Nutrition Research 146 (2026) 40–52

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/NTR

Original Research

Moderate intake of bitter foods is associated with lower risk of type 2 diabetes: A secondary analysis of the 2011-12 National Nutrition and Physical Activity Survey

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ARTICLE INFO

Article history:
Received 8 September 2025
Revised 15 December 2025
Accepted 16 December 2025

Key words:
Dietary bitterness
Bitterness score
Cardiometabolic risk factors
Type 2 diabetes
Australian adults
Taste 2 receptors

ABSTRACT

The sensory properties of food are increasingly recognized for their potential role in regulating blood glucose and cardiometabolic risk factors. This cross-sectional secondary analysis of the 2011–2012 National Nutrition and Physical Activity Survey (NNPAS) aimed to examine the association between dietary bitterness and cardiometabolic risk in Australian adults aged ≥19 years. We hypothesize that higher food and beverage bitterness scores might be associated with lower risk of cardiometabolic risk factors. The study used data from 8975 participants; 225 (~2%) of the population had type 2 diabetes. Dietary bitterness was calculated as a "bitterness score" by multiplying sensory panel–assigned bitter taste values (from the Commonwealth Scientific and Industrial Research Organisation Sensory Diet Database) by the grams of each item consumed, and then summing these values separately for solid foods and for beverages within the NNPAS. Differences across dietary bitterness tertiles were assessed using analysis of variance for continuous variables and chi-squared tests for categorical variables. Multinomial logistic regression was used to assess the association between tertiles of bitterness score and cardiometabolic outcomes, including type 2 diabetes,

Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; GLP-1, glucagon-like peptide 1; HbA1c, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; NNPAS, National Nutrition and Physical Activity Survey; RRR, relative risk ratio; TAS2R, taste 2 receptor; TC, total cholesterol.

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https://doi.org/10.1016/j.nutres.2025.12.007

dyslipidemia, and blood pressure. Participants in the second tertile, but not the highest tertile of bitterness score from foods had lower risk of type 2 diabetes, as defined by fasting plasma glucose (relative risk ratio [RRR] = 0.30; 95% confidence intervals [CI], 0.15–0.60), and glycated hemoglobin (HbA1c) (RRR = 0.43; 95% CI, 0.25–0.75) compared to the lowest tertile. No significant associations were observed between bitterness scores derived from beverages and cardiometabolic risk factors. Consumption of foods with a medium bitterness score was inversely associated with type 2 diabetes risk in Australian adults and may have potential to be included in diabetes prevention efforts.

1. Introduction

Cardiometabolic diseases including obesity, type 2 diabetes, and cardiovascular disease are among the leading causes of noncommunicable diseases [1] and represent a growing global health and economic challenge [2]. In Australia, an estimated 47% of the population (>11 million people) live with 1 or more chronic health conditions, and noncommunicable diseases are responsible for 9 of 10 preventable deaths. Adherence to healthy dietary patterns such as the Australian dietary guidelines [3] or the Mediterranean diet [4] reduces the risk of chronic health conditions and represents one of the most important modifiable strategies for preventing cardiometabolic diseases [5,6]. Beyond nutritional composition, sensory attributes of food, such as bitterness, are increasingly recognized as important influences on food preferences, eating behaviors, and potentially cardiometabolic health [7,8].

Bitter compounds are an overarching name for chemicals that occur naturally in a range of foods and beverages such as brassica vegetables, dark chocolate, nuts, cheese, cocoa, some culinary plants, tea, coffee, beer, and wine [9]. The bitter taste of these foods is primarily attributed to the presence of alkaloids (in coffee, tea, chocolate, and soft drinks), polyphenols (in many fruits, vegetables, beans, and red wine), terpenoids (in citrus fruits), sulfur-containing amino acids, and some aromatic amino acids (in plant and animal proteins).

Emerging evidence suggests that bitter compounds may influence metabolic health through their interaction with bitter taste 2 receptors (TAS2Rs). Activation of TAS2Rs is reported to stimulate the release of gastrointestinal hormones, such as glucagon-like peptide 1 (GLP-1), ghrelin, and cholecystokinin [10–12], enhance insulin secretion, slow gastric emptying, and decrease gastrointestinal motility, which increases satiety [7,13–15]. Animal studies have demonstrated improved glucose tolerance and lowered blood lipids following bitter food consumption, without affecting total food intake [16,17]. Small human intervention studies further support that bitter compound-rich diets (e.g., high polyphenol diet [18] and bitter, strong-tasting vegetables [19]), improve glucose tolerance, insulin sensitivity, body fat mass, and blood pressure among adults at high risk of (or with) type 2 diabetes [19]. Epidemiologic research has also indicated that higher intake of certain bitter foods and beverages enriched in bitter compounds [20–28], including brassica/cruciferous and dark-green leafy vegetables [21–24], bitter beverages [20,25], grape products [27,28], dark chocolate, and cocoa [26], is associated with a reduced risk for type 2 diabetes and cardiovascular disease.

Although these studies provide evidence on the health effects of individual bitter foods, they do not reflect the health effects of dietary bitterness across the overall diet. Dietary bitterness refers to the overall intensity of the bitter taste experience from consumed foods and beverages. Therefore, it is unclear whether the overall sensory characteristics of diet, especially bitterness, contribute independently to cardiometabolic health. To address this gap, dietary analyses that integrate consumption data with a sensory database have been published [29–31]. Understanding the sensory determinants of eating patterns may reveal new approaches for public health nutrition programs, informing strategies beyond conventional nutrient advice to enhance diet quality and reduce the burden of disease. To build the evidence, the aim of this study was to cross-sectionally examine the association between dietary bitterness score and cardiometabolic risk factors including diabetes, dyslipidemia, and blood pressure, in a representative sample of Australian adults aged ≥19 years. We hypothesized that higher food and beverage bitterness scores might be associated with lower risk of cardiometabolic risk factors.

