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Assessment of Fecal Microflora, Short-chain Fatty Acids and Key Metabolic Biomarkers in Adult Males from Pekanbaru

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ABSTRACT

Background: Recent studies have investigated the influence of the gut microbiota on various health determinants. The interaction between gut microbiota and human metabolism influences blood cholesterol, glucose, uric acid and short-chain fatty acids (SCFAs) production.

Methods: This study investigates the correlation between microflora and blood and fecal short-chain fatty acids (SCFAs), total cholesterol, glucose and uric acid levels in adult men. Thirty males aged 18-21 from Pekanbaru, Indonesia, participated. Total plate count (TPC), lactic acid bacteria (LAB) and Bifidobacteria were analyzed using specific media. Total blood cholesterol, glucose and uric acid were quantified with KITs, while SCFAs were assessed using GC-MS. A bivariate analysis explored the correlations between fecal microbiota, blood cholesterol, glucose, uric acid and SCFAs.

Result: The results showed variability in TPC, LAB and Bifidobacteria counts, with correlations between microflora quantity and blood and fecal SCFAs, cholesterol, glucose and uric acid. While most participants had normal cholesterol and glucose, 30% had elevated uric acid levels. Bivariate analysis revealed no correlation between TPC, LAB and Bifidobacteria counts, but microflora may support cholesterol and glucose metabolism. Bifidobacteria abundance was linked to blood uric acid levels. SCFA concentrations correlated with TPC, LAB and Bifidobacteria, with the most prevalent acetic, propionic and butyric acids. Microbiota composition influenced cholesterol, glucose, uric acid and SCFA levels.

Key words: Adult male, Fecal scfas, Glucose, Microflora, Total cholesterol, Uric acid.

INTRODUCTION

The human gut microflora is a complex ecosystem of microorganisms, including bacteria, fungi, viruses and protozoa, that plays a crucial role in digestion, immune modulation, vitamin synthesis and disease prevention (Han *et al.,* 2021). It also involves metabolic processes and mental health (Hasibuan and Kolondam, 2017; Rowland *et al.,* 2018). As research continues to uncover the intricate interactions between diet and gut microbiota, there is growing interest in personalized nutrition strategies that cater to individual microbiome compositions. By optimizing dietary choices, individuals can positively modulate their gut microbiota, leading to improved digestive health, enhanced immunity and reduced risk of chronic diseases (Rathod *et al.,* 2025; Zheng *et al.,* 2024). Gut bacteria ferment dietary fibers into short-chain fatty acids (SCFAs), such as acetate, propionate and butyrate, which influence lipid metabolism, insulin sensitivity and glucose uptake, helping maintain metabolic homeostasis (Morrison and Preston, 2016; Canfora *et al.,* 2015). SCFAs, particularly propionate, can reduce blood cholesterol by inhibiting hepatic cholesterol production, with high-fiber diets enhancing SCFA synthesis (Koh *et al.,* 2016).

Dietary lipids, particularly saturated fats from red meat, butter and full-fat dairy, raise blood cholesterol, while unsaturated fats from olive oil, nuts and fatty fish can lower LDL cholesterol and improve lipid profiles (Mensink *et al.*, 2003; Mozaffarian *et al.,* 2010). Soluble fiber from oats, barley, legumes, fruits and vegetables helps reduce cholesterol by binding to bile acids in the colon (Brown *et al.,* 1999). Lipid digestion and cholesterol metabolism require gut bacteria to deconjugate and modify bile acids (Pato *et al.,* 2005; Pato *et al.,* 2004). Recent studies indicate that gut microbiota may influence cholesterol metabolism through bile acid modification and lipid absorption (Zhang *et al*., 2022).

Fat-free fluid milk increases muscle mass and decreases fat mass. Additionally, higher dietary calcium intakes were substantially associated with lower serum parathyroid hormone and higher serum 25 hydroxy vitamin D levels in milk, which may have a beneficial influence on bone turnover. It was discovered to be linked to a decreased risk of cardiovascular illnesses, type 2 diabetes and weight

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loss (Aggarwal *et al.,* 2023). Fruits, vegetables and their processed products also play a crucial role in human health by providing essential nutrients, fiber, antioxidants and bioactive compounds that contribute to overall wellbeing. Their consumption is associated with various health benefits, including disease prevention and improved bodily functions (Mahapatra and Das, 2022).

