



## COMPARITIVE ANALYSIS OF BASAL METABOLIC RATE BETWEEN DIABETIC AND NON-DIABETIC PATIENTS

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### ABSTRACT

#### Background

Basal Metabolic Rate (BMR) refers to the energy required for vital body functions at rest. This study aims to compare metabolic rate and body composition between diabetic and non-diabetic patients.

#### Methodology

A prospective comparative study was conducted on 155 diabetic and 155 non-diabetic patients in a tertiary care hospital in South India. Body composition parameters, including BMR, Skeletal Muscle Mass (SMM), Body Fat Mass (BFM), and Percentage Body Fat (PBF), were assessed using an InBody Compact Body Composition Analyzer. Diabetic patients on treatment for more than four weeks were included, while pregnant women, individuals with prosthetic implants, and pacemaker users were excluded. Statistical analysis was performed using descriptive statistics and an Independent T-Test.

#### Results

The mean age of participants was  $53.15 \pm 14.4$  years, and the mean BMI was  $27.59 \pm 4.9$  kg/m<sup>2</sup>. Diabetic patients had significantly lower BMR ( $P = 0.15$ ) and SMM ( $P = 0.002$ ), while they exhibited higher BFM ( $P = 0.006$ ) and PBF ( $P = 0.000$ ). BMR and SMM decreased with age ( $P = 0.000$ ) and diabetes duration ( $P = 0.002$ ,  $P = 0.000$  respectively). A higher BMR correlated with a lower PBF ( $P = 0.000$ ). we also additionally found that there was decrease in BMR ( $P = 0.002$ ) and SMM ( $P =$

0.000) with increase in the age and duration of diabetes while there was an increase in Percent Body Fat ( $P = 0.003$ ).

**Conclusion** Diabetic patients exhibit lower BMR and SMM with increased fat accumulation, emphasizing the importance of early lifestyle interventions to improve metabolic health and quality of life.

## INTRODUCTION

An individual's total daily energy expenditure consists of three primary components, which are the Basal Metabolic Rate (BMR), the thermic effect of food, and the physical activity-induced energy expenditure [1]. Of these, the BMR is the highest portion composing the individual's daily energy expenditure, which represents the minimum level of energy expenditure to support the body's vital functions, including respiration, circulation, metabolism, and regulation of temperature, in a rested condition, when the individual is awake and sedentary [2]. BMR is measured when the individual, after an overnight fast and after enough sleep, is placed in a thermo-neutral condition, as this represents the energy requirements of the human body when the energy balance is in a constant, non-changing condition [3]. It represents a constant proportion of an individual's total daily energy expenditure, which is approximately 60-70%, and hence a critical component for the estimation of the individual's requirements for daily energy balance [4].

Factors which affect BMR include aging, sex, genetic makeup, hormonal secretions, and body composition, including the percentage of actual anatomic tissue mass or lean body mass. Changes in BMRs, considering the significance of this process in relation to the internal regulation of metabolism, have become of concern in the emergence of various chronic illnesses. Current scientific discoveries reveal the potential of BMRs, aside from being a marker of metabolism, in being an independent predictor of morbidity and mortality in various illnesses [5]. Current studies, including a prospective and genetic study, concluded a direct correlation of high BMRs and an elevated predisposition in individuals toward acquiring T2DM, a similar process in most cases [6].

In recent decades, the global prevalence of the burden of T2DM has increased alarmingly. In fact, the global prevalence of those with diabetes has risen to more than 830 million in 2022 from the 200 million reported in 1990. In many low- and middle-income countries, the increase in the prevalence of diabetes has accelerated [7]. Moreover, the global mortality rates of those with T2DM across all ages have risen by more than 200% since 1990. In the same period, the spread of the disease has more than doubled in the form of disability-adjusted life years (DALYs). The burden is also estimated to spread even more by the year 2045 [8]. The global prevalence of the overall impact of the disease is linked to issues such as obesity, lack of exercise, unhealthy dietary habits, and socio-economic changes [9].

Lifestyle management strategies focusing on changes to diet, physical activity levels, and weight reduction to improve energy balance play a crucial role in preventing or delaying the onset of diabetes among people at high risk for the disease [10]. However, a substantial number of diabetes patients are physically inactive or inadequately active, potentially leading to changes in energy balance and expenditure patterns [11].