2. Methods

2.1. *Study design and populations*

This study is a secondary analysis of the 2011–12 National Nutrition and Physical Activity Survey (NNPAS), which is part of the 2011–2013 Australian Health Survey. The method for data collection in the Australian Health Survey is reported elsewhere [32]. Briefly, the NNPAS used a stratified multistage probability cluster sampling design to select a nationally representative sample of Australian households across urban and rural households in all states and territories [32]. One adult (≥18 years) and, if applicable, 1 child (2–17 years) from each selected household were randomly chosen to participate. A total of 12,153 Australians from 9519 households completed face-to-face interviews (response rate: 77%) [32]. Household and person weights were applied to ensure representativeness. Demographic variables included age, sex, and area-level socioeconomic status measured by the Socio-Economic Indexes for Areas. Lifestyle factors included smoking status (categorized as current smoker or never smoker) and physical activity level (categorized as high, moderate, or low/ sedentary,

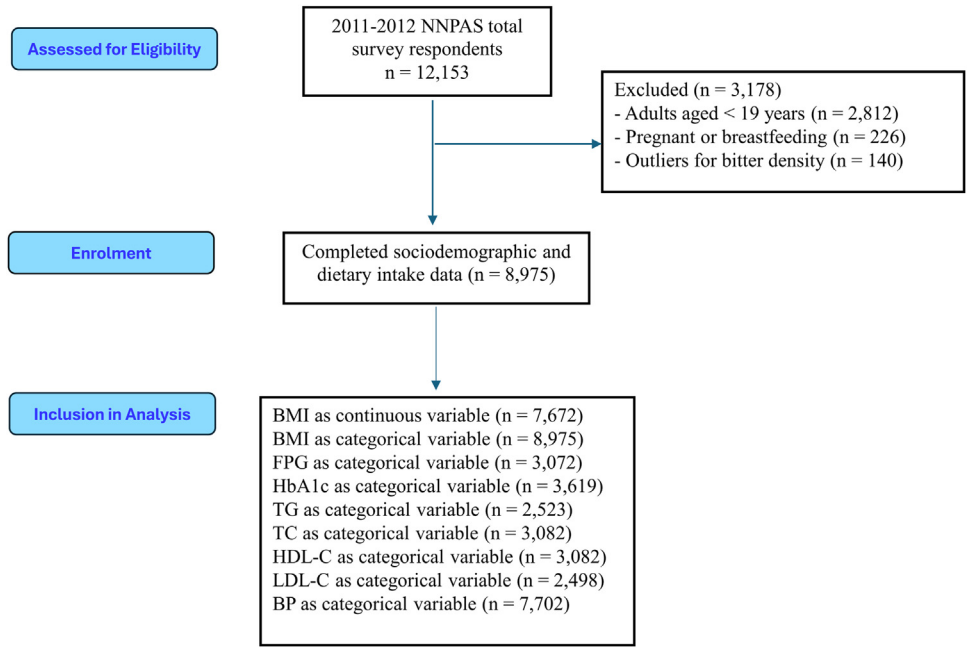

Fig. 1 – Strengthening the Reporting of Observational Studies in Epidemiology diagram of respondents included in the present study.

based on intensity and duration). Anthropometric measurements (height, weight, waist circumference, and body mass index [BMI]) were collected by trained personnel [32]. For this secondary analysis, individuals were excluded if they: (1) were younger than age 19 years, (2) were pregnant or lactating, or (3) were identified as misreporters based on statistical methods identifying extreme outliers for bitter density (Figure 1). The Strengthening the Reporting of Observational Studies in Epidemiology-nutritional epidemiology statement (Supplemental file 1) was used to report this study [33].

2.2. *Dietary intake assessment*

Dietary intake data were collected using two 24-hour dietary recalls conducted by trained interviewers using the Automated Multiple-Pass Method [32]. The first dietary recall was conducted through a face-to-face interview ($n = 12,153$), whereas the second recall, conducted via telephone at least 8 days later, was completed by 60% of the eligible participants ($n = 7735$). This analysis used data from the first dietary recall only to reduce potential nonresponse bias.

Dietary data were analyzed using the AUSNUT 2011–13 food composition database developed by Food Standards Australia and New Zealand. AUSNUT follows a hierarchical classification system, categorizing foods into major (2-digit), submajor (3-digit), minor (5-digit), and descriptive (8-digit) food groups. Based on the 3-digit food codes, food items were categorized as beverages, including drinking water, tea and coffee, fruit and vegetable juices, milk and liquid milk substitutes, soft drinks and other prepared beverages, and alcoholic beverages unless consumed as food. This separation was made due to anticipated differences in sensory and metabolic profiles between solid and liquid dietary components.

2.3. *CSIRO Sensory Diet database*

The CSIRO Sensory Diet database includes sensory characteristics of 724 foods and beverages and is described in detail elsewhere [29]. The bitter taste values of the tested foods and beverages ranged from 0 to 73.80 on a scale of 0 to 100 [34]. Bitter taste intensity, or value, is defined as the rate of bitterness perceived by the sensory panel. A systematic protocol was established to assign sensory values from tested foods to similar nontested foods using a best match approach to ensure a comprehensive coverage of all foods and beverages consumed by the population within Australia, suitable for use with NNPAS [34]. There were no sensory profiles for alcoholic beverages, salt, sugar, and stock because they were not tested by the sensory panel.

2.4. *Integrated NNPAS and CSIRO Sensory Diet database*

The food-level information from the NNPAS was integrated with the CSIRO Sensory Diet database using an 8-digit code. Each food and beverage entry in the database was linked to a respondent-unique identifier, which served as an identifier to connect all data across NNPAS files to individual participants. This unique identifier was then used to merge demographic information and health outcomes with food and beverage intake data including sensory properties, resulting in the final database.

2.5. *Measurement of dietary bitterness*

This study quantified the sensory dimension of diet independent of nutrient composition. For each participant, a bitterness score for each consumed item was calculated by multiplying the bitterness value by the quantity of food consumed.

The dietary bitterness score was calculated as the sum of the bitter scores for all consumed foods and beverages across the day of the survey. To account for variation in total dietary intake, a "bitter density'' value was also calculated by dividing the dietary bitterness score by total energy intake. To avoid computational errors, foods with zero bitterness values were assigned a value of 1, and the bitter density was recalculated [35].

2.6. Outcome measures

The National Health Survey included fasting blood samples for the measurement of cardiometabolic risk biomarkers. These biomarkers include fasting plasma glucose, HbA1c, serum total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, and systolic and diastolic blood pressure (BP) measurements. Detailed classifications of these biomarkers can be found elsewhere [32].

2.7. Statistical analysis

Statistical analyses were conducted using Stata (version 18, StataCorp, 2023). To account for the survey design and sampling methodology of the NNPAS, replicate weights were applied using the jack-knife method. Full details of the relevant Stata commands and replicate weight procedures have been published elsewhere [36,37]. Total energy and bitter density variables were assessed for normality using skewness and kurtosis. Given the large sample size ($n = 8975$) and absolute skewness <2 and kurtosis <7, the data were considered normally distributed per established benchmarks [38].

