BMC Cardiovascular Disorders

RESEARCH

Open Access

# Association of dietary index of gut microbiota with cardiovascular disease risk: new evidence from NHANES 2007–2018

Jiameng Jin[1], Xingang Sun[2*] and Lihong Wang[2*]

## Abstract

**Background** The dietary index of gut microbiota (DI-GM) is a newly proposed index for assessing dietary quality, and studies on its association with cardiovascular disease (CVD) are limited. This study aimed to investigate the association between DI-GM and the prevalence of CVD.

**Methods** We utilized data from the National Health and Nutrition Examination Survey (NHANES). Logistic regression analyses were performed to examine the association between DI-GM and CVD. Smoothed curve fitting was employed to explore potential nonlinear relationships. Additionally, subgroup analyses were conducted to assess the stability of the results.

**Results** The study included 22,590 participants, of whom 20,216 had no CVD and 2,374 had CVD. After adjusting for all covariates, the DI-GM score was significantly negatively associated with CVD risk, with a 4% reduction in CVD risk for each unit increase in DI-GM score (OR = 0.96, 95% CI: 0.94–0.99, $P = 0.015$). Notably, the highest DI-GM score group (6–12) had a 13% lower risk of CVD compared to the lowest DI-GM score group (0–3) (OR = 0.87, 95% CI: 0.76-1.00, $P = 0.048$).

**Conclusion** The research results indicate that a higher DI-GM score protects against CVD, providing crucial empirical support for dietary intervention strategies based on gut microbiota modulation.

**Clinical trial number** Not applicable.

**Keywords** Dietary Index of Gut Microbiota (DI-GM), Cardiovascular disease (CVD), Lipopolysaccharide (LPS), National health and nutrition examination survey (NHANES)

*Correspondence:
Xingang Sun
17816890299@163.com
Lihong Wang
wanglhnew@126.com
[1]Second Clinical Medical College, Zhejiang Chinese Medical University, Hangzhou 310053, China
[2]Department of Cardiology Zhejiang Provincial People's Hospital, People's Hospital of Hangzhou Medical College, 158 Shangtang Road, Hangzhou, Zhejiang Province, China

© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by-nc-nd/4.0/.

## Introduction

Cardiovascular disease (CVD) represents the leading cause of morbidity and mortality globally [1, 2], which significantly impacts public health and healthcare systems. The aging population is experiencing a dramatic increase in CVD prevalence and incidence [3], placing further strain on resources and highlighting the urgent need for preventative strategies and novel therapeutic targets. While traditional risk factors such as hypertension, dyslipidemia, and diabetes are well-established contributors to CVD [4, 5], unhealthy dietary patterns also play a significant role. Specifically, diets high in processed foods and animal fats, combined with low intake of plant-based foods, can disrupt the balance of the gut microbiota. This dysbiosis compromises intestinal barrier integrity, leading to increased intestinal inflammation and translocation of bacterial endotoxins, such as lipopolysaccharide (LPS), into the bloodstream. Subsequently, systemic low-grade inflammation ensues. The resultant inflammatory milieu activates immune cells, promoting atherogenesis. Concurrently, altered gut microbial metabolism produces detrimental metabolites, such as trimethylamine N-oxide (TMAO), which directly impair vascular endothelial function. Furthermore, neurohumoral pathways modulated by gut microbiota dysbiosis influence cardiovascular activity. These interconnected processes culminate in an increased risk of cardiovascular disease, manifested by vascular dysfunction, hypertension, and thrombosis [6, 7].

The intricate relationship between dietary patterns and gut microbiota is increasingly recognized as a crucial determinant of overall health, including cardiovascular well-being [8, 9]. While specific dietary components, such as fiber [10], fermented foods [11], and diverse plant-based food [12], have demonstrated the capacity to positively modulate gut microbial composition and function, traditional dietary indices, including the Healthy Eating Index (HEI) and the Mediterranean Diet Score (MDS), despite their established value in assessing diet quality, have shown inconsistent associations with specific markers of gut microbial diversity and richness [13, 14]. For example, Bowyer et al. [15], utilising data from the TwinsUK cohort, compared the ability of HEI, MDS, and other indices to explain inter-individual variations in gut microbiota. They observed that while the HEI exhibited superior performance in capturing overall microbial community variance, the associations of these indices with specific gut bacterial taxa were not consistently aligned with expectations. Furthermore, Del Chierico et al. [16] postulated that MDS should correlate well with gut microbial composition, given its established links with health. However, the observed associations of MDS with health parameters and the gut microbiota were surprisingly weak. In contrast, the HEI showed a stronger association with the gut microbiota. This suggests that traditional dietary indices may not fully capture the complex interplay between diet and gut microbial ecosystems. Moreover, the generalizability of these indices across diverse populations is limited. For instance, the HEI has been shown to perform less effectively than the MDS in older populations [17]. These findings underscore that existing dietary indices may not adequately capture the relevant dietary factors associated with alterations in gut microbial composition, thus highlighting the need for more targeted dietary assessment tools.