It has been demonstrated by various studies that the basal or resting energy expenditure in individuals with T2DM is elevated compared with non-diabetic individuals, and their BMR found an increase by approximately 5–7% [12, 13]. The hypermetabolic state seen in T2DM patients may be explained by several factors. One potential cause may be the stimulation caused by hyperglycemia in an energy-expensive metabolic pathway such as gluconeogenesis in diabetic patients [14]. Substrate cycling, the wasting effects of glucose intolerance, and protein and fat catabolism may all contribute in an additive way to increased energy expenditure in individuals with T2DM.

Hormonal changes, which are a routine feature in T2DM patients, like the changes in levels of insulin, insulin resistance, altered levels of glucagon, and thyroid hormones, also play a particularly important role in regulating metabolism. Despite the lack of consistent findings in the entire range of T2DM patients, a consistent relationship is observed between poor diabetic control and increased rates of

BMR, along with a reduction in BMR after improving diabetic control, whether through pharmacological intervention or diet, to variable extent [12-14].

Another important factor in which BMR is implicated is body composition. The primary component in determining BMR is lean mass, while obesity, which is a comorbidity often found in T2DM, can affect BMR by altering fat mass, muscle mass, inflammation, and adipokines [4, 15]. Low-grade inflammation and insulin resistance, which often accompany obesity, can further fuel differences in BMR in diabetic patients.

In spite of mounting evidence showing increased BMR in subjects with T2DM, there have been inconsistencies in the magnitude and its association with glycemic control and body composition status. Additionally, there is a scarcity of data from the population of India itself, as there is a factor involving ethnic differences in body composition.

## 2. Materials and Methods

### 2.1 Study Design and Setting

This was a **prospective, hospital-based, comparative observational study** conducted at a tertiary care teaching hospital in South India. The study aimed to evaluate and compare **basal metabolic rate (BMR) and body composition parameters** between individuals with Type 2 Diabetes Mellitus (T2DM) and non-diabetic controls.

### 2.2 Study Population and Sample Size

A total of **310 adult participants** were enrolled and equally divided into two groups:

- **Diabetic group:** 155 individuals diagnosed with Type 2 Diabetes Mellitus
- **Non-diabetic group:** 155 age- and sex-matched individuals without diabetes

Participants were recruited consecutively from outpatient clinics during the study period to minimize selection bias.

### 2.3 Eligibility Criteria

#### 2.3.1 Inclusion Criteria

- Adults aged  $\geq 18$  years
- Diagnosed cases of Type 2 Diabetes Mellitus receiving stable antidiabetic treatment for at least **four weeks**
- Non-diabetic individuals with no prior diagnosis of diabetes
- Ability to stand independently for body composition assessment

#### 2.3.2 Exclusion Criteria

- Pregnant or lactating women
- Individuals with **pacemakers, metallic prosthetic implants**, or limb amputations
- Patients with acute illness, advanced renal or hepatic disease, malignancy, or endocrine disorders affecting metabolism
- Individuals unwilling to provide informed consent

### 2.4 Ethical Approval

The study protocol was reviewed and approved by the **Institutional Ethics Committee**. The study adhered to the principles outlined in the **Declaration of Helsinki**, and **written informed consent** was obtained from all participants prior to enrollment.

### 2.5 Data Collection and Measurements

#### 2.5.1 Demographic and Clinical Variables

Data collected included age, sex, body weight, height, body mass index (BMI), and duration of diabetes (years). BMI was calculated as weight (kg) divided by height squared ( $m^2$ ).

#### 2.5.2 Body Composition Analysis

Body composition parameters were measured using the **InBody Compact Body Composition Analyzer**, which utilizes **bioelectrical impedance analysis (BIA)** to estimate body composition by measuring impedance across multiple frequencies.

The following parameters were recorded:

- **Basal Metabolic Rate (BMR)** (kcal/day)
- **Skeletal Muscle Mass (SMM)** (kg)
- **Body Fat Mass (BFM)** (kg)
- **Percentage Body Fat (PBF)** (%)

To ensure consistency, all measurements were conducted under standardized conditions:

- Participants refrained from eating, caffeine intake, and strenuous physical activity for at least **4 hours** prior to assessment
- Measurements were performed barefoot and in light clothing

## 2.6 Statistical Analysis

Statistical analysis was performed using standard statistical software. Continuous variables were expressed as **mean  $\pm$  standard deviation (SD)**.

