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Research Article

Assessment of Insulin Resistance Using Fasting Biochemical Markers and its Association with Basal Metabolic Rate Variations

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ABSTRACT

Objective: To assess insulin resistance using fasting biochemical markers and to evaluate its association with variations in basal metabolic rate.

Methods: This cross-sectional analytical study was conducted on 120 participants aged 20–60 years. Fasting blood samples were collected to measure glucose, insulin and lipid profile parameters. Insulin resistance was calculated using the Homeostatic Model Assessment (HOMA-IR). Basal metabolic rate was estimated using the Harris-Benedict equation. Participants were categorized into insulin-sensitive and insulin-resistant groups. Statistical analysis was performed using SPSS version 22 and correlations between HOMA-IR and BMR were assessed.

Results: The mean age of participants was 38.6 ± 10.2 years with a mean BMI of 26.1 ± 4.3 kg/m^2. Insulin resistance was present in 61.7% of participants. The insulin-resistant group showed a significantly higher BMR (1622.8 ± 245.7 kcal/day) compared to the insulin-sensitive group (1485.6 ± 210.4 kcal/day) ($p = 0.012$). A moderate positive correlation was observed between HOMA-IR and BMR ($r = 0.34$, $p = 0.004$).

Conclusion: Insulin resistance is highly prevalent and is significantly associated with increased basal metabolic rate. Fasting biochemical markers provide a simple and effective method for its assessment. Understanding this relationship may help in early identification and management of metabolic disorders.

Keywords: Insulin Resistance, Basal Metabolic Rate, Homa-Ir, Fasting Glucose, Fasting Insulin, Metabolic Health.

INTRODUCTION

Insulin resistance (IR) is considered as one of the most significant metabolic disturbances of the present time. It is closely associated with the rising global prevalence of obesity as well as lifestyle-related disorders. (Wilcox, 2005)Insulin Resistance is defined as a condition in which the cells of the body particularly those in muscle, liverand adipose tissue exhibit a diminished response to the normal actions of insulin. As a result, higher levels of insulin are required to achieve glucose homeostasis.(Petersen and Shulman, 2018)With time, this compensatory hyperinsulinemia may fail which leads to impaired glucose tolerance and eventually results in Type 2 Diabetes Mellitus. Beyond glucose metabolism, insulin resistance also contributes to a broad range of metabolic abnormalities which include dyslipidemia, hypertension and endothelial dysfunction, thereby increase the risk of cardiovascular diseases.(Gast et al., 2012)

In recent years, the prevalence of insulin resistance has increased at a concerning rate, especially in developing countries. This increasing rate is due to rapid urbanization, dietary habits and reduced physical activity that has greatlytransformed lifestyle habits. (Dengel et al., 1996)The condition is often considered clinically silent in its early stages which makes early detection difficult and necessary.(Ryan, 2000) Identifying insulin resistance before the onset of irreversible metabolic damage allows for timely intervention through lifestyle modification and

treatment approaches. Therefore, there is a need for simple, reliableand cost-effective methods for its assessment in standard clinical practice.(Tso et al., 2004)

Traditionally, the hyperinsulinemic-euglycemic clamp technique has been regarded as the gold standard for measuring insulin sensitivity. (Faghihi-Kashani et al., 2016)However, its complexity, invasiveness and high cost limit its widespread use, especially in large-scale studies and resource-limited settings. Consequently, surrogate markers based on fasting biochemical parameters have gained considerable attention. (Song et al., 2007) Among these, the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) which is calculated using fasting plasma glucose and insulin levelsis one of the most commonly used indices. It provides a practical and relatively accurate estimation of insulin resistance. (Haffner et al., 1996)

Additionally, Other fasting biochemical markers such as fasting insulin levels, fasting blood glucose and components of the lipid profilesuch as triglycerides and high-density lipoprotein cholesterolhave emerged as indirect indicators of insulin sensitivity.(Kosmas et al.) These markers are not only easily measurable but also provide valuable informationregardingthe overall metabolic state of an individual. (Bosy-Westphal et al., 2015) The use of such biochemical indicators is particularly important in clinical settings where advanced diagnostic tools may not be easily accessible. Moreover, combining multiple markers may improve the predictive accuracy for identifying individuals at risk of metabolic disorders. (Clausen et al., 1996)