Novel dietary indices are emerging to address the limitations of current approaches and provide a more nuanced understanding of the complex interactions between diet and gut microbiota. The dietary index of the gut microbiome (DI-GM) was designed to quantify dietary intake patterns related to the composition and function of the gut microbiota. Such a tool could be handy for unraveling the intricate relationship between diet and shedding light on the specific functions of gut microbiota. Although associations between the DI-GM index and various health outcomes have been explored, investigations into its relationship with CVD risk remain relatively scarce. Moreover, given that dietary interventions are more straightforward to implement, more cost-effective, and generally better tolerated by individuals, we therefore aim to investigate the association between DI-GM scores and CVD risk. This investigation seeks to provide further insights into the role of diet and gut microbiome modulation in influencing CVD. Ultimately, it may help identify potential dietary interventions for preventing and managing CVD.

### Study population

The data for this study were sourced from the National Health and Nutrition Examination Survey (NHANES), a nationally representative study that evaluated the nutritional status and general health of adults and children in the US. The NCHS Institutional Review Board authorised NHANES, all procedures complied with applicable regulations, and each participant gave written informed permission [18]. We performed a preliminary analysis of the data of 59,864 participants from 2007 to 2018. After meticulously excluding 3,963participants due to missing DI-GM data, 25,416 participants missing CVD data, 286 participants missing body mass index (BMI) data, 3160 participants with missing alcohol use data, and 4449 participants with missing poverty income ratio data, we ultimately included 22,590 participants in this study. The study procedure is illustrated in Fig. 1.

### Definition of CVD

A diagnosis of CVD was confirmed by self-report collected during a structured interview. That is, whether a

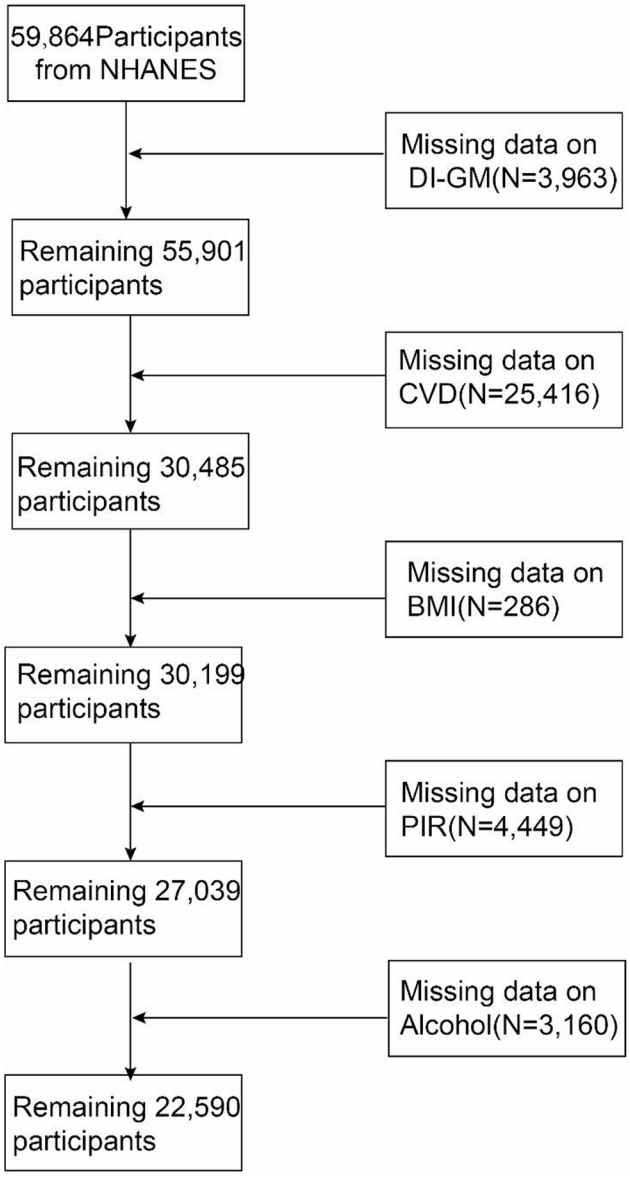


**Fig. 1** Flowchart showing the selection of the studied population

healthcare professional had diagnosed them with congestive heart failure, myocardial infarction, angina, or coronary artery disease. An affirmative response to any of these inquiries identified the participant as having CVD.

### Definition of DI-GM

DI-GM is a literature-based dietary assessment tool designed to quantify the impact of specific foods or nutrients on the gut microbiota. This index incorporates 14 components, categorised as either beneficial (e.g., fermented dairy products, chickpeas, whole grains) or detrimental (e.g., red meat, processed meats, refined grains) based on their potential effects on gut microbial diversity, short-chain fatty acid (SCFA) production, and the Firmicutes-to-Bacteroidetes ratio. The DI-GM scoring method utilises dietary data from the NHANES survey. It employs a weighted scoring system based on whether an individual's intake meets or exceeds sex-specific medians, resulting in a cumulative score ranging from 0 to 13 [19, 20].

## Covariates

To control for potential confounding, we adjusted for gender, age, race, educational level, marital status, physical exercise, medium movement, smoking and alcohol use, poverty income ratio, BMI, total cholesterol, high-density lipoprotein, chronic kidney disease, hypertension, and alcohol use. Diabetes status was self-reported and professionally verified. Further details on covariate definitions are available in the NHANES documentation.

