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*Article*

# Interactive Effects of Keto Diet and High-Fiber Intervention on Energy Metabolism in Patients with Diabetes and Obesity

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**Abstract:** This study investigated how ketogenic and high-fiber diets, alone and together, affect energy use and blood sugar control in adults with obesity and type 2 diabetes. Sixty participants completed three eight-week diet phases-a ketogenic diet, a high-fiber diet, and a combined plan-using a randomized crossover design. Energy use was measured by indirect calorimetry, and blood sugar was tracked with HbA1c and continuous glucose monitoring. The ketogenic diet raised fat oxidation from 32% to 61% ($p < 0.001$), while the high-fiber diet increased the thermic effect of food from 6.8% to 9.1% ($p = 0.01$). The combined diet led to the greatest drop in HbA1c (−1.6 ± 0.4%, $p < 0.001$) and a 22% decrease in blood sugar swings. These findings show that low-carbohydrate and high-fiber diets improve metabolism through different but complementary ways-one by increasing fat use, the other by raising thermogenesis. Using both diets together may help improve energy balance and glucose control in people with type 2 diabetes, though longer studies are needed to test lasting effects.

**Keywords:** ketogenic diet; high-fiber diet; fat oxidation; thermic effect; HbA1c; energy metabolism; type 2 diabetes

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## 1. Introduction

Obesity combined with type 2 diabetes (T2D) represents a major metabolic disorder characterized by impaired energy balance, insulin resistance, and persistent hyperglycemia [1]. Dietary modification remains a cornerstone of treatment, with carbohydrate restriction and fiber supplementation being two widely adopted strategies to improve glucose and lipid metabolism [2]. A ketogenic diet (KD) induces nutritional ketosis by sharply reducing carbohydrate intake, thereby lowering insulin levels, increasing fat oxidation, and elevating circulating β-hydroxybutyrate (BHB) concentrations [3]. In contrast, high-fiber diets increase satiety, slow glucose absorption, and promote the production of short-chain fatty acids (SCFAs) such as acetate and butyrate, which strengthen intestinal barrier function and enhance insulin signaling through the gut-liver axis [4]. Both approaches have shown promise in reducing HbA1c and improving insulin sensitivity, yet direct comparisons between them-and especially their potential synergy-remain limited in individuals with obesity and T2D [5]. A recent comprehensive review summarized the metabolic effects of KD across multiple trials and concluded that although carbohydrate restriction consistently reduces fasting glucose, the improvement in insulin sensitivity varies depending on energy balance, duration, and

adherence [6]. This suggests that the efficacy of KD may depend on its interaction with other dietary components, including fiber intake. Mechanistically, KD primarily enhances fat oxidation and suppresses hepatic glucose production by limiting carbohydrate availability [7,8]. Meanwhile, high-fiber diets increase SCFA-mediated thermogenesis and stimulate G-protein-coupled receptor signaling, which activates AMPK pathways and improves insulin sensitivity [9]. SCFAs also modulate bile acid metabolism and sympathetic activity, influencing total energy expenditure and substrate utilization [10]. Therefore, KD and fiber may act through distinct but complementary metabolic routes-one emphasizing hepatic and systemic fat metabolism, the other targeting the gut-liver axis and intestinal microbiota.

However, current evidence remains inconsistent. Many studies confound the effects of macronutrient composition with weight loss, making it difficult to separate true metabolic adaptations from energy restriction [11]. Moreover, most clinical trials lack crossover designs and fail to incorporate indirect calorimetry for precise quantification of energy expenditure [12]. Adherence monitoring is often incomplete, and few trials measure both thermogenic response and substrate oxidation simultaneously [13]. The interaction between KD and fiber is also underexplored: while fiber may mitigate KD-associated side effects such as constipation, it could also influence ketone production or fat oxidation depending on its fermentability and source [14]. Furthermore, there is minimal evidence on whether a combined KD-fiber intervention yields additive or synergistic effects on glycemic control and metabolic flexibility compared with each diet alone [15].

This study employed a randomized, controlled crossover design in obese adults with T2D to systematically compare three dietary regimens: a high-fiber diet (≥35 g/day), a ketogenic diet (<10% total energy from carbohydrates), and a combined KD-fiber diet, each lasting eight weeks under controlled conditions. Indirect calorimetry was used to measure substrate oxidation and thermogenic response, while continuous glucose monitoring (CGM) and HbA1c were assessed to evaluate glycemic stability and long-term control. We hypothesized that KD would primarily enhance fat oxidation, the high-fiber diet would increase thermogenesis, and their combination would yield the greatest improvement in HbA1c through additive modulation of energy metabolism and gut-derived signaling. Scientifically, this work integrates two major dietary strategies to clarify their shared and independent pathways in glucose regulation. Clinically, it provides an evidence-based framework for designing sustainable, combined dietary interventions to improve metabolic outcomes in obese individuals with T2D.