The basal metabolic rate (BMR) represents as another important component of human metabolism. BMR refers to the amount of energy used by the body at rest to maintain essential physiological functions like respiration, blood circulation, cellular metabolism and thermoregulation. (Møller et al., 2014) It represents the largest component of total daily energy expenditure, accounting for approximately 60–75% of the overall energy requirement in most individuals. BMR is affected by factors including age, gender, body size, lean body mass, hormonal balance and genetic factors. (Maciak et al., 2020)

Variations in basal metabolic rate have important implications for energy balance and body weight regulation. (Piaggi, 2019)Individuals with a relatively lower BMR might be more susceptible to weight gain since their bodies use less energy at rest while those with a higher BMR usually burn more calories even in a resting state. Hormonal factors, particularly those involving thyroid function and insulin function play a crucial role in regulating metabolic rate. (Atamni et al., 2016)

Insulin resistance can have a direct or indirect impact on basal metabolic rate. One possible mechanism includeschanges in substrate metabolism. In insulin-sensitive individuals, glucose is efficiently used as a primary source of energy. (Gillies et al., 2007) However, in the presence of insulin resistance, there is a transition towards increased lipid utilization and reduced glucose oxidation. This metabolic inflexibility may affect overall energy expenditure and lead to variations in BMR. (Bjornstad and Eckel, 2018) Additionally, insulin resistance has been associated with mitochondrial dysfunction which can further affect cellular energy production and metabolic efficiency.(Derakhshan et al., 2015)

Another key factor is the role of body composition in influencing the relationship between insulin resistance and BMR. Lean body mass particularly skeletal muscle is a major component that determine of basal metabolic rate.(Hamilton et al., 2007)Insulin resistance is often associated with increased fat accumulation and reduced muscle mass. (Upchurch, 2007) But changes in body composition may helpto observe variations in BMR among individuals with varying levels of insulin sensitivity. Furthermore, chronic low-grade inflammation which is commonly seen in insulin-resistant conditions may also influence metabolic processes and energy expenditure. (Hirano, 2018)

Despite the importance of both insulin resistance and basal metabolic rate in metabolic health, the relationship between these two factors remains complex and not fully understood. Some studies have reported an increased BMR in insulin-resistant individuals is possibly due to compensatory mechanisms such as increased sympathetic activity or increased levels of circulating insulin. (Hollstein et al., 2019) In contrast, other studies have suggested a reduced or unchanged BMR which highlights the heterogeneity of metabolic reactions among different populations. (Abdesselam et al., 2021)

In clinical practice, understanding the relationship between insulin resistance and basal metabolic rate may have significant

consequences. It can help in identifying individuals at higher risk of metabolic disorders and in tailoring personalized interventions thatimprove the metabolic health.(Gutch et al., 2015) For instance, interventions that improve insulin sensitivitysuch as physical activity, dietary changes and weight managementmay also have beneficial effects on energy metabolism and BMR. (Borai et al., 2011)Similarly, assessing BMR alongside biochemical markers of insulin resistance could provide a more comprehensive assessment of an individual's metabolic condition.(Borai et al., 2007)

Due to the increasing prevalence of metabolic diseases and the limitations of existing diagnostic methods, there is an increasing demand to explore accessible and reliable methods for assessing insulin resistance. (Antuna-Puente et al., 2011)Fasting biochemical markers provide a practical solution in this regard particularly in settings with limited resources. At the same time, investigating their association with basal metabolic rate may provide valuable insights into the underlying mechanisms of metabolic regulation and energy balance.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted to assess insulin resistance using fasting biochemical markers and to evaluate its association with variations in basal metabolic rate (BMR). This multicenter study was conducted over a period of six months from July 2025 to January 2026.The study was conducted after approval from the institutional ethical review committee. All participants were enrolled after obtaining informed written consent.

A total of 120 participants were included in the study using a non-probability consecutive sampling technique. Adult individuals aged between 20 and 60 years of both genders were recruited from outpatient departments and general health check-up clinics. Participants with previously diagnosed Type 2 Diabetes Mellitus, thyroid disorders, chronic liver disease, renal impairment or those on medications affecting glucose metabolism such as corticosteroids were excluded. Pregnant women and individuals with acute illness were also excluded to avoid confounding effects on metabolic parameters.

After enrollment, detailed demographic and clinical information was recorded including age, gender, medical history and lifestyle factors. Anthropometric measurements were obtained using standardized procedures. Body weight was measured in kilograms using a calibrated weighing scaleand height was measured in centimeters using a stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m²). Waist circumference was also measured to assess central obesity.

