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Effect of a probiotic bacteria and yeast mixture on lipid metabolism and gut microbiota in triton X-100-induced hypercholesterolemic rats

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Abstract

Background: Hypercholesterolemia is a major risk factor for cardiovascular disease and is implicated in numerous other metabolic disorders. Several studies have demonstrated that probiotic supplementation can improve lipid metabolism by reducing serum lipid concentrations and lowering cholesterol levels.

Objectives: This study aimed to evaluate the effects of a probiotic mixture (PM) comprising the bacterium *Lactobacillus rhamnosus* yoba and the yeast *Saccharomyces boulardii* on the serum lipid profile, gut microbiota composition, and associated metabolic parameters in hypercholesterolemic rats.

Methods: Hypercholesterolemia was induced by a single intraperitoneal injection of Triton X-100 combined with a high-fat animal diet over two weeks. Rats were assigned to four groups: normal diet (ND), high-fat diet with Triton X-100 (HFD), HFD with low-dose probiotic mixture (HFD+PWL), and HFD with high-dose probiotic mixture (HFD+PWH). Probiotic doses were 30 mg/kg body weight (low) and 40 mg/kg body weight (high). Body weight, blood glucose levels, serum lipid profiles, and gut microbiota composition were assessed.

Results: Triton X-100 and high-fat diet significantly increased serum total cholesterol (TC), triacylglycerols (TG), and low-density lipoprotein cholesterol (LDL-C) ($p<0.05$), with a non-significant increase in high-density lipoprotein cholesterol (HDL-C). Administration of the probiotic mixture (HFD + PWL) significantly reduced TC and LDL-C to 155.11±19.1 mg/dL and 89.25±15.47 mg/dL, respectively, from 226.29±3.47 mg/dL (TC) and 125.16±14.25 mg/dL (LDL-C) in the HFD group ($p<0.05$). TG levels were most effectively reduced by HFD+PWH to 199.58 ± 19.17 mg/dL compared to 342.92 ± 18.76 mg/dL in the HFD group. HDL-C increased to 62.89±8.39 mg/dL in HFD+PWH versus 36.22 ± 8.88 mg/dL in HFD. The probiotic mixture also enhanced yeast and lactic acid bacteria counts while reducing coliform populations from $4.8 \pm 0.89\times10^2$ and $3.53\pm1.46 \times 10^2$ CFU/mL in the stomach and intestine, respectively (HFD), to $1.70\pm0.5 \times 10^2$ and $1.80\pm0.36 \times 10^2$ CFU/mL in HFD+PWH.

Conclusion: These findings indicate that the probiotic mixture of *L. rhamnosus* yoba and *S. boulardii* effectively modulates lipid metabolism and gut microbiota, supporting its potential use as a dietary supplement for managing hypercholesterolemia.

Introduction

Hypercholesterolemia is a well-established, modifiable risk factor contributing to the global burden of cardiovascular diseases (CVDs), type 2 diabetes mellitus, and metabolic syndrome.[1] By 2030, CVDs are projected to account for approximately 40% of all deaths worldwide, affecting an estimated 23.6 million individuals.[2] A central determinant of CVD risk is an imbalanced plasma lipid profile, which not only predisposes to various heart diseases but is also associated with colon cancer.[3] Hypercholesterolemia is characterized by elevated levels of total cholesterol (TC), triacylglycerols (TG), and low-

density lipoprotein cholesterol (LDL-C), accompanied by reduced high-density lipoprotein cholesterol (HDL-C).[4] Individuals with hypercholesterolemia exhibit a threefold higher risk of myocardial infarction compared with normolipidemic counterparts.[5] This elevated risk is often linked to unhealthy dietary patterns, including excessive consumption of processed and high-salt foods, unhealthy fats, added sugars, and insufficient intake of fruits, vegetables, and complex carbohydrates.[6] Even a 1% increase in serum cholesterol has been associated with a 2%–3% increase in CVD incidence, underscoring the need for effective preventive and therapeutic strategies.[7]

