ORIGINAL RESEARCH

Akkermansia muciniphila 001 (AKK001™) Postbiotic and Butyrate (Tributyrin) Impact on Body Morphology Measures and Metabolic Indicators in an Overweight Population: A Randomized, Controlled Trial

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ABSTRACT

Background: Globally, overweight and obesity are a rising health challenge, with their global costs predicted to reach $3 trillion per year by 2030 and increase further. Interestingly, studies have reported that pasteurized *Akkermansia muciniphila* (AKK) can promote weight loss and improve metabolic parameters.

Primary Study Objective: To investigate the impact of pasteurized AKK001 and tributyrin supplementation on overweight individuals.

Methods/Design: Retrospective analysis of data derived from a 90-day randomized, double-blind, placebo-controlled clinical intervention.

Setting: The study site was Sir Run Run Shaw Hospital, School of Medicine, Zhejiang University, Bangzhou, China, from April to September 2025.

Participants: Sixty overweight or obese (BMI≥28kg/m^2) but otherwise healthy participants; intervention group (N=30; 16 males, 14 females; age: 35.2 ± 5.8 years), control group (N=30; 15 males, 15 females; age: 34.8 ± 6.1 years).

Intervention: Random assignment to placebo group or intervention group, with the latter consuming one capsule daily containing 1.5 × 10^{10} TFU pasteurized AKK001 postbiotic (AKK001™) and 150 mg of butyrate as tributyrin.

Outcome Measures: Primary outcomes: Differences in body morphology (weight, body fat percentage, BMI, waist circumference, hip circumference, basal metabolic rate, and ultrasound subabdominal/subcutaneous/visceral fat thickness). Secondary outcomes: metabolic indicators (fasting blood glucose, four kinds of blood lipids (total cholesterol, triglycerides, high-density lipoprotein, and low-density lipoprotein, and insulin level). Tertiary outcomes: liver and kidney function and thyroid function (alanine aminotransferase, aspartate aminotransferase, and serum creatinine), and thyroid function indicators (thyroid-stimulating hormone and free thyroxine).

Results: The intervention group showed statistically significant improvements compared to the control group after 1 and 3 months in primary (P < .001-.05 and <.001, respectively) and secondary outcomes (P < .001-.011 and <.001, respectively). Tertiary outcomes were not significantly different compared to the control group after 1 or 3 months.

Conclusion: Findings suggest that the pasteurized AKK001™/ tributyrin combination may be a viable strategy for weight management. (*Altern Ther Health Med.* [E-pub ahead of print.])

Keywords: Obesity, pasteurized *Akkermansia muciniphila* postbiotic, tributyrin, body fat percentage, abdominal subcutaneous fat, total cholesterol

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INTRODUCTION

Globally, 43% of adults aged 18 years and above were found to be overweight and 16% obese in 2022,[1] with projections of overweight prevalence reaching 43.97% and obesity reaching 38.96% by 2040.[2] In 2021, higher-than-optimal body mass index (BMI) caused an estimated 3.7 million deaths from noncommunicable diseases including cardiovascular diseases, diabetes, cancers, neurological disorders, chronic respiratory diseases, and digestive disorders.[3] The global costs of overweight and obesity are predicted to reach $3 trillion per year by 2030 and more than $18 trillion by 2060.[4] In consideration of these facts, it is evident that safe and effective methods to promote weight loss are needed.[1]

One potential method involves using *Akkermansia muciniphila* (AKK) as a postbiotic, and butyrate as tributyrin. AKK is a human intestinal microorganism (aka, a probiotic). It colonizes the gastrointestinal tract of humans and other animals, and can be found within the large intestinal mucosal layer of intestinal epithelial cells as well as in the caecum.[5]

AKK influences the integrity of the gut-brain axis[8] and is associated with less metabolic disease[9-12]. Previous studies have shown that the postbiotic form of AKK (i.e., a probiotic that has been pasteurized or heat-treated, yet still provides the previously cited health benefits) promotes weight loss, as discussed below.

