Published in final edited form as:
Obesity (Silver Spring). 2022 September ; 30(9): 1887–1897. doi:10.1002/oby.23507.

Associations between ultra- or minimally processed food intake and three adiposity indicators among US adults: NHANES 2011 to 2016

Zefeng Zhang[1], **Henry S. Kahn**[2], **Sandra L. Jackson**[1], **Euridice Martinez Steele**[3,4], **Cathleen Gillespie**[1], **Quanhe Yang**[1]

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

[2]Emory University School of Medicine, Atlanta, Georgia, USA

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

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

Abstract

Objective: Ultraprocessed food (UPF) intake is associated with BMI, but effects on regional adipose depots or related to minimally processed food (MPF) intake are unknown.

Methods: Data included 12,297 adults in the National Health and Nutrition Examination Survey (NHANES), 2011 to 2016. This study analyzed associations between usual percentage of kilocalories from UPFs and MPFs and three adiposity indicators: supine sagittal abdominal diameter to height ratio (SADHtR, estimates visceral adiposity); waist circumference to height ratio (WHtR, estimates abdominal adiposity); and BMI, using linear and multinomial logistic regression.

Results: Standardized β coefficients per 10% increase in UPF intake were 0.0926, 0.0846, and 0.0791 for SADHtR, WHtR, and BMI, respectively (all $p < 0.001$; $p > 0.26$ for pairwise differences). For MPF intake, the β coefficients were −0.0901, −0.0806, and −0.0688 (all $p < 0.001$; $p > 0.18$ pairwise). Adjusted odds ratios (95% CI) for adiposity tertile 3 versus tertile 1 (comparing UPF intake quartiles 2, 3, and 4 to quartile 1) were 1.33 (1.22-1.45), 1.67 (1.43-1.95), and 2.24 (1.76-2.86), respectively, for SADHtR; 1.31 (1.19-1.44), 1.62 (1.37-1.91), and 2.13

Correspondence Zefeng Zhang, Division for Heart Disease and Stroke Prevention, Centers for Disease Control and Prevention, 4770 Buford Highway NE, Mail Stop S107-1,Atlanta, GA 30341, USA. hwv0@cdc.gov.

AUTHOR CONTRIBUTIONS

Zefeng Zhang, Henry S. Kahn, and Quanhe Yang conceived and designed the study. Zefeng Zhang and Quanhe Yang analyzed the data; Zefeng Zhang and Quanhe Yang produced the figures and tables. Zefeng Zhang, Henry S. Kahn, Sandra L. Jackson, Euridice Martinez Steele, Cathleen Gillespie, and Quanhe Yang interpreted the data. Zefeng Zhang and Henry S. Kahn searched the literature and wrote the first draft, with insightful contributions from Sandra L. Jackson, Euridice Martinez Steele, Cathleen Gillespie, and Quanhe Yang. 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 declared no conflict of interest.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

(1.63-2.78), respectively, for WHtR; and 1.27 (1.16-1.39), 1.53 (1.31-1.79), and 1.96 (1.53-2.51), respectively, for BMI. MPF intake showed inverse associations with similar trends in association strength.

Conclusions: Among US adults, abdominal and visceral adiposity indictors were positively associated with UPFs and inversely associated with MPFs.

INTRODUCTION

The Nova food classification system identifies four mutually exclusive categories of foods and food products according to the extent and purpose of the industrial processing they undergo [1]. Nova class 1 includes unprocessed or minimally processed foods (MPFs). Since 2001, among United States (US) adults, the consumption of MPFs has represented about 30% of total calories, and it is declining [2]. Nova class 4 includes ultraprocessed foods (UPFs), the consumption of which has represented about 55% of total caloric intake; this fraction is rising [2]. UPF consumption is generally high in high-income countries and is increasing in low- and middle-income countries [3].

As recently summarized [4], a growing body of international evidence has shown that high intake of UPFs among adults is associated cross-sectionally with increased BMI and categorical obesity [5-7]. These observations have been supported by prospective demonstrations that higher consumption of UPFs confers a higher risk of future weight gain [8, 9]. Cross-sectional and prospective associations of UPF intake, also with increased waist circumference (WC), have been reported by studies from the US [5, 10], the UK [7, 9], Australia [6], Brazil [8], Spain [11], and the Republic of Korea [12].

