HHS Public Access

Author manuscript

J Womens Health (Larchmt). Author manuscript; available in PMC 2024 November 01.

Published in final edited form as:

J Womens Health (Larchmt). 2024 May ; 33(5): 613–623. doi:10.1089/jwh.2023.0739.

Relationship Between Ultra- and Minimally-Processed Food Intake and Cardiovascular Health Among US Women of Reproductive Age

Zefeng Zhang[1], **Sandra L. Jackson**[1], **Euridice Martinez Steele**[2,3], **Donald K. Hayes**[1], **Quanhe Yang**[1]

[1]Division for Heart Disease and Stroke Prevention, Centers for Disease Control and Prevention, Atlanta, Georgia

[2]Department of Nutrition, School of Public Health, University of São Paulo, Brazil

[3]Center for Epidemiological Studies in Health and Nutrition, University of São Paulo, Brazil

Abstract

Objectives: Ultra-processed food (UPF) intake is associated with worse cardiovascular health (CVH), but associations between unprocessed/minimally-processed foods (MPFs) and CVH are limited, especially among women of reproductive age (WRA).

Methods: For 5773 WRA (20–44 years) in NHANES 2007–2018, we identified UPFs and MPFs using the Nova classification and based on 24-hour dietary recalls. We calculated usual percentages of calories from UPFs and MPFs using the National Cancer Institute's usual intake method. Seven CVH metrics were scored, and CVH levels were grouped by tertile. We used multivariable linear and multinomial logistic regression to assess associations between UPFs and MPFs and CVH.

Results: The average usual percentage of calories from UPFs and MPFs was 57.2% and 29.3%, respectively. There was a graded, positive association between higher UPFs intake and higher odds of poor CVH: adjusted odds ratios for the lowest vs highest CVH were 1.74 (95% CI: 1.51–2.01), 2.67 (2.07–3.44) and 4.66 (3.13–6.97), respectively, comparing quartile 2 (Q2)–Q4 to the lowest quartile (Q1) of UPF intake. Higher MPFs intake was associated with lower odds of poor CVH: adjusted odds ratios for the lowest CVH were 0.61 (0.54–0.69), 0.39 (0.31–0.50), and 0.21 (0.14–0.31). Patterns of association remained consistent across subgroups and in sensitivity analyses.

Corresponding Author: Zefeng Zhang, MD, PhD, 4770 Buford Highway NE, Mail Stop S107-1, Chamblee, GA 30341, hwv0@cdc.gov.

AUTHOR CONTRIBUTIONS

ZZ and SJ conceived and designed the study. ZZ and QY analyzed the data; ZZ produced the figures and tables. ZZ, SJ, EM, DH, and QY interpreted the data. ZZ searched the literature and wrote the first draft with insightful contributions from SJ, EM, HD and QY. All authors contributed to the revision of the first draft and reviewed and approved the final version of the paper.

Conflict of interest: The authors report no conflicts of interest or financial disclosures.

Disclaimer: The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention. Code book and data publicly available on NHANES website.

Conclusions: Higher UPF intake was associated with worse CVH, while higher MPF intake was associated with better CVH among WRA in the United States. Our analyses highlight an opportunity for WRA to improve nutrition and their CVH.

Keywords

ultra-processed foods; unprocessed/minimally-processed foods; usual percentage of calories; NHANES; odds ratio; women of reproductive age

INTRODUCTION

Foods can be classified by systems such as Nova, which categorizes four food groups according to the level of commercial processing.[1] Unprocessed/minimally-processed foods (MPFs) are foods without processing other than techniques such as removal of unwanted parts, drying, crushing, and grinding, and only rarely contain additives. Ultra-processed foods (UPFs) are industrially-created products that have been substantially altered.[1] High UPF intake is associated with cardiovascular disease (CVD)[2] as well as obesity, diabetes, dyslipidemia, hypertension, and metabolic syndrome.[3–11] Higher UPF is also associated with higher all-cause mortality.[12–14] Research regarding the effects of MPFs classified by Nova on CVD risk factors is limited.[14,15]

The American Heart Association's (AHA) "Strategic Impact Goal Through 2020 and Beyond," highlighted a set of seven cardiovascular health (CVH) metrics (body mass index [BMI], smoking, physical activity, dietary intake, total cholesterol, blood pressure, and fasting glucose) to prioritize in order to improve heart health.[16] More ideal CVH metrics have been associated with lower CVD incidence and mortality.[17,18] UPF and the AHA's CVH metrics have been shown to have an inverse relationship among US adults and adolescents.[19,20] In 2022, the AHA updated the CVH classification by adding sleep health as an additional metric and a new scoring system, the Life's Essential 8 (LE-8) score.[21]

Among US women, CVD is the leading cause of death,[22] with over 441 000 deaths and an estimated overall cost of $153.5 billion in 2020. Previous epidemiologic studies indicated that cardiovascular risk factors such as unhealthy diet[23,24] were associated with decreased risk of pregnancy-related complications, which in turn could lower subsequent CVD risk among women of reproductive age (WRA). As UPF and MPF intake is a modifiable risk factor of CVD, understanding the association of UPFs and MPFs and CVH among WRA may help WRA and their clinicians understand nutritional choices and opportunities to improve CVH. We used data from the National Health and Nutrition Examination Survey (NHANES) 2007–2018, and examined the association between usual percentage of total daily calories (%kcal) from UPFs and MPFs and CVH, which was assessed by the AHA's LE-8.[21]

PARTICIPANTS AND METHODS

Data source and participants

NHANES is a nationally representative, cross-sectional survey of the civilian noninstitutionalized US population conducted by the National Center for Health Statistics

(NCHS), CDC. It uses a multistage probability sampling design to collect data on nutrition and health via household interviews and physical examinations.[25] Among 7156 non-pregnant WRA aged 20 to 44 years in NHANES 2007–2018 cycles, 6328 had a reliable first 24-hour dietary recall. We sequentially excluded 545 participants with missing data on CVH and 10 participants with missing information on any covariates. The final sample for our analysis was 5773. NHANES protocols were approved by the NCHS Ethics Review Board, and participating adults provided informed consent.

Estimated UPF and MPF intakes

UPFs and MPF intake were estimated using up to two 24-hour dietary recalls. Participants completed the first recall in person, and followed by a phone call for the second recall 3–10 days later.