Butyrate (more precisely, butyric acid/butanoate) is a short-chain fatty acid (SCFA). In humans, butyrate is produced largely by gut bacteria in the colon, through microbial fermentation of dietary fiber and other non-digested carbohydrates.[6] Once it is produced in the colon, butyrate is absorbed by the cells lining the colon (colonocytes), where it serves as a key energy substrate and is metabolized (via β-oxidation/mitochondrial oxidation) to generate ATP.[7] Tributyrin is a stable, slower-release form of butyrate.

In three previous randomized controlled trials[13,14,15] involving overweight and obese participants, 10[10] of pasteurized AKK improved metabolic health parameters and decreased body weight compared to placebo and/or positive control over 8 weeks to 3 months.

It should be noted that Amuc_1100 is a key outer-membrane protein of AKK, which interacts with Toll-like receptor 2 (TLR2) on host intestinal epithelial cells and helps improve gut barrier function.[16,17] *In vivo* research has shown that AKK supplementation increases expression of mucin genes (e.g., *Muc1, Muc5, Muc13*) and boosts goblet cell density.[18] Consequently, a lack of AKK is associated with low goblet cell production.[19] Additionally, preclinical evidence shows that Amuc_1100 promotes lipolysis and "browning" of white adipocytes via the AC3/PKA/HSL pathway. This suggests Amuc_1100 could be a molecular effector by which AKK regulates body fat and energy metabolism.[20]

YouThorin (a biotechnology research organization) has an AKK001 strain derived from a group of centenarians in Rugao, China, which has 134 centenarians per 1 million people, far exceeding the longevity standard (75 centenarians per 1 million people) prescribed by the United Nations.[21] YouThorin conducted an internal (unpublished) *in vitro* study to investigate the expression of Amuc-1100 secreted by their live AKK001 strain compared to the model species AKK-BAA835, via quantitative real-time PCR (qPCR). Results showed that protein expression of AKK001 was 3.3 times that of the model species.

The relevance of this to pasteurized AKK is research demonstrating that Amuc_1100 "is stable at temperatures used for pasteurization" and that pasteurized AKK retains beneficial effects.[16] Wang et al.[22] directly compared pasteurized AKK and purified Amuc_1100 *in vivo* and reported that both pasteurized bacteria and the Amuc_1100 protein show protective effects, consistent with Amuc_1100 being present/functional after pasteurization.

The colon's epithelial cells preferentially use butyrate (over glucose or amino acids), making butyrate central for colon health and epithelial maintenance,[7] reducing pathogen translocation, supporting mucus and tight-junction maintenance,[23] and supporting immune regulation.[24] In addition, while colonocytes metabolize much of butyrate locally, a portion can enter the portal circulation and reach the liver (and possibly beyond), influencing liver metabolism (fatty acid metabolism, lipogenesis, and glucose regulation), and contributing to systemic metabolic regulation.[25]

Since obesity is linked with altered microbial SCFAs, which are a signature of gut dysbiosis and inflammation, a study[26] was conducted to investigate whether tributyrin, a stable, slower-release form of butyrate, could improve metabolic and inflammatory profiles in diet-induced obese mice. Results showed that obese mice treated with tributyrin had lower body weight gain and improved insulin responsiveness and glucose metabolism, partly via reduced hepatic triglyceride content. Additionally, tributyrin induced an anti-inflammatory state in adipose tissue by reducing *Il-1β* and *Tnf-a* and increasing *Il-10*, Tregs cells, and M2-macrophages.

An *in vitro* study[27] in human visceral adipose tissue demonstrated that tributyrin effectively reduced the production of lipopolysaccharide-induced inflammatory cytokines and chemokines, suggesting that tributyrin might alleviate the chronic inflammation associated with obesity. Additionally, a human randomized, single-blinded, two-arm, 28-day parallel design pilot study[28] was conducted in 24 adults with two dose levels of tributyrin, 200 and 400 mg, using questionnaires, blood, stool, and urine samples to evaluate the effect of tributyrin on tolerability. Results showed that tributyrin was well-tolerated, with reduced fecal acetic ($P = .03$) and propionic ($P = .03$) acids, compared to baseline. Trends also showed reduced high-sensitivity C-reactive protein (hs-CRP) after 200 mg ($P = .08$) and 400 mg ($P = .07$), and glucose ($P = .10$) after 200 mg supplementation.