Conventional dietary interventions, such as low-fat diets, have been widely employed to lower blood cholesterol levels; however, their effectiveness is frequently limited by suboptimal patient compliance.[8] Interest in probiotics as modulators of lipid metabolism emerged following Mann and Spoerry's 1974 observation, which demonstrated an 18% reduction in plasma cholesterol after three weeks of Maasai warriors consuming 4–5 liters of fermented milk daily.[9] Since then, numerous studies have elucidated the mechanisms through which probiotics reduce lipid levels. These mechanisms include the adsorption and adhesion of active bacterial cells, decreased reabsorption of bile acids, production of short-chain fatty acids (SCFAs), and inhibition of lipoprotein lipase activity.[10] Importantly, cholesterol-lowering effects are highly strain- and growth-dependent,[6] highlighting the necessity of targeted probiotic interventions.

While many studies have explored the cholesterol-lowering capacity of probiotic bacteria, comparatively fewer have investigated the role of probiotic yeasts. For example, supplementation with *Saccharomyces cerevisiae* ARDMC1, derived from traditional rice beer starter cake, significantly reduced plasma TG, LDL-C, and TC in Wistar rats over 42 days.[11] Similarly, Kim et al.,[12] demonstrated that a mixture of three *Bifidobacterium* species (*B. longum*, *B. breve*, *B. lactis*) and two *Lactobacillus* species (*L. plantarum*, *L. reuteri*) significantly decreased LDL-C, TC, and TG while increasing HDL-C. In a pioneering human study, daily supplementation with *S. cerevisiae* var. *boulardii* CNCM I-1079 for eight weeks significantly reduced remnant lipoprotein particles and triglyceride-rich lipoproteins, although no significant changes were observed in TC, HDL-C, or LDL-C.[13] These findings underscore the critical importance of selecting probiotic strains with high cholesterol-assimilation capacity,[14] as efficacy varies markedly between strains.[15] Additionally, each strain requires specific effective clinical dosages to achieve hypocholesterolemic effects, necessitating human studies to determine optimal therapeutic concentrations.[6]

Objectives

The present study was designed to evaluate the cholesterol-lowering potential of a combined probiotic intervention using the bacterium *Lactobacillus rhamnosus* yoba and the yeast *Saccharomyces boulardii* in a hypercholesterolemic rat model. *L. rhamnosus* yoba, a generic variant of the widely studied *L. rhamnosus* GG, is globally recognized for its documented health benefits.[16,17] Its affordability and availability as a starter culture also make it suitable for nutritional interventions in developing countries.[18,19] Previous studies have shown that *L. rhamnosus* yoba effectively modulates LDL-C without adverse effects while elevating HDL-C.[20] *S. boulardii* remains the only yeast with established clinical efficacy in double-blind studies.[21,22] Administration of *S. boulardii* in hypercholesterolemic models has been associated with significant reductions in TC, LDL-C, and TG, along with favorable modulation of gut microbiota.[23,24]

Methods

Probiotics and culture

The probiotic mixture used in this study comprised the commercially available bacterium *Lactobacillus rhamnosus* yoba (Yoba Foundation, Uganda) and the yeast *Saccharomyces boulardii* (Klaires Lab, USA). Each probiotic was dissolved in peptone water and serially diluted to achieve a final concentration of 0.9×10^{10} CFU/day, corresponding to a stock solution of 10 mg/mL. The administered dose of the probiotic mixture was calculated based on the rats' body weight, with the low dose set at 30 mg/kg and the high dose at 40 mg/kg. The probiotic mixture was administered orally in a 1:1 ratio continuously for two weeks.

Animals and study design

Male Wistar rats, 8 weeks of age, were housed individually in a controlled environment maintained at 25 °C with 55±5% humidity and a 12-hour light/dark cycle. Rats were acclimatized for seven days with ad libitum access to standard chow and water. During the study, rats were provided unrestricted access to food and water, and body weight was recorded weekly.