## Statistical analysis

Data were analysed using Empower Stats v2.0 and R v3.4.3. Participant differences were assessed using descriptive statistics; continuous variables (mean ± standard deviation [SD] or standard error [SE]) were compared using t-tests, whereas categorical variables (classified as proportions) were analysed using chi-square tests. Multivariate logistic regression models examining the independent association of DI-GM with CVD risk reported odds ratios (ORs) with 95% confidence intervals (CIs) to quantify the strength of the effect. To assess the robustness of the observed associations and potential sex, age, BMI, hypertension, and diabetes-specific impact, we performed stratified analyses and interaction analyses to explore the association of DI-GM with CVD in populations with different characteristics. In addition, we investigated the nonlinear association of DI-GM with CVD using smoothed curve fitting. Statistical significance was defined as $P < 0.05$.

## Results

### Description of participants

Table 1 presents the baseline characteristics of the 22,590 NHANES participants, categorised by the presence or absence of cardiovascular disease. The demographic composition of the participants was as follows: 15.19% Mexican American, 10.06% other Hispanic, 44.26% non-Hispanic White, 20.00% non-Hispanic Black, and 10.49% from other racial groups. Among a range of variables, including gender, age, race, educational level, marital status, diabetes, physical exercise, medium movement, smoking, and alcohol use, hypertension, chronic kidney disease, PIR (poverty income ratio), High-density lipoprotein (HDL), TC (total cholesterol), DI-GM, poverty income ratio, BMI, total cholesterol, high-density lipoprotein, chronic kidney disease, hypertension, there were significant differences between the two groups ($P < 0.001$).

**Table 1** Characteristics of NHANES participants, 2007–2018

| Variables | Without CVD | With CVD | *P*-value |
|---|---|---|---|
| | *N* = 20,216 | *N* = 2,374 | |
| **Gender**, **%** | | | < 0.001 |
| Male | 10,018 (49.55%) | 1,408 (59.36%) | |
| Female | 10,198 (50.45%) | 964 (40.64%) | |
| **Age**, **%** | | | < 0.001 |
| <60 | 15,043 (74.41%) | 15,043 (74.41%) | |
| ≥ 60 | 5,173 (25.59%) | 1,677 (70.70%) | |
| **Race**, **%** | | | < 0.001 |
| Mexican American | 3,070 (15.19%) | 197 (8.31%) | |
| Other Hispanic | 2,034 (10.06%) | 180 (7.59%) | |
| Non-Hispanic White | 8,948 (44.26%) | 1,348 (56.83%) | |
| Non-Hispanic Black | 4,044 (20.00%) | 512 (21.54%) | |
| Other Race - Including Multi-Racial | 2,120 (10.49%) | 136 (5.73%) | |
| **Educational level**, **%** | | | < 0.001 |
| Less Than 9th Grade | 1,587 (7.85%) | 316 (13.31%) | |
| 9-11th Grade (Includes 12th grade with no diploma) | 2,680 (13.25%) | 395 (16.65%) | |
| High School Grad/GED or equivalent | 4,622 (22.86%) | 620 (26.14%) | |
| Some College or AA degree | 6,250 (30.92%) | 672 (28.33%) | |
| College Graduate or above | 5,077 (25.11%) | 369 (15.56%) | |
| **Marital status**, **%** | | | < 0.001 |
| Married | 10,373 (51.31%) | 1,226 (51.69%) | |
| Widowed | 1,190 (5.89%) | 429 (18.09%) | |
| Divorced | 2,222 (10.99%) | 356 (15.01%) | |
| Separated | 664 (3.28%) | 84 (3.54%) | |
| Never married | 3,959 (19.58%) | 173 (7.29%) | |
| Living with partner | 1,808 (8.94%) | 104 (4.38%) | |
| **BMI**, **%** | | | < 0.001 |
| <25 | 5,948 (29.42%) | 508 (21.42%) | |
| ≥ 25 | 14,268 (70.58%) | 1,864 (78.58%) | |
| **Diabetes**, **%** | | | < 0.001 |
| Yes | 2,121 (10.49%) | 818 (34.49%) | |
| No | 18,095 (89.51%) | 1,554 (65.51%) | |
| **Physical exercise**, **%** | | | < 0.001 |
| Yes | 4,409 (21.81%) | 326 (13.74%) | |
| No | 15,807 (78.19%) | 2,046 (86.26%) | |
| **Medium Movement**, **%** | | | < 0.001 |
| Yes | 8,111 (40.12%) | 762 (32.12%) | |
| No | 12,105 (59.88%) | 1,612 (67.88%) | |
| **Smoke**, **%** | | | < 0.001 |
| Yes | 9,117 (45.10%) | 1,514 (63.83%) | |
| No | 11,099 (54.90%) | 858 (36.17%) | |
| **Alcohol use**, **%** | | | 0.1 |
| Yes | 9,628 (47.63%) | 1,172 (49.41%) | |
| No | 10,588 (52.37%) | 1,200 (50.59%) | |
| **Hypertension**, **%** | | | < 0.001 |
| Yes | 6,286 (31.09%) | 1,758 (74.11%) | |
| No | 13,930 (68.91%) | 614 (25.86%) | |
| **Chronic kidney disease**, **%** | | | < 0.001 |
| Yes | 439 (2.17%) | 254 (10.71%) | |
| No | 19,777 (97.83%) | 2,118 (89.29%) | |
| **PIR** | 2.59 ± 1.64 | 2.23 ± 1.49 | < 0.001 |
| **HDL** | 53.21 ± 16.15 | 49.83 ± 15.88 | < 0.001 |