None of the aforementioned human clinical studies included a combination of pasteurized AKK and tributyrin. Considering the previously cited data, this study aimed to investigate the impact of pasteurized AKK001 and tributyrin supplementation on overweight human participants.

METHODS

Study Design and Participants

This study was conducted as a randomized, double-blind, placebo-controlled dietary intervention at Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, from which the participants were recruited. The present manuscript reports a retrospective analysis of data generated during this intervention, which was not registered as a prospective clinical trial under Chinese regulatory requirements since researchers did not originally intend to publish. Ethical approval was obtained before participant enrollment, and all participants provided written informed consent. The study was conducted from April to September 2025, with screening of 68 eligible overweight and obese volunteers. Participant inclusion and exclusion criteria are detailed in Table 1. After excluding 8 cases (3 due to personal reasons, 3 non-willingness to be compliant, and 2 with contraindicated medications), 60 participants were included and randomly assigned to an

Table 1. Inclusion/Exclusion Criteria

Main inclusion criteria:
• Age 25-45 years • BMI 25-34.9 • Subject agreed to maintain a regular diet (1800-2000 kcal, carbohydrate, protein, and fat energy ratio of 55%, 20%, and 25%, respectively). • Subject agreed to refrain from consuming other probiotics or weight-loss medications. • Subject agreed to maintain their existing dietary patterns throughout the study period. • Subject was willing and able to comply with the study protocol. • Subject gave voluntary, written, informed consent to participate in the study.
Main exclusion criteria:
• Individuals who were determined to have liver, renal, cardiovascular, or other metabolic disease. • Use of any dietary supplements or medications impacting weight loss or cardiometabolic parameters which may confound the study or its endpoints. • Clinically significant abnormal laboratory results (i.e., outside of the normally accepted range for blood panels) at screening. • Allergy or sensitivity to study product ingredients. • Individuals who were cognitively impaired and/or unable to give informed consent. • Any other condition which, in the primary investigator's opinion, might adversely affect the subject's ability to complete the study, its measurements, or would pose significant risk to the subject.

intervention group (n=30) and a placebo group (n=30). Both groups maintained complete data with no dropouts. All labs were performed internally at the hospital.

The primary outcomes were the morphological measures, weight, body fat percentage, BMI, waist circumference, hip circumference, and abdominal subcutaneous fat thickness (2 cm above, below, and at the umbilical level), as well as abdominal visceral fat thickness (2 cm above, below, and at the umbilical level).

The secondary outcomes were metabolic biomarkers of cardiovascular health, including glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low- density lipoprotein cholesterol (LDL-C), and insulin.

For safety considerations, the tertiary outcomes were liver, kidney, and thyroid function parameters, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin, creatinine and urea nitrogen, thyroid-stimulating hormone, free triiodothyronine and free thyroxine.

Both groups were monitored via weekly telephone follow-up and monthly on-site review to ensure that the subjects maintained regular diet and physical activity levels without other probiotics or weight-loss drugs. Dietary compliance rate was ≥88% in both groups.

Detection Methods and Quality Control

Testing equipment and reagents: Height measuring tape (accuracy 0.1 cm), electronic scale (accuracy 0.1 kg), InBody 770 bioelectrical impedance analyzer (measures basal metabolic rate simultaneously as an estimated value, accuracy 5 kcal/day), color Doppler ultrasound system (GE Logiq E9 with probe frequency 3.5-5.0 MHz), fully automated biochemical analyzer (Roche Cobas c702), and chemiluminescent immunoassay system (Abbott i2000SR). All machines were calibrated regularly, and standard protocols and measurement techniques were used. All reagents were original manufacturer-provided and used within their expiration dates.