All reported food items (8-digit food codes) were classified according to Nova[1] into four groups based on the nature, extent, and purpose of industrial processing: (1) MPFs (e.g., fresh or frozen fruits or vegetables, grains, meats, fish, milk and plain yogurt); (2) processed culinary ingredients (e.g., table sugar, plant oils, animal fats, and salt); (3) processed foods (e.g., cheese, canned fruits and vegetables, canned/smoked/cured meats and fishes, and salted nuts); and (4) UPFs (e.g., industrial grain foods sweet or savory packaged snacks, sausages, chicken nuggets and other reconstituted meats, sugar-sweetened beverages, and other ready-to-eat/heat formulations of several ingredients). US Department of Agriculture (USDA) Food and Nutrient Database for Diet Studies (FNDDS) provided cycle-specific energy and nutrient contents for food codes.[26] For potential handmade recipes, it was the underlying standard reference (SR) codes that were classified according to Nova. FNDDS and USDA National Nutrient Database for SR databases[27] were used to calculate the energy of underlying SR codes. We sorted food items (Food Codes or SR Codes, as appropriate) into mutually exclusive Nova groups and estimated the daily %kcal from UPFs and MPFs, respectively, using two 24-hour dietary recalls when available, and one 24-hour dietary recall otherwise. We did not include processed culinary ingredients or processed foods in the analysis because they accounted for only about 13.5% of total caloric intake. Nova's detailed procedures to classify food items and estimation of calories are described elsewhere.[28]

Data from a single 24-hour dietary recall does not adequately represent a participant's usual intake because of day-to-day variations. The National Cancer Institute developed methodology to estimate the usual %kcal from UPFs and MPFs. Multiple days of dietary intake data among some participants are required in order to estimate the within- and between-individual variations.[29] The percent of the first- and second-day dietary recall data were 100% and 86.9%, respectively, in our study. We used MIXTRAN and DISTRIB macros[29] to estimate the usual intake distribution adjusting for age, sex, race and Hispanic origin, the first- or second-day dietary recalls, and whether the recall was administered on a weekday or the weekend. We used MIXTRAN and INDIVINT macros to fit nonlinear mixed regression models and generate the individual-level predicted usual intake of UPFs and MPFs for association analyses.[29,30]

CVH metrics

CVH metrics were based on the AHA's LE-8, released in 2022 (Supplemental Table 1).[21] Hemoglobin A1c values <5.7%, 5.7%–6.4%, and ≥6.5% were used as a proxy of fasting plasma glucose levels <100 mg/dL, 100 to -<126 mg/dL, and ≥126 mg/dL, respectively,[31] because hemoglobin A1c was available for nearly all participants, while fasting plasma glucose was only collected among half of participants. Use of medications for hypertension, diabetes, and dyslipidemia were self-reported. BMI was based on objectively-measured weight and height. Mean blood pressure was obtained at mobile examination center under standard conditions, and calculated using up to three readings.

Because UPFs and MPFs were derived from dietary data and both UPFs and MPFs are correlated with dietary scores, our main analysis excluded dietary scores from the LE-8 and used tertiles of scores from the remaining seven components. Each LE-8 component score ranged from 0 to 100, with a higher score indicating better health. In a supplemental analysis, the LE-8 summary score (the mean of eight metric components) was categorized as low (<50), moderate (50–79) and high (≥80) CVH.[21]

Covariates

Demographic and socioeconomic variables included age, race and Hispanic origin (non-Hispanic White, non-Hispanic Black, Hispanic, or others); education (<12, 12, or >12 years); marital status (never married, married/living with partner, or divorced/separated/widowed); poverty-to-income ratio [PIR, a ratio of household income to the poverty threshold, accounting for family size: <1.30, 1.30–3.49, and ≥3.50, and missing ($n = 452$)]; alcohol consumption (no use: 0 drinks per day; moderate use: <1 drink per day, and heavy use: ≥1 drink per day, and missing [$n = 450$]); and health insurance (yes/no).

Statistical analyses

We used SUDAAN version 11 (RTI International, Research Triangle Park, NC) for the statistical analyses. We compared characteristics across tertiles of CVH score (excluding the diet component) by analyses of variance for continuous variables and χ^2 for categorical variables. We used the combined 12-year first 24-hour dietary sampling weights (divided by 6, for the 6 combined 2-year cycles) to represent the non-institutionalized US population and account for sampling probability, differential nonresponse and noncoverage, and the day of the week. All statistical tests were 2-tailed, and $p < 0.05$ was considered significant.

Restricted cubic spline models with four knots (20th, 40th, 60th and 80th percentiles) were used to examine departure from a linear relationship between usual %kcal from UPFs and MPFs and CVH.[32] There was no evidence of departure from linearity relationships between CVH and UPFs or MPFs (p values for non-linearity: 0.321 and 0.468, respectively). For each UPF and MPF intake percentage category, we calculated the adjusted differences in CVH scores by using the mid-point of the first quartile (Q1) of intake (43.8% of calories from UPFs and 17.9% of calories from MPFs) as the reference.[30]

Multivariable linear regression was used to estimate the change in CVH scores per 10% increase in %kcal from UPFs and MPFs. Multinomial logistic regression was used to estimate the adjusted odds ratios (aORs) for tertile 1 (T1) and T2 vs. T3 CVH comparing quartiles 2 to 4 vs. quartile 1 of usual %kcal from UPFs or MPFs. The base model adjusted for age, and race and Hispanic origin; the fully adjusted model was additionally educational attainment, marital status, PIR, alcohol consumption, and health insurance. Multiplicative interactions were tested between UPFs or MPFs and age (<30 or ≥30 years), race and Hispanic origin, marital status, education years, and PIR based on the Wald-F test.

Sensitivity analyses

The CVH metrics differ in their potential roles linking UPFs and MPFs and CVD. Four CVH health factors, i.e., BMI, non-high-density lipoprotein cholesterol, blood pressure, and fasting plasma glucose are four potential mechanisms, and overall diet quality could be a fifth. Sleep, smoking, and physical activity are not known to be mechanisms linking UPFs and MPFs to CVD.

We performed five sensitivity analyses to test the robustness of the results. (1) We examined the association between quartiles of %kcal from UPFs and MPFs and tertiles of the four CVH health factors (Supplemental Tables 2 and 3). (2) We examined the association between quartiles of %kcal from UPFs and MPFs and tertiles of the four CVH health factors plus the dietary component score (Supplemental Tables 4 and 5). We used the USDA healthy eating index-2015 scores[33] as the proxy for healthy diet scores, which were calculated using the first and second 24-hour dietary recalls in NHANES (Supplemental Table 1). (3) We examined the association between quartiles of %kcal from UPFs and MPFs and the LE-8 (including diet component) categories (low, moderate, and high) (Supplemental Tables 6 and 7). (4) We examined the association between the percentage quartiles of UPFs and MPFs by weight and tertiles of seven CVH metric score (excluding diet component) because some studies have demonstrated that the proportion of UPFs out of the total weight of foods and beverages consumed may better account for UPFs that do not contribute to energy intake (e.g., artificially sweetened drinks), as well as for properties directly related to food processing rather than those related to their nutritional characteristics (Supplemental Tables 8 and 9).[11] (5) We examined the association between quartiles of %kcal from UPFs and MPFs and tertiles of the seven CVH metric score (excluding diet component) excluding participants with total energy intake <500 or >3500 (Supplemental Tables 10 and 11).[34]

RESULTS

The mean age of women of reproductive age was 32.1 years. Over half (58.7%) of the participants were non-Hispanic White, and about two-thirds (67.5%) had an education level of college or above. WRA who were younger, non-Hispanic White, or never married were more likely to have higher CVH scores; so were WRA who had the following characteristics: a higher education level, higher PIR, moderate alcohol consumption, and health insurance (table 1).

Figures 1 and 2 show the distribution of usual %kcal from UPFs and MPFs and adjusted differences in CVH scores across the range of UPF and MPF intake. The mean usual %kcal was 57.2% and 29.3%, respectively, for UPF and MPF. The mid-points of %kcal of quartiles 1 through 4 were 43.8%, 53.5%, 60.9%, and 70.6% for UPF intake, respectively, and 17.9%, 25.2%, 31.7%, and 41.7% for MPF intake, respectively. Every 10% increase in calories from UPFs was associated with a 3.6 point lower CVH score (*P* < .001), whereas every 10% increase in calories from MPFs was associated with a 4.1 point higher CVH score (*P* <.001).