**Table 1** (continued)

| Variables | Without CVD | With CVD | *P*-value |
|---|---|---|---|
| | *N* = 20,216 | *N* = 2,374 | |
| **TC** | 194.51 ± 41.05 | 179.66 ± 44.61 | < 0.001 |
| **DI-GM** | 4.95 ± 1.71 | 4.81 ± 1.72 | < 0.001 |

DI-GM, the dietary index for gut microbiota. PIR, poverty income ratio. BMI, body mass index. CVD, cardiovascular disease.TC, total cholesterol. HDL, high-density lipoprotein. Continuous variables were expressed as weighted means and standard errors, while categorical variables were expressed as weighted percentages. For continuous variables, the *p*-value was based on the analysis of variance (ANOVA), and for categorical variables, the *p*-value was based on the chi-square test. A higher DI-GM score indicates a healthier gut microbiota

**Table 2** Association between DI-GM and CVD, NHANES 2007–2018

| CVD | OR (95% CI), *P*-value | | |
|---|---|---|---|
| | Model I | Model II | Model III |
| **DI-GM** | 0.95 (0.93, 0.98) 0.0002 | 0.92 (0.89, 0.94) < 0.0001 | 0.96 (0.94, 0.99) 0.015 |
| **DI-GM group** | | | |
| 0–3 | 1.00(Reference) | 1.00(Reference) | 1.00(Reference) |
| 4–5 | 0.90 (0.81, 1.01) 0.078 | 0.88 (0.78, 0.99) 0.0364 | 0.94 (0.83, 1.06) 0.307 |
| 6–12 | 0.84 (0.75, 0.95) 0.004 | 0.72 (0.63, 0.81) < 0.0001 | 0.87 (0.76, 1.00) 0.048 |
| **P for trend** | 0.96 (0.93, 0.99) 0.004 | 0.92 (0.80, 0.95) < 0.0001 | 0.97 (0.93, 1.00) 0.045 |

DI-GM is the dietary index for gut microbiota, CVD, and cardiovascular diseases

Model I adjusted for none;

Model II adjusted for gender, age, and race;

Model III adjusted for gender, age, race, educational level, marital status, diabetes, physical exercise, medium movement, smoking, and alcohol use, poverty income ratio, body mass index, total cholesterol, high-density lipoprotein, chronic kidney disease, hypertension, and alcohol use

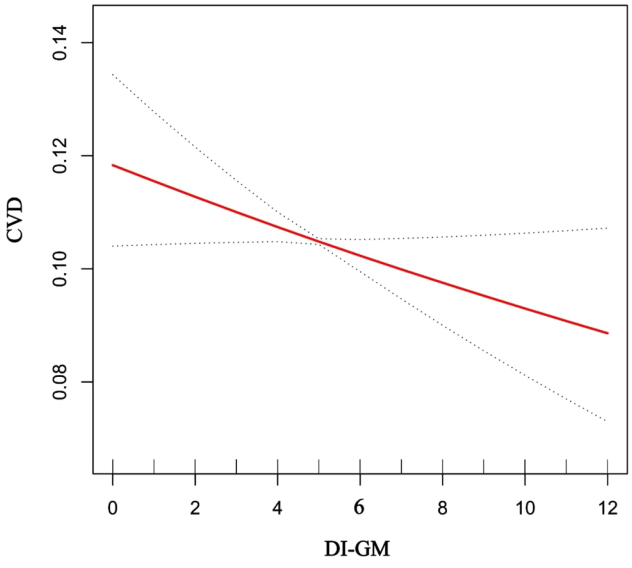


**Fig. 2** Correlation of DI-GM with Cardiovascular Disease

### Correlation between DI-GM and CVD

Logistic regression analysis (Table 2) showed a significant positive correlation between DI-GM and CVD with or without adjustment for covariates.

### Association of DI-GM with CVD

Multivariate analysis (Model III) revealed that the highest tertile of DI-GM was associated with a 4% lower CVD risk compared to the lowest tertile (OR = 0.96, 95% CI: 0.94–0.99, $P = 0.015$).Notably, Trend analysis showed a 3% reduction in CVD risk for each additional tertile group of DI-GM scores (OR = 0.97, 95% CI:0.93-1.00; $P$ for trend = 0.045).

### Smoothed curve fitting

We also visualised and analysed the results by smoothing curve fitting. As shown in Fig. 2, after adjusting for all covariates, there was a trend toward decreasing CVD with increasing DI-GM score (overall $P < 0.001$).