Carbohydrates significantly affect blood glucose levels, with simple carbohydrates raising blood glucose quickly and complex carbohydrates providing a slower, controlled release (Jenkins *et al.,* 1981). High-glycemic foods raise blood glucose and insulin levels, while low-glycemic foods improve glycemic control and reduce diabetes risk (Barclay *et al.,* 2008). Soluble fiber in foods like oats and beans slows glucose absorption and improves long-term glycemic control (Slavin, 2013). Additionally, purine-rich diets and sugary foods, particularly fructose, are linked to elevated uric acid levels, leading to hyperuricemia and gout (Choi *et al.,* 2004; Johnson *et al.,* 2007). Gut microbiota plays a significant role in uric acid metabolism by breaking down purines, reducing absorption and influencing liver uric acid synthesis (Tong *et al.,* 2022; Feng *et al.,* 2024; Vernocchi *et al.,* 2016; Zhang *et al.,* 2022). Dysbiosis may contribute to elevated uric acid levels and associated health conditions (Vernocchi *et al.,* 2016). Research on gut microflora and its role in human metabolism has rapidly evolved. Gut microbiota contributes to producing SCFAs, which play a crucial role in metabolic homeostasis, including regulating cholesterol, blood glucose and uric acid levels. However, most studies have been conducted in countries with dietary patterns different from those of the Indonesian population. The preset study aimed to explore the relationship between gut microbiota and metabolic parameters in young adult males in Pekanbaru, Indonesia, who exhibit distinct dietary consumption patterns.

MATERIALS AND METHODS

Place and time of research

This research was conducted at the Agricultural Product Analysis Laboratory, Faculty of Agriculture, Universitas Riau, Pekanbaru, Vahana Scientific Laboratory, Padang, West Sumatra and Nano Center Indonesia, Tangerang, Banten, Indonesia. The research was carried out in July and August of 2024.

Subjects

The study included 30 adult male participants aged 18-21 from Pekanbaru, Indonesia, as part of the initial phase of intervention research on fecal microbiota, SCFA profiles, blood cholesterol, glucose, uric acid levels and immune regulation. Participants received either UHT milk or UHT milk containing probiotics. Selection criteria excluded individuals with lactose sensitivity. Participants completed a questionnaire on dietary habits, including protein, carbohydrate, fat, vegetable, fruit intake and probiotic or functional food consumption containing LAB and Bifidobacteria-data on eating frequency and food types consumed the month before stool sample collection were gathered. Stool samples were collected on day 0, pre-intervention and stored in sterile vials at 4-6°C. Written informed consent was obtained, ensuring voluntary participation. The research protocol was approved by the Faculty of Nursing Ethics Committee, Universitas Riau, Pekanbaru, Indonesia.

Chemical and media

The media used were PCA medium to calculate TPC, MRS Agar to calculate LAB and MRS-LC Agar to calculate Bifidobacteria. The chemicals used were sodium chloride, formic acid, ethyl acetate and Na_2SO_4 anhydrate.

Observation

The tested fecal SCFAs and fecal microflora profile parameters include TPC, LAB and Bifidobacteria.

Preparation of medium

The directions for creating media following the guidelines provided by the relevant media manufacturer (Merck, Indonesia) are to prepare PCA, MRS agar and MRS-LC agar.

Analysis of the number of fecal microflora

The number of fecal microflora calculations refers to Sutrisna *et al.* (2017) using the spread surface plate method (microflora counted and inoculated on PCA, MRS agar and MRS-LC agar media). The growing microbial colonies were counted using a colony counter. The number of microflora is counted and expressed in CFU/g feces.

Analysis of fecal SCFAs by GC-MS

SCFAs fecal sample analysis was conducted using the 6890 Gas Chromatograph with autosamplers and the 5973 Mass Selective Detector, provided by Agilent Technologies, Singapore, along with the Chemo Station Data System. The data was analyzed using Agilent Technologies' Mass Hunter.

Analysis of blood total cholesterol, glucose and uric acid

The total blood cholesterol, glucose and uric acid levels were measured for each participant. All tests were performed using the total cholesterol Test Strip, blood sugar Test Strip and uric acid Test Strip (multi-monitoring system, auto-check).

Data analysis

The correlation between the number of microflora and blood total cholesterol, sugar, uric acid and fecal SCFAS of the subjects was analyzed using bivariate analysis by the SPSS version 26 application and visualized using graph pad prism version 10.2.1.