A high-fat diet (HFD) was prepared by blending animal fat (tallow, 36%) with standard chow (64%) containing maize, fish meal, and soybean oil. After the adaptation period, rats were randomly assigned to four experimental

groups, each comprising six animals: 1) normal diet (ND), 2) high-fat diet with Triton X-100 (HFD), 3) HFD with low-dose probiotic mixture (HFD+PWL), and 4) HFD with high-dose probiotic mixture (HFD+PWH). Hypercholesterolemia was induced via a single intraperitoneal injection of Triton X-100 (Sigma Aldrich, USA) at 100 mg/kg body weight on day zero, prior to probiotic treatment. Control and HFD+Triton X-100 groups received normal saline.

Measurement of body weight

Rat body weight was recorded prior to treatment initiation and weekly throughout the study using a 1-kg electronic compact scale. The administered dose of the probiotic mixture was adjusted according to body weight. Body weight gain was calculated as follows:

Body weight gain (g) = Final body weight – Initial body weight.

Glycemia assessment

Fasting blood glucose was measured at weeks 1 and 2 following a 6-hour fast using the tail-prick method. Blood glucose levels were determined with a Fine-Test Blood Glucose Monitor and reported in mg/dL.

Serum lipid analysis

At the end of the experiment, rats were anesthetized with chloroform, and blood samples were collected via the abdominal vein. Serum was separated by centrifugation at 1,500×g for 15 minutes and analyzed for total triglycerides (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C) using Randox enzymatic kits. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula.[25] Aliquots of serum were incubated with kit reagents at 37 °C for 10 minutes, and absorbance was measured at 546 nm for TC and TG. Results were expressed in mg/dL.[26,27]

Gut microbiota analysis

Gastrointestinal tract (GIT) contents were aseptically removed post-mortem. Samples were homogenized in normal saline, serially diluted, and plated on selective media. Lactic acid bacteria (LAB) were enumerated on de Man-Rogosa-Sharpe (MRS) agar, total coliforms on MacConkey agar, and total yeasts on Sabouraud dextrose agar (SDA).

Gastric acidity, volume, and pH determination

Gastric contents were collected, and gastric juice volume (mL/100 g), total acidity (mg/L), and pH were measured. Total titratable acidity was assessed following the method of Osilo et al.[28] Briefly, gastric samples were titrated with 0.1 M sodium hydroxide (NaOH) using two drops of phenolphthalein as an indicator, and titratable acidity was expressed as a percentage of lactic acid. pH was measured directly using a calibrated digital pH meter.

Statistical analysis

The continuous variables were expressed as the mean ± SD, and the categorical variables were presented as a percentage and frequency. One-way analysis of variance (ANOVA) followed by Tukey's multiple comparison test was used to evaluate differences among groups. All statistical analyses were performed with SPSS (version 16.0, SPSS Inc, Chicago, IL, USA). A "P-value" less than 0.05 was considered significant.

Ethical considerations

The study was conducted in accordance with the Declaration of Helsinki. All animal experiments were approved by the Animal Research Ethics Committee of Nnamdi Azikiwe University (Approval number: NAU/AREC/2024/0075), Awka, Nigeria, and were conducted in accordance with institutional and international guidelines for the care and use of laboratory animals.

Results

Effect of the cholesterol-lowering probiotic mixture on body weight

The body weight of all rats was monitored throughout the two-week treatment period with the probiotic mixture [Table-1]. Rats in the ND group consistently exhibited lower body weight (55.00–68.00 g) compared with other groups. All groups demonstrated an increase in body weight from day 0 to day 14. On day 0, there was no significant difference in body weight between the HFD+PWL (67.33±6.66 g) and HFD (67.00±7.81 g) groups ($p<0.05$). However, HFD+PWH rats showed a significant increase in body weight throughout the study (94.67±3.06 g, 99.67±3.06 g, and 105.67±5.13 g on days 0, 7, and 14, respectively) relative to ND and HFD groups. Body weight gain was lower in the probiotic-treated groups compared with HFD alone, with HFD+PWH exhibiting the lowest gain (11.00±2.65 g). Nevertheless, differences in body weight gain among groups were not statistically significant ($p < 0.05$).

