMR than similar studies in Pima Indians or Caucasians (3, 4). However, no study has examined whether BMR is higher in Japanese subjects with type 2 diabetes compared to subjects without diabetes.

As BMR may be different between individuals with non-diabetes, pre-diabetes or diabetes, some adjustments may be necessary when BMR is calculated in these groups. As the majority of clinical facilities are unable to carry out indirect calorimetry, BMR is usually estimated from predictive equations using data including age, sex, height, and weight (11). Previous studies indicate that predictive equations derived mainly from measurements in Caucasian subjects tend to overestimate BMR in both Asians (11, 12) and Caucasians (11, 13–17). We recently developed new predictive equations for BMR in the Japanese population (18). One of these equations was shown to be the best predictor of BMR amongst several predictive equations in healthy Japanese subjects (19). However, no study has investigated the validity of several of these published equations in Japanese subjects with type 2 diabetes.

The purpose of the present study was therefore to compare BMR between subjects with non-diabetes, pre-diabetes or diabetes in the obese Japanese population.
The second aim of the study was to examine the validity of several predictive equations for BMR in these subjects.

**MATERIALS AND METHODS**

*Subjects.* The subjects in the study were 50- to 59-year-old obese subjects who resided in Saku City (Nagano Prefecture in Japan). The subjects were selected randomly from participants in the Saku Control Obesity Program (SCOP). The details of SCOP are described elsewhere (20). Thirty obese Japanese adults without diabetes (*n* = 10), or with pre-type 2 diabetes (*n* = 7) or type 2 diabetes (*n* = 13) participated in this study. Two diabetic patients were treated by diet and exercise prescription, and one diabetic patient by metformin or glibenclamide therapy. Another diabetic patient who had experienced a diabetes patient education program in the past was included also, whereas those on insulin therapy were excluded. The subjects were instructed to eat a usual diet and carry out normal, but not vigorous physical activity beginning 1 d before the measurements. All the investigations were carried out in the Saku Central Hospital. This study was conducted according to the guidelines of the Declaration of Helsinki and all procedures involving human subjects were approved by the Ethical Committee of the National Institute of Health and Nutrition in Tokyo, Japan and the Ethical Committee of the Saku Central Hospital. The study protocol was explained to the subjects prior to enrollment, and all the subjects signed an informed consent form.

**Anthropometric and body composition.** The physical characteristics of the subjects are summarized in Table 1. Body weight was measured to the nearest 0.1 kg and body height to the nearest 0.1 cm using an automatic scale (Tanita, BF-220, Tokyo, Japan). The measurements were performed in light clothing and underwear. The light clothing was then weighed and subtracted from the total to obtain body weight with minimal clothing (underwear). Body mass index (BMI: kg/m²) was calculated as body weight (kg) divided by square of body height (m²). Percentage body fat was measured using a bioelectrical impedance technique (Tanita, BF-220). Fat-free mass (FFM) and fat mass (FM) were calculated from percentage body fat and body weight.

