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The Effects of Periodized Traditional and Circuit-Based Resistance Training on Branched-Chain and Aromatic Amino Acid Metabolism and Ceramide Levels in Overweight and Obese Men

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A B S T R A C T

Objective: Amino acids (AAs) and their metabolites are altered with obesity and recognized as one of the predisposing factors for the development of insulin resistance. This study aimed to examine the impact of two types of resistance training on AAs, ceramide metabolism, and insulin resistance in overweight and obese men.

Methods and Materials: A total of 33 overweight and obese men were randomly divided into three control, circuit resistance training (CRT) and traditional resistance training (TRT) groups and the number of subjects in each group was 11 participants. The Training intervention consisted of periodized TRT and CRT training with the wave patterns and was conducted three sessions a week for three months (36 sessions). Serum levels of AAs and ceramides were measured using high-performance liquid chromatography (HPLC) at baseline and post-intervention.

Findings: The CRT group showed a notable reduction in total branched-chain amino acids (BCAAs), aromatic amino acids (AAAs), and ceramides compared to the control group (P = 0.001), indicating a meaningful metabolic improvement. Similarly, the TRT group exhibited moderate decreases in total BCAAs (P = 0.006) and AAAs (P = 0.017) relative to controls, changes that are inversely associated with insulin sensitivity. However, the differences between the CRT and TRT groups did not reach statistical significance, suggesting comparable effects between these two training protocols.

Conclusion: According to the results, the CRT group's changes were more significant than the TRT group. Therefore, circuit resistance training may prevent obesity-induced metabolic disorders.

Keywords: *Amino acids, ceramide, insulin resistance, and periodized resistance training*

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1. Introduction

Obesity represents a major global health concern, with profound implications for morbidity, mortality, and healthcare systems worldwide. Excess adiposity, particularly its distribution across visceral depots, is closely linked to the heightened risk of developing metabolic disturbances, including insulin resistance (IR) and type 2 diabetes mellitus (T2D) (1). The early detection of metabolic biomarkers associated with these disorders may offer critical avenues for the prevention and management of obesity-related complications (2).

Insulin resistance, a hallmark of metabolic syndrome and a pivotal contributor to T2D pathogenesis, manifests as a diminished biological response to insulin, impairing glucose uptake and utilization. Mounting evidence implicates perturbations in amino acid and lipid metabolism—particularly involving branched-chain amino acids (BCAAs) and aromatic amino acids (AAAs)—in the etiology of obesity-associated IR (3, 4). Elevated circulating levels of BCAAs and their catabolic intermediates have been consistently reported in obese individuals and are predictive of future T2D independent of traditional risk factors such as body mass index (BMI) and fasting glucose (5, 6).

Although BCAAs are essential for muscle protein synthesis, glucose uptake, and energy provision (7). their metabolic dysregulation—particularly the accumulation of toxic intermediates due to impaired oxidation—may contribute to systemic insulin resistance (8). Furthermore, obesity-induced mitochondrial dysfunction may exacerbate incomplete fatty acid oxidation, thereby influencing amino acid catabolic pathways (9).

In parallel, dysregulated lipid metabolism in obesity fosters the accumulation of bioactive sphingolipids, notably ceramides, which are synthesized from saturated fatty acids such as palmitate. Ceramides act as critical signaling molecules that modulate cellular processes including apoptosis, inflammation, and insulin signal transduction (10, 11). Elevated ceramide levels in skeletal muscle and plasma have been robustly associated with impaired insulin sensitivity and heightened cardiometabolic risk(12, 13).

Collectively, these findings underscore the need for interventions targeting amino acid and lipid metabolic pathways to mitigate obesity-related metabolic dysfunctions (14, 15). Exercise training, particularly resistance training, has emerged as an effective strategy to enhance mitochondrial oxidative capacity, modulate substrate metabolism, and improve insulin action (16). The rationale for comparing traditional and circuit resistance training protocols in metabolic assessment is to evaluate their differing effects on exercise intensity, energy expenditure, and muscle recovery, which lead to distinct changes in body composition and metabolic performance. However, the effects of different resistance training modalities on simultaneous alterations in circulating BCAAs, AAAs, and ceramides remain inadequately explored.