### Subgroup analysis

Subgroup analyses were performed to assess the stability of the relationship between DI-GM and CVD. As shown in Fig. 3, no significant interactions were found between gender, age, BMI, diabetes mellitus, and hypertension in the subgroup analyses ($P > 0.05$ for interaction).

Figure 3. Subgroup analysis between DI-GM and CVD

## Discussion

The present study observed a negative association between DI-GM scores and CVD risk, suggesting that a dietary pattern that promotes healthy gut microbiota, such as plant foods, whole grains, and fermented foods, may have protective effects on cardiovascular health. This finding is consistent with the association of gut flora dysbiosis with increased CVD risk in previous studies [21, 22]. In addition, our findings further support the notion that modulating gut flora through dietary interventions may improve cardiovascular health [23, 24]. Specifically, nutritional components covered by the DI-GM Index, such as fibre, polyphenols, and dietary precursors of SCFA, have been shown to have the ability to modulate the structure and function of the gut flora, which in turn influences host metabolic and immune responses [25, 26]. These alterations may reduce the risk of CVD through various pathways, including lowering systemic inflammation, improving lipid metabolism, and regulating blood pressure [27]. While our findings suggest an

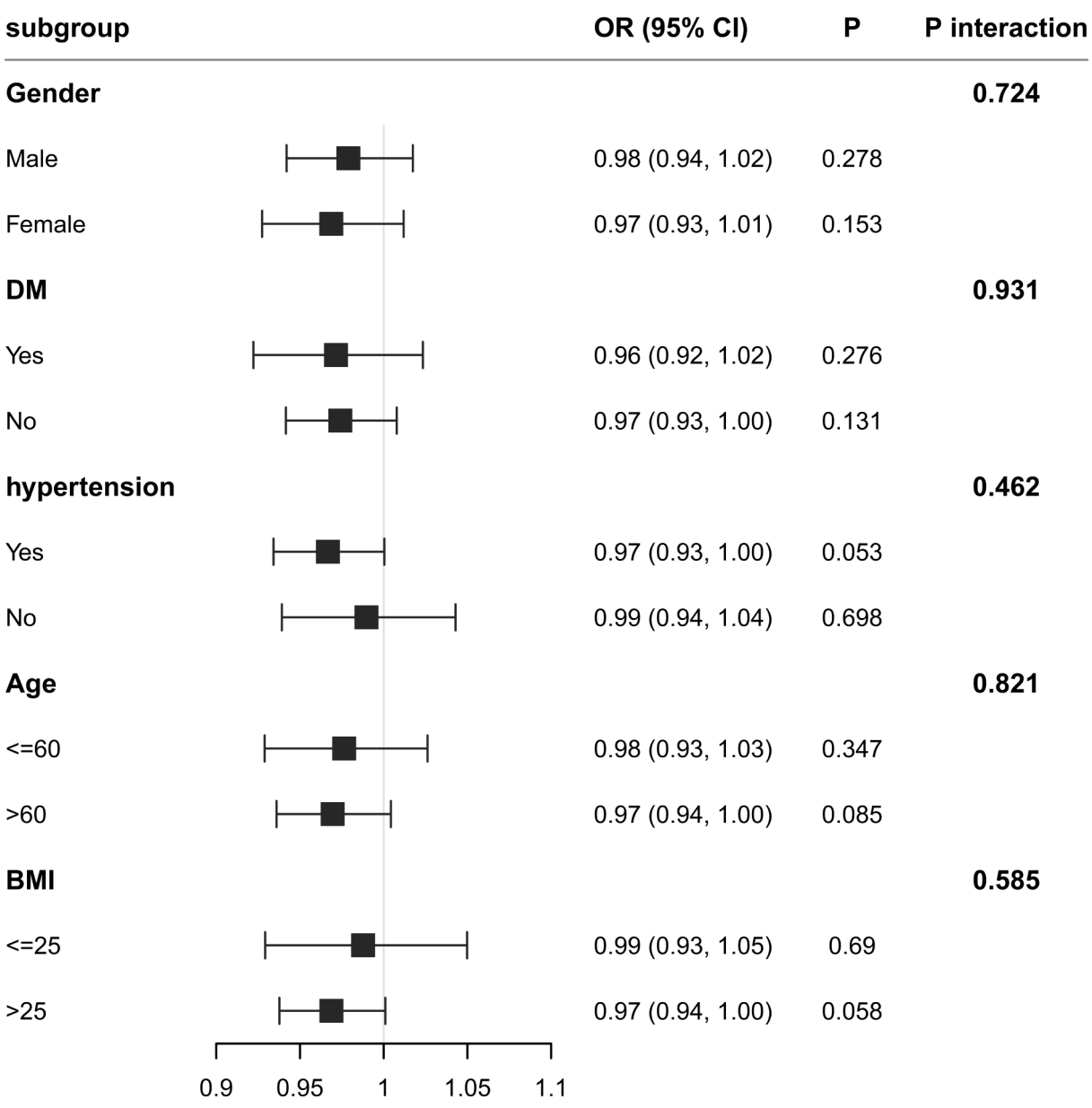


**Fig. 3** Correlation of DI-GM with Cardiovascular Disease

association between DI-GM and CVD risk, the underlying mechanism requires further investigation.