RESULTS AND DISCUSSION

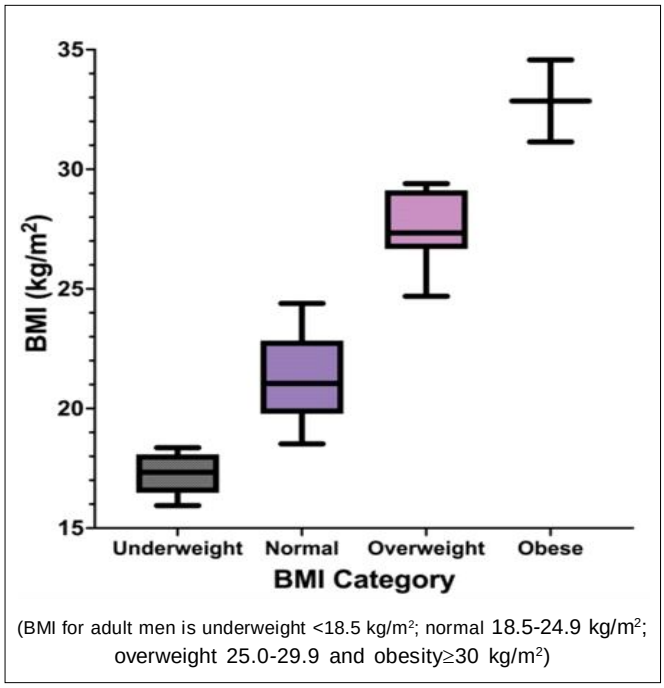
Profile of subjects

The initial data in this study were male adults aged between 18-21 years, with a body weight of 45-112 kg, height of

159-180 cm and the BMI distribution is presented in (Fig 1). Fig 1 shows that 6.67% of subjects are underweight, 13.33% are overweight, 6.67% are obese and 73.33% have a normal BMI. BMI variations were primarily influenced by dietary intake and eating frequency. Secondary data indicate that overweight and obese subjects consumed more food and ate more frequently than those with normal BMI. Additionally, these subjects were less physically active, leading to higher calorie intake than expenditure. Excess calorie intake is converted into fat through lipogenesis and stored in adipocytes. Without adequate physical activity, this process repeats, contributing to weight gain. The human body's evolutionary tendency to store fat as an energy reserve, coupled with modern food abundance, increases the prevalence of overweight and obesity (Bray and Bouchard, 2020; Hall and Guo, 2017). Furthermore, sedentary lifestyles exacerbate energy imbalance by reducing calorie expenditure, while diets high in sugar, fat and simple carbohydrates further elevate fat storage risks (Hill and Wyatt, 2005).

Profile of fecal microflora

Fecal total plate count (TPC) measures viable bacteria in fecal samples, reflecting gut microbial load, including



(BMI for adult men is underweight <18.5 kg/m^2; normal 18.5-24.9 kg/m^2; overweight 25.0-29.9 and obesity≥30 kg/m^2)

Fig 1: BMI distribution by category of adult male subjects.

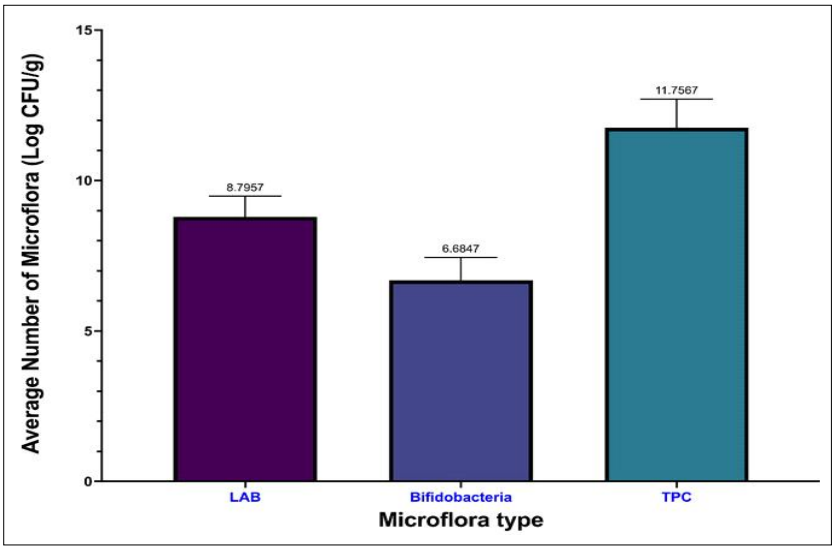


Fig 2: The average number of microflora-type.

probiotics and pathogens. Lactic acid bacteria (LAB) and Bifidobacteria play a critical role in gut health by balancing harmful bacteria and supporting digestion and immunity. Fig 2 presents TPC, LAB and Bifidobacteria levels in the study subjects' feces, showing variations likely influenced by diet.

The findings suggest that diverse dietary sources impact TPC levels, including fruits, vegetables, carbohydrates, proteins, fats and fermented milk. Diets high in animal-based protein and fat may favor species like Bacteroides, which efficiently metabolize proteins and lipids (Rinninella *et al.,* 2019). These results highlight the influence of dietary composition on gut microbiota. While TPC offers valuable insights into microbial burden, further studies are needed to identify specific dietary patterns or nutrients that significantly impact TPC and gut health. Understanding these relationships will help optimize gut microbiota for better digestion, immunity and overall health.