Regarding amino acid levels during or after exercise, studies have shown varying results, including no change, a decrease, or an increase in their concentrations (5, 6, 17–19). In the study of Deberzan and Koriski (2001), Helg et al. (2004), Bruce et al. (2006), Dubai et al. (2011), and Bergman et al. (2016) by examining endurance exercises on ceramide content in skeletal muscle and human plasma and Rats reported total ceramide levels and glucose tolerance improved significantly (17-21). Rivas et al. (2012) did not observe significant changes in ceramide content by examining ceramide content and anabolic cycling after high-intensity acute resistance training (10 repetitions and 80% 1RM) with standard meals in obese young and elderly people (22). Sogard et al. (2019) investigated the effect of HIIT training on diacylglycerol and ceramide and reported that ceramide content decreased in both young and old overweight and obese groups and improved metabolism at the level of glucose (23).

Thus, the present study seeks to address these knowledge gaps by evaluating the comparative effects of periodized traditional and circuit-based resistance training on BCAA, AAA, and ceramide metabolism, as well as insulin resistance, in overweight and obese young men. By integrating a comprehensive biochemical analysis with structured exercise interventions, this study aims to provide novel insights into the optimization of exercise prescriptions for metabolic health improvement.

2. Methods and Materials

2.1 Study Design and Participants

This applied, quasi-experimental study employed a randomization using a computer-generated randomization sequence, controlled, pretest-posttest design to investigate the effects of two distinct resistance training modalities—periodized traditional resistance training (TRT) and circuit resistance training (CRT)—on branched-chain amino acids (BCAAs), aromatic amino acids (AAAs), ceramide metabolism, and insulin resistance in overweight and obese young men.

The study recruited sedentary men aged 17–21 years from the local community, who had not participated in regular physical activities in the six months preceding the study. Interested participants were invited through public announcements and asked to complete a questionnaire detailing their medical history, demographic characteristics, and physical activity background.

A total of 33 participants were randomly assigned to one of three groups: control, circuit resistance training (CRT), and traditional resistance training (TRT). The inclusion criteria were: (1) age between 17–21 years, (2) no regular participation in sports or exercise within the last six months, (3) absence of developmental or motor disorders, (4) no cardiovascular diseases, and (5) male gender Figure 1. Exclusion criteria included unwillingness to continue participation, engagement in other training programs during the study, or missing more than one session during the intervention period. Furthermore, participants were instructed to consume the same meal with identical composition and portion size as they did before the pretest, the evening before each blood sampling session, to ensure consistency between pretest and posttest conditions and minimize variability in fasting metabolic measurements (24).