Higher UPF intake was associated with higher odds of poor CVH. Comparing quartiles 2, 3, and 4 with the lowest quartile (quartile 1) of UPF intake, the fully aORs for T1 (compared with T3 CVH) were 1.74 (95% CI: 1.51–2.01), 2.67 (95% CI: 2.07–3.44), and 4.66 (95% CI: 3.13–6.97), respectively. The corresponding aORs for T2 vs. T3 CVH were 1.23 (95% CI: 1.10–1.38), 1.44 (95% CI: 1.17–1.76), and 1.77 (95% CI: 1.29–2.44) (Table 2). In contrast, higher MPFs intake was related to lower odds of poor CVH. Comparing quartiles 2, 3, and 4 with the lowest quartile (quartile 1) of MPF intake, the fully aORs for T1 (compared with T3 CVH) were 0.61 (95% CI: 0.54–0.69), 0.39 (95% CI: 0.31–0.50), and 0.21 (95% CI: 0.14–0.31), respectively. The corresponding aORs for T2 vs. T3 CVH were 0.80 (95% CI: 0.72–0.88), 0.65 (95% CI: 0.54–0.78), and 0.49 (95% CI: 0.36–0.67) (Table 3).

The associations were consistent across age (<30 and ≥30 years), marital status, PIR, and education levels (Figures 3 and 4). The association differed by race and Hispanic origin (*P* value for the interaction <0.001), with associations observed in non-Hispanic White, Hispanic and other race WRA, and no association observed for non-Hispanic Black WRA (Figures 3 and 4). The pattern of association remained largely consistent across sensitivity analyses (Supplemental Tables 2–11). When we performed an analysis excluding participants with self-reported history of cardiovascular disease, the association between UPFs and MPFs and CVH remained consistent (results not shown).

DISCUSSION

Among US WRA, more than half of calories consumed were derived from UPFs and nearly 30% of their energy from MPFs. Higher %kcal from UPFs were associated with worse CVH. In contrast, CVH improved with higher MPF consumption. These results remained consistent when stratified by sociodemographic subgroups and in sensitivity analyses.

Increasing evidence has demonstrated the association between UPF intake and worse health outcomes,[2–14,35–38] and one study has shown high intake of MPFs was inversely related to adiposity indicators among adults.[15] However, little is known about the association between UPFs and MPFs and health among WRA. Our results are consistent with prior findings indicating that healthy dietary patterns could benefit CVH of women overall, which is important to consider across the lifespan in relation to optimal health before[39,40] and after pregancy.[41–44] Prior to pregnancy, healthy diet (characterized by consumption of fruits, vegetables, legumes, nuts, and fish, as well as limited consumption of red meat) are associated with lower risks of hypertensive disorders of pregnancy, gestational diabetes, and preterm delivery.[23] In addition, in the year prior to conception, a mother’s diet may influence fetal growth, gestational age, and infant birth weight.[39] High protein and fruits intake may lower risk of preterm delivery, whereas high fat and sugar intake may increase risk of preterm delivery.[39] Our analyses demonstrated the inverse association of UPFs and positive associations of MPFs with CVH among WRA. Future research could explore whether increased caloric intake from UPFs might be associated with increased pregnancy-related complications, or whether replacing UPFs with MPFs might reduce pregnancy-related complications.

The association between UPFs and CVH may be driven by multiple mechanisms.[45,46] First, UPFs are typically calorically dense, with large quantities of added sugar, fats, and salt.[4] Combinations of sweetness, fat, and salt, as well as high-intensity flavorings, make UPFs highly palatable, which may override natural satiety mechanisms.[47] Second, UPFs generally lack dietary fiber, vitamins, and minerals,[4] so they are not as filling as MPFs.[48] Third, UPFs also may speed the rate of eating compared with MPFs,[48] potentially resulting in quicker caloric consumption, and a higher quantity of total energy intake if individuals consume more food before satiety signals can reach the brain. Fourth, UPF intake is associated with higher exposure to chemicals from food packaging (i.e., bisphenol and phthalates), which have been linked to adiposity, diabetes, hypertension, and coronary artery disease.[49,50] We are not aware of any studies that may explain the mechanisms underlying the relationship between MPFs and CVH. Benefits may be derived from greater satiety (such as via higher fiber or protein content of MPFs), lower energy density of MPFs, or other pathways by which consumption of MPFs may lead to less food intake overall. Alternatively, consumption of MPFs may affect health indirectly by replacing intake of UPFs; lowered intake of UPFs could impact CVH via the mechanisms discussed above.

This work is the first, to our knowledge, to analyze the association between UPFs and MPFs and CVH using the AHA's LE-8 framework among WRA in the United States. Dietary intake data was obtained from a large, representative sample of US WRA, and measurement error models were used to account for individual-level dietary variation.

This study has limitations. First, causal associations between dietary intake and CVH could not be inferred from cross-sectional data. Second, if people who developed cardiovascular risk factors or cardiovascular disease subsequently took steps to improve their diets, reverse causality could have led to an underestimate of the association between UPFs and CVH. Third, NHANES did not consistently collect data about extent and purpose of food processing for all foods, which could have led to misclassification of UPFs and MPFs. Fourth, confounding from unmeasured factors could have influenced our results. For example, higher UPF or lower MPF might serve as a proxy for worse dietary quality, health-related behaviors, or unmeasured social determinants of health, which could have inflated the observed associations between UPFs and MPFs and CVH.[51] Fifth, recall bias may have biased our results toward the null if persons with obesity or other poorer CVH factors were more likely to under-report unhealthy food intake.[52,53] Finally, UPFs and MPFs were relying on 1–2 days of recall, which could cause nondifferential misclassification leading to an underestimate of the strength of association.

Among women of reproductive age in the United States, UPFs represented more than half of total calorie intake, while MPFs accounted for less than one third. Our study is the first to report a graded inverse association between UPFs and CVH and a positive association between MPFs and CVH in this population. Our analyses highlight intersections between nutrition and CVH and potential opportunities for clinical and public health efforts to support healthier food consumption among WRA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGEMENT

We would like to thank the staff and participants in the 2007–2018 NHANES for their hard work and dedication. We also warmly thank our colleagues from Brazil for the development of the Nova classification system.