| Table 1. Physical characteristics and metabolic parameters in subjects with non-diabetes, pre-diabetes, or diabetes. |
|-------------------------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Non-diabetes (*n* = 10) | Pre-diabetes (*n* = 7) | Diabetes (*n* = 13) | ANOVA p value |
| Mean±SD | Range | Mean±SD | Range | Mean±SD | Range |
| Male/female | | 5/5 | 3/4 | 8/5 | 0.760 |
| Age (y) | | 54±3 | 51–59 | 53±3 | 50–57 | 52±3 | 50–59 | 0.826 |
| Height (cm) | | 165±10.0 | 151.5–179.2 | 162.9±7.7 | 155.0–174.0 | 165.3±8.7 | 152.0–176.6 | 0.317 |
| Weight (kg) | | 81±7.2 | 69.3–93.5 | 82.0±10.7 | 67.0–97.7 | 87.4±12.4 | 70.2–116.5 | 0.211 |
| Body mass index (kg/m²) | | 29.7±1.7 | 27.7–33.2 | 30.9±3.3 | 27.9–38.0 | 32.0±3.5 | 28.1–39.2 | 0.206 |
| Body fat (%) | | 33.7±7.4 | 24.0–45.4 | 36.2±8.9 | 23.4–45.4 | 36.5±6.6 | 24.0–46.0 | 0.727 |
| FM (kg) | | 26.9±4.5 | 21.2–34.5 | 29.5±8.0 | 20.9–44.6 | 31.7±6.2 | 23.1–45.7 | 0.808 |
| FFM (kg) | | 54.1±10.2 | 41.6–71.1 | 52.4±11.4 | 40.8–68.5 | 55.8±11.2 | 41.6–73.0 | 0.001 |
| Fasting glucose (mg/dL) | | 99±5 | 92–109 | 111±10 | 90–121 | 130±17 | 100–168 | <0.001 |
| Loge HbA1c | | 1.8±0.0 | 1.7–1.8 | 1.8±0.1 | 1.7–1.9 | 1.9±0.2 | 1.6–2.5 | 0.016 |
| [HbA1c (%)] | | 5.9 | 5.7–6.0 | 5.9 | 5.4–6.4 | 7.1 | 5.0–12.6 | 0.019 |
| Loge fasting insulin | | 1.8±0.4 | 1.0–2.3 | 2.5±0.7 | 1.5–3.3 | 2.3±0.6 | 1.5–3.5 | 0.339 |
| [Fasting insulin (μU/mL)] | | 6.5 | 2.8–10.1 | 14.9 | 4.4–27.4 | 12.1 | 4.3–33.8 | 0.030 |
| HOMA-R | | 1.6±0.6 | 0.7–2.5 | 4.1±2.4 | 1.3–8.2 | 3.8±2.7 | 1.5–11.8 | 0.321 |
| Triglycerides (mg/dL) | | 149±66 | 76–268 | 221±119 | 87–410 | 153±89 | 51–184 | 0.221 |
| Free fatty acid (mEq/L) | | 0.4±0.2 | 0.1–0.7 | 0.5±0.1 | 0.4–0.7 | 0.5±0.2 | 0.3–1.0 | 0.339 |

FM: fat mass. FFM: fat-free mass. HbA1c: glycosylated hemoglobin. HbA1c and fasting insulin were log transformed. HOMA-R = fasting insulin×fasting glucose/405. Differences between the non-diabetes, pre-diabetes and diabetes groups were evaluated by one-way ANOVA and Bonferroni post hoc test. *p<0.05 vs. non-diabetes. †p<0.05 vs. pre-diabetes.
determined using a dry gas volume meter (Shinagawa, DC-5, Tokyo, Japan) and then converted to the volume under conditions of standard temperature, pressure, and dry gas (STPD). The gas exchange results were converted to BMR (kcal/d) using Weir’s equation (22).

**Predictive equations of BMR.** Predictive BMR was calculated using the Ganpule (18), Japan-DRI (23), Harris-Benedict (24, 25), Schofield (26), Owen (14, 15), and Mifflin (16) equations (Table 2). The Japan-DRI provided the BMR standards (standard BMR per unit weight) according to age and sex category, with the data for these standards being obtained from a Japanese BMR database (21, 23). The Owen and Mifflin equations were developed using data obtained from adults including obese subjects.

**Blood samples.** Venous blood samples were collected after a fast of at least 12 h for measurement of fasting glucose, glycosylated hemoglobin (HbA1c), insulin, triglycerides, and free fatty acid. The value of the internationally used HbA1c (%) (HbA1c [NGSP]) defined by the NGSP (National Glycohemoglobin Standardization Program), was calculated by adding 0.4% to the obtained HbA1c (JDS) (%) defined by the Japan Diabetes Society (JDS) (27). Insulin and free fatty acids were examined using the laboratory testing services provided by SRL Inc. (Tokyo, Japan). Insulin (µIU/mL) was measured using CLEIA (Lumipulse Presto Insulin, Fujirebio Inc.), which has a minimal detection limit of 0.3 µIU/mL. Free fatty acid (mEq/L) was determined using an enzymatic assay (NEFA-SS ‘Elken,’ Elken Chemical Co. Ltd., Tokyo, Japan) with a sensitivity of 0.005 mEq/L. Other blood parameters were analyzed in the clinical laboratory of Saku Central Hospital. HOMA-R was calculated as fasting insulin (µIU/mL) × fasting glucose (mg/dL)/405.

