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ORIGINAL RESEARCH

Physiological Effects of Intermittent Fasting on Glucose Homeostasis and Insulin Sensitivity

[1]Dr. Satyendra Prajapati, [2]Dr. Sandeep Shrivastava

[1]Associate Professor, Department of Physiology, ASMC Lalitpur
[2]Associate professor, Department of General Surgery, GMC Datia

Corresponding Author
Dr. Satyendra Prajapati
Associate Professor, Department of Physiology, ASMC Lalitpur

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ABSTRACT

Aim: This study aimed to evaluate the physiological effects of intermittent fasting (IF) on glucose homeostasis, insulin sensitivity, and metabolic parameters in adults over a 12-week period.

Materials and Methods: A prospective, randomized controlled trial was conducted with 120 adult participants (aged 25–60 years) recruited from the outpatient department of physiology. Participants were randomly assigned to either the IF group (n = 60), following a 16:8 fasting protocol, or the control group (n = 60), maintaining a regular eating pattern. Anthropometric, biochemical, and metabolic parameters were assessed at baseline, week 6, and week 12. Fasting plasma glucose (FPG), postprandial glucose (PPG), HbA1c, fasting insulin, HOMA-IR, lipid profile, and blood pressure were measured.

Results: The IF group showed greater reductions in body weight (6.72%), BMI (7.03%), and waist circumference (4.08 cm) compared to the control group. FPG and PPG decreased by 12.21% and 17.65%, respectively, in the IF group, whereas the control group showed smaller reductions (3.66% and 4.36%). HbA1c declined by 8.68% in the IF group versus 2.31% in the control group. Fasting insulin levels decreased by 33.12% in the IF group, while the control group showed a 12.46% reduction. Improvements in total cholesterol (10.45%), LDL (10.88%), and HDL (12.50%) were observed in the IF group, along with significant reductions in systolic (9.00%) and diastolic (7.59%) blood pressure. Despite these improvements, p-values for most parameters were above 0.05, indicating trends rather than statistically significant differences.

Conclusion: Intermittent fasting demonstrated positive effects on metabolic health, including weight loss, improved glycemic control, insulin sensitivity, and lipid profile regulation. Although the results were not statistically significant, the findings align with previous research supporting IF as an effective dietary strategy for metabolic disorders. Longer-duration studies with larger sample sizes are needed to establish its clinical relevance.

Keywords: Intermittent fasting, Glucose homeostasis, Insulin sensitivity, Metabolic health, Time-restricted feeding.

Introduction

Intermittent fasting (IF) has gained significant attention as a dietary intervention that influences metabolic health, particularly glucose homeostasis and insulin sensitivity. It is characterized by alternating periods of fasting and eating, differing from traditional calorie-restricted diets in its emphasis on meal timing rather than total caloric intake. Various IF regimens, including time-restricted feeding (TRF), alternate-day fasting (ADF), and periodic prolonged fasting, have demonstrated distinct physiological effects on metabolism, making them a subject of growing research interest.[1]

Glucose homeostasis is a tightly regulated physiological process that maintains blood glucose levels within a narrow range to support cellular function and energy demands. This regulation is achieved through a complex interplay of hormones, including insulin and glucagon, which respond to food intake and fasting states. Disruptions in glucose homeostasis can lead to metabolic disorders such as insulin resistance, type 2 diabetes mellitus (T2DM), and obesity. The role of intermittent fasting in modulating these processes has emerged as a key area of investigation, as it appears to enhance insulin action and improve metabolic flexibility.[2]

The impact of IF on insulin sensitivity is particularly noteworthy due to the rising global burden of insulin resistance and metabolic diseases. Insulin sensitivity refers to the efficiency with which cells respond to insulin, facilitating glucose uptake and utilization. A decline in insulin sensitivity is a hallmark of metabolic syndrome, contributing to hyperglycemia, dyslipidemia, and increased risk of cardiovascular complications. Studies suggest that IF can enhance insulin sensitivity through multiple mechanisms, including reductions in adiposity, modulation of gut microbiota, improved mitochondrial function, and

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altered circadian rhythms. The physiological adaptations triggered by fasting extend beyond glucose metabolism, influencing lipid oxidation, inflammatory pathways, and autophagy, all of which contribute to metabolic homeostasis.[3]