Abbreviations:

AHA	American Heart Association
BMI	body mass index
CI	confidence interval
CVD	cardiovascular disease
CVH	cardiovascular health
FNDDS	Food and Nutrient Database for Dietary Studies
LE-8	Life’s Essential 8
MPFs	unprocessed/minimally-processed foods
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
OR	odds ratio
SR	Standard Reference
WRA	women of reproductive age
UPF	ultra-processed foods
USDA	U.S. Department of Agriculture
%kcal	percentage of calories

References

1. Monteiro CA, Cannon G, Moubarac JC, et al. The UN Decade of Nutrition, the NOVA food classification and the trouble with ultra-processing. Public Health Nutr 2018;21:5–17. doi: 10.1017/S1368980017000234 [PubMed: 28322183]
2. Srour B, Fezeu LK, Kesse-Guyot E, et al. Ultraprocessed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). BMJ 2019;365:l1451. doi: 10.1136/bmj.l1451 [PubMed: 31142457]

3. Mendonca RD, Pimenta AM, Gea A, et al. Ultraprocessed food consumption and risk of overweight and obesity: the University of Navarra Follow-Up (SUN) cohort study. Am J Clin Nutr 2016;104:1433–1440. doi: 10.3945/ajcn.116.135004 [PubMed: 27733404]

4. Canhada SL, Luft VC, Giatti L, et al. Ultra-processed foods, incident overweight and obesity, and longitudinal changes in weight and waist circumference: the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). Public Health Nutr 2020;23:1076–1086. doi: 10.1017/S1368980019002854 [PubMed: 31619309]

5. Juul F, Martinez-Steele E, Parekh N, et al. Ultraprocessed food consumption and excess weight among US adults. Br J Nutr 2018;120:90–100. doi: 10.1017/S0007114518001046 [PubMed: 29729673]

6. Nardocci M, Leclerc BS, Louzada ML,et al. Consumption of ultra-processed foods and obesity in Canada. Can J Public Health 2019;110:4–14. doi: 10.17269/s41997-018-0130-x [PubMed: 30238324]

7. Rauber F, Campagnolo PD, Hoffman DJ, et al. Consumption of ultra-processed food products and its effects on children's lipid profiles: a longitudinal study. Nutr Metab Cardiovasc Dis 2015;25:116–122. 10.1016/j.numecd.2014.08.001 [PubMed: 25240690]

8. Mendonca RD, Lopes AC, Pimenta AM, et al. Ultra-processed food consumption and the incidence of hypertension in a Mediterranean cohort: the Seguimiento Universidad de Navarra Project. Am J Hypertens 2017;30:358–366. 10.1093/ajh/hpw137 [PubMed: 27927627]

9. Martinez Steele E, Juul F, Neri D, et al. Dietary share of ultra-processed foods and metabolic syndrome in the US adult population. Prev Med 2019;125:40–48. doi: 10.1016/j.ypmed.2019.05.004 [PubMed: 31077725]

10. Tavares LF, Fonseca SC, Garcia Rosa ML, et al. Relationship between ultra-processed foods and metabolic syndrome in adolescents from a Brazilian family doctor program. Public Health Nutr 2012;15:82–87. doi: 10.1017/s1368980011001571 [PubMed: 21752314]

11. Srour B, Fezeu LK, Kesse-Guyot E, et al. Ultraprocessed food consumption and risk of type 2 diabetes among participants of the NutriNet-Santé prospective cohort. JAMA Intern Med 2020;180:283–291. doi: 10.1001/jamainternmed.2019.5942 [PubMed: 31841598]

12. Schnabel L, Kesse-Guyot E, Alles B, et al. Association between ultraprocessed food consumption and risk of mortality among middle-aged adults in France. JAMA Intern Med 2019;179:490–498. doi: 10.1001/jamainternmed.2018.7289 [PubMed: 30742202]

13. Rico-Campa A, Martinez-Gonzalez MA, Alvarez-Alvarez I, et al. Association between consumption of ultra-processed foods and all cause mortality: SUN prospective cohort study. BMJ 2019;365:l1949. doi: 10.1136/bmj.l1949 [PubMed: 31142450]

14. Blanco-Rojo R, Sandoval-Insausti H, Lopez-Garcia E, et al. Consumption of ultra-processed foods and mortality: a national prospective cohort in Spain. Mayo Clin Proc 2019;94:2178–2188. doi: 10.1016/j.mayocp.2019.03.035 [PubMed: 31623843]

15. Zhang Z, Kahn HS, Jackson SL, et al. Associations between ultra- or minimally processed food intake and three adiposity indicators among US adults: NHANES 2011 to 2016. Obesity (Silver Spring) 2022;30:1887–1897. doi: 10.1002/oby.23507 [PubMed: 35924441]

16. Lloyd-Jones DM, Hong Y, Labarthe D, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. Circulation 2010;121:586–613. doi: 10.1161/circulationaha.109.192703 [PubMed: 20089546]

17. Folsom AR, Yatsuya H, Nettleton JA, et al. , Atherosclerosis Risk in Communities (ARIC) Study Investigators. Community prevalence of ideal cardiovascular health, by the American Heart Association definition, and relationship with cardiovascular disease incidence. J Am Coll Cardiol 2011;57:1690–1696. doi: 10.1016/j.jacc.2010.11.041 [PubMed: 21492767]

18. Yang Q, Cogswell ME, Flanders WD, et al. Trends in cardiovascular health metrics and associations with all-cause and CVD mortality among US adults. JAMA 2012;307:1273–1283. doi: 10.1001/jama.2012.339 [PubMed: 22427615]

19. Zhang Z, Jackson S, Martinez E, et al. Association between ultra-processed food intake and cardiovascular health among U.S. adults: a cross-sectional analysis of the NHANES 2011–2016. Am J Clin Nutr 2021;113:428–436. doi: 10.1093/ajcn/nqaa276 [PubMed: 33021623]

20. Zhang Z, Jackson SL, Steele EM, et al. Relationship between ultraprocessed food intake and cardiovascular health among U.S. adolescents: results from the National Health and Nutrition Examination Survey 2007–2018. J Adolesc Health 2022;70:249–257. doi: 10.1016/j.jadohealth.2021.09.031 [PubMed: 34716093]
21. Lloyd-Jones DM, Allen NB, Anderson CAM, et al. Life's Essential 8 — Updating and enhancing the American Heart Association's construct of cardiovascular health: A Presidential Advisory from the American Heart Association. Circulation 2022;146:e18–e43. doi: 10.1161/cir.0000000000001078 [PubMed: 35766027]
22. Tsao CW, Aday AW, Almarzooq ZI, et al. Heart disease and stroke statistics-2023 update: a report from the American Heart Association. Circulation 2023;147:e93–e621. doi: 10.1161/cir.0000000000001123 [PubMed: 36695182]
23. Stephenson J, Heslehurst N, Hall J, et al. Before the beginning: nutrition and lifestyle in the preconception period and its importance for future health. Lancet 2018;391:1830–1841. doi: https://doi.org/10.1016%2FS0140-6736(18)30311-8 [PubMed: 29673873]
24. Courtney AU, O'Brien EC, Crowley RK, et al. DASH (Dietary Approaches to Stop Hypertension) dietary pattern and maternal blood pressure in pregnancy. J Hum Nutr Diet 2020;33:686–697. doi: 10.1111/jhn.12744 [PubMed: 32196793]
25. CDC National Health and Nutrition Examination Survey. Available from: https://www.cdc.gov/nchs/nhanes/index.htm [Last accessed: July 7, 2023].
26. US Department of Agriculture, Agriculture Research Service. Food and Nutrient Database for Dietary Studies. Available from: https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/food-surveys-research-group/docs/fndds/ [Last accessed: July 7, 2023].
27. US Department of Agriculture, Agricultural Research Service. Nutrient data laboratory. USDA national nutrient database for standard reference. 2016. Available from: http://www.ars.usda.gov/nea/bhnrc/mafcl [Last accessed: July 7, 2023].
28. Steele EM, O'Connor LE, Juul F, et al. Identifying and estimating ultraprocessed food intake in the US NHANES according to the Nova classification system of food processing. J Nutr 2022;153:225–241. doi: 10.1016/j.tjnut.2022.09.001 [PubMed: 36913457]
29. Guenther PM, Kott PS, Carriquiry AL. Development of an approach for estimating usual nutrient intake distributions at the population level. J Nutr 1997;127:1106–1112. doi: 10.1093/jn/127.6.1106 [PubMed: 9187624]
30. Kipnis V, Midthune D, Buckman DW, et al. Modeling data with excess zeros and measurement error: Application to evaluating relationships between episodically consumed foods and health outcomes. Biometrics 2009;65:1003–1010. doi: 10.1111/j.1541-0420.2009.01223.x [PubMed: 19302405]
31. American Diabetes Association. Standards of medical care in diabetes 2010. Diabetes Care 2010;33:S11–S61. doi: 10.2337/dc10-S011 [PubMed: 20042772]
32. Desquilbet L, Mariotti F. Dose-response analyses using restricted cubic spline functions in public health research. Stat Med 2010;29:1037–1057. doi: 10.1002/sim.3841 [PubMed: 20087875]
33. Krebs-Smith SM, Pannucci TE, Subar AF, et al. Update of the Healthy Eating Index-2015. J Acad Nutr Diet 2018;118:1591–1602. doi: 10.1016/j.jand.2018.05.021 [PubMed: 30146071]
34. Banna JC, McCroy MA, Fialkowski MK, et al. Examining plausibility of self-reported energy intake data: Considerations for method selection. Front Nutr 2017;4:45. doi: 10.3389/fnut.2017.00045 [PubMed: 28993807]
35. Askari M, Heshmati J, Shahinfar H, et al. Ultra-processed food and the risk of overweight and obesity: a systematic review and meta-analysis of observational studies. Int J Obes (Lond) 2020;44(10):2080–2091. doi: 10.1038/s41366-020-00650-z. [PubMed: 32796919]
36. Mambrini SP, Menichetti F, Ravella S, et al. Ultra-Processed Food Consumption and Incidence of Obesity and Cardiometabolic Risk Factors in Adults: A Systematic Review of Prospective Studies. Nutrients. 2023;5(11):2583. doi: 10.3390/nu15112583.
37. Pagliai G, Dinu M, Madarena MP, et al. Consumption of ultra-processed foods and health status: a systematic review and meta-analysis. Br J Nutr 2021;125(3):308–318. doi: 10.1017/S0007114520002688. [PubMed: 32792031]