Dietary bitterness scores for foods and beverages were ranked both separately and grouped into tertiles (low, which represents low bitterness; moderate; and high, which represents high bitterness) for analysis. Descriptive statistics (frequencies, mean, and standard error) were used to describe variables and participant characteristics across tertiles of bitterness scores for foods and beverages. Comparisons among tertiles of the dietary bitterness score were conducted using the one-way analysis of variance test for continuous variables and chi-squared analyses for categorical variables. Tukey's post hoc test was used to identify pairwise differences following significant analysis of variance results for dietary intake. Multinomial logistic regression was used to assess the association between dietary bitterness score and cardiometabolic factors across these tertiles with effect estimates reported as relative risk ratio. In the analyses of cardiovascular risk factors, participants taking medications for dyslipidemia were excluded to avoid confounding from pharmacological lipid-lowering effects.

Covariates identified from expert knowledge and from published literature were included in the regression models because of their influence on exposure and outcome measures. Three models were adopted in the multinomial logistic regression analysis: model 1 adjusted for age, sex at birth, area-level disadvantage, smoking, and physical activity; model 2 additionally adjusted for dietary fiber for foods analysis and alcohol intake for beverages analysis; and model 3 additionally adjusted for BMI. Based on our predefined analysis plan **(Supplemental file 2)**, model 3 was considered the main model. To control for multiple comparisons, a Bonferroni correction was applied. For the diabetes outcomes (fasting blood glucose and HbA1c [diabetes vs. no diabetes/prediabetes vs. nondiabetes]), an adjusted significance threshold of $P < .0083$ was used (based on 6 comparisons: 3 models × 2 outcomes). For all other outcomes, a Bonferroni correction across the 3 models resulted in an adjusted threshold of $P < .0167$ was applied.

3. Results

Table 1 summarizes the demographic, lifestyle, and cardiometabolic characteristics of the 8975 participants across tertiles of dietary bitterness scores, analyzed separately for foods and beverages. Mean bitterness scores were 1173, 2743, and 5762 across food tertiles and 11,437, 32,205, and 66,294 across beverage tertiles.

For the food bitterness score, participants in the highest tertile were older ($P < .0001$) and more likely to have higher educational attainment (33% with a university degree in highest tertile vs. 19% in lowest; $P < .0001$). Females were more represented in the lower food bitterness tertiles ($P = .001$). The higher food bitterness score was associated with lower smoking prevalence ($P < .0001$) and greater physical activity ($P < .0001$). Socioeconomic status followed a similar pattern, with those less disadvantaged more likely to fall in the highest food bitterness tertile (26% in highest tertile vs. 19% in lowest; $P < .0001$). When analyzing participants' cardiometabolic factors, those in the highest food bitterness tertile were less likely to be obese ($P = .0005$), have central obesity ($P = .003$), and abnormal triglycerides concentration ($P = .02$). The number of diabetes cases as indicated by HbA1c or fasting plasma glucose, or dyslipidemia as indicated by abnormal TC, LDL-C, HDL-C, or BP did not statistically differ between food bitterness score tertiles ($P > .05$ for all).

For the beverage bitterness score, participants in the highest tertile were older (mean age: 52 years in highest tertile vs. 41 years in lowest; $P < .0001$) and more likely to have higher educational attainment (26% with a university degree in the highest tertile vs. 25% in the lowest; $P = .001$). A higher beverage bitterness score was associated with higher smoking prevalence (20% smokers in highest tertile vs. 19% in lowest; $P = .03$) and less physical activity (12% highly active in highest tertile vs. 16% in lowest; $P = .018$). Participants living in outer regional areas were more likely to fall in the highest beverage bitterness tertile (18% in highest tertile vs. 15% in the lowest; $P = .01$). When analyzing participants' cardiometabolic factors, participants in the highest beverage bitterness tertile were more likely to be overweight ($P = .01$), have central obesity ($P < .0001$), dyslipidemia ($P < .0001$), abnormal TC ($P < .0001$), LDL-C ($P < .0001$), and BP ($P < .0001$). The number of diabetes cases as indicated by HbA1c or fasting plasma glucose, HDL-C, and TG concentration did not statistically differ between beverage bitterness score tertiles ($P > .05$ for all).

Participants' dietary compositions across tertiles of food and beverage bitterness scores are presented in Table 2. Participants in the highest food bitterness tertile had higher energy intake, higher intakes of green/brassica vegetables (servings),

Table 1 – The characteristics of the sample population participating in the National Nutrition and Physical Activity Survey.