All subjects underwent a 75-g oral glucose tolerance test. The subjects were divided into three groups according to the Diagnosis Criteria Exploratory Committee of the Japan Diabetes Society (2010) (27): non-diabetes (n=10), pre-diabetes (n=7), and diabetes (n=13).

**Statistical analysis.** The results are expressed as the mean±standard deviation (SD). Statistical significance was set at p<0.05. The Kolmogorov-Smirnov test was used for statistical testing of normality. HbA1c and fasting insulin were log transformed as the data were not normally distributed. Differences in body composition, blood parameters, and BMR (kcal/d, kcal/kg weight/d and kcal/kg FF/d) among the three groups were evaluated using one-way analysis of variance (ANOVA) and the Bonferroni post hoc test. Analysis of covariance (ANCOVA) with BMR as the dependent variable and FFM, FM, age, and sex as covariates was carried out. In order to examine the mechanism for differences in BMR, the blood sample measurements such as fasting glucose were added to FFM, FM, age, and sex in ANCOVA. The interaction terms with sex and body composition variables were examined in these analyses. Multiple linear regression models were also constructed using BMR as the dependent variable and FFM, FM, age, and sex as the independent variables. Gender was treated as a binomial variable (0 for male subjects, 1 for female subjects). Body height was not adjusted for, as it did not contribute significantly to BMR in the models (p>0.05). The relationships between the residual (measured BMR versus BMR after adjustment for FFM, FM, age, and sex) and fasting glucose, log, HbA1c, log, fasting insulin, HOMA-R, triglycerides, and free fatty acid were examined using Pearson’s correlation coefficients. The statistical significance of differences between measured BMR and predicted equation BMR was analyzed by one-way ANOVA with repeated measurements and Dunnett’s post hoc test, while differences between predicted and measured BMR values among non-diabetes, pre-diabetes, and diabetes were evaluated by one-way ANOVA and Bonferroni’s post hoc test. The statistical analyses were performed using SPSS for Windows (version 18.0; SPSS Inc., Chicago, IL, USA).

**RESULTS**

No significant difference was observed in body composition among the three groups (Table 1). The subjects with diabetes had significantly higher fasting glucose and log, HbA1c levels than subjects with non-diabetes. There was no interaction between sex and diabetes diagnosis in the relationship to BMR (F=2.166, p=0.137). Moreover the interaction terms with sex and body composition variables in ANCOVA with BMR as the dependent variable were not significant. Therefore, both sexes were combined in all analyses. After adjustment for FFM, FM, age, and sex the BMR in subjects with diabetes was 7.1% higher than in non-diabetic subjects (Table 3). The ANCOVA showed fasting glucose

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**Table 2. Predictive equations for basal metabolic rate used in the present study.**

| Predictive equations (kcal/d) | Males | Females |
|------------------------------|-------|---------|
| **Ganpule**                  | (0.0481×W+0.0234×H−0.0138×A−0.4235)×1,000/4.186 | (0.0481×W+0.0234×H−0.0138×A−0.9708)×1,000/4.186 |
| **Japan-DRI (2010)**         | 21.5×W | 20.7×W |
| **Harris-Benedict**          | 66.4730+13.7516×W+5.0033×H−6.7550×A | 655.0955+9.5634×W+1.8496×H−4.6756×A |
| **Schofield**                | (0.048×W+3.653)×1.000/4.186 | (0.034×W+3.538)×1.000/4.186 |
| **Owen**                     | 879+(10.20×W) | 795+(7.18×W) |
| **Mifflin**                  | 5+(9.99×W)+(6.25×H)+(4.92×A) | −161+(9.99×W)+(6.25×H)+(4.92×A) |

W: weight (kg), H: height (cm), A: age (y). Predictive equations for 50- to 59-y old obesity subjects were used.
was an independent determinant of BMR, in addition to FFM, FM, age, and sex. After adjusting for fasting glucose in addition to FFM, FM, age and sex, there were no significant differences in BMR among the three groups.