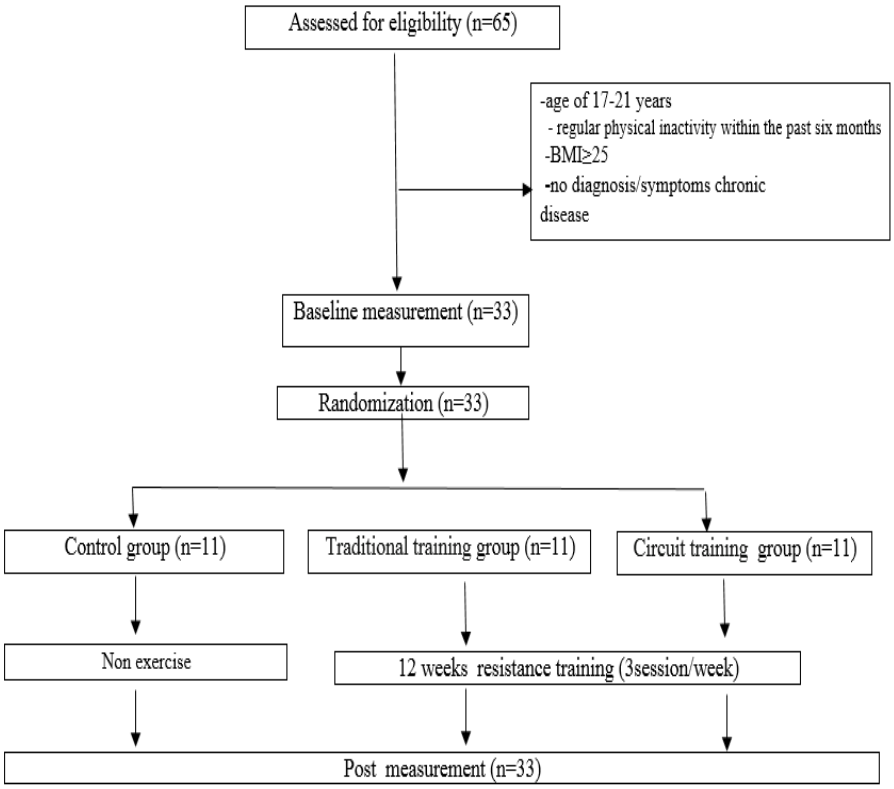

Figure 1. Flow diagram of the progress

2.2 Measures

Prior to the intervention, anthropometric measurements, including height, weight, body fat percentage, body mass index (BMI), and lean body mass, were collected. Strength assessments were performed to determine the one-repetition maximum (1RM) for key resistance exercises, establishing individualized training intensities. The 1RM tests were repeated at the beginning of each mesocycle to adjust exercise loads and adhere to the principle of progressive overload.

2.3 Intervention

The intervention involved periodized CRT and TRT programs incorporating wave patterns, conducted three times a week for 12 weeks. Training intensity ranged from 50–80% of 1RM and included 3 sets per exercise, with 8–24 repetitions and 8–14 exercises per session (Figure 2).

Exercises included the leg press, bench press, step-ups, incline bench press, lat pulldown, crunches, lunges, shoulder press, leg curls, seated row, leg extensions, reverse fly, knee-hip raises on parallel bars, and barbell curls. The intensity of each session was carefully selected based on multiple sources to ensure appropriate metabolic pressure and maximize the metabolic effects of the training. The exercises were performed at an intensity optimized for maximum effectiveness(17, 25).

It was critical to take the necessary precautions in increasing the intensity of the exercises for the overweight and obese participants during the preparatory stage so that they would have enough time to recuperate. In the third week, a "shock stage" was introduced by increasing the intensity and variety of training movements per microcycle to prevent physiological adaptation to monotonous training stimuli. However, the volume of the exercises was not manipulated in the fourth week to reduce injuries and allow recovery in the participants

While the two protocols in the present study were completely similar in terms of the training volume, they differed in terms of implementation and rest time. There was no rest between circuit exercises. The rest time was the shortest period between the changes of movement. Nevertheless, we considered three minutes of rest between each circuit. In contrast, the traditional resistance training (TRT) protocol included 90 seconds of rest between sets and three minutes between different exercises.

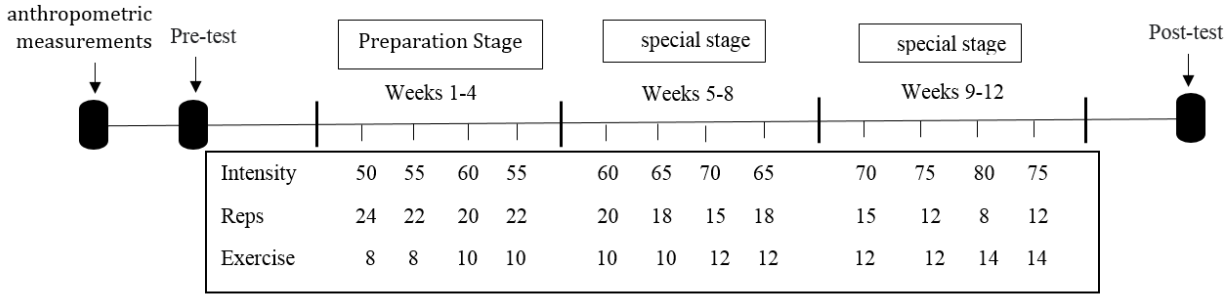

Figure 2. Resistance training program

Blood samples were collected from participants twice: once before the start of the exercise protocol and again at the end of the intervention. A total of 8 milliliters of blood was drawn from the antecubital vein using venipuncture, performed by a trained phlebotomist while participants were seated. The samples were collected in EDTA tubes, stored at -80°C, transported to the laboratory, and centrifuged at 3,000 rpm (1,000 g) for 15 minutes at room temperature. The biochemical variables were analyzed using the high-performance liquid chromatography (HPLC) method (26). Serum insulin levels were measured using the ELISA method, following the instructions provided by the Monobond kit. Blood glucose levels were determined using the glucose oxidase method. Fasting insulin (microunits per milliliter) and fasting glucose (milimoles per liter) / 22.5 were used to calculate the insulin resistance index (HOMA-IR) (27, 28).