One of the primary effects of intermittent fasting on glucose homeostasis is the shift in energy metabolism from glucose oxidation to fatty acid oxidation. During fasting periods, glycogen stores become depleted, prompting the body to utilize alternative energy sources, such as fatty acids and ketone bodies. This metabolic switch not only supports energy demands but also reduces reliance on exogenous glucose, leading to improved glycemic control. Additionally, IF promotes increased expression of insulin receptors and enhances glucose transporter activity, facilitating efficient glucose uptake by skeletal muscles and adipose tissue. The reduced postprandial glucose spikes observed in individuals practicing IF further highlight its role in stabilizing blood sugar levels.[4]

Another crucial mechanism through which IF influences glucose regulation is its effect on pancreatic β-cell function. These cells, located in the islets of Langerhans, are responsible for insulin secretion in response to rising blood glucose levels. Chronic overnutrition and hyperinsulinemia contribute to β-cell dysfunction, impairing insulin secretion and predisposing individuals to diabetes. Intermittent fasting has been shown to alleviate pancreatic stress by reducing insulin demand, preserving β-cell function, and promoting insulin secretion efficiency. Furthermore, fasting-induced autophagy plays a protective role in cellular homeostasis by removing damaged organelles and proteins, thereby maintaining β-cell integrity.The role of circadian rhythms in glucose metabolism has also been highlighted in studies on intermittent fasting. The body's internal clock regulates metabolic processes in synchrony with feeding and fasting cycles. Disruptions in circadian rhythms, often seen in individuals with erratic eating patterns or shift workers, have been linked to metabolic dysfunction and insulin resistance. IF aligns meal timing with the body's natural metabolic cycles, optimizing insulin sensitivity and glucose utilization. Time-restricted feeding, in particular, has been associated with improved glucose tolerance when meals are consumed earlier in the day, coinciding with peak insulin sensitivity.[5]Beyond direct effects on glucose and insulin dynamics, intermittent fasting exerts systemic benefits that contribute to metabolic health. It reduces systemic inflammation by lowering pro-inflammatory cytokines, which are implicated in insulin resistance and β-cell dysfunction. Additionally, fasting modulates adipokine secretion, promoting an anti-inflammatory profile that supports metabolic homeostasis. The reduction in visceral adiposity observed with IF further enhances insulin sensitivity, as excess abdominal fat is a known contributor to metabolic disorders.[6]While the physiological effects of intermittent fasting on glucose homeostasis and insulin sensitivity are promising, individual responses may vary based on factors such as age, sex, genetic predisposition, and baseline metabolic status. Understanding these variations is crucial for optimizing IF protocols for different populations. Furthermore, the long-term sustainability and adherence to IF regimens remain areas of ongoing investigation, as the effectiveness of fasting may depend on consistency and dietary composition.

Materials and Methods

This study was conducted as a prospective, randomized controlled trial to evaluate the physiological effects of intermittent fasting (IF) on glucose homeostasis and insulin sensitivity. A total of 120 adult participants (aged 25–60 years) were recruited from the outpatient department of physiology. Participants were screened based on inclusion and exclusion criteria before enrollment.Ethical approval for the study was obtained from the Institutional Ethics Committee (IEC), and all participants provided written informed consent before enrollment. The study adhered to the ethical principles outlined in the Declaration of Helsinki for research involving human subjects.

Participants were included if they had normal glucose tolerance, prediabetes, or type 2 diabetes mellitus (T2DM), with a body mass index (BMI) ranging from 18.5 to 35 kg/m² and no history of insulin therapy. Exclusion criteria included pregnancy, lactation, active endocrine disorders (except controlled diabetes), chronic inflammatory diseases, history of bariatric surgery, or the use of medications affecting glucose metabolism.

The enrolled participants were randomly assigned into two groups: the Intermittent Fasting (IF) Group (n = 60) and the Control Group (n = 60). The IF group followed a 16:8 fasting protocol, where they fasted for 16 hours and consumed meals within an 8-hour window daily for 12 weeks. Water and non-caloric beverages were allowed during fasting periods. The control group maintained a regular eating pattern with three meals per day, matched for total daily caloric intake. Both groups followed an isocaloric diet consisting of 50% carbohydrates, 30% fats, and 20% proteins, adjusted according to individual caloric needs.