- **Independent Student's t-test** was used to compare BMR and body composition parameters between diabetic and non-diabetic groups
- **Pearson correlation analysis** assessed associations between age, duration of diabetes, BMR, SMM, and PBF
- A **p-value  $< 0.05$**  was considered statistically significant

## 3. Results

A total of **310 participants** were included in the final analysis, comprising **155 individuals with Type 2 Diabetes Mellitus (T2DM)** and **155 non-diabetic controls**. The overall mean age of the study population was  **$53.15 \pm 14.4$  years**, with a mean body mass index (BMI) of  **$27.59 \pm 4.9$  kg/m<sup>2</sup>**. Baseline demographic characteristics were comparable between the two groups.

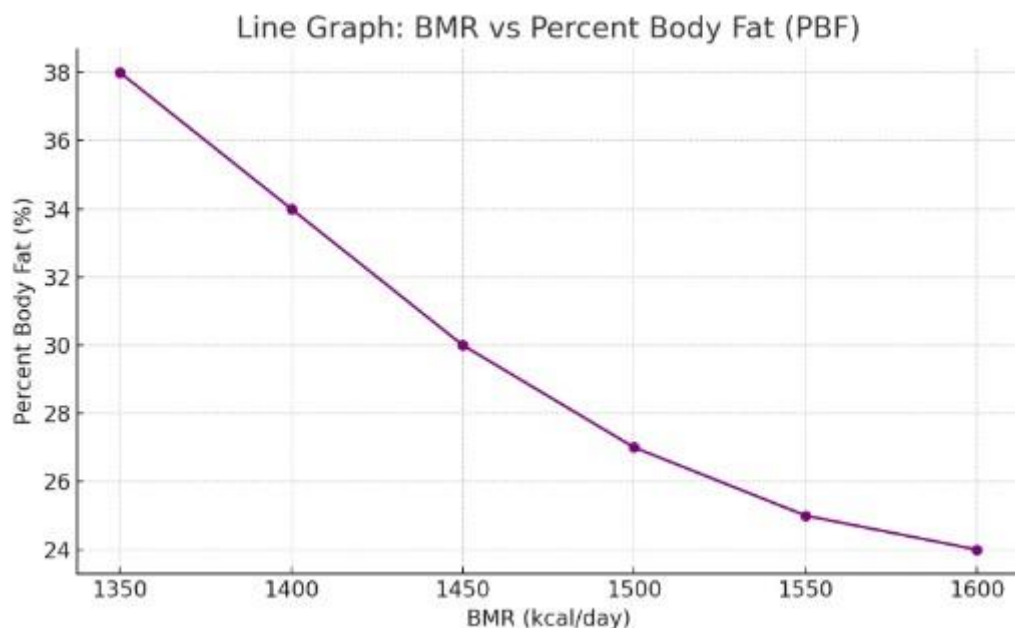
### 3.1 Comparison of Basal Metabolic Rate and Body Composition Parameters

Patients with Type 2 Diabetes Mellitus exhibited a **numerically lower basal metabolic rate (BMR)** compared to non-diabetic individuals ( $1428 \pm 185$  vs.  $1486 \pm 192$  kcal/day); however, this difference did not reach statistical significance ( $p = 0.15$ ).

In contrast, **skeletal muscle mass (SMM)** was **significantly lower** among diabetic participants compared to non-diabetic controls ( $23.4 \pm 4.1$  kg vs.  $25.8 \pm 4.3$  kg;  $p = 0.002$ ).

Diabetic individuals also demonstrated **significantly greater adiposity**, with higher **body fat mass (BFM)** ( $29.6 \pm 6.8$  kg vs.  $26.1 \pm 6.2$  kg;  $p = 0.006$ ) and a markedly elevated **percentage body fat (PBF)** ( $35.9 \pm 7.1\%$  vs.  $31.2 \pm 6.4\%$ ;  $p < 0.001$ ).

These findings indicate a distinct **shift toward reduced metabolically active tissue and increased fat accumulation** in individuals with Type 2 Diabetes Mellitus.



### 3.2 Association of Age with Metabolic and Body Composition Parameters

Advancing age was significantly associated with adverse changes in metabolic parameters. **Basal metabolic rate declined progressively with increasing age** ( $p < 0.001$ ), accompanied by a significant reduction in skeletal muscle mass ( $p < 0.001$ ).