Effect of the probiotic mixture on fasting blood glucose

Fasting blood glucose levels were highest in the HFD group (90.33±4.04 mg/dL). Supplementation with the probiotic mixture significantly reduced fasting glucose ($p<0.05$), with HFD+PWH and HFD+PWL achieving 64.33±1.53 mg/dL and 65.00±6.08 mg/dL, respectively, compared with HFD [Figure-1].

Table-1. Changes in the body weight of the rats

Groups	Days			Body weight gain
	0	7	14	
ND	55.00^{a}±4.36	60.00^{a}±7.00	68.00^{a}±7.21	13.00^{a}±4.58
HFD	67.00^{a}±7.81	79.67^{b}±10.02	89.00^{ab}±9.85	22.00^{a}±3.46
HFD +PWL	67.33^{a}±6.66	73.00^{ab}±7.21	80.33^{a}±10.79	13.00^{a}±9.85
HFD+PWH	94.67^{b}±3.06	99.67^{c}±3.06	105.67^{b}±5.13	11.00^{a}±2.65

Different superscript letters within the same column represent the statistical significance ($p<0.05$) (mean ± SD).

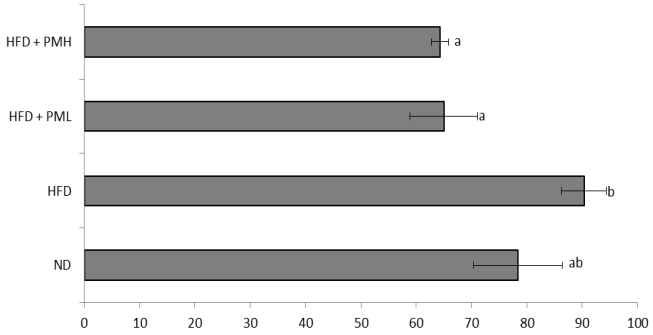

Figure-1. Fasting blood glucose of hypercholesterolemic rats treated with probiotic mixture. Different superscript letters represent statistical significance difference ($p<0.05$) (mean±SD).

Effect of the probiotic mixture on serum lipid profiles

The HFD group displayed significantly elevated serum TG, TC, and LDL-C levels relative to all probiotic-treated groups ($p<0.05$). TG was highest in HFD rats (342.92±18.76 mg/dL) and was significantly reduced in HFD+PWL and HFD+PWH groups to 237.08±12.58 mg/dL and 199.58±19.14 mg/dL, respectively [Figure-2A].

Similarly, TC and LDL-C concentrations were highest in HFD rats (226.29±3.47 mg/dL and 125.16±14.25 mg/dL, respectively). HFD+PWL treatment significantly lowered TC (155.11±19.10 mg/dL) and LDL-C (89.25±15.47 mg/dL), while HFD+PWH maintained low TC levels (158.3±1.66 mg/dL) [Figures 2B and 2C]. HDL-C levels increased in the probiotic-treated groups, with HFD+PWL and HFD+PWH reaching 56.33±12.47 mg/dL and 62.89±8.39 mg/dL, respectively, compared to HFD (36.22±8.88 mg/dL). However, these changes were not statistically significant ($p<0.05$) [Figure-2D].