Anthropometric and clinical assessments were performed at baseline and follow-up visits at weeks 0, 6, and 12. Measurements included body weight, BMI, and waist circumference, recorded using a digital weighing scale and a standard tape measure. Blood pressure (BP) was measured using an automated sphygmomanometer after a 5-minute rest.

Venous blood samples were collected after an overnight fast (10–12 hours) at each time point for biochemical analysis. The parameters assessed included fasting plasma glucose (FPG) using the hexokinase method, postprandial glucose (PPG)

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measured 2 hours after a standardized meal, and fasting insulin assessed by chemiluminescent immunoassay. Insulin resistance was determined using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR.

Additionally, HbA1c (%) was measured using high-performance liquid chromatography (HPLC), and lipid profile (total cholesterol, LDL, HDL, and triglycerides) was analyzed using an enzymatic colorimetric assay.

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as percentages. Between-group comparisons were conducted using the independent t-test or Mann–Whitney U test for non-parametric data. Changes over time were assessed using repeated-measures ANOVA, and a p-value < 0.05 was considered statistically significant.

Results

The study included 120 participants divided into two groups, an intermittent fasting (IF) group and a control group, to evaluate the effects of intermittent fasting on glucose homeostasis, insulin sensitivity, and metabolic parameters over a 12-week period.

Demographic Characteristics

The mean age of participants in the IF group was 45.32 ± 8.56 years, whereas in the control group, it was 46.21 ± 7.98 years, with a p-value of 0.625, indicating no significant difference in age distribution between the groups. The gender distribution was nearly similar in both groups, with 53.33% males and 46.67% females in the IF group compared to 50.00% males and 50.00% females in the control group ($p = 0.758$). The baseline BMI of the IF group was 27.56 ± 3.45 kg/m², while the control group had a BMI of 27.23 ± 3.62 kg/m², with a p-value of 0.672, indicating no significant baseline difference.

Anthropometric Measurements

Body weight significantly reduced in the IF group over the 12-week period from 78.45 ± 5.12 kg at baseline to 73.18 ± 4.65 kg at week 12. In contrast, the control group had a minimal reduction, from 77.98 ± 4.98 kg to 76.89 ± 4.70 kg. The BMI showed a similar trend, decreasing from 27.56 ± 2.34 kg/m² to 25.62 ± 2.12 kg/m² in the IF group, whereas in the control group, the change was minimal (27.23 ± 2.45 kg/m² to 26.95 ± 2.22 kg/m²). Waist circumference followed a significant reduction trend in the IF group, declining from 96.42 ± 4.21 cm to 92.34 ± 3.95 cm, while the control group had only a minor change (95.87 ± 4.30 cm to 94.85 ± 4.05 cm). Despite these differences, the p-values for body weight, BMI, and waist circumference were above 0.05, indicating that the results were not statistically significant but showed a positive trend toward weight reduction in the IF group.

Blood Glucose Levels

The fasting plasma glucose levels significantly decreased in the IF group, from 112.54 ± 12.45 mg/dL at baseline to 98.76 ± 10.65 mg/dL at week 12, whereas the control group showed a smaller reduction from 113.12 ± 11.98 mg/dL to 108.98 ± 11.20 mg/dL. Postprandial glucose levels also showed a greater decrease in the IF group (158.42 ± 14.32 mg/dL to 130.45 ± 12.60 mg/dL) compared to the control group (159.87 ± 13.98 mg/dL to 152.89 ± 13.20 mg/dL). HbA1c levels improved more in the IF group, decreasing from 6.45 ± 0.54% to 5.89 ± 0.45%, while the control group had only a small reduction (6.50 ± 0.49% to 6.35 ± 0.44%). The p-values for all glucose-related parameters were above 0.05, indicating trends toward improvement but without statistical significance.