2.4 Data Analysis

Data analysis included descriptive statistics, such as mean and standard deviation, and inferential statistics. The Shapiro-Wilk test was used to evaluate the normal distribution of the data. To analyze the measurement stages in each group separately, we performed repeated -measures analysis of variance with an intergroup factor test (for differences between training and control groups).

3. Results

Table 1 shows the descriptive statistics of the findings. The results of the Shapiro-Wilk test indicated that the data had a normal distribution. After 12 weeks, the subjects in the circuit and traditional training groups ($P < 0.001$) were aligned with the pretest phase, experiencing significant weight loss ($P < 0.001$ and $P = 0.001$) and a significant reduction in BMI ($P < 0.001$), respectively. Furthermore, the intergroup and intragroup changes in the traditional resistance training group before and after the intervention included an increase in HDL ($P = 0.01$) and a significant decrease in LDL ($P = 0.001$), cholesterol ($P = 0.007$), glucose ($P < 0.001$), and valine ($P = 0.004$).

In the circuit resistance training group, an increase was observed in HDL ($P < 0.001$) and a reduction was observed in glucose ($P < 0.001$), LDL ($P = 0.004$), TG ($P = 0.01$), and isoleucine ($P < 0.001$). Moreover, significant differences

were observed in the levels of valine ($P < 0.001$), phenylalanine ($P < 0.001$), tyrosine ($P < 0.001$), and tryptophan ($P < 0.001$) in this group.

According to the results of ANOVA, levels of isoleucine ($P = 0.005$) and valine ($P = 0.005$) differed notably among the circuit resistance training, control, and traditional training groups. Specifically, isoleucine levels showed a modest but statistically meaningful change in the control group ($P = 0.038$), whereas no meaningful changes were observed in the traditional resistance training group. These findings suggest differential effects of the interventions on specific branched-chain amino acids.

Table 1. Subject characteristics.

	Control		Circuit training		Traditional training	
	Pre	Post	Pre	Post	Pre	Post
n	11	11	11	11	11	11
Age (years)	17.7±0.88	-	17.9±0.78	-	17.65±0.6	-
Height (cm)	173.45±5.02	-	174.63±5.4	-	174.54±5.64	-
Weight (kg)	91.38±17.4	91.87±17.2	93.41±10.84	91.83±11.50*	90.74±13.47	89.38±14.26*
BMI (kg/m2)	30.41±4.6	30.53±4.7	30.55±4	30.07±4.1*	30.7±4.3	30.33±4.5*
Fasting glucose (mg/dl)	94.18±9.4	90.27±5.7	96.09±6.2	88±6*	100.18±9.7	89.64±3.7*
Fasting insulin (IU/mL)	5.17±1.3	5.98±1.9	5.32±2.3	4.5±0.6	5.9±1.3	5.07±1.2
HDL-C (mg/dL)	34.64±4.4	37.27±4.7	36.36±3.2	42.71±6.1*	38.27±7.4	42.27±6.7*
LDL-C (mg/dL)	93±18.7	87.7±21.9	118.1±29.7	104±27.1*	110±22.3	92.7±23*
TG (mg/dL)	98.18±66.2	104.54±36	121.7±43.8	91.6±13.1*	111.3±35.6	94.91±20
Cholesterol (mg/dL)	143.3±25.8	147±24.38	175.82±31.5	169.27±38.1	170.73±30.6	154±24.4*
Leucine (nmol/mL)	233.18±45.9	236.6±29.1	228.1±43.3	212.2±47.2	209.33±43.1	206.7±40.8
Isoleucine (nmol/mL)	181.2±49.8	161±59	162.8±31.2	73.9±18.8*†	142.6±38	119.7±53.1†
Valine (nmol/mL)	232.8±20.7	227.2±37.7	223.2±22.9	166±52.1*†	234.1±33.6	186.9±30.4*
Phenylalanine (nmol/mL)	55.8±4.2	59.8±8.7	61.7±9.7	51±1.7*	62.8±8.6	57.4±8
Tyrosine (nmol/mL)	58.6±9	54.2±3.7	59.8±9.4	50±3.6*	59.6±10.7	55.9±7.2
Tryptophan (nmol/mL)	102.3±22.7	95±29.3	100.9±37.6	66.8±13.9*	94.9±27.3	78.7±17.1
Histidine	59±12.6	58.27±10	64.2±9.6	60.45±9.6	61.73±5.6	62±10.5