In this report, we use inexpensive adiposity indicators other than BMI and WC to help clarify how dietary intakes of UPFs and MPFs might be associated with regional accumulations of adipose tissue. Our novel analyses focus on the sagittal abdominal diameter to height ratio (SADHtR) and the WC to height ratio (WHtR).

The US National Health and Nutrition Examination Survey (NHANES) during only 2011 to 2016 measured the supine sagittal abdominal diameter (SAD, also called “abdominal height”) using a sliding-beam caliper [13]. Studies using abdominal magnetic resonance imaging or computed tomography images have demonstrated that visceral fat areas are more strongly correlated with SAD than with WC [14, 15]. Abdominal subcutaneous fat contributes relatively little to the variation in SAD because the subcutaneous depot falls away toward the sides of a supine belly. Visceral fat, by contrast, remains near the midline. Thus, SAD serves as an anthropometric estimator specifically of visceral adiposity. Early studies have demonstrated that supine SAD is strongly linked to cardiometabolic risk factors for men and women; SADHtR showed a stronger association with some of the risk factors than uncorrected SAD [16].

Our analyses focus also on the WHtR, an abdominal estimator of cardiometabolic risk possibly superior to the uncorrected WC [17]. Obtained in the standing position, WC captures abdominal adipose tissue from both subcutaneous and visceral depots. As applied in clinical anthropometry, the correction of WC for height tends to reduce the sex difference found for the uncorrected WC. Another advantage of using WHtR is that increased height usually identifies adults with relatively longer legs, and greater leg length can support enlarged adipose depots in the lower extremities, which are associated with reduced cardiometabolic risk [18, 19].

An earlier report based on data from NHANES compared SADHtR, WHtR, and BMI for identifying adults with elevated concentrations of fasting triglycerides or levels of insulin resistance [20]. In adjusted models, SADHtR significantly outperformed the BMI for identifying persons located in the top quartile of the cardiometabolic biomarkers. The risk ratios for the WHtR consistently fell midway between those for SADHtR and BMI.

METHODS

Data source and participants

NHANES is a series of cross-sectional, nationally representative surveys of the US noninstitutionalized civilian population conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC) [21]. We report here on NHANES examinations conducted from 2011 through 2016. Participants were recruited using a complex, stratified, multistage probability cluster-sampling design. They completed personal structured interviews at home and then had a physical examination at a mobile examination center. Among 17,048 participants aged ≥20 years, a total of 14,865 had a complete first 24-hour dietary recall. We sequentially excluded 174 pregnant women, 849 participants who had missing data on any adiposity indicators, 130 participants who had missing information on any covariates, and 1,415 participants with implausible energy intake (defined as consumption of <800 kcal/d or >4,200 kcal/d in men and <500 kcal/d or >3,500 kcal/d in women) [22]. The final sample for the analyses was 12,297 (Figure 1). The NCHS Research Ethics Review Board reviewed and approved NHANES, and all adult participants provided written informed consent.

Adiposity indicators

The SAD was measured by a portable, sliding-beam caliper using a standardized protocol. Supine participants rested on a lightly padded exam table with their hips in a flexed position as the examiner marked the level of their iliac crests. The lower arm of the caliper was then positioned under the small of the back, and the upper arm was raised above the belly in alignment with the iliac-crest level [13]. The examiner asked the participant to inhale gently, slowly let the air out, and then relax. The examiner then lowered the caliper's upper arm, letting it lightly touch the abdomen but without compressing it. The SAD value, recorded to the nearest 0.1 cm, was read directly from a tape measure fixed to the caliper shaft. For 94.4% of adults, we defined SAD as the mean of two initial measurements; when the difference between the first and second measurements exceeded 0.5 cm (5.6%), we used the mean of up to four measurements [23]. SADHtR was calculated as SAD in centimeters divided by height in centimeters.

Weight, height, and WC (measured in a standing position by tape measure just above the uppermost lateral border of the ilium) were obtained by established methods [24]. WHtR was calculated as WC in centimeters divided by height in centimeters. BMI was calculated as weight in kilograms divided by height in meters squared. The SADHtR, WHtR, and BMI were each scaled to their sex-specific tertile distributions.