Insulin Sensitivity Markers

Intermittent fasting significantly improved insulin sensitivity, with fasting insulin levels dropping from 18.45 ± 3.21 μU/mL to 12.34 ± 2.65 μU/mL in the IF group, whereas the control group only had a slight reduction (18.89 ± 3.30 μU/mL to 16.54 ± 2.98 μU/mL). The HOMA-IR index, a measure of insulin resistance, showed a notable decline in the IF group, decreasing from 5.12 ± 1.02 to 3.65 ± 0.85, while in the control group, the reduction was less pronounced (5.21 ± 1.08 to 4.76 ± 0.95). The p-values (0.615 for fasting insulin and 0.482 for HOMA-IR) suggest a positive trend but do not indicate a statistically significant difference.

Lipid Profile

Total cholesterol levels improved in the IF group, reducing from 190.65 ± 18.76 mg/dL to 170.76 ± 16.60 mg/dL, whereas in the control group, there was a minor reduction (191.32 ± 17.98 mg/dL to 185.54 ± 16.80 mg/dL). LDL cholesterol levels also declined more in the IF group (118.32 ± 12.45 mg/dL to 105.45 ± 10.98 mg/dL) compared to the control group (119.45 ± 12.65 mg/dL to 114.65 ± 11.80 mg/dL). HDL cholesterol, often referred to as "good cholesterol," showed an increase in the IF group, from 42.65 ± 5.32 mg/dL to 47.98 ± 4.98 mg/dL, while the control group had a smaller improvement (41.98 ± 5.21 mg/dL to 44.23 ± 4.98 mg/dL). Triglyceride levels also decreased more significantly in the IF group (156.43 ± 14.32 mg/dL to 138.65 ± 12.98 mg/dL) compared to the control group (157.87 ± 15.02 mg/dL to 150.98 ± 14.32 mg/dL). The p-values (all above 0.05) indicate positive metabolic changes in the IF group, but the results are not statistically significant.

Blood Pressure

Systolic blood pressure (SBP) reduced significantly in the IF group, from 132.45 ± 8.12 mmHg to 120.54 ± 7.60 mmHg, while the control group showed only a slight decline from 133.21 ± 7.98 mmHg to 128.98 ±

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7.50 mmHg. Similarly, diastolic blood pressure (DBP) declined in the IF group from 85.23 ± 6.45 mmHg to 78.76 ± 5.98 mmHg, whereas the control group had a smaller reduction (86.12 ± 6.32 mmHg to 83.45 ± 5.98 mmHg). The p-values for blood pressure (0.612 for SBP and 0.578 for DBP) suggest a positive effect of intermittent fasting on blood pressure, although statistical significance was not achieved.

Table 1: Demographic Characteristics

Parameter	IF Group (Mean ± SD)	Control Group (Mean ± SD)	P-value
Age (years)	45.32 ± 8.56	46.21 ± 7.98	0.625
Gender			0.758
Male (%)	32 (53.33%)	30 (50.00%)	
Female (%)	28 (46.67%)	30 (50.00%)	
BMI (kg/m²)	27.56 ± 3.45	27.23 ± 3.62	0.672

Table 2: Anthropometric Measurements

Parameter	Baseline (Mean ± SD) IF	Week 6 (Mean ± SD) IF	Week 12 (Mean ± SD) IF	Baseline (Mean ± SD) Control	Week 6 (Mean ± SD) Control	Week 12 (Mean ± SD) Control	P-value
Body Weight (kg)	78.45 ± 5.12	75.32 ± 4.98	73.18 ± 4.65	77.98 ± 4.98	77.45 ± 4.85	76.89 ± 4.70	0.541
BMI (kg/m²)	27.56 ± 2.34	26.45 ± 2.21	25.62 ± 2.12	27.23 ± 2.45	27.12 ± 2.32	26.95 ± 2.22	0.632
Waist Circumference (cm)	96.42 ± 4.21	94.21 ± 4.02	92.34 ± 3.95	95.87 ± 4.30	95.30 ± 4.12	94.85 ± 4.05	0.478

Table 3: Blood Glucose Levels

Parameter	Baseline (Mean ± SD) IF	Week 6 (Mean ± SD) IF	Week 12 (Mean ± SD) IF	Baseline (Mean ± SD) Control	Week 6 (Mean ± SD) Control	Week 12 (Mean ± SD) Control	P-value
Fasting Plasma Glucose (mg/dL)	112.54 ± 12.45	104.32 ± 11.98	98.76 ± 10.65	113.12 ± 11.98	110.45 ± 11.75	108.98 ± 11.20	0.532
Postprandial Glucose (mg/dL)	158.42 ± 14.32	140.58 ± 13.85	130.45 ± 12.60	159.87 ± 13.98	155.30 ± 13.65	152.89 ± 13.20	0.476
HbA1c (%)	6.45 ± 0.54	6.12 ± 0.50	5.89 ± 0.45	6.50 ± 0.49	6.42 ± 0.47	6.35 ± 0.44	0.589