38. Santos FSD, Dias MDS, Mintem GC, et al. Food processing and cardiometabolic risk factors: a systematic review. Rev Saude Publica 2020;54:70. doi: 10.11606/s1518-8787.2020054001704 [PubMed: 32725096]
39. Grieger JA, Grzeskowiak LE, Clifton VL. Preconception dietary patterns in human pregnancies are associated with preterm delivery. J Nutr 2014;144:1075–1080. doi: 10.3945/jn.114.190686 [PubMed: 24790026]
40. Lang AY, Boyle JA, Fitzgerald GL, et al. Optimizing preconception health in women of reproductive age. Minerva Ginecol 2018;70:99–119. doi: 10.23736/s0026-4784.17.04140-5 [PubMed: 28895680]
41. Stang J, Huffman LG. Position of the Academy of Nutrition and Dietetics: obesity, reproduction, and pregnancy outcomes. J Acad Nutr Diet 2016;116:677–691. doi: 10.1016/j.jand.2016.01.008 [PubMed: 27017177]
42. Spring B, Moller AC, Colangelo LA, et al. Healthy lifestyle change and subclinical atherosclerosis in young adults: Coronary Artery Risk Development in Young Adults (CARDIA) study. Circulation 2014;130(1):10–17. doi: 0.1161/CIRCULATIONAHA.113.005445. [PubMed: 24982115]
43. Thangavel P, Puga-Olguín A, Rodríguez-Landa JF, et al. Genistein as potential therapeutic candidate for menopausal symptoms and other related diseases. Molecules 2019;24(21):3892. doi: 10.3390/molecules24213892. [PubMed: 31671813]
44. Guzek D, Bska dg, Groele B, et al. Fruit and Vegetable Dietary Patterns and Mental Health in Women: A Systematic Review. Nutrition Reviews 2022;80(6): 1357–1370. doi: 10.1093/nutrit/nuab007 [PubMed: 34041545]
45. Juul F, Vaidean G, Parekh N. Ultra-processed foods and cardiovascular diseases: Potential mechanisms of action. Adv Nutr 2021;12:1673–1680. doi: 10.1093/advances/nmab049 [PubMed: 33942057]
46. Ludwig DS. Technology, diet, and the burden of chronic disease. JAMA 2011;305:1352–1353. doi: 10.1001/jama.2011.380 [PubMed: 21467290]
47. Holt SH, Miller JC, Petocz P, et al. A satiety index of common foods. Eur J Clin Nutr 1995;49:675–690. [PubMed: 7498104]
48. Hall KD, Ayuketah A, Brychta R, et al. Ultra-processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of ad libitum food intake. Cell Metab 2019;31:67–77. doi: https://doi.org/10.1016%2Fj.cmet.2019.05.008
49. Buckley JP, KIM H, Wong E, et al. Ultra-processed food consumption and exposure to phthalates and bisphenols in the US National Health and Nutrition Examination Survey, 2013–2014. Environ Int 2019;131:105057. doi: 10.1016/j.envint.2019.105057 [PubMed: 31398592]
50. Rancière F, Lyons JG, Loh VHY, et al. Bisphenol A and the risk of cardiometabolic disorders: a systematic review with meta-analysis of the epidemiological evidence. Environ Health 2015;14:46. doi: 10.1186/s12940-015-0036-5 [PubMed: 26026606]
51. Schuit AJ, van Loon AJ, Tijhuis M, et al. Clustering of lifestyle risk factors in a general adult population. Prev Med 2002;35:219–224. doi: 10.1006/pmed.2002.1064 [PubMed: 12202063]
52. Lichtman SW, Pisarska K, Berman ER, et al. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. N Engl J Med. 1992;327(27):1893–1898. doi: 10.1056/NEJM199212313272701. [PubMed: 1454084]
53. Wehling H, Lusher J. People with a body mass index ⩾30 under-report their dietary intake: A systematic review. J Health Psychol. 2019;24(14):2042–2059. doi: 10.1177/1359105317714318 [PubMed: 28810493]