CVD is characterised by complex pathophysiological mechanisms, and intestinal dysfunction and its association with systemic inflammation are becoming increasingly prominent. Deng et al. demonstrated that patients with congestive heart failure (CHF) suffer from significant abnormalities of intestinal structure and function, which contribute to the progression of CVD through multiple mechanisms [28]. Specifically, impaired intestinal barrier functionthe, commonly referred to as "leaky gut," leads to the translocation of LPS into the bloodstream, triggering systemic inflammation and exacerbating vascular injury. Meanwhile, impaired intestinal microcirculation impairs intestinal barrier function and affects nutrient absorption. In addition, Wang et al. [29]. Showed that patients with CHF often suffer from intestinal dysbiosis, which is characterised by changes in the abundance of specific flora, such as an increase in Gram-negative bacteria and a decrease in SCFA-producing bacteria [30]. This dysbiosis affects CVD in several ways. Firstly, it exacerbates a "leaky gut," activates immune cells, and promotes vascular inflammation and atherosclerosis. Secondly, the gut flora is involved in the metabolism of dietary components (e.g., choline and carnitine), producing trimethylamine (TMA), which is oxidised in the liver to trimethylamine oxide (TMAO), which promotes thrombosis and increases the risk of CVD [31]. Furthermore, gut flora may indirectly

affecting influence host metabolism by affecting bile acid metabolism and SCFA production, thereby impacting cardiovascular health [30]. It is worth noting that the association between gut microbiota and CVD is not a simple linear causality but relatively complexly regulated by various factors such as host genetics, immune status, and lifestyle [32, 33]. Although studies targeting specific flora and metabolites have provided important clues, the particular mechanisms of gut flora in the development of CVD still need to be explored in depth to develop more effective CVD prevention and treatment strategies based on intestinal microecological regulation.

Hou et al. demonstrating that the dynamic balance of intestinal flora is intricately regulated by both environmental and host factors [34]. Environmental factors such as dietary patterns, geographic migration, and antibiotic use significantly affect the composition of the flora. In contrast, host phenotypes such as BMI, metabolic indicators, and disease states are also associated with dysbiosis. Genetic factors and family cohabitation also have an impact. Among these factors, diet is a key modifiable factor [35]. To quantify dietary patterns and their health effects, researchers have but developed a variety of dietary assessment tools, including the HEI and the MDS, which are widely used. However, these traditional dietary indices do not specifically assess the effects of diet on the gut microbiota. Their correlations with indicators of the diversity and abundance of the intestinal flora have been inconsistent [15, 36]. DI-GM, as a novel dietary index, offers several key advantages. Firstly, it is constructed based on evidence from the literature and focuses on a wide range of indicators, including gut microbiota diversity, SCFA production, and specific bacterial changes. Secondly, it is more targeted by including particular foods rather than food groups. Additionally, it incorporates beneficial components, such as fermented dairy and chickpeas, and distinguishes between unfavourable components. Furthermore, studies have confirmed that DI-GM correlates with biomarkers of gut microbiota diversity and is comparable, providing a reasonable basis for dietary assessment [36].

This study observed a negative correlation between DI-GM scores and CVD risk, a finding highlighting the role of diet as a potentially modifiable factor in regulating gut health and cardiovascular health. Several beneficial elements in the DI-GM, such as green tea and coffee, have been investigated and shown to have cardiovascular protective effects. Specifically, moderate coffee and green tea intake reduces the risk of CVD events such as coronary heart disease, heart failure, and stroke and positively affects metabolic syndrome [37, 38]. In addition, fermented foods are an essential component of DI-GM, and several studies have shown that fermented foods can potentially benefit cardiometabolic health by modulating gut flora, reducing inflammation and oxidative stress, and enhancing gut barrier function [39]. The mechanisms underlying these benefits may involve bioactive compounds produced during fermentation. These compounds can activate the Nrf2 pathway, which exerts cytoprotective effects and attenuates chronic inflammatory responses associated with obesity, atherosclerosis, and others [40]. Therefore, in-depth studies incorporating gut microbiome data are essential to comprehensively assess the practical value of DI-GM in the prevention and management of cardiovascular diseases. Future studies should focus on exploring the interactions between different food components in DI-GM and specific gut microbiota, as well as the effects of the resulting metabolites on the cardiovascular system, to provide the scientific basis for developing more precise and individualised dietary interventions.

## Strengths and limitations

Strengths of this study include the large and representative sample size, which allowed subgroup analyses by sex, age, body mass index, blood pressure, and blood glucose to assess the robustness of DI-GM to CVD. However, the cross-sectional study design limited the ability to infer a causal relationship between DI-GM and CVD. In addition, residual confounders may remain despite full covariate adjustment.

## Conclusion

Our findings suggest that DI-GM is significantly associated with increased CVD. These results provide a scientific basis for nutritional intervention strategies targeting CVD.