variables	Food bitterness score (3199 ± 2279)			[a]P value	Beverage bitterness score (36867 ± 26118)			[a]P value
	T1 Lowest (*n* = 2992)	T2 (*n* = 2992)	T3 Highest (*n* = 2991)		T1 Lowest (*n* = 2992)	T2 (*n* = 2992)	T3 Highest (*n* = 2991)	
Age (y) (*N* = 8975)	47.9 ± 0.3	49.6 ± 0.3	50.6 ± 0.3	**<.0001**	40.8 ± 0.4	48.3 ± 0.4	52.1 ± 0.4	**<.0001**
Sex				**.001**				**.009**
Male	1389 (46)	1341 (45)	1507 (50)		1473 (49)	1410 (47)	1354 (45)	
Female	1603 (54)	1651 (55)	1484 (50)		1519 (51)	1582 (53)	1637 (55)	
SEIFA index				**<.0001**				**.008**
First (most disadvantaged)	654 (22)	547 (18)	481 (16)		596 (20)	505 (17)	581 (19)	
Second	648 (22)	610 (20)	591 (20)		639 (21)	586 (20)	624 (21)	
Third	612 (20)	576 (19)	592 (20)		563 (19)	615 (21)	602 (20)	
Fourth	495 (17)	553 (18)	549 (18)		541 (18)	549 (18)	507 (17)	
Fifth (least disadvantaged)	583 (19)	706 (24)	778 (26)		653 (22)	737 (25)	677 (23)	
Remoteness area				.11				**.001**
Major cities	1878 (62)	1953 (65)	1960 (66)		2010 (67)	1992 (67)	1789 (60)	
Inner regional	588 (20)	570 (19)	567 (19)		525 (18)	554 (19)	646 (22)	
Outer regional	526 (18)	469 (16)	464 (15)		457 (15)	446 (15)	556 (18)	
Highest levels of education				**<.0001**				**.001**
University degree	569 (19)	751 (26)	959 (33)		730 (25)	798 (27)	751 (26)	
Diploma	1063 (36)	1018 (35)	980 (33)		989 (34)	1068 (36)	1004 (34)	
Nonschool qualification	1321 (45)	1173 (40)	999 (34)		1229 (42)	1075 (37)	1189 (40)	
Smoking status				**<.0001**				**.03**
Yes	792 (27)	512 (17)	391 (13)		565 (19)	525 (18)	605 (20)	
No	2200 (73)	2480 (83)	2600 (87)		2427 (81)	2467 (83)	2386 (80)	
Physical activity level				**<.0001**				**.018**
High	300 (10)	420 (14)	558 (19)		460 (16)	432 (14)	386 (12)	
Moderate	756 (26)	814 (28)	889 (30)		777 (26)	813 (28)	869 (30)	
Low	1899 (64)	1726 (58)	1510 (51)		1719 (58)	1712 (58)	1704 (58)	
Cardiometabolic factors								
BMI (*N* = 7672) kg/m^2	27.7 ± 0.2	27.3 ± 0.1	26.9 ± 0.1	**.0005**	26.9 ± 0.2	27.4 ± 0.1	27.6 ± 0.2	**.001**
BMI categories (*N* = 8975)				**.0005**				**.001**
Underweight	41 (1)	40 (1)	32 (1)		52 (2)	36 (1)	25 (1)	
Normal	811 (27)	841 (28)	945 (32)		935 (31)	832 (28)	830 (28)	
Overweight	891 (30)	931 (31)	1004 (34)		866 (29)	944 (32)	1016 (34)	
Obese	1249 (42)	1180 (39)	1010 (34)		1139 (38)	1180 (39)	1120 (37)	
Waist circumference (*N* = 8975)				**.003**				**<.0001**
Normal	826 (33)	834 (33)	931 (36)		984 (39)	829 (33)	778 (30)	
At risk of central obesity	546 (22)	577 (23)	621 (24)		549 (22)	583 (23)	612 (23)	
Central obesity	1125 (45)	1149 (45)	1044 (40)		977 (39)	1124 (44)	1217 (47)	
Fasting plasma glucose (*N* = 3072)				.112				.869
No diabetes	761 (88)	960 (91)	1054 (91)		759 (91)	938 (90)	1078 (90)	
At risk of diabetes	50 (6)	51(5)	63 (5)		40 (5)	55 (5)	69 (6)	
Diabetes	50 (6)	41 (4)	42 (4)		34 (4)	48 (5)	51 (4)	
HbA1c (*N* = 3619)				.064				.809
No diabetes	867 (86)	1080 (87)	1207 (88)		874 (88)	1057 (87)	1223 (87)	
At risk of diabetes	76 (8)	109 (9)	93 (7)		71 (7)	94 (8)	113 (8)	
Diabetes	66 (7)	56 (5)	65 (5)		48 (5)	69 (6)	70 (5)	
Dyslipidemia (*N* = 2483)				.891				**<.0001**
No	259 (37)_	315 (37)	349 (38)		310 (44)	306 (37)	307 (33)	
Yes	450 (63)	533 (63)	577 (62)		398 (56)	532 (63)	630 (67)	
Total cholesterol (*N* = 3082)				.725				**<.0001**
Normal total cholesterol	507 (58)	636 (60)	690 (60)		588 (67)	614 (59)	631 (54)	
Abnormal total cholesterol	362 (42)	425 (40)	462 (40)		296 (33)	423 (41)	530 (46)	
Low-density lipoprotein (*N* = 2498)				.957				**<.0001**
Normal low-density lipoprotein	421 (59)	503 (59)	548 (59)		473 (66)	489 (58)	510 (54)	
Abnormal low-density lipoprotein	288 (41)	352 (41)	386 (41)		255 (34)	378 (42)	470 (46)	

(*continued on next page*)

Table 1 (*continued*)

variables	Food bitterness score (3199 ± 2279)			[a]*P* value	Beverage bitterness score (36867 ± 26118)			[a]*P* value
	T1 Lowest (*n* = 2992)	T2 (*n* = 2992)	T3 Highest (*n* = 2991)		T1 Lowest (*n* = 2992)	T2 (*n* = 2992)	T3 Highest (*n* = 2991)	
Triglycerides (N = 2523)				**.002**				.077
Normal triglycerides	590 (82)	742 (86)	829 (88)		636 (88)	717 (84)	808 (85)	
Abnormal triglycerides	129 (18)	121 (14)	112 (12)		86 (12)	134 (16)	142 (15)	
High-density lipoprotein (N = 3082)				.069				.865
Normal high-density lipoprotein	667 (77)	822 (77)	929 (81)		693 (78)	819 (79)	906 (78)	
Abnormal high-density lipoprotein	202 (23)	239 (23)	223 (19)		191 (22)	218 (21)	255 (22)	
Blood pressure (N = 7702)				.531				**<.0001**
Normal blood pressure	1913 (76)	1944 (76)	2009 (77)		2030 (80)	1915 (75)	1921 (73)	
High blood pressure	599 (24)	631 (25)	606 (23)		494 (20)	625 (25)	717 (7)	

Abbreviations: BMI, body mass index; SEIFA: Socio-Economic Indexes for Areas.
BMI categories were classified as: underweight BMI < 18.5, normal BMI 18.5 – 24.9, overweight BMI 25 –29.9, and obese BMI ≥ 30; waist circumference categories were classified as: normal waist circumference <94 (men) or < 80 (women), at risk of central obesity waist circumference ≥ 94 (men) or ≥ 80 (women), and central obesity waist circumference ≥ 102 (men) or ≥ 88 (women); diabetes status was defined using HbA1c: <6% classified as normal, 6–<6.5% as at risk of diabetes, and >6.5% as diabetes or fasting plasma glucose concentration (<6.1 mmol/L as normal, 6.1–<7.0 mmol/L as at risk, and ≥7.0 mmol/L as diabetes); cardiovascular and blood pressure biomarkers were classified as "normal" or "abnormal": abnormal was defined as total cholesterol ≥5.5 mmol/L, fasting low-density lipoprotein ≥3.5 mmol/L, high-density lipoprotein <1.0 mmol/L (men) or <1.3 mmol/L (women), fasting triglycerides ≥2.0 mmol/L and blood pressure ≥140/90 mm Hg); dyslipidemia was defined as taking cholesterol-lowering medication or meeting any abnormal lipid criteria after excluding individuals taking dyslipidemia medications. The lowest tertile (T1: reference) indicates the lowest bitterness score of the diet, and the highest (T3) indicates the highest bitterness score of diet. Data reported as mean ± standard error for continuous variables. Data reported as *N* (%) for categorical variables. Significant *P* values are shown in bold. Population size for age: 16,150,093; for BMI: 14,021,046.
[a] *P* value to compare the variables between tertiles of the food and beverage bitterness score using chi-squared analysis for categorical data and the *t*-test for continuous data.

total sugar, dietary fiber, less processed red meat (grams), a higher percentage of energy from sugar and fat, and a lower percentage of energy from saturated fat. Participants in the highest beverage bitterness tertile had higher energy intake from beverages, a higher percentage of energy from fat, and a higher intake of coffee and tea.