Estimated UPF and MPF intakes

We used 24-hour dietary recalls to estimate the intakes of UPFs and MPFs. NHANES collected up to two 24-hour dietary recall data among participants. The first recall was administered in person at the mobile examination center, and the second recall occurred via phone 3 to 10 days later. The US Department of Agriculture (USDA) Food and Nutrient Database for Diet Studies (FNDDS) converts consumed foods and beverages into gram amounts and determines their nutrient values by using eight-digit food codes.

All recorded food codes were classified according to Nova [25]. The MPFs (Nova class 1, including unprocessed foods) are defined as foods that may be altered only by processes such as removal of inedible or unwanted parts, drying, crushing, grinding, fractioning, roasting, boiling, pasteurization, refrigeration, freezing, placing in containers, vacuum packaging, or nonalcoholic fermentation. None of these processes adds salt, sugar, oils or fats, or other food substances to the original food. Examples of MPFs include fresh, dry, or frozen fruit or vegetables, grains, legumes, nuts, meat, fish, and milk. The processed culinary ingredients (Nova class 2) include table sugar, oils, fats, and salt. The processed foods (Nova class 3) include foods such as canned fish and vegetables and artisanal cheeses, which are manufactured by adding salt, sugar, oil, or other processed culinary ingredients to minimally processed foods. The UPFs (Nova class 4) are defined as industrial formulations made mostly or entirely from substances extracted from foods or derived from food constituents with little, if any, intact food. These items often contain added flavors, colors, emulsifiers, and other cosmetic additives. Examples of UPFs include 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. For food codes judged to be a homemade recipe, the classification was applied to the underlying standard reference (SR) codes or ingredient codes obtained from the cycle-specific USDA FNDDS data-bases [26]. SR code energy values were obtained using the cycle-specific USDA FNDDS [26] and SR databases [27]. We sorted food items (food codes or SR codes) into mutually exclusive Nova groups and estimated the percentage of kilocalories from UPFs and from MPFs.

We did not address Nova class 2 or class 3, which together accounted for only about 16% of adult total caloric intake [2]. Further details on classification procedures of the Nova system [1] and estimation of Nova calorie contributions have been described elsewhere [25].

Dietary data from a single 24-hour recall may not adequately represent a participant's usual intake because of day-to-day variations in diet. Because the use of single-day data may bias our estimates, we therefore used the National Cancer Institute method to estimate the usual percentage of kilocalories from UPFs and MPFs. This method requires at least some respondents to have multiple days of dietary intake in order to estimate the within- and between-individual variations [28]. For estimating the distribution of usual intake, we used MIXTRAN and DISTRIB macros and adjusted for age, sex, race-Hispanic origin, the first- or second-day dietary recalls (all participants had a first-day and 87.3% had a second-day dietary recall), and the day of the week when 24-hour recall was collected (weekday [Monday-Thursday] vs. weekend [Friday-Sunday]). For the association study, we fitted the nonlinear mixed regression models and generated the predicted individual's usual intake of UPFs and MPFs given the reported intakes and other covariates using MIXTRAN and INDIVINT macros. Although these predicted usual intakes may still have errors, under the regression calibration assumptions, these errors may not affect the estimated association between UPFs and MPFs and adiposity indicators [29].

Covariates

For base-model analyses, our analytic covariates included categorical age (20-29, 30-39, 40-49, 50-59, 60-69, and >70 years), sex, and a five-level ancestral marker based on race-Hispanic origin (non-Hispanic White, non-Hispanic Black, non-Hispanic Asian, Hispanic, and others [including mixed races]). This "race-Hispanic origin" marker was derived from participant responses to the questions regarding "Hispanic/Latino or Spanish origin" and self-identified race(s). Although the responses can be imprecise, they may reflect social and cultural patterns related to participants' methods of food preparation, migration history, size of household, occupational opportunities, etc. The marker is not intended to indicate genetic or biological distinctions.

Our fully adjusted analyses included additional covariates describing educational attainment (less than high school, high school graduate, and more than high school), physical activity (ideal, intermediate, and poor), smoking status (never, former, and current), and awareness of a diabetes diagnosis. Ideal physical activity was defined as ≥150 minutes of moderate activity or ≥ 75 minutes of vigorous activity or an equivalent combination of moderate and vigorous activity per week; intermediate physical activity was defined as 1 to 149 minutes of moderate activity or 1 to 74 minutes of vigorous activity or an equivalent combination of moderate and vigorous activity per week; poor physical activity was defined as no daily moderate or vigorous activity (30). Participants were categorized as a current smoker if they smoked at least 100 cigarettes in life and currently smoked; a former smoker if they smoked at least 100 cigarettes in life and did not currently smoke; and a nonsmoker if they smoked less than 100 cigarettes in life. Awareness of diagnosed diabetes was established based on the participant's response to the question "Have you ever been told by a doctor or health professional that you have diabetes or sugar diabetes?"