BMI = body mass index; HOMA-IR = homeostasis model assessment of insulin resistance: LDL: low-density lipoprotein; HDL: high-density lipoprotein; * Significant difference compared to pre-test; † Significant difference compared to control group.

After 12 weeks, serum ceramides in the circuit resistance training group decreased markedly ($P < 0.001$), accompanied by substantial reductions in total BCAAs ($P < 0.001$), total amino acids (AAs) ($P < 0.001$), and insulin resistance, indicating meaningful metabolic improvements. In the traditional training group, moderate decreases were observed in total BCAAs ($P = 0.006$) (Figure 3a), total AAs ($P = 0.017$) (Figure 3b), and insulin resistance ($P = 0.005$) (Figure 3c). However, ceramide levels remained largely unchanged in this group ($P = 0.050$) (Figure 3d), suggesting a differential effect of the training modalities on lipid metabolism.

According to the results of ANOVA, total BCAAs ($P = 0.001$) were significant in the circuit resistance training group and the control group, as well as between the traditional training group and the control group ($P = 0.005$).

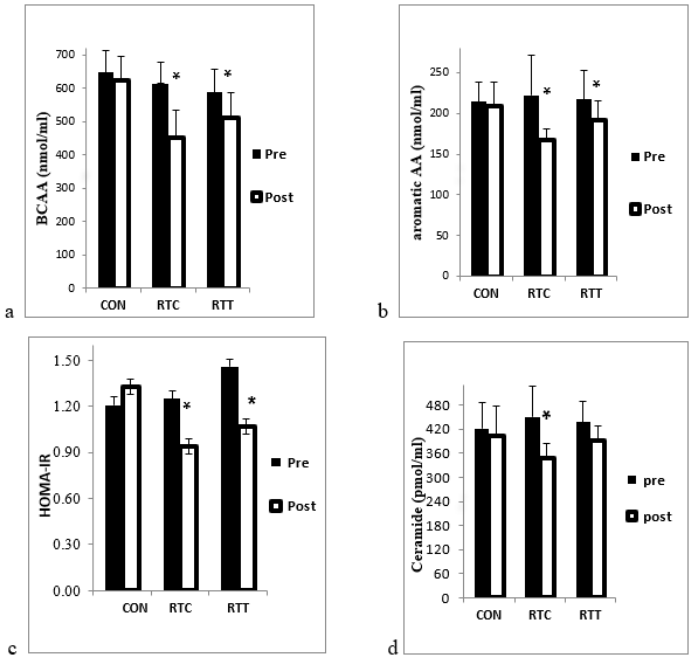

Figure 3. Changes in total BCAA (including leucine, isoleucine, and valine) (a), total AAA (including phenylalanine, tryptophan, and tyrosine) (b), HOMA-IR (C), and ceramide levels before and after exercise training.

4. Discussion and Conclusion

This study aimed to investigate the effects of two resistance training modalities—circuit resistance training (CRT) and traditional resistance training (TRT)—on amino acid metabolism, lipid profiles, ceramides, and insulin resistance in obese young men. Both training interventions led to significant reductions in insulin resistance, with CRT and TRT decreasing HOMA-IR scores by 23% and 26.7%, respectively. These improvements align with previous findings demonstrating that resistance training enhances insulin sensitivity and modifies lipid metabolism in obese adolescents (29, 30).

Furthermore, both CRT and TRT elicited comparable reductions in LDL, triglycerides, and total cholesterol, reflecting enhanced lipid profiles post-training. Such improvements are consistent with evidence suggesting that resistance training contributes to favorable cardiometabolic outcomes through mechanisms including improved lipid oxidation and increased GLUT-4 translocation (31).