Study Setting and Intervention

The study site was Sir Run Run Shaw Hospital, School of Medicine, Zhejiang University, Bangzhou, China. Participants were randomly assigned to a placebo group or intervention group, with one capsule daily of AKKBoom, containing 1.5×10^{10} TFU pasteurized *Akkermansia muciniphila* 001 postbiotic (AKK001™) and 150 mg of butyrate as tributyrin microencapsulated powder. The capsule also included microcrystalline cellulose, beta-cyclodextrin, silica, and magnesium stearate as inactive excipients. The placebo contained only the same inactive excipients and was indistinguishable by sight or smell.

Ethical Approval

In accordance with local regulatory requirements, this study was documented as a retrospective clinical investigation and therefore was not prospectively registered in a public clinical trial registry. The study was approved by the Shao Yifu Hospital Ethics Committee of Zhejiang University School of Medicine (Approval No.: 2025-2179-01). All participants signed informed consent forms, and throughout its duration, the study strictly adhered to the ethical principles of the 2013 Helsinki Declaration.

Data Analysis

Statistical analysis was performed using SPSS 26.0 software. Numerical data are presented as "mean ± standard deviation (x±s)", with independent samples *t* tests used for inter-group comparisons and paired *t* tests for intra-group intervention comparisons. Categorical data were expressed as "count (%)", with inter-group comparisons conducted using χ^2 chi-square tests. All statistical analyses were double-sided, with significance level set at $P < .05$.

RESULTS

The study was completed with 60 subjects. Baseline characteristics showed no significant differences in age or gender distribution between the intervention and placebo groups ($P \geq .05$), Table 2. The intervention group had an average age of 35.2 ± 5.8 years with a male-to-female ratio of 16:14, while the control group averaged 34.8 ±6.1 years with a male-to-female ratio of 15:15.

Primary Outcomes

For the primary outcomes, Table 3 provides body morphology measures at baseline, month 1, and month 3, while Table 4 provides abdominal fat measures at baseline, month 1, and month 3.

After one month, the intervention group showed statistically significant differences compared to the control group in weight, body fat percentage, BMI, waist circumference, hip circumference, abdominal subcutaneous fat thickness (2 cm above, below, and at the umbilical level), as well as

Table 2. Baseline Comparison of Age and Gender Distribution

Group	Number of Cases	Age (years)	Gender (male/female)
Intervention group	30	35.2±5.8	16/14
Placebo group	30	34.8±6.1	15/15

Table 3. Comparison of Body Morphology Measures

Variable	Group	Baseline	Month 1	Month 3	*p*-value (Month 1/Month 3)[a]
Height (cm)	Intervention	168.5±7.2	168.5±7.2	168.5±7.2	Not Applicable
	Placebo	167.8±6.9	167.8±6.9	167.8±6.9	
Weight (kg)	Intervention	82.9±7.9	79.8±7.9	75.3± 8.1	.019/.001
	Placebo	83.2±7.7	83.2±7.7	82.5±7.8	
Body fat %	Intervention	30.2±3.5	28.1±3.3	25.6±3.2	.027/.001
	Placebo	30.5±3.4	30.5±3.4	29.8±3.5	
BMI (kg/m^2)	Intervention	29.7±2.1	28.2±2. 4	26.5±2. 3	.007/.001
	Placebo	29.5±2.5	29.5±2. 5	29. 1±2. 5	
Waistline (cm)	Intervention	93.7±5.9	89.5±5.8	85.2±5.6	.05/.001
	Placebo	93.8±6.0	93.8±6.0	92.5± 6.1	
Hip (cm)	Intervention	97.6±5.2	95.6±5. 0	92.3±4. 8	.001/.001
	Placebo	97.8±5.1	97.8±5. 1	98.6±5. 2	