All potential confounders were included in the multivariate analyses to examine the association between tertiles of food and beverage bitterness scores, and the risk of type 2 diabetes mellitus, dyslipidemia, and BP (Table 3). In the fully adjusted model, there was no association between tertile of food bitterness score and risk of prediabetes. However, there was a lower risk of type 2 diabetes as assessed by fasting plasma glucose (RRR = 0.30; 95% CI, 0.15–0.60; *P* for trend = .001) or HbA1c (RRR= 0.43; 95% CI, 0.25–0.75; *P* for trend = .003) among participants in the second compared to the lowest tertile of bitterness score. Participants in the highest tertile also had lower risk of type 2 diabetes compared to the lowest tertile, but the association was not statistically significant. In the analyses of cardiovascular risk factors, after excluding individuals taking medications for dyslipidemia, there was no association between food bitterness score and likelihood of abnormal cardiovascular risk factors across any of the models (Table 3). In the adjusted models, there was no association between tertile of beverage bitterness score and risk of prediabetes or overt type 2 diabetes as defined by fasting plasma glucose and HbA1c (Table 3). No significant associations were observed between the beverage bitterness score and the likelihood of abnormal cardiovascular risk factors across any of the models.

4. Discussion

This study investigated the association between overall dietary bitterness and several cardiometabolic risk factors in a representative sample of Australian adults. Participants whose diets had a moderate food bitterness score had lower risk of type 2 diabetes compared to those with the lowest food bitterness score. No association was observed between food or beverage bitterness score and risk of prediabetes or abnormal cardiovascular risk factors.

The association between moderate food bitterness score and lower risk of type 2 diabetes is consistent with those reported in several intervention trials, which collectively suggest that diets rich in bitter-tasting compounds are associated with decreased risk of type 2 diabetes [23,39–45]. Unlike prior studies that focused on specific bitter foods or beverages, such as brassica vegetables, tea and coffee, or polyphenol-rich diets, this study evaluated the collective contribution of all consumed foods and beverages based on their bitterness, controlling for total energy intake. The overall dietary bitterness score was primarily driven by foods with high or medium bitterness intensity that were consumed most frequently. The main food groups contributing to dietary bitterness are bras-

Table 2 – Dietary intakes for participating adults in the National Nutrition and Physical Activity Survey across tertiles of food and beverage bitterness score.

Diet variables	Food bitterness score			[d]P value	Beverage bitterness score			[e]P value
	T1	T2	T3		T1	T2	T3	
Bitterness of diet	1173 ± 13	2743 ± 12	5762 ± 54	**<.0001**	11,437 ± 161	32,205 ± 171	66,294 ± 555	**<.0001**
Energy and nutrients								
Energy intake (Kcal)	1393 ± 15	1706 ± 17	2112 ± 18	**<.0001**	326 ± 8	354 ± 8	374 ± 7	**<.0001**
% energy from protein	18.2 ± 0.2	18.5 ± 0.1	18.5 ± 0.1	.25	18.6 ± 0.18	18.30 ± 0.16	18.27 ± 0.16	.17
% energy from carbohydrate	43.7 ± 0.3	43.1 ± 0.2	43.3 ± 0.2	.29	44.2 ± 0.3	43.1 ± 0.3 [c]	42.7 ± 0.3	**.001**
% energy from fat	30.5 ± 0.2[a]	30.9 ± 0.2[c]	31.2 ± 0.2	**.02**	30.2 ± 0.2	31.0 ± 0.2	32.0 ± 0.2	**<.0001**
% energy from saturated fat	12.4 ± 0.14	12.1 ± 0.11	11.6 ± 0.09	**<.0001**				
% energy from sugar	18.7 ± 0.2[a]	18.8 ± 0.2	19.6 ± 0.2	**.03**	18.5 ± 0.2 [a]	19.3 ± 0.2	19.6 ± 0.2	**.005**
Total sugar (added, free, from food) (g)	200 ± 4[a]	204 ± 4	232 ± 5	**<.0001**	204 ± 4[a]	209 ± 4	226 ± 5	**.002**
Added sugar (g)	53.2 ± 1.2	49.1 ± 1.4	51.3 ± 1.3	.30	50 ± 1[ab]	50 ± 1	54 ± 1	**.03**
Free sugar (g)	60.1 ± 1.1	56.4 ± 1.2	59.3 ± 1.1	.80	57 ± 1	57 ± 1	60 ± 1	.09
Sugar from food and beverages (g)	88.2 ± 1.2	99.3 ± 2.4	122.1 ± 2.4	**<.0001**	98 ± 2[a]	102 ± 2	111 ± 2	**<.0001**
Dietary fiber (g)	15.1 ± 0.2	22 ± 0.2	32 ± 0.4	**<.0001**	-	-	-	-
Salt (mg)	1,490 ± 10	1,510 ± 10[c]	1,540 ± 20	**.01**	-	-	-	-
Food groups								
Green/brassica vegetables (serves)	0.3 ± 0.01	0.6 ± 0.01	1.07 ± 0.03	**<.0001**	-	-	-	
Processed red meat (g)	0.03 ± 0.004	0.03 ± 0.004	0.01 ± 0.003	**.006**	-	-	-	
Alcoholic beverages (g)	-	-	-	-	402 ± 21	398 ± 19	370 ± 21	.20
Tea/coffee (g)	-	-	-	-	217 ± 7	733 ± 11	1,420 ± 17	**<.0001**

Data reported as mean ± standard error. The lowest tertile (T1: reference) indicates the lowest bitterness score of the diet, and the highest (T3) indicates the highest bitterness score of diet. Significant *P* values are shown in bold.

[a] Results from the Tukey post hoc test when T1 vs. and T2 are not significant.

[b] Results from the Tukey post hoc test when T1 vs. and T3 are not significant.

[c] Results from the Tukey post hoc test when T2 vs. and T3 are not significant.

[d] *P* value comparing the diet variables between tertiles of food bitterness score.