Statistical analyses

Statistical analyses were performed using R version 4.0.3, SAS version 9.4 (SAS Institute Inc.), and SUDAAN version 11 (RTI International), accounting for the complex sampling design. Characteristics were expressed as means for continuous variables or as percentages for categorical variables. We used biannual sampling weights for the firstday 24-hour dietary recall, dividing them by three (data from three biannual cycles in NHANES) to represent the noninstitutionalized US population and account for sampling probability and nonresponse. All statistical tests were two-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 percentage of kilocalories from UPFs and MPFs and the three adiposity indicators [31]. There was no evidence of nonlinearity when modeling SADHtR, WHtR, and BMI either for UPFs (p values for nonlinearity, respectively: 0.146, 0.131, and 0.131) or for MPFs (p values for nonlinearity: 0.184, 0.243, and 0.132). We then calculated, for each percentage of UPF/MPF intake category in the histogram, the adjusted differences in SADHtR, WHtR, and BMI by using the midpoint of the lowest quartile (Q1) of intake (40.2% of calories from UPFs and 18.1% of calories from MPFs) as the reference.

We used multivariable linear regression to estimate the change in each of three adiposity indicators per 10% increase of percentage of kilocalories from UPFs and MPFs. To compare whether the associations between UPFs or MPFs and three adiposity indicators differed significantly, we calculated the standardized β coefficients. The standardized β coefficients can be interpreted as standard deviation (SD) changes of adiposity indicators per 10% increase of percentage of kilocalories from UPF or MPF intake. We first used R to calculate SD of the three adiposity indicators for a complex survey and then standardized SADHtR using the following formula: (SADHtR – mean of SADHtR)/SD of SADHtR. The same standardization formulas were used for WHtR and BMI. We then used standardized adiposity indicators as the dependent variable and included UPFs (or MPFs), categorical age, sex, race-Hispanic origin, education, smoking, physical activity, and awareness of diabetes in the multivariable linear regression model in SUDAAN to get the standardized β coefficients. A more positive standardized β coefficient indicates a stronger association of adiposity with the consumed food items (UPFs or MPFs), and a more negative standardized β coefficient indicates a stronger inverse association with accumulated adiposity. We used the z test to determine whether the difference between the standardized β coefficients was statistically significant between pairs of compared adiposity indicators.

We used multinomial logistic regression models to estimate the adjusted odds ratios (OR) for tertile 3 and tertile 2 versus tertile 1 of adiposity indicators comparing quartiles 2, 3, and 4 versus quartile 1 of usual percentage of kilocalories from UPFs or MPFs. We tested the multiplicative interaction between UPFs or MPFs and age (<50 or ≥ 50 years) and sex on the three indicators.

Because we excluded 17.3% of NHANES participants (1,415 participants with implausible energy intake and 1,153 participants who were pregnant, with missing adiposity indicators, or missing other covariates), we compared the weighted distributions of anthropometry, demographic, and clinical characteristics between the analytic sample and all participants with complete dietary recall to assess the representativeness of the analytic sample. We also conducted a sensitivity analysis to examine the association between UPFs and MPFs and three adiposity indicators by including the participants with implausible total energy intake (92.2% of all participants).

RESULTS

The simple distributions in our analytic sample of adiposity indicators, age, demographic, and clinical variables are presented as weighted estimates in Table 1. Further detail related to the tertile cut points of the adiposity indicators is presented in Table 2. For each anthropometric variable (excepting height) the inter-tertile range was greater for women than for men. This observation supports our analytic strategy to employ sex-specific anthropometric tertiles for scaling the adiposity indicators in our multinomial logistic regression analyses.