Fecal counts of LAB and Bifidobacteria reflect the abundance of lactic acid-producing bacteria crucial for gut health. These microbes convert carbohydrates into lactic acid, maintaining an acidic pH that inhibits pathogen growth (Sarkar and Mandal, 2016). Their abundance is influenced by dietary factors, particularly high-fiber diets rich in fruits, vegetables, legumes, whole grains and fermented foods. Prebiotics, such as non-digestible carbohydrates, support their growth by promoting lactic acid and SCFA production. Multivariate analysis showed that frequent consumption of high-fiber foods correlated with higher fecal counts of LAB and Bifidobacteria. Fermented foods, like yoghurt and kefir, directly introduce LAB, enhancing their populations. Bifidobacteria, key anaerobic bacteria, support the immune system, maintain the microbiome and aid digestion (Slavin, 2013). *Salmonella typhimurium* is prevented and the intestinal tract is repaired by a consortium of probiotic fermented milk that contains Bifidobacterium sp. and *Lactobacillus acidophilus* (Rathod *et al.,* 2025). Diets rich in fiber and fermented foods are essential for sustaining healthy LAB and Bifidobacteria populations and promoting gut health (Zheng *et al.,* 2024).

Table 1: Fecal short-chain fatty acids of adult male subjects.

Fecal samples of subjects*	Acetic acid (µmol/g)	Propionic acid (µmol/g)	Butyric acid (µmol/g)	Total SCFA (µmol/g)
S1	38.65	5.77	3.58	44.42
S2	76.85	6.29	3.78	86.92
S3	68.70	3.61	4.74	77.05
S4	58.39	4.06	7.36	69.81
S5	87.56	4.16	4.21	95.93
S6	60.93	3.05	2.93	66.91
S7	78.79	5.30	4.14	88.23
S8	63.01	3.56	3.25	69.82
S9	105.89	2.62	2.18	110.69
S10	85.65	4.14	4.76	94.55
S11	76.77	5.64	2.65	85.06
S12	60.36	1.29	1.60	63.25
S13	76.10	5.48	1.89	83.47
S14	53.58	1.09	0.91	55.58
S15	76.59	3.04	9.70	89.33
S16	68.57	5.57	3.96	78.10
S17	62.90	2.92	1.76	67.58
S18	89.13	8.61	2.46	100.2
S19	44.29	2.49	3.41	50.19
S20	80.48	4.31	2.97	87.76
S21	55.17	2.69	2.57	60.43
S22	41.15	2.18	1.45	44.78
S23	47.25	1.07	0.92	49.24
S24	103.20	9.44	4.15	116.79
S25	62.15	4.41	4.43	70.99
S26	125.85	4.57	2.41	132.83
S27	123.29	2.34	4.65	130.28
S28	58.49	4.72	7.49	70.70
S29	72.01	2.40	1.78	76.19
S30	54.89	2.96	2.13	59.98
Average	71.89	3.99	3.47	79.23

*S1- 30 = Subject number 1 to 30.

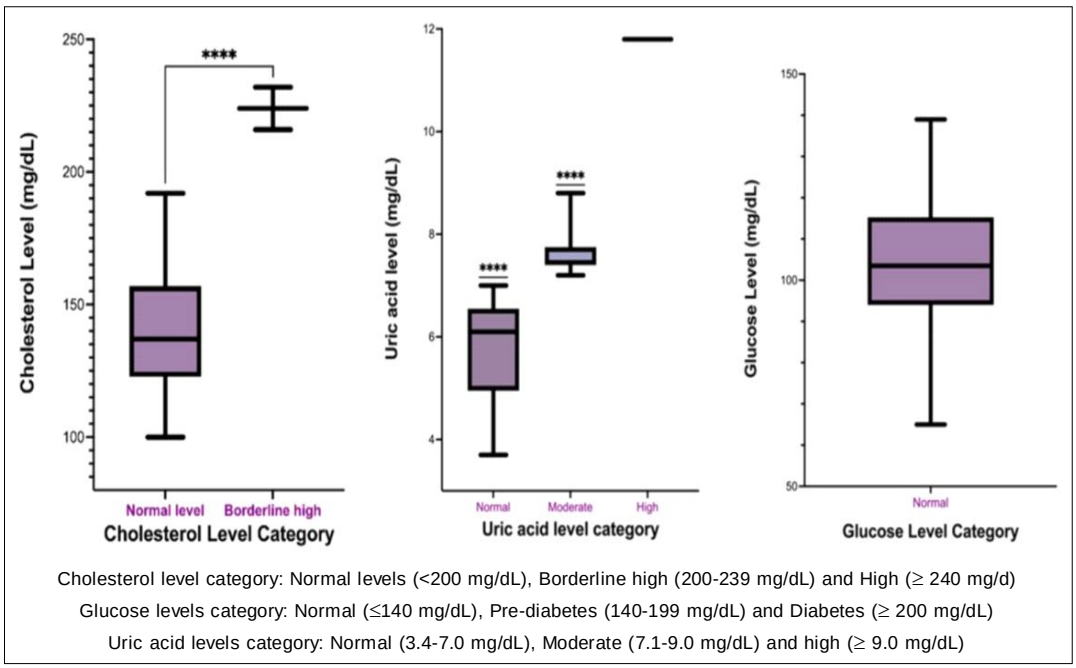
Fecal short-chain fatty acids of subjects