Effect of the probiotic mixture on gut microbiota

The probiotic mixture favorably modulated gut microbial populations in hypercholesterolemic rats [Table-2]. HFD disrupted gut microbial balance relative to ND rats. In probiotic-treated groups, both fungal and LAB counts in the stomach and intestine increased, while coliform counts decreased. Stomach coliform counts decreased from 4.8±0.89 CFU/mL (HFD) to 1.80±0.1 CFU/mL (HFD+PWL) and 1.70±0.5 CFU/mL (HFD+PWH), though the reduction was not statistically significant. Notably, intestinal yeast counts significantly increased in the HFD+PWH group (7.17±1.50 CFU/mL)

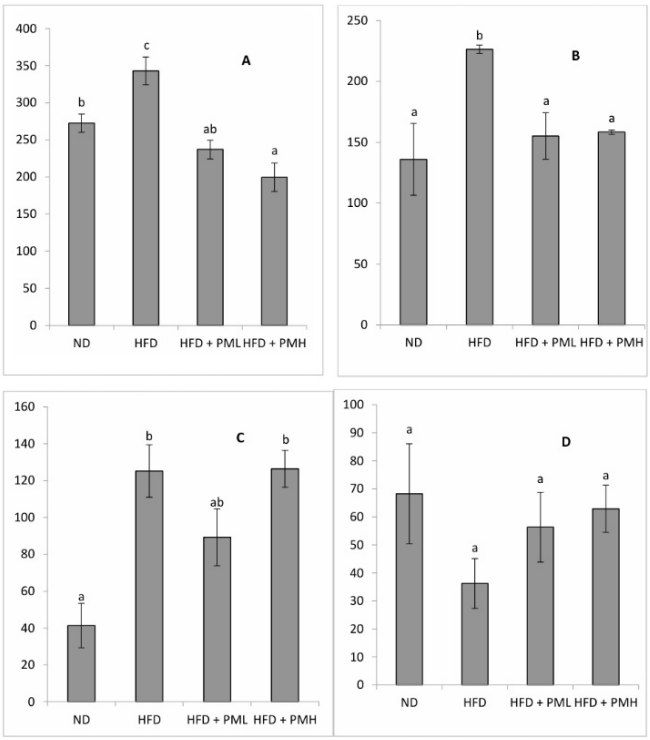

Figure-2. Effect of probiotic mixture on A) total triglycerides, B) total cholesterol, C) low-density lipoproteins, D) high-density lipoproteins. Different superscript letters represent statistical significance difference ($p<0.05$) (mean ± SD).

Gastric volume, pH, and acidity

Gastric pH remained acidic across all groups (1.26±0.02 to 1.89±0.09). Probiotic treatment elevated pH levels, with a significant increase observed in HFD+PWH rats (1.89±0.09, $p<0.05$). Total acidity increased in probiotic-treated rats (354.17±14.43 mg/L for HFD+PWL and 425.00±66.14 mg/L for HFD+PWH) relative to HFD (295.83±71.25 mg/L), though differences were not statistically significant. Gastric juice volume was lower in probiotic-treated groups (2.33±0.32 mL/100 g for HFD+PWL and 3.43±0.57 mL/100 g for HFD+PWH) compared with HFD (4.03±2.07 mL/100 g), but the reduction did not reach statistical significance [Table-3].

Table-2. Effect of the probiotic mixture on the gut microbiota of hypercholesterolemic rats

Groups	Stomach			Intestine		
	Fungi count (CFU/ml) X 10^2	Coliform count (CFU/ml) X 10^2	LAB count (CFU/ml) X 10^2	Fungi count (CFU/ml) X 10^2	Coliform count (CFU/ml) X 10^2	LAB count (CFU/ml) X 10^2
ND	1.30^{a}±0.20	3.73^{a}±3.04	3.17^{a}±2.37	2.90^{a}±1.25	3.2^{a}±1.41	2.87^{a}±1.63
HFD	1.17^{a}±0.06	4.8^{a}±0.89	2.57^{a}±1.21	2.90^{a}±1.08	3.53^{a}±1.46	2.33^{a}±1.25
HFD+PML	1.23^{a}±0.31	1.80^{a}±0.1	4.5^{a}±2.56	3.73^{a}±0.49	2.13^{a}±0.21	3.77^{a}±1.80
HFD+PMH	1.80^{a}±0.46	1.70^{a}±0.5	4.4^{a}±0.95	7.17^{b}±1.50	1.80^{a}±0.36	3.5^{a}±0.44

Different superscript letters within the same column represent the statistical significance ($p<0.05$) (mean ± SD).