### Abbreviations

| | |
|---|---|
| DI-GM | Dietary Index of Gut Microbiota |
| CVD | Cardiovascular disease |
| LPS | Lipopolysaccharide |
| TMAO | Trimethylamine N-oxide |
| HEI | Healthy Eating Index |
| MDS | Mediterranean Diet Score |
| NHANES | National Health and Nutrition Examination Survey |
| SD | Standard deviation |
| SE | Standard error |
| ORs | Odds ratios |
| CIs | Confidence intervals |
| SCFA | Short-chain fatty acids |
| CHF | Congestive heart failure |
| TMA | Trimethylamine |
| TMAO | Trimethylamine oxide |
| PIR | Poverty income ratio |
| TC | Total cholesterol |
| HDL | High-density lipoprotein |

### Acknowledgements

Not applicable.

### Author contributions

J.J.: Project administration, Writing—review & editing, Writing—original draft, Investigation. X.S.: Validation, Conceptualization. L.W.: Funding acquisition, Project administration, Supervision, Writing—review & editing, Resources. All authors reviewed the manuscript.

**Funding**
This study was supported by the National Natural Science Foundation of China (No. 81670447), the Medicine and Health Project of Zhejiang Province (No. 2023KY531), the Traditional Chinese Medicine Program of Zhejiang Provincial (No. 2022ZZ003, No. 2023ZL248, 2022ZB024), and the Ten-thousand Talents Program of Zhejiang Province (No. 2021R52025).

**Data availability**
Publicly available datasets were analyzed in this study. This data can be found here: https://www.cdc.gov/nchs/nhanes/index.htm.

## Declarations

**Ethical approval**
The studies involving human participants were reviewed and approved by the NCHS Research Ethics Review Board (ERB). All participants provided written informed consent.

**Competing interests**
The authors declare no competing interests.

Received: 25 February 2025 / Accepted: 17 April 2025
Published online: 28 April 2025