All participants were instructed to undergo overnight fasting for at least 8–10 hours prior to blood sampling. Venous blood samples were collected under aseptic conditions in the morning. Fasting plasma glucose was measured using the glucose oxidase methodwhile fasting serum insulin levels were determined using enzyme-linked immunosorbent assay (ELISA). Lipid profile parameters including total cholesterol, triglycerides, high-density lipoprotein (HDL) and low-density lipoprotein (LDL)were analyzed using standard automated biochemical analyzers.

Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) whichwas calculated using the formula:

HOMA-IR = (Fasting Insulin [μIU/mL] × Fasting Glucose [mg/dL]) / 405

$$\text{HOMA-IR} = \frac{\text{Fasting Insulin} \times \text{Fasting Glucose}}{405}$$

Participants were categorized into insulin-sensitive and insulin-resistant groups based on established HOMA-IR cutoff values.Basal metabolic rate (BMR) was estimated using the Harris-Benedict equation taking into account age, gender, height and weight of each participant. This method provides an indirect but widely accepted estimation of resting energy expenditure in clinical and research settings. Participants were stratified into groups based on their BMR values to assess variations across different levels of insulin resistance.

Data was entered and analyzed using Statistical Package for Social Sciences (SPSS) version 22. Quantitative variables such as age, BMI, fasting glucose, insulin levels, HOMA-IR and BMR were expressed as mean ± standard deviation. Qualitative variables such as gender and insulin resistance status were presented as frequencies and percentages. The independent sample t-test was used to compare mean values between groupswhile Pearson correlation analysis was applied to determine the relationship between HOMA-IR

and BMR. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 120 participants were included in the study. It comprised of both males and females within the age range of 20 to 60 years. The analysis focused on evaluating fasting biochemical markers, insulin resistance using HOMA-IR and their association with basal metabolic rate (BMR).

Table 1: Demographic and Anthropometric Characteristics of Study Participants

Variable	Mean ± SD
Age (years)	38.6 ± 10.2
Weight (kg)	71.4 ± 12.5
Height (cm)	165.2 ± 8.7
BMI (kg/m²)	26.1 ± 4.3
Waist Circumference (cm)	92.5 ± 10.8

The study population showed a mean age of 38.6 years with a slightly overweight average BMI. Waist circumference values indicated a considerable proportion of participants with central obesity, which is a known risk factor for insulin resistance.

Table 2: Fasting Biochemical Parameters of Participants

Parameter	Mean ± SD
Fasting Glucose (mg/dL)	102.3 ± 14.6
Fasting Insulin (µIU/mL)	14.8 ± 6.2
Total Cholesterol (mg/dL)	188.5 ± 32.1
Triglycerides (mg/dL)	156.7 ± 40.3
HDL Cholesterol (mg/dL)	41.2 ± 7.5
LDL Cholesterol (mg/dL)	115.6 ± 28.4

Fasting glucose levels were observed to be in the borderline range while fasting insulin levels showed variability among participants. Lipid profile analysis revealed mildly elevated triglycerides and relatively low HDL levels, suggesting an underlying metabolic imbalance in a subset of individuals.

Table 3: Distribution of Insulin Resistance Based on HOMA-IR

Category	Frequency (n)	Percentage (%)
Insulin Sensitive	46	38.3%
Insulin Resistant	74	61.7%

Based on HOMA-IR values, 61.7% of participants were classified as insulin resistant, indicating a high prevalence of metabolic dysfunction within the study population.

Table 4: Comparison of BMR between Insulin-Sensitive and Insulin-Resistant Groups

Group	BMR (kcal/day) Mean ± SD	p-value
Insulin Sensitive	1485.6 ± 210.4	
Insulin Resistant	1622.8 ± 245.7	0.012

Participants with insulin resistance demonstrated a significantly higher basal metabolic rate compared to insulin-sensitive individuals ($p < 0.05$). This suggests a possible compensatory increase in energy expenditure in insulin-resistant states.

Table 5: Correlation between HOMA-IR and Basal Metabolic Rate (BMR)

Variable	Correlation Coefficient (r)	p-value
HOMA-IR vs BMR	+0.34	0.004

A moderate positive correlation was observed between HOMA-IR and BMR, which was statistically significant ($p < 0.05$). This indicates that higher levels of insulin

resistance are associated with increased basal metabolic rate.

DISCUSSION

The study was conducted to assess insulin resistance using fasting biochemical markers and to explore its association with variations in basal metabolic rate (BMR). The findings demonstrate a high prevalence of insulin resistance in the study population with more than half of the participants classified as insulin resistant based on HOMA-IR values. In addition, a statistically significant positive association was observed between insulin resistance and basal metabolic rate. This suggests that metabolic alterations in insulin-resistant individuals may influence resting energy expenditure.