Conversely, **percentage body fat increased significantly with age** ( $p = 0.003$ ), reflecting an age-related redistribution of body composition toward adiposity. This pattern was observed across both diabetic and non-diabetic participants but was more pronounced in individuals with diabetes.

### 3.3 Impact of Duration of Diabetes on Metabolic Health

Among individuals with Type 2 Diabetes Mellitus, **longer duration of diabetes was significantly associated with worsening metabolic outcomes**. Basal metabolic rate showed a significant inverse association with duration of diabetes ( $p = 0.002$ ).

Similarly, skeletal muscle mass declined significantly with increasing duration of diabetes ( $p < 0.001$ ), while percentage body fat demonstrated a significant positive association ( $p = 0.003$ ). These findings suggest a **progressive deterioration of body composition with chronicity of diabetes**, characterized by muscle loss and fat accumulation.

### 3.4 Correlation Between Basal Metabolic Rate and Body Composition

Correlation analysis revealed a **strong positive association between basal metabolic rate and skeletal muscle mass** ( $r = 0.62$ ,  $p < 0.001$ ), indicating that higher muscle mass was associated with higher resting metabolic expenditure.

In contrast, basal metabolic rate demonstrated a **moderate to strong negative correlation with percentage body fat** ( $r = -0.55$ ,  $p < 0.001$ ), suggesting that increased adiposity was associated with reduced metabolic efficiency. These correlations emphasize the central role of **muscle-fat balance** in determining basal metabolic rate in individuals with Type 2 Diabetes Mellitus.

### 3.5 Consolidated Summary of Findings

Overall, individuals with Type 2 Diabetes Mellitus demonstrated a distinct alteration in body composition characterized by reduced skeletal muscle mass and basal metabolic rate, accompanied by increased body fat mass and percentage body fat. Advancing age and longer duration of diabetes were independently associated with a progressive decline in metabolically active tissue, alongside a concomitant increase in adiposity. Furthermore, a strong inverse relationship was observed between adiposity and basal metabolic rate, indicating reduced metabolic efficiency in individuals with higher

fat accumulation. Collectively, these findings suggest the development of a sarcopenic–adiposity phenotype in Type 2 Diabetes Mellitus, which may have important implications for metabolic control, disease progression, and long-term complication risk.

**Table 1. Baseline Characteristics of the Study Population (n = 310)**

| Variable                                            | Value            |
|-----------------------------------------------------|------------------|
| Age (years), mean $\pm$ SD                          | 53.15 $\pm$ 14.4 |
| Body Mass Index (kg/m <sup>2</sup> ), mean $\pm$ SD | 27.59 $\pm$ 4.9  |
| Participants with Type 2 Diabetes Mellitus, n (%)   | 155 (50.0)       |
| Non-diabetic participants, n (%)                    | 155 (50.0)       |

**Table 2. Basal Metabolic Rate and Body Composition Parameters: Group Comparison and Associations**

| Parameter                       | Diabetic (n = 155)<br>Mean $\pm$ SD | Non-Diabetic (n = 155)<br>Mean $\pm$ SD | p-value           | Change with Age | Change with Diabetes Duration | Correlation with BMR (r, p) |
|---------------------------------|-------------------------------------|-----------------------------------------|-------------------|-----------------|-------------------------------|-----------------------------|
| Basal Metabolic Rate (kcal/day) | 1428 $\pm$ 185                      | 1486 $\pm$ 192                          | 0.15              | ↓ (p < 0.001)   | ↓ (p = 0.002)                 | —                           |
| Skeletal Muscle Mass (kg)       | 23.4 $\pm$ 4.1                      | 25.8 $\pm$ 4.3                          | <b>0.002*</b>     | ↓ (p < 0.001)   | ↓ (p < 0.001)                 | <b>+0.62, &lt;0.001</b>     |
| Body Fat Mass (kg)              | 29.6 $\pm$ 6.8                      | 26.1 $\pm$ 6.2                          | <b>0.006*</b>     | ↑               | —                             | -0.48, <0.001               |
| Percentage Body Fat (%)         | 35.9 $\pm$ 7.1                      | 31.2 $\pm$ 6.4                          | <b>&lt;0.001*</b> | ↑ (p = 0.003)   | ↑ (p = 0.003)                 | <b>-0.55, &lt;0.001</b>     |

Values expressed as mean  $\pm$  SD.

Independent Student's *t*-test used for group comparisons.