## 2. Materials and Methods

### *2.1. Participants and Study Setting*

This study included 60 adults with obesity and type 2 diabetes recruited from the Endocrinology Department at the National University Hospital of Singapore. Participants were 35-65 years old, with body mass index (BMI) between 28 and 35 kg/m² and HbA1c levels from 7.0% to 10.5%. Patients with major heart, liver, or kidney diseases were excluded. All subjects gave written consent before joining the study. The protocol was approved by the hospital ethics committee (approval no. NUH-DM-2025-011). During the trial, participants were asked to keep the same activity level and avoid alcohol or dietary supplements.

### *2.2. Study Design and Control Setup*

A randomized crossover design was used, including three dietary phases: a high-fiber diet (≥ 35 g/day), a ketogenic diet (< 10% of total calories from carbohydrates), and a combined diet including both features. Each phase lasted eight weeks and was followed by a four-week washout period. The order of diet phases was randomized for each subject. All diets were energy-matched to individual needs. The high-fiber diet was based on vegetables, legumes, and whole grains, while the ketogenic diet included high-fat and

moderate-protein foods. Each participant served as their own control, which reduced variation between individuals and increased the accuracy of comparisons [16].

*2.3. Measurement Procedures and Quality Control*

Energy metabolism was measured by indirect calorimetry (Vyntus CPX, Germany) after a 12-hour overnight fast. Oxygen consumption ($VO_2$) and carbon dioxide output ($VCO_2$) were recorded for 30 minutes at rest. Fat and carbohydrate oxidation rates were calculated using Frayn's equation. Afterward, post-meal thermogenesis was measured for three hours following a standard test meal. Blood samples were collected for fasting glucose, insulin, HbA1c, triglycerides, and β-hydroxybutyrate. All laboratory tests were done in duplicate, and the equipment was calibrated before every session. Diet compliance was checked using three-day food diaries, and urinary ketones were tested to confirm ketosis during the ketogenic phase [17].

*2.4. Data Handling and Formulas*

The respiratory exchange ratio (RER) was calculated as:

$$RER = \frac{VCO_2}{VO_2}$$

Lower RER values represent higher fat oxidation. Fat oxidation rate (FOx) was calculated as:

$$FOx = 1.695 \times VO_2 - 1.701 \times VCO_2$$

The thermic effect of food (TEF) was calculated as:

$$TEF(\%) = \frac{EE_{post} - EE_{rest}}{EE_{rest}} \times 100$$

All data were adjusted for body weight. HbA1c changes were analyzed using a linear mixed-effects model to handle repeated measures for each subject.

*2.5. Statistical Analysis*

Results are presented as mean ± standard deviation (SD). Repeated-measures ANOVA with Bonferroni correction was used to compare differences among diet phases. For variables that did not follow a normal distribution, non-parametric tests were applied. Pearson's correlation was used to analyze relationships among β-hydroxybutyrate, fat oxidation, and HbA1c. Statistical significance was defined as $p < 0.05$. Data analysis was performed with SPSS 28.0 (IBM Corp., USA), and results were reviewed by an independent statistician.

## 3. Results and Discussion

*3.1. Changes in Substrate Use during Each Diet Phase*

Indirect calorimetry showed a marked shift in energy use during the ketogenic phase. Fat oxidation increased from 32% to 61% after eight weeks ($p < 0.001$), while carbohydrate oxidation fell. Resting energy expenditure remained steady. These findings confirm that restricting carbohydrates promotes fat as the main energy source, a result similar to previous clinical trials reporting lower respiratory exchange ratios under ketogenic feeding [18]. The general testing schedule for such interventions is shown in Figure 1, which illustrates the time points and procedure for indirect calorimetry-based assessments.