The use of fasting biochemical markers particularly the Homeostatic Model Assessment (HOMA-IR)proved to be a practical and reliable method for evaluating insulin resistance in this study. This is consistent with the findings of Matthews DR et al., who originally developed the HOMA model and demonstrated its effectiveness as a surrogate marker for insulin sensitivity in epidemiological and clinical research.(Matthews DR, 1985) Similarly, studies by Wallace TM and Levy JC have further validated HOMA-IR as a useful tool for large-scale metabolic studies due to its simplicity and reproducibility.(Wallace et al., 2004)

The prevalence of insulin resistance observed in this study aligns with global trends, particularly in populations with increasing rates of obesity and sedentary lifestyles. Research conducted by Reaven GM highlighted insulin resistance as a central feature of metabolic syndrome which link it to a cluster of conditions including dyslipidemia, hypertension and impaired glucose tolerance.(Reaven, 1988) The elevated triglyceride levels and reduced HDL cholesterol observed in our study further support this association, indicating an underlying metabolic imbalance among insulin-resistant individuals.

One of the key findings of this study is the significantly higher basal metabolic rate observed in participants with insulin resistance compared to insulin-sensitive individuals. This finding is in agreement with studies conducted by Segal KR et al., who reported increased resting energy expenditure in insulin-resistant individuals possibly due to compensatory mechanisms such as hyperinsulinemia and increased sympathetic nervous system activity. Elevated insulin levels may stimulate thermogenesis and increase metabolic activity contributing to a higher BMR.(Segal et al., 1991)

The positive correlation between HOMA-IR and BMR observed in this study further supports the hypothesis that insulin resistance is associated with alterations in energy metabolism. Similar findings have been reported by Weyer C et al., who demonstrated that individuals with insulin resistance exhibit increased energy expenditure potentially as a compensatory response to reduced metabolic efficiency. This phenomenon may reflect the body's attempt to maintain energy balance despite impaired glucose utilization.(Weyer et al., 1999)

However, the relationship between insulin resistance and BMR is complex and not entirely consistent across all studies. For instance, research by Ravussin E and colleagues has shown that lower metabolic rates may predispose individuals to weight gain and insulin resistance over time. These contrasting findings suggest that the direction of the relationship may depend on various factors, including the stage of metabolic dysfunction, body composition and genetic predisposition.(Ravussin et al., 1988)

Body composition plays a crucial role in determining basal metabolic rate, as lean body mass is the primary contributor to resting energy expenditure. Insulin resistance is often associated with increased adiposity and reduced muscle mass which may influence BMR in different ways. In early stages of insulin resistance, increased fat mass and hyperinsulinemia may elevate BMR whereas in later stages, metabolic adaptations and reduced lean mass may lead to a decline in energy expenditure. This dynamic interplay may explain the variability observed in different studies.

Another important mechanism linking insulin resistance to changes in BMR is mitochondrial dysfunction. Impaired mitochondrial activity in insulin-resistant individuals can reduce the efficiency of oxidative phosphorylation that leads to altered energy production and increased metabolic demand. Studies by Petersen KF and Shulman GI have emphasized the role of mitochondrial dysfunction in the pathogenesis of insulin resistance which highlight its impact on cellular energy metabolism.(Petersen and Shulman, 2006)

The findings of this study have important clinical implications. The significant association

between insulin resistance and basal metabolic rate suggests that assessment of BMR, along with fasting biochemical markers which may provide a more comprehensive understanding of an individual's metabolic status. This could aid in early identification of individuals at risk of metabolic disorders and facilitate the development of personalized interventions aimed at improving insulin sensitivity and metabolic health.

CONCLUSION

This study showed that insulin resistanceis highly prevalent and shows a significant positive association with basal metabolic rate variations when assessed through fasting biochemical markers such as HOMA-IR. Individuals with higher levels of insulin resistance exhibited increased resting energy expenditure which suggested that the underlying metabolic adaptations may be related to hyperinsulinemia and altered substrate utilization. These findings highlight the importance of simple, cost-effective biochemical markers in the early detection of metabolic dysfunction and emphasize the potential value of incorporating basal metabolic rate assessment for a more comprehensive evaluation of metabolic health. Further large-scale and longitudinal studies are recommended to better understand the causal relationship and underlying mechanisms linking insulin resistance with energy metabolism.

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