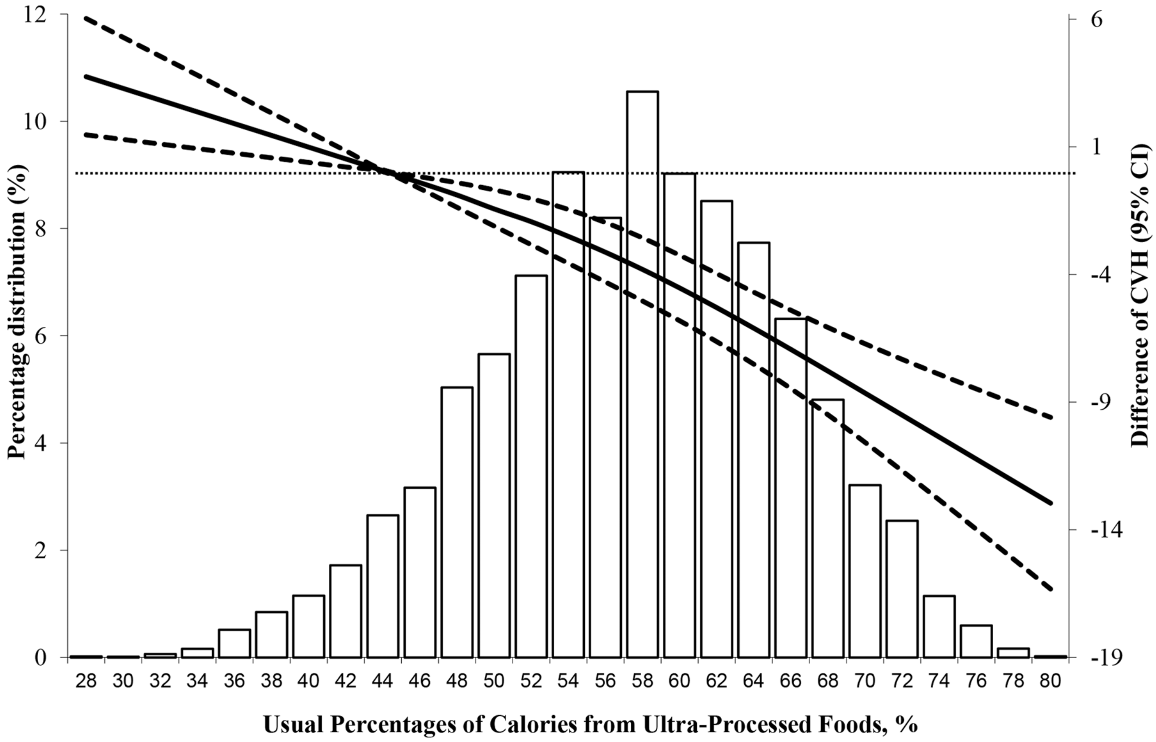

FIG. 1.

Distributions of usual percentage of calories from ultra-processed foods and adjusted differences in cardiovascular health scores (43.8% as reference), US women of reproductive age, National Health and Nutrition Examination Survey 2007–2018[1,2]

1 Adjusted by age, sex, race and Hispanic origin, education, poverty income ratio, marital status, alcohol use, and health insurance.

2 CI: confidence interval.

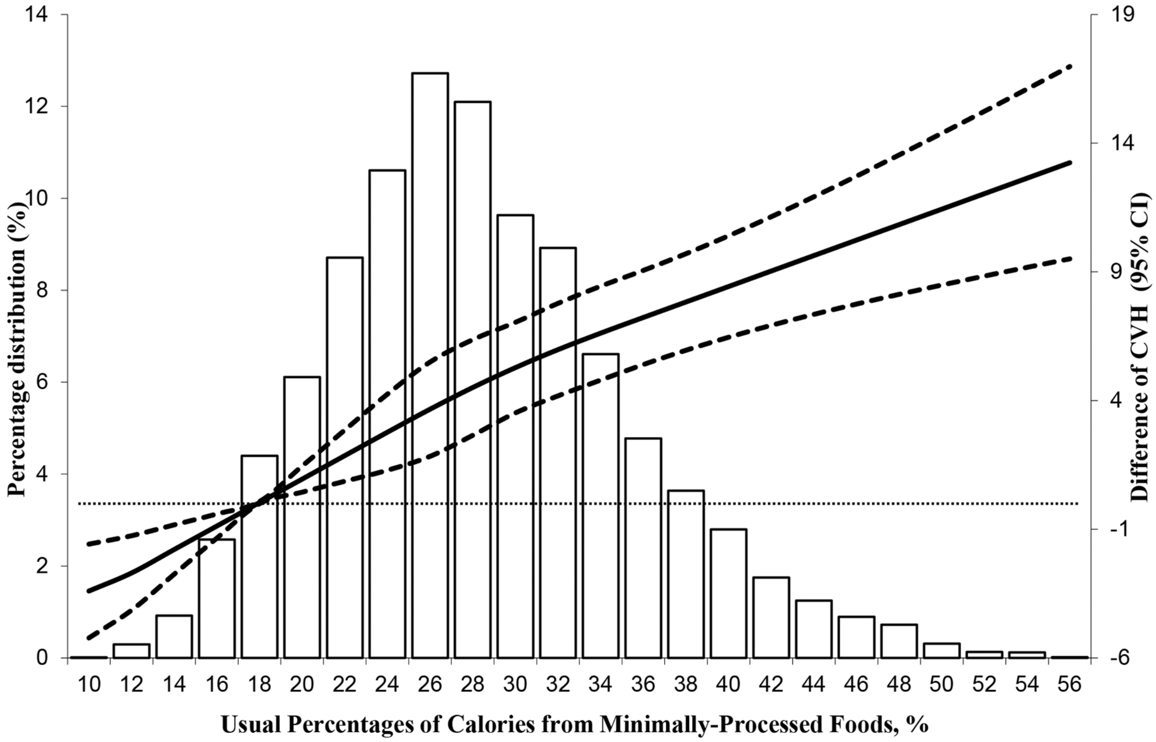

FIG. 2.

Distributions of usual percentage of calories from minimally-processed foods and adjusted differences in cardiovascular health scores (17.9% as reference), US women of reproductive age, National Health and Nutrition Examination Survey 2007–2018[1,2]

1 Adjusted by age, sex, race and Hispanic origin, education, poverty income ratio, marital status, alcohol use, and health insurance.