The gut microbiota is pivotal in digesting complex carbohydrates and fibers, fermented into SCFAs like acetate, propionate and butyrate (Table 1). The range of the amount of each of these SCFAs is slightly different from the normal SCFAs range in adults, namely acetic acid, propionic acid and butyric acid in adult feces. However, the average value is generally in the range of acetic acid, around 40-60 µmol/g, propionic acid around 10-20 µmol/g and butyric acid around 5-15 µmol/g feces. However, the total SCFAs range in the study ranged from 44.42-132.83 µmol/g, almost the same as the normal total SCFAs range in adults, which is 50-150 µmol/g feces. Acetic acid is generally the most dominant SCFAs, followed by propionate and butyrate (den Besten *et al.,* 2013). Similar results were found in the subjects of the present study. The composition of SCFAs can change based on factors such as diet, fiber consumption, gut health status and microbiota composition.

According to Koh *et al.* (2016), SCFAs, primarily acetate, propionate and butyrate, are metabolites produced by gut microbiota fermentation of dietary fibers, including LAB. LAB primarily produces lactic acid through the fermentation of carbohydrates and this lactate can serve as a substrate for other gut microbes to produce SCFAs, particularly butyrate. This process often involves cross-feeding interactions within the gut microbiota (Louis and Flint, 2009). Bifidobacteria are known for their beneficial effects on human health, including their ability to produce SCFAs. Bifidobacteria produce SCFAs primarily through the fermentation of dietary fibers and prebiotics, such as inulin, oligofructose and other prebiotics and resistant starches as substrates for fermentation. The fermentation results in the production of lactate, which can be further converted to acetate (Louis and Flint, 2009). Short-chain fatty acids (SCFAs), such as acetate, propionate and butyrate, are produced by various microbes other than LAB and Bifidobacteria. These microbes are TPCs that play an essential role in the gut ecosystem and produce SCFAs that support gut health and overall human metabolism. Adequate production of SCFAs is associated with numerous health benefits, including reduced risk of colorectal cancer, improved gut health and better metabolic profiles. Conversely, reduced SCFAs production has been linked to inflammatory diseases, metabolic syndrome and other health issues (Blaak *et al.,* 2020).

The blood total cholesterol, glucose and uric acid of subject

The relationship between blood cholesterol, glucose, uric acid and gut microflora is a growing research focus. Fig 3 shows blood cholesterol, glucose and uric acid levels in non-fasting male subjects, with 93.33% having normal cholesterol. Bivariate analysis found no correlation between cholesterol and gut microflora, likely due to similar diets and environments. At ages 18-21, hormonal peaks, such as testosterone, influence efficient cholesterol metabolism, which increases HDL and reduces total cholesterol (Robinson *et al*., 2021). Probiotics, particularly Bifidobacteria, may lower cholesterol by binding it in the intestines and promoting fecal excretion (Brown *et al*., 1999; Pato *et al*., 2004, 2005). Soluble fibers in the dietary food bind to bile acids in the intestine, preventing their reabsorption. Since bile acids are made from cholesterol, their increased excretion forces the liver to use more cholesterol to



Cholesterol level category: Normal levels (<200 mg/dL), Borderline high (200-239 mg/dL) and High (≥ 240 mg/d)
Glucose levels category: Normal (≤140 mg/dL), Pre-diabetes (140-199 mg/dL) and Diabetes (≥ 200 mg/dL)
Uric acid levels category: Normal (3.4-7.0 mg/dL), Moderate (7.1-9.0 mg/dL) and high (≥ 9.0 mg/dL)

Fig 3: Blood total cholesterol, uric acid and glucose levels category of adult male subjects.

synthesize new bile acids, thereby reducing circulating LDL-cholesterol levels. The viscous gel formed by soluble fibers can trap cholesterol and other lipids, reducing their absorption in the small intestine (Gunness and Gidley, 2010). These findings underscore the potential of probiotics in managing cholesterol and highlight the need for further exploration of gut microbiome-metabolism interactions.