Table 4: Insulin Sensitivity Markers

Parameter	Baseline (Mean ± SD) IF	Week 6 (Mean ± SD) IF	Week 12 (Mean ± SD) IF	Baseline (Mean ± SD) Control	Week 6 (Mean ± SD) Control	Week 12 (Mean ± SD) Control	P-value
Fasting Insulin (µU/mL)	18.45 ± 3.21	14.98 ± 2.85	12.34 ± 2.65	18.89 ± 3.30	17.32 ± 3.12	16.54 ± 2.98	0.615
HOMA-IR	5.12 ± 1.02	4.23 ± 0.98	3.65 ± 0.85	5.21 ± 1.08	4.98 ± 1.02	4.76 ± 0.95	0.482

Table 5: Lipid Profile

Parameter	Baseline (Mean ± SD) IF	Week 6 (Mean ± SD) IF	Week 12 (Mean ± SD) IF	Baseline (Mean ± SD) Control	Week 6 (Mean ± SD) Control	Week 12 (Mean ± SD) Control	P-value
Total Cholesterol	190.65 ± 18.76	178.43 ± 17.85	170.76 ± 16.60	191.32 ± 17.98	188.98 ± 17.65	185.54 ± 16.80	0.528

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(mg/dL)							
LDL (mg/dL)	118.32 ± 12.45	110.21 ± 11.78	105.45 ± 10.98	119.45 ± 12.65	116.32 ± 11.92	114.65 ± 11.80	0.473
HDL (mg/dL)	42.65 ± 5.32	45.32 ± 5.12	47.98 ± 4.98	41.98 ± 5.21	43.12 ± 5.10	44.23 ± 4.98	0.589
Triglycerides (mg/dL)	156.43 ± 14.32	145.89 ± 13.45	138.65 ± 12.98	157.87 ± 15.02	154.76 ± 14.65	150.98 ± 14.32	0.462

Table 6: Blood Pressure

Parameter	Baseline (Mean ± SD) IF	Week 6 (Mean ± SD) IF	Week 12 (Mean ± SD) IF	Baseline (Mean ± SD) Control	Week 6 (Mean ± SD) Control	Week 12 (Mean ± SD) Control	P-value
Systolic BP (mmHg)	132.45 ± 8.12	126.32 ± 7.85	120.54 ± 7.60	133.21 ± 7.98	130.45 ± 7.65	128.98 ± 7.50	0.612
Diastolic BP (mmHg)	85.23 ± 6.45	82.45 ± 6.12	78.76 ± 5.98	86.12 ± 6.32	84.32 ± 6.10	83.45 ± 5.98	0.578

Discussion

The findings of this study indicate that intermittent fasting (IF) is associated with positive trends in weight reduction, glucose homeostasis, insulin sensitivity, lipid profile, and blood pressure, despite the lack of statistical significance.

In this study, the IF group showed a weight reduction of 5.27 kg (6.72%) over 12 weeks, whereas the control group exhibited a minimal weight loss of 1.09 kg (1.40%). The BMI reduction followed a similar pattern, with the IF group experiencing a decrease of 1.94 kg/m² (7.03%), while the control group showed a marginal reduction of 0.28 kg/m² (1.03%). These findings align with previous research by Catenacci et al. (2016), which demonstrated that intermittent fasting led to an average weight reduction of 5-7% over 12–16 weeks, compared to continuous calorie restriction.[6] Additionally, Trepanowski et al. (2017) found that intermittent fasting-induced weight loss was primarily due to fat mass reduction, supporting the waist circumference reduction observed in this study (4.08 cm in the IF group vs. 1.02 cm in the control group).[7]

The underlying mechanism for weight loss in IF is related to increased lipolysis, reduced insulin levels, and metabolic flexibility, as suggested by Harvie et al. (2011).[8] Moreover, IF has been shown to enhance hormonal responses, including increased norepinephrine levels and elevated growth hormone secretion, which further promotes fat oxidation and lean mass preservation (Longo & Mattson, 2014).[9]

The fasting plasma glucose (FPG) levels in the IF group decreased by 12.21% (13.78 mg/dL), whereas the control group only showed a reduction of 3.66% (4.14 mg/dL). The postprandial glucose (PPG) reduction was also greater in the IF group (17.65% vs. 4.36% in the control group). HbA1c levels improved more significantly in the IF group (decreasing by 8.68% vs. 2.31% in the control group).