[e] *P* value comparing the diet variables between tertiles of beverage bitterness score.

sica vegetables among solid foods and tea and coffee among beverages [35]. The ATTICA cohort study found that consuming at least 4 servings of cruciferous vegetables per day over 10 years was associated with a 58% lower risk of developing type 2 diabetes compared with lower intakes [45]. Similarly, a UK Biobank analysis reported that higher tea consumption over 14 years was associated with a modestly reduced type 2 diabetes risk, with 7% lower risk for 2 to 3 cups/day compared with nonconsumption [44]. In addition, a pooled analysis of three large US cohorts found that each additional cup of coffee consumed without additives was associated with a 10% lower risk of type 2 diabetes [43]. By assessing total dietary bitterness rather than individual foods or beverages, the present study observed a slightly greater reduction in the likelihood of elevated glycemic markers, suggesting that a comprehensive assessment of bitter compounds across the diet may better capture potential protective effects. However, because this analysis is cross-sectional, the findings cannot establish causality and should be interpreted with caution compared with evidence from longitudinal studies.

The observed association between food bitterness score and lower risk of type 2 diabetes may be mechanistically explained by the activation of TAS2Rs, which are distributed throughout the oral cavity and gastrointestinal tract and trigger the release of incretin hormones such as GLP-1 and GIP. These hormones bind to their respective receptors on pancreatic β cells, activating intracellular signaling pathways that enhance glucose-stimulated insulin secretion and ultimately the reduction of circulating blood glucose concentration [46]. GLP-1 can also suppress glucagon release, impacting hepatic glucose production, and further lowering blood glucose [15]. Incretin hormones additionally slow gastric emptying by inhibiting the gastric inhibitory vagal motor circuit and the vagus nerve and nucleus tractus solitarius, minimizing postmeal glucose spikes. Altogether, this cascade, initiated by bitter compound sensing, supports improved glucose homeostasis and provides a plausible mechanism by which bitter foods influence glycemic health [15]. Moderate intake of hormetic nutrients, including polyphenol-rich foods, may promote gut and brain health through activation of the Nrf2 signaling pathway and upregulation of stress-resilience vitagenes. These pathways enhance antioxidant defence, and modulate inflammation, potentially contributing to improved insulin sensitivity and β-cell protection [47,48].

Participants with the most bitter diets (highest tertile) showed similar associations as those with moderate intake in

Table 3 – Logistic regression analysis testing the association between food and beverage bitterness score and cardiometabolic factors among participating adults in the National Nutrition and Physical Activity Survey.

Variable		Food bitterness score					Beverage bitterness score				
		T1	T2		T3		T1	T2		T3	
			RRR (CI)	*P* trend	RRR (CI)	*P* trend		RRR (CI)	*P* trend	RRR (CI)	*P* trend
Prediabetes											
Fasting plasma glucose											
Case	Total: 164	50	51		63		40	55		69	
	M:90, F:74	M:26, F:24	M:25, F:26		M:39, F:24		M:22, F:18	M:28, F:27		M:40, F:29	
	Model 1 [a]	1 REF	0.90 (0.51-1.61)	.727	1.10 (0.65-1.85)	.714	1 REF	0.91 (0.48-1.72)	.774	0.81 (0.44-1.50)	.489
	Model 2 [b]	1 REF	0.96 (0.51-1.81)	.901	1.31 (0.59-2.90)	.506	1 REF	0.91 (0.49-1.72)	.775	0.81 (0.44-1.49)	.488
	Model 3 [c]	1 REF	1.01 (0.51-2.04)	.966	1.41 (0.60-3.33)	.428	1 REF	0.94 (0.46-1.94)	.868	0.92 (0.48-1.79)	.795
HbA1c											
Cases	Total: 278	76	109		93		71	94		113	
	M:133, F:145	M:39, F:37	M:53, F:56		M:41, F:52		M:38, F:33	M:44, F:50		M:51, F:62	
	Model 1	1 REF	1.28 (0.78-2.12)	.325	1.03 (0.64-1.65)	.907	1 REF	0.63 (0.38-1.01)	.057	0.64 (0.39-1.06)	.080
	Model 2	1 REF	1.32 (0.79-2.22)	.280	1.11 (0.65-1.90)	.686	1 REF	0.62 (0.38-1.01)	.056	0.64 (0.39-1.05)	.077
	Model 3	1 REF	1.26 (0.73-2.17)	.408	1.06 (0.62-1.81)	.823	1 REF	0.57 (0.33-0.95)	.033	0.59 (0.35-0.99)	.048
Diabetes											
Fasting plasma glucose											
Case	Total: 133	50	41		42		34	48		51	
	M:84, F:49	M:28, F:22	M:25, F:16		M:31, F:11		M:21, F:13	M:29, F:19		M:34, F:17	
	Model 1	1 REF	0.38 (0.21-0.70)	**.002**	0.52 (0.33-0.80)	**.004**	1 REF	0.58 (0.27-1.23)	.154	0.54 (0.31-0.94)	.030
	Model 2	1 REF	0.36 (0.19-0.68)	**.002**	0.42 (0.21-0.83)	.013	1 REF	0.58 (0.27-1.22)	.149	0.53 (0.30-0.93)	.029
	Model 3	1 REF	0.30 (0.15-0.60)	**.001**	0.44 (0.22-0.87)	.020	1 REF	0.49 (0.25-0.99)	.046	0.55 (0.28-1.06)	.075
HbA1c											
Cases	Total: 187	66	56		65		48	69		70	
	M:106, F:81	M:35, F:31	M:30, F:26		M:41, F:24		M:25, F:33	M:40, F:29		M:41, F:29	
	Model 1	1 REF	0.44 (0.28-0.69)	**.001**	0.60 (0.37-0.97)	.036	1 REF	0.64 (0.34-1.20)	.166	0.60 (0.34-1.06)	.076
	Model 2	1 REF	0.43 (0.26-0.69)	**.002**	0.57 (0.31-1.04)	.068	1 REF	0.64 (0.34-1.20)	.158	0.59 (0.34-1.03)	.062
	Model 3	1 REF	0.43 (0.25-0.75)	**.003**	0.61 (0.32-1.17)	.136	1 REF	0.58 (0.28-1.19)	.133	0.61 (0.33-1.15)	.128
Abnormal triglycerides											
Cases	Total: 362	129	121		112		86	134		142	
	M:226, F:136	M:74, F:55	M:75, F:46		M:77, F:35		M:51, F:35	M:90, F:44		M:85, F:57	
	Model 1	1 REF	0.91 (0.66-1.26)	.570	0.81 (0.57-1.15)	.242	1 REF	1.23 (0.86-1.75)	.255	1.09 (0.73-1.63)	.680
	Model 2	1 REF	0.91 (0.65-1.23)	.606	0.82 (0.53-1.26)	.360	1 REF	1.23 (0.86-1.74)	.252	1.09 (0.73-1.62)	.681
	Model 3	1 REF	0.90 (0.64-1.29)	.579	0.80 (0.52-1.23)	.304	1 REF	1.20 (0.72-1.69)	.331	1.10 (0.72-1.69)	.643
Abnormal total cholesterol											
Cases	Total: 1249	362	425		462		296	423		530	
	M:529, F:720	M:148, F:214	M:174, F:251		M:207, F:255		M:136, F:160	M:194, F:229		M:199, F:331	
	Model 1	1 REF	1.02 (0.80-1.30)	.865	0.92 (0.72-1.17)	.487	1 REF	1.31 (1.01-1.69)	.039	1.35 (1.02-1.79)	.039
	Model 2	1 REF	1.09 (0.84-1.42)	.512	1.08 (0.79-1.49)	.596	1 REF	1.30 (1.01-1.68)	.045	1.36 (1.02-1.81)	.038
	Model 3	1 REF	1.09 (0.84-1.44)	.486	1.08 (0.78-1.49)	.647	1 REF	1.31 (1.00-1.73)	.052	1.38 (1.03-1.86)	.031