Furthermore, multiple regression analysis demonstrated 81% of the variability ($R^2$) in BMR was explained by FFM, FM, age, and sex, while fasting glucose as an additional independent variable explained
values by Ganpule, Owen and Mifflin equations were non-diabetes, pre-diabetes, or diabetes. Predicted BMR from six equations and measured BMR in subjects with insulin, HOMA-R, triglycerides, or free fatty acid. Significant differences were found between predicted and sex (Table 3). For the other equations, no significant correlation between residual BMR and loge fasting glucose, HbA1c, and fasting insulin, HOMA-R, triglycerides, and free fatty acids are shown in Fig. 1. Residual BMR correlated significantly with fasting glucose ($r=0.391$, $p=0.032$) and loge HbA1c ($r=0.414$, $p=0.023$), although there was no significant correlation between residual BMR and loge fasting insulin, HOMA-R, triglycerides, or free fatty acid.

Table 4 shows differences between BMR predicted from six equations and measured BMR in subjects with non-diabetes, pre-diabetes, or diabetes. Predicted BMR values by Ganpule, Owen and Mifflin equations were not significantly different from the measured BMR in non- or pre diabetes. On the other hand, for diabetes there was no significant difference between measured and predicted BMR calculated by Harris-Benedict and Schofield equations. The differences between BMR predicted by Ganpule and Mifflin equations and measured BMR was significant lower in subjects with diabetes than in subjects without diabetes. The prediction error by Ganpule and Mifflin equations were similar to that calculated when BMR was adjusted for FM, FFM, age, and sex (Table 3). For the other equations, no significant differences were found between predicted and measured BMR.

**DISCUSSION**

This study compared BMRs among subjects with non-diabetes, pre-type 2 diabetes and type 2 diabetes in the obese Japanese population. The results showed that obese Japanese subjects with type 2 diabetes had significantly higher BMR than obese Japanese without diabetes. A similar trend has been demonstrated in previous studies. Furthermore, given the significant relationship observed between residual BMR and fasting glucose, it is possible that fasting glucose level may be a factor in the higher BMR found in obese subjects with type 2 diabetes.

Several previous studies have examined whether or not BMR in patients with type 2 diabetes is higher than in non-diabetic subjects. Huang et al. (28) reported that BMR in these patients was 8.4% higher in females and 4.6% higher in males than in the corresponding non-diabetic subjects. Maiolo et al. (29) also reported that BMR was 35% higher in diabetic patients. It is important to note that BMR was not adjusted for body composition in these studies which may explain a large portion of the increase in BMR. On the other hand, two previous studies performed similar comparisons after adjustment for BMR. Fontvieille et al. (3) showed in Pima Indians that the BMR in patients with type 2 diabetes (weight: $107\pm33$ kg, body fat: $32\pm9\%$) was 5.2% higher than in non-diabetic subjects (weight: $99\pm24$ kg, body fat: $39\pm7\%$). Bitz et al. (4) also compared BMRs between subjects with or without type 2 diabetes in Caucasians and showed that BMR in the diabetic subjects (BMI: $35.5\pm3.7$ kg/m$^2$) was $6.9\%$ higher than in non-diabetic subjects (BMI: $34.1\pm4.7$ kg/m$^2$). In the another 3% of the variability in BMR.

The relationships among residual BMR (measured BMR minus BMR after adjustment for FM, FFM, age, and sex) and fasting glucose, loge HbA1c, loge fasting insulin, HOMA-R, triglycerides, and free fatty acids are shown in Fig. 1. Residual BMR correlated significantly with fasting glucose ($r=0.391$, $p=0.032$) and loge HbA1c ($r=0.414$, $p=0.023$), although there was no significant correlation between residual BMR and loge fasting insulin, HOMA-R, triglycerides, or free fatty acid.