Consuming meals high in fiber, such as soluble fibers and resistant starch, is crucial to increasing the production of SCFA. Additionally, fermented foods may provide healthy bacteria that support the synthesis of SCFAs. The gut microbiota ferments complex carbohydrates and fibers into SCFAs, such as acetate, propionate and butyrate, influencing blood glucose regulation. All subjects had normal blood glucose levels (<140 mg/dL), but bivariate analysis showed no correlation between blood glucose and fecal microbes (TPC, LAB and Bifidobacteria). Other gut microbes, including Bacteroides, *Akkermansia muciniphila* and *Faecalibacterium prausnitzii*, regulate glucose metabolism through SCFA production and improving insulin sensitivity (Cani *et al.,* 2007; Duncan *et al.,* 2009; Everard *et al.,* 2013; Qin *et al.,* 2012). Regarding uric acid, 70% of subjects had normal levels, while 30% had elevated levels, likely due to high-purine diets. Bivariate analysis showed a correlation between Bifidobacteria and uric acid levels, suggesting their role in uric acid metabolism. Bifidobacteria produce enzymes that degrade uric acid into allantoin, preventing crystal formation (Cao *et al.,* 2017; Wang *et al.,* 2021). SCFA levels in this study were within normal ranges, with acetic acid (71.89 µmol/g), propionic acid (3.99 µmol/g) and butyric acid (3.47 µmol/g). SCFA production is influenced by fiber intake, gut health and microbiota composition, with LAB and Bifidobacteria contributing to butyrate and other SCFAs (Louis and Flint, 2009). SCFAs benefit health by reducing inflammation and improving metabolism (Blaak *et al.,* 2020; Lopez-Siles *et al.,* 2017). Additionally, *Akkermansia muciniphila* and *Ruminococcus* spp. produce acetate and propionate, enhancing metabolic health (Derrien *et al.,* 2010; Flint *et al.,* 2012).

CONCLUSION

The findings showed individual variations in TPC, LAB and Bifidobacteria levels among subjects. A correlation was observed between microflora counts, blood and fecal SCFA levels and total cholesterol, glucose and uric acid. While most subjects had average glucose and cholesterol levels, 30% had moderate to high uric acid. Although no direct association was found between TPC, LAB and Bifidobacteria in bivariate analysis, microflora plays a key role in cholesterol and glucose metabolism. Notably, Bifidobacteria were correlated with uric acid levels. SCFA levels varied, with acetic acid being the most abundant, followed by propionic and butyric acids. The study suggests that microflora composition influences cholesterol, glucose, uric acid and SCFA levels.

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Disclaimers

The views and conclusions expressed in this article are solely those of the authors and do not necessarily represent the views of their affiliated institutions. The authors are responsible for the accuracy and completeness of the information provided but do not accept any liability for any direct or indirect losses resulting from using this content.

Informed consent

The Committee of Experimental Animal Care approved all animal procedures for experiments and handling techniques were approved by the University of Animal Care Committee.

Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this article. No funding or sponsorship influenced the study's design, data collection, analysis, publication decision, or manuscript preparation.

REFERENCES

Aggarwal, R., Bains, K. and Jain, R. (2023). Effect of milk or milk derived food supplementation on body composition of young Indian women. *Asian Journal of Dairy and Food Research*. **42(3):** 348-354. doi: 10.18805/ajdfr.DR-2066.

Barclay, A.W., Petocz, P., McMillan-Price, J., Flood, V. M., Prvan, T., Mitchell, P. and Brand-Miller, J. C. (2008). Glycemic index, glycemic load and chronic disease risk-A meta-analysis of observational studies. *The American Journal of Clinical Nutrition*. **87(3):** 627-637.

Blaak, E.E., Canfora, E.E., Theis, S., Frost, G., Groen, A.K., Mithieux, G., Nauta, A., Scott, K., Stahl, B., van Harsselaar, J., van Tol, R., Vaughan, E.E. and Verbeke, K. (2020). Short-chain fatty acids in human gut and metabolic health. *Beneficial Microbes*. **11(5):** 411-455.

Bray, G.A. and Bouchard, C. (2020). The biology of human overfeeding: A systematic review. *Obesity Reviews*. **21(9):** e13040.

Brown, L., Rosner, B., Willett, W.W. and Sacks, F.M. (1999). Cholesterol-lowering effects of dietary fiber: A meta-analysis. *The American Journal of Clinical Nutrition*. **69(1):** 30-42.

Canfora, E.E., Jocken, J.W. and Blaak, E.E. (2015). Short-chain fatty acids in control of body weight and insulin sensitivity. *Nature Reviews Endocrinology*. **11(10):** 577-591.

Cani, P.D., Amar, J., Iglesias, M.A., Poggi, M., Knauf, C., Bastelica, D., *et al.* (2007). Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes*. **56(7):** 1761-1772.