(*continued on next page*)

Table 3 (*continued*)

Variable	Food bitterness score						Beverage bitterness score					
		T1	T2		T3		T1	T2		T3		
			RRR (CI)	*P* trend	RRR (CI)	*P* trend		RRR (CI)	*P* trend	RRR (CI)	*P* trend	
Abnormal low-density lipoprotein												
Cases	Total: 1026	288	352		386		242	351		433		
	M:479, F:547	M:128, F:160	M:156, F:196		M:195, F:191		M:122, F:120	M:182, F:169		M:175, F:258		
	Model 1	1 REF	1.04 (0.78-1.39)	.790	1.06 (0.83-1.35)	.647	1 REF	1.41 (1.05-1.87)	.021	1.44 (1.05-1.98)	.027	
	Model 2	1 REF	1.10 (0.81-1.49)	.526	1.23 (0.89-1.69)	.191	1 REF	1.41 (1.05-1.87)	.021	1.44 (1.05-1.99)	.026	
	Model 3	1 REF	1.14 (0.84-1.55)	.406	1.23 (0.88-1.72)	.230	1 REF	1.41 (1.03-1.91)	.031	1.46 (1.06-2.02)	.021	
Abnormal high-density lipoprotein												
Cases	Total: 664	247	295		270		191	218		255		
	M:250, F:414	M:74, F:128	M:76, F:163		M:100, F:123		M:62, F:129	M:86, F:132		M:102, F:153		
	Model 1	1 REF	0.90 (0.66-1.22)	.474	0.94 (0.72-1.25)	.682	1 REF	1.05 (0.81-1.37)	.687	1.00 (0.77-1.30)	.961	
	Model 2	1 REF	0.84 (0.62-1.16)	.285	0.81 (0.59-1.13)	.208	1 REF	1.07 (0.82-1.40)	.616	0.99 (0.76-1.31)	.983	
	Model 3	1 REF	0.86 (0.62-1.19)	.365	0.79 (0.58-1.09)	.154	1 REF	1.03 (0.78-1.38)	.813	1.01 (0.76-1.35)	.943	
Dyslipidemia												
Cases	Total: 1,60	450	533		577		398	532		630		
	M:691, F:869	M:188, F:262	M:223, F:310		M:280, F:297		M:178, F:220	M:254, F:278		M:259, F:371		
	Model 1	1 REF	0.94 (0.71-1.23)	.644	1.04 (0.79-1.39)	.744	1 REF	1.27 (0.93-1.73)	.125	1.18 (0.81-1.70)	.380	
	Model 2	1 REF	0.91 (0.67-1.22)	.513	0.96 (0.70-1.31)	.793	1 REF	1.28 (0.94-1.74)	.119	1.17 (0.81-1.69)	.393	
	Model 3	1 REF	0.96 (0.70-1.31)	.781	0.00 (0.72-1.38)	.964	1 REF	1.19 (0.80-1.77)	.222	1.19 (0.80-1.77)	.374	
Abnormal blood pressure												
Cases	Total: 1836	599	631		606		494	625		717		
	M:960, F:876	M:333, F:266	M:310, F:321		M:317, F:289		M:286, F:208	M:335, F:290		M:339, F:378		
	Model 1	1 REF	0.99 (0.81-1.22)	.946	0.80 (0.63-1.02)	.069	1 REF	0.92 (0.74-1.14)	.446	0.94 (0.75-1.17)	0556	
	Model 2	1 REF	1.00 (0.81-1.25)	.985	0.82 (0.62-1.09)	.167	1 REF	0.92 (0.74-1.14)	.449	0.95 (0.76-1.18)	.611	
	Model 3	1 REF	1.01 (0.81-1.26)	.779	0.81 (0.62-1.07)	.139	1 REF	0.87 (0.70-1.08)	.212	0.93 (0.75-1.16)	.527	

Abbreviations: F, female; M, male; REF, reference.

Significant *P* values are shown in bold. *N* and population-weighted equivalence. fasting plasma glucose: *N* and population size for model 1: 3047 and 5,157,925; model 2: 3047 and 5,157,925; model 3: 2921 and 4,958,907. HbA1c: *N* and population size for model 1: 3591 and 6,087,145; model 2: 3591 and 6,087,145; model 3: 3438 and 5,838,362. Triglycerides: *N* and population size for model 1: 2498 and 4,317,531; model 2: 2498 and 4,317,531; model 3: 2407 and 4,168,942. Total cholesterol: *N* and population size for model 1: 3054 and 5,274,108; model 2: 3054 and 5,274,108; model 3: 2935 and 5,073,919. Low-density lipoprotein: *N* and population size for model 1: 2473 and 4,262,366; model 2: 2473 and 4,262,366; model 3: 2382 and 4,113,777. High-density lipoprotein: *N* and population size for model 1: 3054 and 5,274,108; model 2: 3054 and 5,274,108; model 3: 2935 and 5,073,919. Dyslipidemia: *N* and population size for model 1: 2458 and 4,235,983; model 2: 2458 and 4,235,983; model 3: 2368 and 4,087,566. Blood pressure: *N* and population size for model 1: 7618 and 13,816,768; model 2: 7618 and 13,816,768; model 3: 7353 and 13,384,882.

[a] Model 1 = adjusted for age, gender, area-level disadvantage, smoking, and physical activity.

[b] Model 2 = additionally adjusted for "fiber intake" for the bitterness score of solid foods and "alcohol intake" for the bitterness score of beverages.