Table-3. Effect of treatment with probiotic mixture on the gastric juice parameters

GROUPS	pH	Total acidity (mg/L)	Volume (ml/100g)
ND	1.26^{a}±0.02	258.33^{a}±14.43	2.07^{a}±0.12
HFD	1.42^{ab}±0.10	295.83^{a}±71.25	4.03^{a}±2.07
HFD + PML	1.72^{ab}±0.35	354.17^{a}±14.43	2.33^{a}±0.32
HFD + PMH	1.89^{b}±0.09	425.00^{a}±66.14	3.43^{a}±0.57

Different superscript letters within the same column represent the statistical significance ($p < 0.05$). (mean ± SD).

Discussion

The cholesterol-lowering potential of probiotics has garnered substantial interest and has been corroborated by numerous studies demonstrating their beneficial effects on hypercholesterolemia. Probiotic mixtures, in particular, have shown combined effects that enhance lipid-lowering activity when administered orally to hypercholesterolemic models.[29]

In this study, two commercially available probiotics, *Lactobacillus rhamnosus* yoba and *Saccharomyces boulardii*, were evaluated for their capacity to reduce cholesterol in Triton X-100-induced, high-fat diet-fed rats. Feeding rats with HFD resulted in significant body weight gain [Table-1], elevated fasting blood glucose [Figure-1], and increased lipid concentrations [Figure-2]. Administration of the probiotic mixture for two weeks mitigated these effects, significantly reducing body weight gain, fasting blood glucose, and serum lipid levels, while enhancing the yeast and LAB populations in the gut microbiota.

All experimental groups, including those receiving probiotics, exhibited body weight increases over the two-week period. This weight gain may reflect lipid accumulation in adipocytes, a recognized precursor of obesity.[30] The observed increase in probiotic-treated groups could also be attributed to the growth-promoting potential of yeast, which are known to secrete bioactive compounds that facilitate cellular differentiation and maturation,[31,32] though these specific metabolites were not assessed in the present study. The reduction in body weight gain in probiotic-treated groups relative to HFD alone aligns with previous findings reported by Tiepma et al.[14]

The significant reduction in fasting blood glucose in the probiotic-treated groups highlights a synergistic effect of *L. rhamnosus* yoba and *S. boulardii* on glucose homeostasis. Improved glycemia may alleviate glucotoxicity and lipotoxicity in target tissues, reflecting the close metabolic interplay between lipid and glucose regulation.[27]

Elevated concentrations of TC, TG, and LDL-C, coupled with reduced HDL-C, are critical risk factors for the development of cardiovascular diseases.[33,11] The probiotic mixture effectively lowered LDL-C, TG, and TC, with HFD+PWH treatment achieving TG levels below those of the ND group and TC levels comparable to normal controls. These results are consistent with those of Puttarat et al.,[34] who reported comparable cholesterol-lowering effects with both single and mixed indigenous probiotic strains.

No significant increase in HDL-C was observed in the probiotic-treated groups compared with HFD. This finding parallels the study by Saika et al.,[11] in which supplementation with *S. cerevisiae* ARDMC1 did not significantly elevate HDL-C. Such outcomes may be due to the short duration of the experiment or limited probiotic efficacy in stimulating HDL-C synthesis over the two-week period.[35,11]

Probiotic supplementation enhanced yeast and LAB populations in the gastrointestinal tract, indicating successful colonization. The concurrent reduction in coliforms suggests antagonistic activity against pathogenic bacteria, potentially via the production of antimicrobial metabolites, though these were not directly measured. These findings confirm the ability of the probiotic mixture to modulate gut microbiota composition and suppress coliform overgrowth.[36]

Similar observations were reported by Xie et al.,[37] who documented increased *Lactobacillus* and *Bifidobacterium* counts and decreased *Escherichia coli* in hypercholesterolemic rats treated with *L. fermentum* and *L. plantarum*, highlighting the capacity of probiotics to survive gastrointestinal conditions and establish within the gut microbiota.