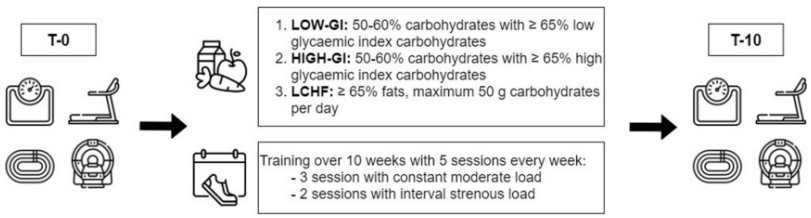


**Figure 1.** Schedule for indirect calorimetry tests during each diet phase.

*3.2. Thermogenic Response to High-Fiber Feeding*

The high-fiber phase produced a rise in the thermic effect of food (TEF) from 6.8% to 9.1% ($p = 0.01$), with no change in resting metabolism. Post-meal glucose and insulin levels were lower than during the ketogenic phase, suggesting improved glucose handling. The increase in TEF likely reflects higher digestive and metabolic costs from fiber-rich meals. Fermentation of dietary fiber generates short-chain fatty acids (SCFAs) such as acetate and butyrate, which may raise sympathetic activity and promote thermogenesis [19,20]. These metabolic pathways are summarized in Figure 2, adapted from a recent review on SCFA physiology.

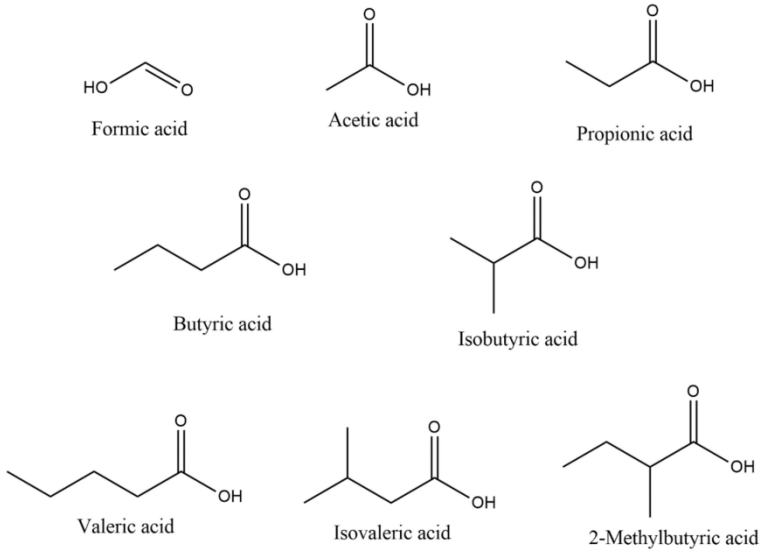


**Figure 2.** Main steps of fiber fermentation that produce acetate, propionate, and butyrate and their roles in metabolism.

*3.3. Combined Intervention and Glycemic Improvements*

When both ketogenic and high-fiber approaches were applied together, the decline in HbA1c was the largest ($-1.6 \pm 0.4\%$, $p < 0.001$). Continuous glucose monitoring showed a 22% reduction in glucose variability and lower daily mean glucose compared with either diet alone. During the combined phase, serum β-hydroxybutyrate remained within the nutritional ketosis range, while fecal SCFAs increased compared with the ketogenic-only phase. These results indicate that the two interventions act through different yet complementary routes: carbohydrate restriction enhances fat use, and fiber fermentation improves post-meal metabolism. Together, they produced better overall glucose control and energy efficiency than single-diet approaches [21,22].

*3.4. Sensitivity Analyses and Comparison with Previous Studies*

The results were consistent after adjustment for sequence, weight change, and washout period effects. Correlation analysis showed that HbA1c reduction was related to both increased fat oxidation ($r = -0.41$, $p = 0.003$) and higher TEF ($r = -0.37$, $p = 0.007$). Compared with earlier studies using single-diet designs, the crossover model in this work minimized between-subject variation and allowed a direct within-person comparison. Previous ketogenic studies have reported lower RER and greater fat oxidation but lacked controlled thermogenic measurements [23]. In contrast, the present data confirm that fiber can raise post-meal thermogenesis without reducing fat oxidation. The effect size, however, depends on fiber type, fermentation rate, and baseline microbiota composition [24]. Longer interventions with tracer-based energy tracking are needed to confirm these short-term metabolic effects and determine their long-term benefits [25].

## 4. Conclusion

This study found that ketogenic and high-fiber diets affect energy metabolism through different but complementary ways in adults with obesity and type 2 diabetes. The ketogenic diet raised fat oxidation from 32% to 61%, while the high-fiber diet increased the thermic effect of food from 6.8% to 9.1%. When both diets were used together, the drop in HbA1c was the greatest (−1.6%), showing stronger control of blood sugar. These findings show that combining low-carbohydrate and high-fiber eating plans can help improve both energy use and glucose regulation more effectively than either approach alone. The results provide a practical direction for dietary therapy targeting both fat metabolism and thermogenesis in metabolic disease. However, this study was limited by its small sample size and short duration. Future research with longer observation and more diverse fiber sources is needed to confirm these benefits and explore how they influence long-term metabolic health.

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