Mean difference: Mean of difference between predicted and measured BMR. ANOVA*: Significance of differences between predicted and measured BMR analyzed by one-way ANOVA with repeated measurements and Dunnett’s post hoc test. Post hoc test: Predicted vs. Measured. ANOVA*: Differences in predicted equation between non-diabetes, pre-diabetes and diabetes evaluated by one-way ANOVA and Bonferroni post hoc test. $^d_{p<0.05}$, Bonferroni post hoc test, non-diabetes vs. diabetes.

|                | Mean±SD (kcal/d) | Mean difference±SD (kcal/d) | ANOVA* p value | Post hoc testb p value | ANOVAc p value |
|----------------|-----------------|-----------------------------|----------------|------------------------|----------------|
| Non-diabetes  |                 |                             |                |                        |                |
| ($n=10$)      |                 |                             |                |                        |                |
| Ganpule       | $1.511\pm194$   | $25\pm119$                  | $<0.001$       | $0.844$                | $0.019^d$      |
| Japan-DRI     | $1.712\pm175$   | $227\pm117$                 | $<0.001$       | $0.222$                |                |
| Harris-Benedict| $1.584\pm196$   | $99\pm127$                  | $0.002$        | $0.061$                |                |
| Schofield     | $1.660\pm209$   | $175\pm117$                 | $<0.001$       | $0.068$                |                |
| Owen          | $1.548\pm219$   | $62\pm123$                  | $0.094$        | $0.075$                |                |
| Mifflin       | $1.499\pm209$   | $13\pm126$                  | $0.990$        | $0.026^d$              |                |
| Pre-diabetes  |                 |                             |                |                        |                |
| ($n=7$)       |                 |                             |                |                        |                |
| Ganpule       | $1.502\pm214$   | $17\pm148$                  | $0.001$        | $0.977$                |                |
| Japan-DRI     | $1.727\pm241$   | $242\pm132$                 | $<0.001$       |                        |                |
| Harris-Benedict| $1.583\pm210$   | $98\pm159$                  | $0.111$        |                        |                |
| Schofield     | $1.648\pm222$   | $163\pm185$                 | $0.002$        |                        |                |
| Owen          | $1.532\pm229$   | $48\pm198$                  | $0.734$        |                        |                |
| Mifflin       | $1.486\pm226$   | $2\pm167$                   | $1.000$        |                        |                |
| Diabetes      |                 |                             |                |                        |                |
| ($n=13$)      |                 |                             |                |                        |                |
| Ganpule       | $1.601\pm237$   | $-110\pm99$                 | $<0.001$       | $<0.001$               |                |
| Japan-DRI     | $1.856\pm290$   | $146\pm147$                 | $<0.001$       |                        |                |
| Harris-Benedict| $1.692\pm253$   | $-19\pm110$                 | $0.898$        |                        |                |
| Schofield     | $1.766\pm263$   | $55\pm98$                   | $0.065$        |                        |                |
| Owen          | $1.649\pm264$   | $-62\pm99$                  | $0.032$        |                        |                |
| Mifflin       | $1.585\pm242$   | $-126\pm100$                | $<0.001$       |                        |                |
present study, BMR adjusted for FFM, FM, age, and sex, was significantly higher in diabetic compared with non-diabetic subjects (7.1%) (Table 3). Surprisingly, the adjusted BMR in patients with diabetes was higher than in non-diabetic subjects. These three studies using adjusted BMR obtained similar results in different ethnicities.

Although the physiological mechanisms responsible for the increased BMR in individuals with type 2 diabetes are poorly understood, several mechanisms have been proposed to explain this increase. These include increased energy costs during hyperglycaemia, for example gluconeogenesis, protein turnover, and sympathetic nervous system activity (3). Bitz et al. (4) reported that free fatty acids may be a potential mediator in several mechanisms associated with increased BMR. Gougeon et al. (30) reported that BMR adjusted for weight, FFM, age, and sex was significantly higher in subjects with type 2 diabetes with a fasting plasma glucose >180 mg/dL than those with a level <180 mg/dL (30). They used a fasting plasma glucose level of 180 mg/dL as it represents the concentration considered to be the glycosuria threshold which reflects poor control. Gougeon et al. (30) also reported that fasting plasma glucose was a significant independent variable and increased the prediction of BMR by more than 3%.