[c] Model 3 = additionally adjusted for body mass index.

model 1. Adjusting for dietary fiber resulted in a loss of significance at the *P* value set but was still marginally significant for those in the highest tertile (model 2). This attenuation likely reflects differences in the sources of dietary bitterness, whereby participants in the highest tertile tended to consume more green and brassica vegetables, which are both bitter and higher in fiber, whereas participants with moderate bitterness scores may have consumed lower-fiber foods such as dark chocolate, fruit and vegetable juices, or olives. This finding of healthier dietary patterns being linked to the highest tertile of bitterness score in this study is supported by previous studies, suggesting that higher diet quality is associated with higher bitterness scores [19,35,49]. Unlike the association observed for type 2 diabetes, there was no association between food bitterness and the likelihood of prediabetes, which may reflect the greater variability and instability of fasting plasma glucose concentration in individuals with prediabetes, reducing the ability to detect associations. Supporting this explanation, a large cohort study found that among individuals with normal fasting glucose at baseline, 40% were reclassified as having prediabetes at subsequent assessments, highlighting the fluid nature of this metabolic state [50]. Additionally, the lack of association with prediabetes does not necessarily mean that dietary bitterness is unimportant, but it may indicate that measuring food bitterness alone is insufficient to predict prediabetes. However, it is plausible that individuals with prediabetes could benefit from consuming a higher intake of bitter foods to potentially reduce their risk of progressing to type 2 diabetes, consistent with the protective associations observed for established diabetes in this study. Foods and beverages were analyzed separately because of the complex metabolic profiles observed across bitterness tertiles, as well as differences in demographic, lifestyle, and dietary patterns identified in the descriptive analysis. Individuals in the highest tertile of food bitterness tended to have a higher level of education, were less socioeconomically disadvantaged, engaged in more physical activity, and had lower rates of smoking, whereas individuals in the highest tertile of beverage bitterness score were more likely to smoke, be less physically active, consume more fat, sugar, tea, and coffee, and have higher numbers of individuals with high BMI and central obesity. These characteristics align with previous research suggesting that health-conscious individuals with higher socioeconomic status consume more brassica vegetables and bitter-tasting foods, although it remains unclear whether these patterns are driven by taste perception or solely by lifestyle and socioeconomic factors [51,52]. Previous studies also show that high coffee intake is associated with higher intake of fat and sugar, which might also be the case in the Australian population [53,54]. Descriptive data from the current study show that higher beverage bitterness scores are associated with higher consumption of coffee or tea and BMI. However, the direction of this association is unclear. It is possible that obesity influences taste sensitivity, which in turn affects food choices, or that high intake of bitter beverages contributes to differences in BMI [9,54]. The observed differences in sociodemographic factors and dietary patterns between individuals with higher food bitterness and those with higher beverage bitterness suggest that bitter food choices may be associated with healthier overall diets, whereas higher beverage bitterness scores were observed in participants with less favorable sociodemographic and dietary characteristics. Similarly, we found a higher prevalence of overweight, central obesity, dyslipidemia, and abnormal TC and LDL-C concentrations in the highest tertile of beverage bitterness. In crude logistic regression models, higher beverage bitterness scores were associated with increased risk of adverse cardiometabolic factors. However, these associations disappeared after adjustment for sociodemographic and dietary covariates, suggesting that the crude associations were likely explained by differences in participant characteristics rather than beverage bitterness itself. Nevertheless, the possibility of residual confounding by unmeasured lifestyle, genetic predisposition, or behavioral factors cannot be excluded.

This analysis benefits from a large and diverse Australian population sample, which enhances the generalizability of the findings. It also introduces a novel approach by examining dietary bitterness scores for beverages and foods that remain understudied in cardiometabolic health research. Additionally, careful adjustment for potential confounders improves the validity and strengthens the reliability of our results. This study has several limitations. Although the association between dietary bitterness score and risk of type 2 diabetes was statistically significant, these findings should be interpreted with caution because of the relatively low number of participants with diabetes, despite the overall large sample size. The limited case distribution may reduce the power to detect associations and affect the stability of the estimates. Additionally, we were underpowered to perform sensitivity analyses stratified by age and gender due to the limited number of cases in each subgroup, which limits the reliability of the conclusions. Another limitation of this study is that alcohol, which is a bitter-tasting beverage was not assigned a bitterness value in the CSIRO database and its potential contribution to the bitterness score and outcome of BMI could not be tested. The study also did not account for genetic variations in the TAS2R bitter taste receptor gene, which has been independently associated with taste preferences, and dietary intake patterns as well as BMI and diabetes [55,56]. However, findings from previous studies suggest that environmental and lifestyle factors have a stronger influence on food preferences and disease risk than the genetic polymorphisms implicated in the taste receptor genes [52,57].

5. Conclusion

Findings from this study suggest that moderate intake of bitter foods but not at the highest level, may be associated with lower relative risk of type 2 diabetes among Australian adults, whereas no such benefit was found for bitter beverages or other cardiometabolic outcomes. However, these associations may be influenced by coexisting dietary patterns. Promoting bitter solid foods may be beneficial to Australian individuals, whereas the promotion of bitter beverages should be approached more cautiously, with emphasis on limiting added sugar and fat. Nevertheless, regular exposure to bitter beverages could help build taste familiarity and encourage the intake of bitter foods.

Sources of support

This work was supported by Adelaide University.

Author declarations

The authors declare no potential conflicts of interest.

CRediT authorship contribution statement

Maedeh Moradi: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Zinat Mohammadpour:** Writing – review & editing, Methodology. **Gilly A. Hendrie:** Writing – review & editing, Methodology. **Paige G. Brooker:** Writing – review & editing, Methodology. **Leonie K. Heilbronn:** Writing – review & editing, Visualization, Supervision, Methodology, Funding acquisition, Conceptualization. **Jessica A. Grieger:** Writing – review & editing, Supervision.

Ethics approval and consent to participate

Ethics approval for the household interview component of the survey was granted under the *Census and Statistics Act 1905*. Written informed consent for participation in the National Health Measures Survey was obtained by trained interviewers from the Australian Bureau of Statistics, as detailed in the *Australian Health Survey User Guide* (https://www.abs.gov.au/ausstats/abs@.nsf/Lookup/4363.0.55.001Chapter2152011-13). The study protocol was reviewed and approved by the Australian Bureau of Statistics.

Availability of data

Restrictions apply to the availability of these data. Data were obtained from the Australian Bureau of Statistics and are available at https://www.abs.gov.au/about/data-services/consultancy-services#whereto-find-abs-data (accessed on November 17, 2023) with the permission of the Australian Bureau of Statistics.

Acknowledgment

The authors have no acknowledgments to declare.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.nutres.2025.12.007.

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