Cao, T., Li, X., Mao, T., Liu, H., Zhao, Q., Ding, X., Li, C., Zhang, L. and Tian, Z. (2017). Probiotic therapy alleviates hyper-uricemia in C57BL/6 mouse model. *Biomedical Research*. **28(5):** 2244-2249.

Choi, H.K., Atkinson, K., Karlson, E.W., Willett, W. and Curhan, G. (2004). Purine-rich foods, dairy and protein intake and the risk of gout in men. *New England Journal of Medicine*. **350(11):** 1093-1103.

Den Besten, G., van Eunen, K., Groen, A.K., Venema, K., Reijngoud, D.J. and Bakker, B.M. (2013). The role of short-chain fatty acids in the interplay between diet, gut microbiota and host energy metabolism. *Journal of Lipid Research*. **54(9):** 2325-2340.

Derrien, M., van Passel, M.W.J., van de Bovenkamp, J.H.B., Schipper, R., de Vos, W. and Dekker, J. (2010). Mucin-bacterial interactions in the human oral cavity and digestive tract. *Gut Microbes*. **1(4):** 254-268.

Duncan, S.H., Louis, P., Thomson, J.M. and Flint, H.J. (2009). The role of pH in determining the species composition of the human colonic microbiota. *Environmental Microbiology*. **11:** 2112-2122.

Everard, A., Belzer, C., Geurts, L., Ouwerkerk, J.P., Druart, C., Bindels, L.B., Guiot, Y., Derrien, M., Muccioli, G.G., Delzenne, N.M., de Vos, W.M. and Cani, P.D. (2013). Cross-talk between *Akkermansia muciniphila* and intestinal epithelium controls diet-induced obesity. *Proceedings of the National Academy of Sciences*. **110(22):** 9066-9071.

Feng, Z., Zhang, Y., Lai, Y., Jia, C., Wu, F. and Chen, D. (2024). Causal relationship between gut microbiota and kidney diseases: A two-sample Mendelian randomization study. *Frontiers in Immunology*. **14:** 1277554.

Flint, H.J., Scott, K.P., Duncan, S.H., Louis, P. and Forano, E. (2012). Microbial degradation of complex carbohydrates in the gut. *Gut Microbes*. **3(4):** 289-306.

Gunness, P. and Gidley, M.J. (2010). Mechanisms underlying the cholesterol-lowering properties of soluble dietary fibre polysaccharides. *Food and Function*. **1:** 149-155.

Hall, K.D. and Guo, J. (2017). Obesity energetics: Body weight regulation and the effects of diet composition. *Gastroenterology*. **152(7):** 1718-1727.e3.

Han, S., Lu, Y., Xie, J., Fei, Y., Zheng, G., Wang, Z., Liu, J., Lv, L., Ling, Z., Berglund, B., Yao, M. and Li, L. (2021). Probiotic gastrointestinal transit and colonization after oral administration: A long journey. In Frontiers in Cellular and Infection Microbiology (Vol. 11). Frontiers Media S.A.

Hasibuan, F.E.B. and Kolondam, B.J. (2017). Interactions between the gut microbiota and the human immune system. *Journal Ilmiah Sains*. **17(1):** 35-42.

Hill, J.O. and Wyatt, H.R. (2005). Role of physical activity in preventing and treating obesity. *Journal of Applied Physiology*. **99(2):** 765-770.

Jenkins, D.J., Wolever, T.M., Taylor, R.H., Barker, H., Fielden, H., Baldwin, J.M., Bowling, A.C., Newman, H.C., Jenkins, A.L. and Goff, D.V. (1981). Glycemic index of foods: A physiological basis for carbohydrate exchange. *The American Journal of Clinical Nutrition*. **34(3):** 362-366.

Johnson, R.J., Segal, M.S., Sautin, Y., Nakagawa, T., Feig, D.I., Kang, D.H., Gersch, M.S., Benner, S. and Sánchez-Lozada, L.G. (2007). Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease and cardiovascular disease. *The American Journal of Clinical Nutrition*. **86(4):** 899-906.

Koh, A., De Vadder, F., Kovatcheva-Datchary, P. and Bäckhed, F. (2016). From dietary fiber to host physiology: Short-chain fatty acids as key bacterial metabolites. *Cell*. **165(6):** 1332-1345.

Lopez-Siles, M., Duncan, S.H., Garcia-Gil, L.J. and Martinez-Medina, M. (2017). *Faecalibacterium prausnitzii*: from microbiology to diagnostics and prognostics. *The ISME Journal*. **11(4):** 841-852.

Louis, P. and Flint, H.J. (2009). Diversity, metabolism and microbial ecology of butyrate-producing bacteria from the human large intestine. *FEMS Microbiology Letters*. **294(1):** 1-8.