In the present study, we showed a significant relationship between residual BMR and fasting glucose (Fig. 1). After adjusting for fasting glucose in addition to FFM, FM, age and sex, there were no significant differences in BMR among the three groups (Table 3). Fasting glucose as an additional independent variable explained another 3% of the variability in BMR by multiple regression analysis. Therefore, the degree to which fasting glucose contributes to BMR was similar in different ethnicities. Weyer et al. (31) reported that a higher endogenous glucose output (EGO) was a relatively late finding in the development of type 2 diabetes and typically was not evident until the transition from impaired glucose tolerance (IGT) to diabetes. The extent to which the energy cost of EGO contributes to increased BMR is, therefore, probably less in individuals with IGT than in those with diabetes. In the present study, BMR in pre-diabetic subjects was not significantly higher than that measured in non-diabetic subjects. Fasting glucose values were also similar in non-diabetic and pre-diabetic subjects, which may have contributed to the similar BMR values we observed between the two groups. This result supports the results of Weyer et al. (31). In summary, higher BMR in obese subjects with type 2 diabetes may be related to fasting glucose level.

One of the limitations of the present study is the relatively small sample size. In the present study, both sexes were combined. Moreover, one diabetic patient who received metformin or glibenclamide therapy and another diabetic patient who had experienced diabetes patient education program in the past were included. However, more detailed analyses with larger samples size are needed for the better understanding of the effects of sex and medication. In particular, there is some possibility that medication affects the relationship between blood glucose and BMR through the suppression of blood fasting glucose.

As the majority of clinical facilities do not have indirect calorimetry, BMR is usually estimated from predictive equations using data such as age, sex, height, and weight (11). A predictive equation of BMR in obese subjects is important to provide the basis for an individualized treatment plan for weight loss (28). In the present study, we examined the validity of six predictive equations for BMR in Japanese subjects with non-diabetes, pre-diabetes or diabetes. The Ganpule (18) and Japan-DRI (21, 23) equations were developed based on data from Japanese subjects. The Harris-Benedict (24, 25), Schofield (26), Owen (14, 15), and Mifflin (16) equations are used internationally. The Harris-Benedict equation is the most common method for calculating BMR (26), while the Owen (14, 15) and Mifflin (16) equations were developed in adults including obese subjects.

Huang et al. (28) demonstrated that the Harris-Benedict equation overestimated BMR in diabetic males and underestimated the value in diabetic females, while Gougeon et al. (30) reported that BMR predicted by the Owen equations did not differ significantly from measured BMR in obese diabetic males. In the present study, the differences between BMR predicted by Ganpule and Mifflin equations and measured BMR was significant lower and negative for most predictive equations in subjects with diabetes than in subjects without diabetes. In the present study, ANCOVA showed that the differences in average prediction error of the Ganpule and Mifflin equation among the three groups were comparable to the differences obtained after adjustment for FM, FFM, age, and sex. BMR was underestimated by 110 and 126 kcal/d in diabetes, while predicted BMR was comparable to measured BMR in the non-diabetes and pre-diabetes groups. Therefore, adjustment should be made for diabetes when predicting BMR.

In conclusion, obese Japanese with type 2 diabetes have higher BMR than obese Japanese without diabetes. This phenomenon appears to be similar in different ethnicities such as Pima Indians, Caucasians, and Asians. Although the physiological mechanisms responsible for the increased BMR in subjects with type 2 diabetes remain unclear, the fasting glucose level could be a major factor contributing to this increase. Furthermore, the difference between the prediction errors of the Ganpule and Mifflin equation in subjects with and without diabetes tended to be significant and was comparable to those when BMR was adjusted for FM, FFM, age, and sex. It is therefore important to pay attention to the prediction error for BMR in diabetic patients in the clinical setting.

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