[a]Compared to placebo; all $P \leq .05$

Figure 1. Body Morphology Measures Difference Between Placebo & AKKBoom Groups

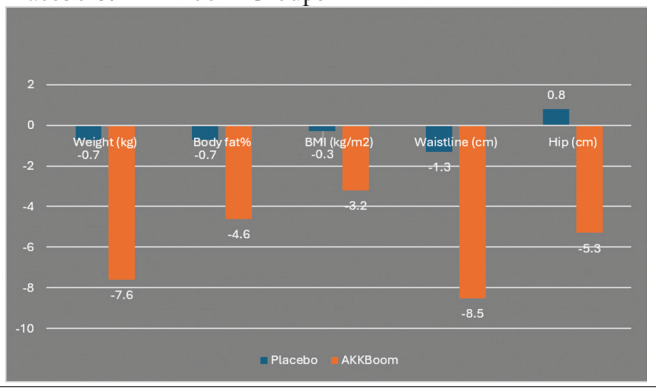

Table 4. Comparison of Abdominal Fat Measures

Variable	Group	Baseline	Month 1	Month 3	*P* value (Month 1/ Month 3)[a]
Subcutaneous abdominal Fat thickness (mm) – 2 cm above umbilicus	Intervention	34.5±4.4	32.1±4.3	28.5±4.2	.024/.001
	Placebo	34.8±4.4	34.8±4.4	35.2±4.5	
Subcutaneous abdominal Fat thickness (mm) – 2 cm below umbilicus	Intervention	32.5±3.9	29.5±3.9	26.3±3.8	.017/.001
	Placebo	32.2±4.0	32.2±4.0	32.8±4.1	
Subcutaneous abdominal Fat thickness (mm) – umbilical level	Intervention	32.2±4.1	30.2±4.1	27.1±4.0	.005/.001
	Placebo	32.8±4.2	32.8±4.2	33.5±4.3	
Abdominal visceral Fat thickness (mm) – 2 cm above umbilicus	Intervention	78.1±7.2	72.5±7.5	65.2±7.3	.048/.001
	Placebo	77.8±7.8	77.8±7.8	78.5±8.1	
Abdominal visceral Fat thickness (mm) – 2 cm below umbilicus	Intervention	72.4±7.8	68.8±7.0	62.5±6.8	.009/.001
	Placebo	72.1±7.3	72.1±7.3	75.2±7.6	
Abdominal visceral Fat thickness (mm) - umbilical level	Intervention	74.7±7.5	70.5±7.2	64.1±7.1	.016/.001
	Placebo	75.2±7.6	75.2±7.6	76.8±7.8	