Notably, body mass and body fat percentage significantly decreased following both exercise protocols, indicating that reductions in adiposity may partly mediate improvements in insulin sensitivity. However, conflicting results in the literature regarding the effects of resistance training on insulin resistance (6, 32, 33) suggest that variations in exercise type, intensity, volume, and participant characteristics could account for discrepancies.

Importantly, the present study observed a trend toward greater reduction in total serum BCAAs and AAAs following CRT compared to TRT, with CRT showing nearly twice the decrease in BCAA and AAA concentrations; however, not all comparisons reached statistical significance. This finding is generally consistent with prior research suggesting that increased catabolism of BCAAs may enhance insulin sensitivity and support mitochondrial health (33, 34).

Nevertheless, other studies have reported divergent outcomes. For instance, Lee et al. (33) demonstrated that 12 weeks of combined endurance and resistance training did not significantly alter plasma BCAA concentrations despite notable improvements in insulin sensitivity.

The bidirectional relationship between BCAA metabolism and insulin sensitivity highlights the complexity of these interactions. Enhanced BCAA catabolism may promote insulin sensitivity, while conversely, improved insulin sensitivity may facilitate BCAA clearance (33, 35).

Additionally, the reduction in serum ceramides following CRT was more pronounced compared to TRT, although the between-group difference was not statistically significant.

This observation is important, considering that ceramides are implicated in impairing insulin signaling and promoting metabolic inflexibility (12, 36). Decreased ceramide levels likely reflect improved mitochondrial fatty acid oxidation and reduced lipotoxicity following resistance training (21, 23).

Increased mitochondrial biogenesis and enhanced beta-oxidation capacity in skeletal muscles, as observed in response to CRT, may underlie the greater ceramide reduction (38). Consequently, these adaptations may alleviate the inhibitory effects of ceramides on amino acid transporters such as SNAT-2, thereby facilitating amino acid uptake and utilization (37).

Previous investigations have shown that resistance and aerobic training both decrease skeletal muscle ceramide content and improve glucose metabolism (21, 23). Our findings further suggest that CRT, with its higher metabolic demand and reduced rest periods, may stimulate greater mitochondrial adaptations, leading to more substantial reductions in ceramides and related lipotoxic intermediates.

Interestingly, decreased ceramide levels following CRT may suggest a potential attenuation of inhibitory effects on amino acid transporters. Previous studies have proposed that ceramides could downregulate SNAT-2, thereby impairing amino acid uptake (37). If supported by future research, the observed reduction in ceramides in our study might hypothetically enhance amino acid transport into skeletal muscle, which could promote protein synthesis and metabolic control. However, this mechanistic link remains speculative, as our study did not directly measure SNAT-2 activity or amino acid transporter function.

Furthermore, the present study supports the hypothesis that resistance training enhances systemic BCAA catabolism by improving mitochondrial function. Enhanced BCAA oxidative potential reduces the accumulation of toxic catabolic intermediates that have been implicated in insulin resistance (35). This mechanism could also explain why CRT was associated with greater reductions in BCAA concentrations and greater improvements in insulin sensitivity compared to TRT.

Overall, CRT appears to confer greater metabolic benefits compared to TRT, possibly due to higher cumulative energy expenditure, shorter rest intervals, and greater engagement of oxidative pathways. These findings provide valuable insights into the optimization of resistance training for improving metabolic health in obese individuals.

Collectively, these adaptations highlight the importance of mitochondrial health and substrate metabolism regulation in mediating the beneficial effects of resistance training on metabolic dysfunction in in obese men after 12 weeks. The integration of circuit-based resistance protocols may therefore represent a more effective strategy for targeting amino acid and lipid dysregulation in obese populations.

Authors' Contributions

M. C. and R. F. contributed equally to the study, with M. C. taking the lead on research design. M. C, R. F, R.A, S.A, and A.KH assisted in data collection, which included anthropometric measurements and body composition analysis. M. C and R. F were also responsible for conducting the statistical analysis using SPSS and contributed to the interpretation of the data. Additionally, M. C. was involved in writing and reviewing the manuscript and provided approval for its final version.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

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Declaration of Interest

The authors report no conflict of interest.

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Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants. The study protocol was reviewed and approved by the Ethics Committee of Mazandaran University (IR.UMZ.REC.1399.017) and registered with the Iranian

Clinical Trial Center (IRCT) under the code IRCT20180928041160N2.

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