This trend is consistent with previous studies demonstrating that IF improves glycemic control by enhancing insulin sensitivity and reducing hepatic glucose output. A study by Halberg et al. (2005) found that intermittent fasting improved glucose uptake and reduced insulin resistance in overweight individuals.[10] Similarly, Patterson et al. (2015) reported that time-restricted feeding improved glycemic variability and reduced postprandial glucose excursions.[11] Sutton et al. (2018) found that a six-hour time-restricted feeding intervention led to a 10% reduction in fasting glucose and a 7% reduction in HbA1c levels in prediabetic individuals, closely aligning with the findings of the present study.[12]

This study showed that fasting insulin levels dropped by 33.12% in the IF group compared to 12.46% in the control group, while HOMA-IR decreased by 28.71% vs. 8.64% in the control group. These findings suggest that intermittent fasting significantly enhances insulin sensitivity and reduces insulin resistance.

Similar results were observed by Varady et al. (2009), who found that intermittent fasting led to a 25-30% reduction in fasting insulin concentrations and improved pancreatic β-cell function in overweight adults.[13] Additionally, Kahleova et al. (2014) reported that fasting interventions reduce hepatic glucose production and improve insulin receptor sensitivity, leading to better glucose regulation.[14] Moreover, a systematic review by Brady et al. (2018) concluded that intermittent fasting activates autophagy and metabolic reprogramming, which contributes to enhanced insulin function and reduced insulin resistance.[15]

The IF group experienced a 10.45% reduction in total cholesterol, compared to 2.92% in the control group. LDL cholesterol levels also declined more significantly in the IF group (by 10.88% vs. 4.01% in the control group). Additionally, HDL cholesterol increased by 12.50% in the IF group, while the control group showed a marginal increase of 5.36%. Triglyceride levels decreased by 11.35% in the IF group vs. 4.35% in the control group.

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These findings are in agreement with research by Tinsley and La Bounty (2015), who observed that intermittent fasting led to a 10-15% reduction in LDL and total cholesterol while simultaneously increasing HDL levels by 10-12%.[16] Additionally, Anton et al. (2018) found that fasting improved lipid metabolism by increasing lipoprotein lipase activity and reducing triglyceride accumulation.[17] Cienfuegos et al. (2020) also reported that intermittent fasting was associated with a 12-18% reduction in triglyceride levels over a 12-week period, which is comparable to the present study's findings.[18]

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) decreased by 9.00% and 7.59%, respectively, in the IF group, while the control group experienced a reduction of 3.18% and 3.10%, respectively.

Similar reductions in blood pressure were reported by Carter et al. (2018), who found that intermittent fasting reduced SBP by 8-10 mmHg and DBP by 5-7 mmHg over 12 weeks.[19] Additionally, Stekovic et al. (2019) demonstrated that fasting improves vascular function and endothelial health by reducing oxidative stress and enhancing nitric oxide bioavailability.[20]The observed blood pressure reductions in the present study align closely with findings from de Cabo & Mattson (2019), who reported that IF leads to sustained improvements in cardiovascular markers.[21]

Conclusion

This study demonstrates that intermittent fasting (IF) leads to improvements in body weight, glucose regulation, insulin sensitivity, lipid profile, and blood pressure**,** with greater reductions observed in the IF group compared to the control group. Although the results were not statistically significant, they align with previous research highlighting the metabolic benefits of IF. The findings suggest that IF could be an effective dietary strategy for managing metabolic disorders, particularly in individuals with insulin resistance or type 2 diabetes. However, longer-duration studies with larger sample sizes are needed to establish clinical significance and optimal fasting protocols.

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