**References**

1. Shilpa Sharma, Mark J. Sarna.Epidemiology.The global burden of reduced GFR: ESRD, CVD and mortality. Nat Rev Nephrol 2017,13(8):447–8.
2. Francesco Paneni CD, Cañestro P, Libby TF, Lüscher. The aging cardiovascular system: Understanding it at the cellular and clinical levels. Am Coll Cardiol 2017,18;69(15):1952–67.
3. RamonLuengo-Fernandez MW-A. et,al.Economic burden of cardiovascular diseases in the European union: a population-based cost study. Eur Heart J 2023,1;44(45):4752–67.
4. Emir Muzurović, Dimitri P, Mikhailidis C, Mantzoros. Non-alcoholic fatty liver disease, insulin resistance, metabolic syndrome, and their association with vascular risk. Metabolism 2021 Jun: 119:154770.
5. Foody J, Huo Y, Ji L, Zhao D, Boyd D, Meng HJ. Susan Shiff, Dayi Hu.Unique and varied contributions of traditional CVD risk factors: A systematic literature review of CAD risk factors in China. Clin Med Insights Cardiol 2013 Apr 4:7:59–86.
6. Chen J, Vitetta L. Gut microbiota metabolites in NAFLD pathogenesis and therapeutic implications. Int J Mol Sci. 2020;21:5214.
7. Claire LBoulangé, Chilloux ALNJ, Nicholson JK. Marc-Emmanuel Dumas. Impact of the gut microbiota on inflammation, obesity, and metabolic disease. Genome Med. 2016;8(1):42.
8. Ana M, Valdes J, Walter E, Segal. Tim D spector.role of the gut microbiota in nutrition and health.bmj.2018. Jun 13:361k2179.
9. Harry JF 1, Sylvia H. Duncan 1, Karen P Scott 1, Petra Louis 1. Linkages between diet, gut microbiota composition, and gut metabolism. Proc Nutr Soc.2015;74(1):13–22.
10. Simpson HL, Campbell BJ. Review Article: dietary fibre-microbiota interaction. Aliment Pharmacol Ther. 2015;42(2):158–79.
11. Stiemsma LT, Nakamura RE, Nguyen JG, Michels KB. Does consumption of fermented foods modify the human gut microbiota?? J Nutr. 2020;150:1680–92.
12. O'Keefe SJD, Li JV, Lahti L, Ou J, Carbonero F, Mohammed K, et al. Fat, fibre and cancer risk in African Americans and rural Africans. Nat Commun. 2015;6:6342.
13. Sofia Reddel L, Putignani. Federica Del Chierico.The impact of Low-FODMAPs, Gluten-Free, and ketogenic diets on gut microbiota modulation in pathological conditions. Nutrients.22019;11(2):373.
14. Sehgal K, Khanna S. Gut microbiota: a target for intervention in obesity. Expert Rev Gastroenterol Hepatol. 2021;15:1169–79.
15. Bowyer RCE, Jackson MA, Pallister T, Skinner J, Spector TD, Welch AA, et al. Use of dietary indices to control for diet in human gut microbiota studies. Microbiome. 2018;6:77.
16. Gianluca Ianiro M, Punčochář N, Karcher S, Porcari. Variability of strain engraftment and predictability of Microbiome composition after fecal microbiota transplantation across different disease. Nat Med. 2022;28(9):1913–23.
17. Hatløy A, Torheim LE. Food variety—a good indicator of nutritional adequacy of the diet? A case study from an urban area in Mali, West Africa. Eur J Clin Nutr, pages891–8.
18. Clifford L, Johnson R, Paulose-Ram CL, Ogde MD. Carroll Nati. Oral health and nutrition examination survey: analytic guidelines, 1999–2010. Vital Health Stat. 2013;2(161):1–24.
19. Zipf G, Chiappa M, Kathryn S. Porter.National health and nutrition examination survey: plan and operations, 1999–2010. Vital Health Stat. 2013;1(56):1–37.
20. Bezawit E, Kase AD, Liese. Jiajia Zhang the development and evaluation of a Literature-Based dietary index for gut microbiota. Nutrients. 2024;16(7):1045.
21. Tang WHW, Bäckhed F, Landmesser U, Hazen SL. Intestinal microbiota in cardiovascular health and disease: JACC State-of-the-Art review. J Am Coll Cardiol. 2019;73:2089–105.
22. Luqman A, Hassan A, Ullah M, Naseem S, Ullah M, Zhang L, et al. Role of the intestinal Microbiome and its therapeutic intervention in cardiovascular disorder. Front Immunol. 2024;15:1321395.
23. Wang Z, Klipfell E, Bennett BJ, Koeth R, Levison BS, Dugar B, et al. Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. Nature. 2011;472:57–63.
24. Marco Witkowski TL, Weeks, Stanley L. Hazen.Gut microbiota and cardiovascular disease. Circ Res. 2020;127(4):553–70.
25. Maryam, Hemmati. Setayesh Kashanipoor.Importance of gut microbiota metabolites in the development of cardiovascular diseases. Life Sci 22023 Sep 15:329:121947.
26. Edward S, Chambers T, Preston G, Fros, Douglas J. Morrison.Role of gut Microbiota-Generated Short-Chain fatty acids in metabolic and cardiovascular health. Curr Nutr Rep. 2018;7(4):198–206.
27. Battson ML, Lee DM, Weir TL, Gentile CL. The gut microbiota is a novel regulator of cardiovascular function and disease. J Nutr Biochem. 2018;56:1–15.
28. Tang WHW, Li DY, Hazen SL. Dietary metabolism, the gut microbiome, and heart failure. Nat Rev Cardiol. 2019;16:137–54.
29. Wang Z, Cai Z, Ferrari MW, Liu Y, Li C, Zhang T, et al. The correlation between gut microbiota and serum metabolomic in elderly patients with chronic heart failure. Mediators Inflamm. 2021;2021:5587428.
30. Douglas J, Morrison. Tom preston.formation of short chain fatty acids by the gut microbiota and their impact on human metabolism. Gut Microbes 2016.7(3):189–200.
31. Tang WHW, Wang Z, Fan Y, Levison B, Hazen JE, Donahue LM, et al. Prognostic value of elevated levels of intestinal Microbe-Generated metabolite Trimethylamine-N-Oxide in patients with heart failure: refining the gut hypothesis. J Am Coll Cardiol. 2014;64:1908–14.
32. Takeshi Kitai J, Kirsop WH, Wilson Tang. Exploring Microbiome Heart Fail Curr Heart Fail Rep. 2016;13(2):103–9.
33. Trøseid M, Ueland T, Hov JR, Svardal A, Gregersen. Microbiota-dependent metabolite trimethylamine-N-oxide is associated with disease severity and survival of patients with chronic heart failure. J Intern Med. 2015;277(6):717–26.
34. Hou K, Wu Z-X, Chen X-Y, Wang J-Q, Zhang D, Xiao C, et al. Microbiota in health and diseases. Signal Transduct Target Ther. 2022;7:135.
35. Hills RD, Pontefract BA, Mishcon HR, Black CA, Sutton SC, Theberge CR. Gut microbiome: profound implications for diet and disease. Nutrients. 2019;11:1613.
36. Cotillard A, Cartier-Meheust A, Litwin NS, Chaumont S, Saccareau M, Lejzerowicz F, et al. A posteriori dietary patterns better explain variations of the gut Microbiome than individual markers in the American gut project. Am J Clin Nutr. 2022;115:432–43.
37. Mesas AE, Leon-Muñoz LM, Rodriguez-Artalejo F, Lopez-Garcia E. Effect of coffee on blood pressure and cardiovascular disease in hypertensive individuals: a systematic evaluation and meta-analysis. Am J Clin Nutr. 2011;94:1113–26.
38. Mesas AE, Leon-Muñoz LM, Rodriguez-Artalejo F, Lopez-Garcia E. The effect of coffee on blood pressure and cardiovascular disease in hypertensive individuals: a systematic review and meta-analysis. Am J Clin Nutr. 2011;94:1113–26.
39. Mafra D, Borges NA, Alvarenga L, Ribeiro M, Fonseca L, Leal VO, et al. Fermented food: should patients with cardiometabolic diseases go back to an early neolithic diet? Crit Rev Food Sci Nutr. 2023;63:10173–96.
40. Mathur H, Beresford TP, Cotter PD. Health Benefits of Lactic Acid Bacteria (LAB) Fermentates. Nutrients. 2020;12:1679.

## Publisher's note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.