[a]Compared to placebo; all $P \leq .05$

Figure 2. Abdominal Fat Measures Difference Between Placebo & AKKBoom Groups

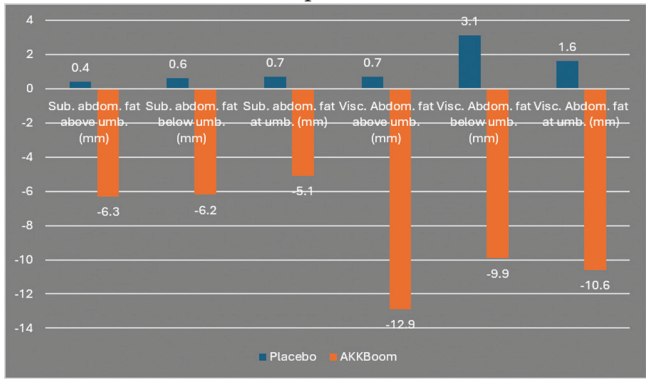

Table 5. Comparison of Metabolic Indicators

Variable	Group	Baseline	Month 1	Month 3	*P* value (Month 1/ Month 3)[a]
Glucose -mmol/L	Intervention	5.9±1.2	5.5±0.6	5.2±0.6	0.237/0.001
	Placebo	5.9±1.0	5.9±0.7	5.8±0.7	
Total Chol. - mmol/L (mg/dL)	Intervention	4.8±0.2 (185.6)	4.7±0.5 (181.7)	4.3±0.5 (166.3)	0.011/0.001
	Placebo	4.8±0.1 (185.6)	4.8±0.6 (185.6)	4.9±0.6 (189.5)	
Triglycerides - mmol/L (mg/dL)	Intervention	1.9±0.8 (168.3)	1.7±0.4 (150.6)	1.5±0. 4 (132.9)	0.024/0.001
	Placebo	2.0±0.1 (177.14)	2.0±0.5 (177.14)	1.9±0.5 (168.3)	
HDL-C - mmol/L (mg/dL)	Intervention	1.3±0.1 (50.3)	1.4±0.2 (54.1)	1.6±0.3 (61.9)	0.165/0.001
	Placebo	1.3±0.1 (50.3)	1.3±0.2 (50.3)	1.3±0.2 (50.3)	
LDL-C - mmol/L (mg/dL)	Intervention	2.9±0.6 (112.1)	2.9±0.4 (112.13)	2.6±0.4 (100.5)	0.006/0.001
	Placebo	2.9±0.3 (112.1)	3.0±0.5 (116.0)	3.1±0.5 (119.9)	
Insulin -mU/L	Intervention	16.8±2.9	14.8±3.0	12.5±2.8	0.003/0.001
	Placebo	16.2±2.7	17.2±3.1	16.8±3.2	
Basal metabolic rate - kcal/d	Intervention	1411.0±78.0	1485.0±85.0	1568.0±92.0	0.001/0.001
	Placebo	1410.0±80.0	1412.0±78.0	1455.0±86.0	

[a]Compared to placebo

abdominal visceral fat thickness (2 cm above, below, and at the umbilical level ($P < .001$-$.051$).

After 3 months, the intervention group showed significantly lower values of weight, body fat percentage, BMI, waist circumference, hip circumference, subcutaneous abdominal fat thickness (2 cm above, below, and at the umbilical level), and visceral abdominal fat thickness (2 cm above, below, and at the umbilical level) than the placebo group ($P < .001$).

Figure 1 shows the differences between the intervention group and the placebo group in body morphology measures, and Figure 2 does the same for abdominal fat measures. The specific data are shown in Table 3 and Table 4.

Secondary Outcomes

Table 5 lists the secondary outcomes for the intervention and placebo groups. One month after the intervention, the intervention group showed statistically significant reductions ($P < .001$-$.024$) in total cholesterol, triglycerides, LDL-C, and insulin levels compared to the placebo, while their basal metabolic rate was higher. However, there were no significant differences in glucose ($P < .237$) and HDL-C ($P < .165$) levels between the two groups.

After 3 months of intervention, the blood glucose, total cholesterol, triglyceride, LDL-C, and insulin levels of the intervention group were significantly lower than those of the placebo group ($P < .05$), while the HDL-C level and basal metabolic rate were significantly higher than those of the placebo group ($P < .001$).

Tertiary Outcomes

Table 6 lists the tertiary outcomes for the intervention and placebo groups. These parameters were used to verify the safety of the intervention. One month after the intervention, the levels of ALT, AST, total bilirubin, serum creatinine, urea nitrogen, thyroid-stimulating hormone, free triiodothyronine, and free thyroxine in both groups were within the normal