Gastric juice analysis revealed higher acidity in probiotic-treated groups compared with HFD, suggesting that these probiotics may enhance gastric acid secretion, potentially through the production of organic acids as metabolic byproducts, although this was not directly measured. Observed reductions in gastric volume alongside increased pH and total acidity in probiotic-treated animals further support a modulatory effect of the probiotic mixture on gastric secretory function.[38]

Despite the observed lipid-modulating effects, this study has several limitations: the absence of single-strain treatment groups prevents delineation of individual probiotic contributions and confirmation of synergistic interactions; while the hypercholesterolemic rat model provides valuable mechanistic insights, interspecies differences in lipid metabolism and gut microbiota composition may limit direct extrapolation to human applications, necessitating clinical trials for validation; minor baseline variability in initial body weight among groups, despite randomization, may have influenced outcomes; and the assessment was confined largely to serum lipid profiles, lacking evaluation of underlying cellular and molecular mechanisms -such as gene expression, regulatory enzymes, or lipid transport proteins- which restricts the depth of mechanistic insight into how the probiotic mediated its lipid-modulating effects.

Conclusions

This study demonstrates the therapeutic potential of an equimolar consortium comprising *S. boulardii* and *L. rhamnosus* yoba as a nutraceutical intervention to mitigate cardiovascular risk and associated metabolic disorders. The combined administration of these microorganisms exhibits enhanced efficacy in attenuating hypercholesterolemia and favorably modulating gut microbiota. These findings support the use of this binary probiotic mixture to achieve superior hypocholesterolemic outcomes relative to single-strain supplementation.

Practical points in Biochemistry/Nutrition:

A synergistic probiotic mixture of *Lactobacillus rhamnosus yoba* and *Saccharomyces boulardii* effectively lowers serum triglycerides, total cholesterol, and LDL-C while improving gut flora balance, offering a promising dietary strategy for managing hypercholesterolemia.

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Competing interests

The authors declare that they have no competing interests.

Abbreviations

CVD: Cardiovascular Disease; GIT: Gastrointestinal Tract; HDL-C: High-Density Lipoprotein Cholesterol; HFD: High-Fat Diet; LAB: Lactic Acid Bacteria; LDL-C: Low-Density Lipoprotein Cholesterol; ANOVA: Analysis of Variance; CFU: Colony Forming Unit; ND: Normal Diet; PWL/H: Probiotic Mixture Low/High Dose; SCFA: Short-Chain Fatty Acid; SDA: Sabouraud Dextrose Agar; TC: Total Cholesterol; TG: Triglycerides.

Authors' contributions

Conceptualization: C.O., T.M.C.A.; Methodology: C.O., T.M.C.A., I.U.N., J.C.O., C.N.O.; Investigation: C.J.U., C.P.I.; Data Curation: C.O., T.M.C.A., C.J.U., C.P.I., J.C.O., C.N.O., I.U.N.; Supervision: C.O., T.M.C.A.; Writing – Review and Editing: C.O., I.U.N. All authors read and approved the final manuscript. All authors take responsibility for the integrity of the data and the accuracy of the data analysis.

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Role of the funding source

None.

Availability of data and materials

The data used in this study are available from the corresponding author on request.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. All animal experiments were approved by the Animal Research Ethics Committee of Nnamdi Azikiwe University (Approval number: NAU/AREC/2024/0075), Awka, Nigeria, and were conducted in accordance with institutional and international guidelines for the care and use of laboratory animals.

Consent for publication

By submitting this document, the authors declare their consent for the final accepted version of the manuscript to be considered for publication.

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