Pearson correlation analysis used for associations.

A *p*-value < 0.05 was considered statistically significant.

#### 4. Discussion:

In this study we found that Type 2 Diabetes Mellitus (T2DM) subjects had a slightly lower basal metabolic rate (BMR) than non-diabetic controls - 1428  $\pm$  185 vs 1486  $\pm$  192 kcal/day. However, this difference wasn't statistically significant - *p* = 0.15. The result suggests that resting energy expenditure might actually be a bit lower in T2DM - but the thing is individual variation, plus factors like body size and composition probably masked the effect. It's also worth noting that BMR is mainly influenced by lean tissue mass, so changes in skeletal muscle mass can have more of a metabolic impact than the absolute BMR value.

Harihar et al found out that - in young-onset T2DM, muscle mass loss is a major player in glycemic dysregulation. They found a strong negative correlation between skeletal muscle mass as a percentage of body weight and HbA1c (*r* = -0.624) and fasting blood glucose (*r* = -0.656) [16]. That matches up pretty well with the results of the current study which showed that diabetic subjects had a significant reduction in skeletal muscle mass compared to non-diabetic controls - 23.4  $\pm$  4.1 kg vs 25.8  $\pm$  4.3 kg (*p* = 0.002). Because skeletal muscle is where insulin drives glucose uptake, losing this sort of tissue probably just makes insulin resistance worse and glucose disposal even lower - all without any obvious changes in total body weight or BMI.

Other research, like Gómez-Ambrosi et al suggest that Body Fat Percentage (BF%) has a really big role to play in diabetes risk [17]. They found out that people with prediabetes or T2DM tend to have higher BF% even if they have similar BMI and waist circumference to the non-diabetic controls. And in this study, we found that our diabetic patients had significantly more body fat - both in terms of mass (29.6  $\pm$  6.8 vs 26.1  $\pm$  6.2 kg; *p* = 0.006) and percentage (35.9  $\pm$  7.1% vs 31.2  $\pm$  6.4%; *p* < 0.001).

Which pretty much challenges the idea that BMI can be used to gauge metabolic dysfunction - and points out the need to use direct measurement of body fat.

Looking at that a bit more closely, Pan et al showed that the risk of diabetes goes up by 4% for every 1% increase in body fat percentage (HR = 1.04, 95% CI: 1.04–1.05), and that it's almost three times higher for people in the top BF% quartile compared with the bottom one (HR = 2.72) [18]. So all of this together gives a pretty clear picture of someone with T2DM looking like they have "sarcopenic obesity" - which is basically poor metabolic efficiency and a high cardiometabolic risk. So this isn't just about being overweight - it's about the actual composition of the body. Which makes it a pretty good case for treatment strategies that focus on both muscle maintenance and fat loss.

In the current study, metabolic changes at advanced age were significantly related to adverse changes in most metabolic parameters such as, a progressive decrease in basal metabolic rate (BMR) ( $p < 0.001$ ) and skeletal muscle mass (SMM) ( $p < 0.001$ ). These results are in line with the physiological alterations with age like decreased anabolic hormone activity, decreased levels of physical activity, and gradual degradation of metabolically active lean tissue. Since skeletal muscle is the major predictor of resting energy expenditure, the concomitant reduction in BMR and SMM with age indicates a biologically plausible pathway in which age is related to metabolic susceptibility.

In line with this observation, Wang et al. have found out that there was an inverse correlation between predicted BMR quartiles and age with the older ones showing a much lower BMR and worse lipid profile in terms of increased total cholesterol and LDL-C (all  $p < 0.05$ ). The findings imply that age-associated decreases of BMR are not independent but associated with more extensive metabolic dysregulation[19].

On the other hand, percentage body fat (PBF) was found to significantly go up as the age got older ( $p = 0.003$ ) indicating a redistribution of body composition at an older age towards being adipose. Dai et al. also showed that the correlation between age and diabetes type 2 mellitus is mediated by the body fat percentage, and the indirect effect of age on the risk of diabetes through increased adiposity is significant (IE = 1.09; 95% CI: 1.07–1.12;  $p < 0.001$ ). Taken together, these observations point to the decisive role of age-related changes in muscle mass and fat gain in the metabolic inefficiency and predisposition to diabetes[20].