2 CI: confidence interval.

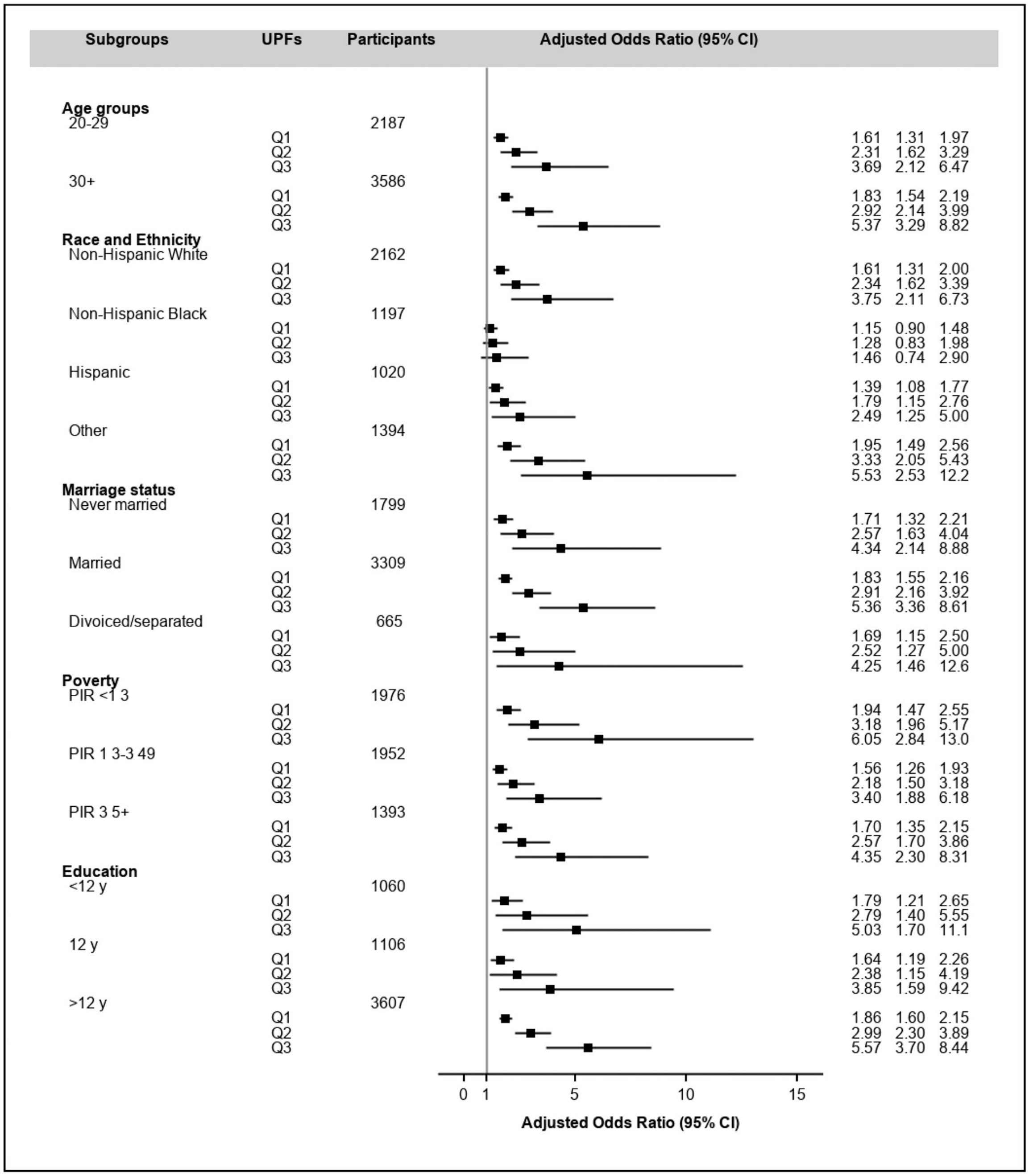

FIG. 3.

Adjusted odds ratios and 95% confidence intervals for cardiovascular health metrics associated with usual percentage of calories from ultra-processed foods according to socio-demographic strata, US women of reproductive age, National Health and Nutrition Examination Survey 2007–2018[1,2,3]

1 CI: confidence intervals; PIR: poverty-income-index; Q: quartile; UPFs: ultra-processed foods.

2 Adjusted odds ratios of tertile 1 (T1) vs T3 of 7 cardiovascular health metric scores (excluding diet component).

Author Manuscript

Author Manuscript

Author Manuscript

3 Adjusted for age as continuous variable, race-Hispanic origin, education, poverty, marital status, alcohol consumption, and health insurance.

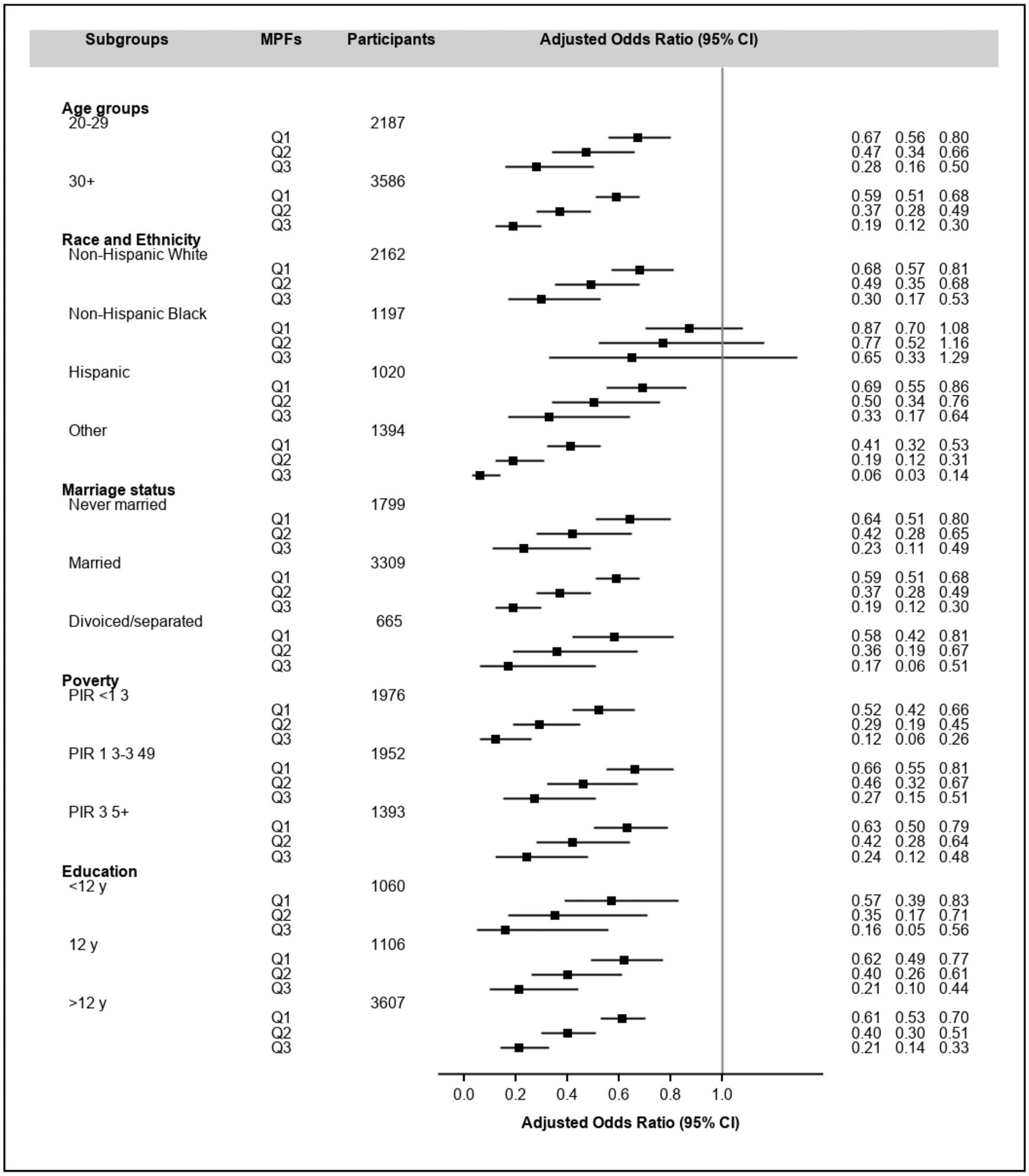

FIG. 4.

Adjusted odds ratios and 95% confidence intervals for cardiovascular health metrics associated with usual percentage of calories from minimally-processed foods according to socio-demographic strata, US women of reproductive age, National Health and Nutrition Examination Survey 2007–2018[1,2,3]

1 CI: confidence intervals; MPFs: minimally-processed foods; PIR: poverty-income-index; Q: quartile.

2 Adjusted odds ratios of tertile 1 (T1) vs T3 of 7 cardiovascular health metric scores (excluding diet component).

3 Adjusted for age as continuous variable, race-Hispanic origin, education, poverty, marital status, alcohol consumption, and health insurance.

Table 1.