Table 6. Comparison of Liver and Kidney Function Parameters

Variable	Group	Baseline	Month 1	Month 3	*P* value (Month 1/Month 3)[a]
ALT (U/L)	Intervention	29.4±5.8	29.1±6.0	28.5±6.2	.374/.630
	Placebo	28.8±6.2	29.5±5.8	29.2±5.9	
AST (U/L)	Intervention	26.9±5.9	26.8±5.9	26.3±5.8	.451/.650
	Placebo	27.1±5.9	27.3±6.0	27.1±6.2	
Total Bilirubin (μmol/L)	Intervention	14.7±3.1	15.0±3.2	15.2±3.1	.332/.543
	Placebo	14.6±3.3	14.9±3.1	14.8±3.3	
Creatinine (μmol/L)	Intervention	75.2±6.1	73.2±8.1	72.5±8.3	.245/.512
	Placebo	76.1±7.8	74.1±7.8	73.8±7.9	
Urea Nitrogen (mmol/L)	Intervention	5.5±1.1	5.3±1.2	5.2±1.1	.504/.504
	Placebo	5.5±1.0	5.5±1.1	5.4±1.2	
Thyroid-stimulating hormone (mIU/L)	Intervention	2.3±0.8	2.3±0.5	2.3±0.5	.195/.375
	Placebo	2.4±0.4	2.4±0.6	2.4±0.6	
Free triiodothyronine (pmol/L)	Intervention	4.5±0.5	4.5±0.4	4.5±0.4	.394/.385
	Placebo	4.5±0.5	4.6±0.5	4.6±0.5	
Free thyroxine (pmol/L)	Intervention	15.4±1.4	15.3±1.2	15.2±1.3	.721/.311
	Placebo	15.4±1.2	15.4±1.3	15.5±1.2	

[a]Compared to placebo

reference range, and there was no significant difference between the groups ($P \geq .05$).

After 3 months of intervention, all liver and kidney parameters remained within the normal reference range, with no significant differences between the groups ($P \geq .05$).

Safety and Adverse Effects

The improved metabolic markers included as secondary outcome measures, along with liver, kidney, and thyroid function tests included in the tertiary measures, show no short-term negative impact of AKKBoom. The intervention group at 1-month and 3-month maintained normal liver, kidney, and thyroid function parameters, with no significant inter-group differences ($P > .05$), further confirming the safety of this intervention. In the short term, AKKBoom appears to pose no potential metabolic burden on the liver and kidneys.

DISCUSSION

Several interesting endpoints made the results particularly noteworthy. In addition to weight reduction, body fat percentage, BMI, waist circumference, hip circumference, subcutaneous abdominal fat thickness, and visceral abdominal fat thickness, the improvements in the metabolic parameters of blood glucose, total cholesterol, triglycerides, LDL-C, HDL-C, and insulin levels suggest that this nutraceutical combination may contribute to these important health benefits along with weight loss. Furthermore, there were significant improvements in basal metabolic rate after both one and three months. This bodes well for potentially increasing total daily energy expenditure as part of an ongoing weight maintenance program.

The epigenetic aspect of pasteurized AKK is likewise interesting to consider as part of its mechanism of action. In mice fed a high-fat diet, pasteurized AKK improved metabolic dysregulation by preventing body weight gain after one week. Additionally, it decreased the weight of the major adipose tissues, reduced adipogenesis/lipogenesis and serum total cholesterol levels, improved glucose homeostasis (balance), and suppressed inflammatory insults. These benefits may be impacted by pasteurized AKK's downregulation of gene expression associated with the inflammatory cytokine TNF-α (tumor necrosis factor alpha) and suppression of TLR4 (Toll-like receptor 4),[26] whose overactivation is otherwise linked to chronic inflammation.[29]

Future human clinical studies using the pasteurized AKK001™/butyrate combination may consider examining this epigenetic activation. In addition, future studies should include proper registration with appropriate agencies such as Clinicaltrials.gov.

CONCLUSION

In this randomized, placebo-controlled intervention, daily supplementation with 1.5×10^{10} TFU pasteurized AKK001™ and 150 mg tributyrin was associated with significant improvements in body morphology measures, body fat distribution, and metabolic indicators over 90 days, while maintaining normal liver, kidney, and thyroid function. These findings support the potential role of the AKK001™/tributyrin combination in weight management and as a complementary strategy for metabolic health in overweight adults. Larger, longer-term, and mechanistic studies are warranted to confirm these effects and explore underlying pathways.

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AUTHOR DISCLOSURE STATEMENT

Gene Bruno is the Chief Scientific Officer for Nutraland USA, a raw material supplier that markets AKK001™, although Nutraland USA is not a study sponsor.

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