Greater years of Type 2 Diabetes Mellitus (T2DM) in the current case had a significant correlation with a gradual decline in metabolic health and body composition. The basal metabolic rate (BMR) showed a strong negative correlation to the duration of diabetes ( $p = 0.002$ ), which showed a decreasing energy expenditure at rest during the progression of chronicity of the disease. This metabolic stress decrease is probably cumulative, caused by decreased physical exercise and progressive depletion of metabolically active lean tissue, all indicative of inadequate metabolic adaptation to long-term T2DM.

At the same time, skeletal muscle mass (SMM) deteriorated seriously ( $p < 0.001$ ), whereas percentage body fat (PBF) grew ( $p = 0.003$ ) as the duration of diabetes progressed, and this is indicative of a shift towards a sarcopenic obesity phenotype. Ko et al. reported that prolonged diabetes duration was associated with poorer glycemic control, and the increased risk of having HbA1c  $\geq 7.0\%$  (53 mmol/mol) despite structured education programs[21]. As a complement to this, Karguppikar et al. showed an increased resting metabolic rate, lean body mass and a reduced fat percentage in subjects with shorter disease period ( $< 5$  years), with diabetes period showing a negative correlation with weight-adjusted RMR ( $p < 0.05$ ). Taken together, these results indicate the accruing metabolic cost of chronic T2DM and the necessity of early intervention aimed at preserving muscle and controlling adiposity[22].

In the current research, correlation analysis showed that the two variables, basal metabolic rate (BMR) and skeletal muscle mass (SMM) showed a positive correlation ( $r = 0.62$ ,  $p < 0.001$ ) meaning that people with extreme muscle mass had higher resting metabolic expenditure. The obtained conclusion supports the key physiological role of skeletal muscle as the main source of resting energy expenditure and supports its relevance in the maintenance of metabolic efficiency in Type 2 Diabetes Mellitus (T2DM) patients.

In this regard, Liu et al. have mentioned that fat-to-muscle ratio (FMR) is related independently with metabolic disorders among people with T2DM and that an adiposity-to-muscle mass imbalance worsens metabolism [23]. These results indicate that absolute muscle mass and its ratio to fat mass are the critical factors of metabolic health.

Additional support of this correlation, Vaughn et al. were able to establish a high linear correlation between skeletal muscle mass and BMR in non-diabetic college-age men ( $r = 0.80$ ;  $r^2 = 0.65$ ) [24]. These findings, although in a non-diabetic group, confirm the underlying biological connection between muscle mass and resting metabolism, indicating the possible existence of the relationships in health and disease conditions.

Basal metabolic rate on the other hand had moderate to strong negative association with percent body fat (PBF) ( $r = -0.55$   $p < 0.001$ ), such that the metabolic efficiency decreases as one gets fatter. Faria et al. found that relative BMR per kilogram body weight was significantly lower in obese individuals than in lean ones ( $18.3 \pm 1.8$  vs.  $24.7 \pm 2.6$  kcal/kg;  $p < 0.001$ ), and the relative BMR had negative relation with PBF ( $r = 0.49$ ) [25]. On the same note, Weyer et al. established that reduced prediction of resting metabolic rate that was adjusted to fat-free mass had a significant relationship with high levels of body fatness ( $p < 0.01$ ) [26].

These results combined highlight the fact that skeletal muscle/adipose tissue balance is a strong determinant of basal metabolic rate. Excessive adiposity and decreased muscle mass in patients with T2DM can potentially have a significant effect on metabolic efficiency, and therefore muscle-preservation and fat-reduction strategies are necessary to maximize basal metabolism and general metabolic well-being.

## Conclusion

This study demonstrates that individuals with Type 2 Diabetes Mellitus exhibit an unfavorable body composition profile characterized by reduced skeletal muscle mass and increased adiposity, despite having no statistically significant difference in absolute basal metabolic rate compared with non-diabetic controls. The strong positive association between BMR and skeletal muscle mass, along with the inverse relationship between BMR and body fat percentage, highlights the central role of muscle–fat balance in determining metabolic efficiency.

Advancing age and longer duration of diabetes were associated with progressive muscle loss, increased adiposity, and declining BMR, supporting the development of a sarcopenic–adiposity phenotype in T2DM. These findings emphasize that metabolic dysfunction in diabetes is driven more by qualitative changes in body composition than by BMR alone. Interventions focusing on muscle preservation and fat reduction may therefore be essential to improve metabolic health and long-term outcomes in individuals with Type 2 Diabetes Mellitus.

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