Comparison of Selected Characteristics by Tertiles of Cardiovascular Health Scores (Except Diet Component)—US Women of Reproductive Age (20–44 years), National Health and Nutrition Examination Survey 2007–2018[1,2]

Characteristic	Overall (n = 5773)	T1 CVH scores (n = 2078)	T2 CVH scores (n = 1907)	T3 CVH scores (n = 1788)	P value[3]
Age, years (mean±se)	32.1±0.20	35.7±0.35	32.9±0.19	30.7±0.31	<0.001
Race and Hispanic origin (%, se)					
Non-Hispanic White	58.7 (1.78)	53.9 (2.41)	55.5 (2.36)	66.1 (1.76)	<0.001
Non-Hispanic Black	13.2 (0.96)	18.7 (1.68)	14.3 (1.25)	7.1 (0.73)	
Hispanic	11.7 (1.03)	12.8 (1.23)	13.0 (1.34)	9.4 (0.99	
Other	16.4 (0.81)	14.5 (1.05)	17.2 (1.28)	17.4 (1.10)	
Education (%, se)					
<12 years	13.6 (0.78)	21.0 (1.17)	14.9 (1.24)	5.7 (0.71)	<0.001
12 years	18.9 (0.82)	26.0 (1.21)	19.0 (1.32)	12.2 (1.01)	
>12 years	67.5 (1.28)	53.0 (1.58)	66.1 (1.88)	82.1 (1.23)	
Poverty-to-income ratio (%, se)					
0–129%	27.4 (1.06)	38.9 (1.41)	25.3 (1.42)	18.9 (1.66)	<0.001
130–349%	33.8 (1.02)	36.0 (1.31)	35.2 (1.68)	30.4 (1.55)	
≥350%	31.8 (1.19)	19.0 (1.35)	31.1 (1.93)	44.3 (1.96)	
Missing	7.0 (0.63)	6.2 (0.68)	8.4 (1.22)	6.5 (0.82)	
Marital status (%, se)					
Married/Living with partner	58.9 (1.21)	56.8 (1.59)	61.4 (1.70)	58.7 (2.04)	<0.001
Never married	30.3 (1.28)	27.6 (1.44)	27.9 (1.43)	35.0 (2.12)	
Divorced, separate or widowed	10.8 (0.62)	15.6 (0.96)	10.8 (1.03)	6.3 (0.78)	
Alcohol consumption (%, se)					
No use	49.1 (1.27)	56.7 (1.72)	50.3 (1.47)	41.0 (1.77)	<0.001
Moderate use	35.8 (1.14)	27.7 (1.48)	34.8 (1.46)	44.1 (1.85)	
Heavy use	8.6 (0.59)	8.7 (1.01)	8.3 (1.03)	8.7 (1.04)	
Missing	6.6 (0.48)	6.9 (0.79)	6.6 (0.82)	6.2 (0.75)	
Health insurance (%, se)					

Characteristic	Overall (*n* = 5773)	T1 CVH scores (*n* = 2078)	T2 CVH scores (*n* = 1907)	T3 CVH scores (*n* = 1788)	*P* value[3]
Yes	77.2 (0.87)	70.8 (1.40)	77.1 (1.25)	83.2 (1.17)	<0.001
No	22.8 (0.86)	29.2 (1.40)	22.9 (1.25)	16.8 (1.17)	

CVH: cardiovascular health; se: standard error.

[1]Tertiles of CVH scores were defined as tertile 1 (T1) CVH <68.7, T2 CVH 68.7–83.8, and T3 CVH ≥83.9 out of a maximum score of 100.

[2]*P* value for trend across CVH metric categories was assessed by analysis of variance for continuous variable, and χ^2 for categorical variables.

TABLE 2.

Odds Ratio and 95% Confidence Intervals for Cardiovascular Health Metrics (Excluding Diet Component) Associated With Ultra-Processed Foods, US Women of Reproductive Age (20–44 Years), National Health and Nutrition Examination Survey 2007–2018, $n = 5773$[1]

	Quartiles of usual percentage of calories from UPFs				***P* value**[3]
	Q1	**Q2**	**Q3**	**Q4**	
Mid-point and range of usual percentage of calories from UPFs	43.8% (30.6%–49.0%)	53.5% (49.1%–56.9%)	60.9% (57.0%–64.7%)	70.6% (64.8%–84.2%)	
Age and race and Hispanic origin adjusted					
T2 vs. T3 CVH scores	1.00	1.35 (1.22–1.49)	1.69 (1.42–2.03)	2.28 (1.72–3.03)	<0.001
T1 vs. T3 CVH scores	1.00	2.15 (1.88–2.46)	3.86 (3.05–4.88)	8.29 (5.74–11.97)	<0.001
Fully adjusted[2]					
T2 vs. T3 CVH scores	1.00	1.23 (1.10–1.38)	1.44 (1.17–1.76)	1.77 (1.29–2.44)	<0.001
T1 vs. T3 CVH scores	1.00	1.74 (1.51–2.01)	2.67 (2.07–3.44)	4.66 (3.13–6.97)	<0.001

CVH, cardiovascular health; Q: quartile; T: tertile; UPF, ultra-processed foods.

[1] Tertiles of CVH scores were defined as T1 CVH <69.7, T2 CVH 69.7–84.1, and T3 CVH ≥84.2.

[2] Adjusted for age as continuous variable, race and Hispanic origin, education, poverty-to-income ratio, marital status, alcohol consumption, and health insurance.

[3] *P*-value of beta-coefficient for percentage of calories (continuous) from UPFs in the multinomial logistic regression models.

TABLE 3.

Odds Ratio and 95% Confidence Intervals for Cardiovascular Health Metrics Associated With Minimally-Processed Foods, US Women of Reproductive Age (20–44 years), National Health and Nutrition Examination Survey 2007–2018, *n* = 5773[1]

	Quartiles of usual percentage of calories from MPFs				***P* value[3]**
	Q1	**Q2**	**Q3**	**Q4**	
Mid-point and range of usual percentage of calories from MPFs	17.9% (9.9%–21.9%)	25.2% (22.0%–28.3%)	31.7% (28.4%–35.8%)	41.3% (35.9%–56.5%)	
Age and race and Hispanic origin adjusted					
T2 vs. T3 CVH scores	1.00	0.77 (0.70–0.84)	0.61 (0.51–0.73)	0.43 (0.32–0.58)	<0.001
T1 vs. T3 CVH scores	1.00	0.54 (0.48–0.60)	0.31 (0.25–0.39)	0.14 (0.10–0.20)	<0.001
Fully adjusted[2]					
T2 vs. T3 CVH scores	1.00	0.80 (0.72–0.88)	0.65 (0.54–0.78)	0.49 (0.36–0.67)	<0.001
T1 vs. T3 CVH scores	1.00	0.61 (0.54–0.69)	0.39 (0.31–0.50)	0.21 (0.14–0.31)	<0.001

CVH, cardiovascular health (diet component excluded); MPFs, minimally-processed foods; Q: quartile; T: tertile.

[1]Tertiles of CVH scores were defined as T1 CVH <69.7, T2 CVH 69.7–84.1, and T3 CVH ≥84.2.

[2]Adjusted for age as continuous variable, race and Hispanic origin, education, poverty-to-income ratio, marital status, alcohol consumption, and health insurance.

[3]*P*-value of beta-coefficient for percentage of calories (continuous) from MPFs in the multinomial logistic regression model