Mahapatra, D. and Das, M. (2022). Health-promoting effects of developed probiotic orange beverage: An *in vitro* study. *Asian Journal of Dairy and Food Research*. **41(4):** 403-409. doi: 10.18805/ajdfr.DR-1690.

Mensink, R.P., Zock, P.L., Kester, A.D. and Katan, M.B. (2003). Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: A meta-analysis of 60 controlled trials. *The American Journal of Clinical Nutrition*. **77(5):** 1146-1155.

Morrison, D.J. and Preston, T. (2016). Formation of short chain fatty acids by the gut microbiota and their impact on human metabolism. *Gut Microbes*. **7(3):** 189-200.

Mozaffarian, D., Micha, R. and Wallace, S. (2010). Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: A systematic review and meta-analysis of randomized controlled trials. *PLoS Medicine*. **7(3):** e1000252.

Pato, U., Ali, M. and Parlindungan, A.K. (2005). Taurocholate deconjugation and cholesterol binding by indigenous dadih lactic acid bacteria. *Hayati*. **12(3):** 103-107.

Pato, U., Surono, I.S., Koesnandar and Hosono, A. (2004). Hypocholesterolemic effect of indigenous dadih lactic acid bacteria by deconjugation of bile salts. *Asian-Australasian Journal of Animal Sciences*. **17(12):** 1741-1745.

Qin, J., Li, Y., Cai, Z., Li, S., Zhu, J., Zhang, F., Liang, S., Zhang, W., Hansen, T., Sanchez, G., Raes, J., Falony, G., Okuda, S. and Almeida, M. (2012). A metagenome-wide association study of gut microbiota in type 2 diabetes. *Nature*. **490(7418):** 55-60.

Rathod, A.C., Khedkar, C.D., Sarode, A.R. and Kalyankar, S.D. (2025). Commercially available probiotic products for wellbeing of mankind, livestock and fishery/: A review. *Asian Journal of Dairy and Food Research*. **44(1):** 1-7.

Rinninella, E., Raoul, P., Cintoni, M., Franceschi, F., Miggiano, G.A.D., Gasbarrini, A. and Mele, M.C. (2019). What is the healthy gut microbiota composition? A changing ecosystem across age, environment, diet and diseases. *Microorganisms*. **7(1):** 14.

Robinson, G.A., Peng, J., Peckham, H., Radziszewska, A., Butler, G., Pineda-Torra, I., Jury, E.C. and Ciurtin, C. (2021). Sex hormones drive changes in lipoprotein metabolism. *Science*. **24(11):** 103257.

Rowland, I., Gibson, G., Heinken, A., Scott, K., Swann, J., Thiele, I., Tuohy, K. and Rowland, I. (2018). Gut microbiota functions: Metabolism of nutrients and other food components. *European Journal of Nutrition*. **57(1):** 1-24.

Sarkar, A. and Mandal, S. (2016). Bifidobacteria-insight into clinical outcomes and mechanisms of its probiotic action. *Microbiological Research*. **192:** 159-171.

Slavin, J. (2013). Fiber and prebiotics: Mechanisms and health benefits. *Nutrients*. **5(4):** 1417-1435.

Sutrisna, R., Ekowati, C.N., Farisi, S. and Setyawan, H.V. (2017). The viability test of lactic acid bacteria from intestine in preparation on poultry ration. *Journal Ilmiah Peternakan Terpadu*. **5(3):** 53-57.

Tong, S., Zhang, P., Cheng, Q., Chen, M., Chen, X., Wang, Z., Lu, X. and Wu, H. (2022). The role of gut microbiota in gout: Is gut microbiota a potential target for gout treatment. *Frontiers in Cellular and Infection Microbiology*. **24(12):** 1051682.

Vernocchi, P., Del Chierico, F. and Putignani, L. (2016). Gut microbiota profiling: Metabolomics based approach to unravel compounds affecting human health. *Frontiers in Microbiology*. **7:** 1144.

Wang, J., Chen, Y., Zhong, H., Chen, F., Regenstein, J. and Hu, X. (2021). The gut microbiota as a target to control hyperuricemia pathogenesis: Potential mechanisms and therapeutic strategies. *Critical Reviews in Food Science and Nutrition*. **62(14):** 3979-3989.

Zhang, W., Wang, T., Guo, R., Cui, W., Yu, W., Wang, Z., Jiang, Y., Jiang, M., Wang, X., Liu, C., Xiao, J., Shang, J., Wen, X. and Zhao, Z. (2022). Variation of serum uric acid is associated with gut microbiota in patients with diabetes mellitus. *Frontiers in Cellular and Infection Microbiology*. **11:** 761757.

Zheng, Y., Qin, C., Wen, M., Zhang, L. and Wang, W. (2024). The effects of food nutrients and bioactive compounds on the gut microbiota: A Comprehensive review. *Foods*. **13(9):** 1345.