Figure 2 shows the distribution of usual percentage of kilocalories from UPFs and MPFs and adjusted differences for each percentage of UPF/MPF intake in three adiposity indicators (the midpoint of the lowest quartile: 40.2% for UPFs and 18.1% for MPFs as reference, respectively). The mean usual percentage of kilocalories from UPF and MPF intake was 55.0% and 30.1%, respectively. The midpoints of percentage of kilocalories of quartiles 1 through 4 were 40.2%, 50.8%, 59.0%, and 69.9% for UPF intake and were 18.1%, 25.8%, 32.7%, and 42.7% for MPF intake, respectively. In linear regression models, every 10% increase in calories from UPFs was associated with an increase of 0.0025 points, 0.0082 points, and 0.52 kg/m^2 in SADHtR, WHtR, and BMI (all $p < 0.001$). In contrast, every 10% increase in calories from MPFs was associated with a decrease of 0.0024 points, 0.0078 points, and 0.46 kg/m^2 in SADHtR, WHtR, and BMI (all $p < 0.001$; Supporting Information Table 1). The standardized β coefficients (SE) per 10% increase of calories from UPFs were 0.0926 (0.0148), 0.0846 (0.0147), and 0.0791 (0.0150; $p > 0.26$ for pairwise differences) and were −0.0901 (0.0162), −0.0806 (0.0162), and −0.0688 (0.0163) per 10% increase of calories from MPFs, respectively, for SADHtR, WHtR, and BMI ($p > 0.18$ for pairwise differences; Supporting Information Table 1).

Figure 3 displays OR for higher tertiles of each adiposity indicator comparing quartiles 2, 3, and 4 to quartile 1 of UPF intake. For our base models (adjusted only for age, sex, and race-Hispanic origin), only for SADHtR did the third adiposity tertile demonstrate increasing OR without any overlap of CI. For our fully adjusted models, the patterns of association strength with UPF intake persisted with slight but nonsignificant differences between adiposity indicators: SADHtR > WHtR> BMI. Figure 4 shows OR for higher tertiles of each adiposity indicator comparing quartiles 2, 3, and 4 to quartile 1 of MPF intake. MPFs showed similar trends but inverse associations with all three adiposity indicators, with nonsignificant differences between adiposity: SADHtR > WHtR> BMI. The associations between percentage of kilocalories from UPFs and MPFs and three adiposity indicators were consistent across age (<50 and ≥50 years) and sex subgroups (all p values for the interaction >0.076).

The characteristics were comparable between weighted estimates of the included participants and the full adult sample (Supporting Information Table 2). The pattern of association between UPFs and MPFs and three adiposity indicators (Supporting Information Tables 3-4) remained consistent with our primary analyses.

DISCUSSION

In this nationally representative sample of US adults, participants consumed, on average, more than half of their energy from UPFs and more than one-fourth of their energy from MPFs. Higher values of percentage of kilocalories from UPFs were associated with greater values of BMI (a whole-body adiposity indicator), WHtR, and SADHtR (alternative abdominal indicators). Increased percentage of energy intake from MPFs, by contrast, showed an inverse association with all three adiposity indicators. The strengths of the relationships were comparable for our alternative adiposity indicators, demonstrating a trend toward stronger relationships for SADHtR > WHtR > BMI. Sensitivity analyses did not materially change these observed trends when participants with implausible energy intake were included.

Our cross-sectional findings related to UPF intake are consistent with previous reports from many countries that found positive associations with the BMI and WC [5-12]. By reporting data on SADHtR, we have established that adiposity associated with adult UPF intake is likely to include increases located specifically in the visceral adipose tissue depot. Although unsurprising, our novel finding is important because visceral adiposity, more specifically than whole-body adiposity, serves as a strong marker of cardiometabolic risk in adults [32, 33]. A recent study of adolescents similarly found that a higher prevalence of UPFs in their diet was more strongly associated with visceral obesity (which was estimated by SAD) than with overall obesity (as estimated by the BMI) [34]. These parallel findings among adults and adolescents suggest that the higher UPF intake among adults would likely be associated with cardiometabolic risk and mortality. Indeed, an increase in cardiovascular events and cardiovascular disease mortality among high consumers of UPFs has been recently reported from adults in the Framingham Offspring Cohort [35].

In our study, higher MPF intake was significantly associated with reduced values for SADHtR, WHtR, and BMI. A recent analysis of 1,774 French adults, to the contrary, found that MPF caloric intake was associated with increased values of BMI and a higher likelihood of categorical obesity. These results are difficult to interpret, however, because they were not adjusted for any demographic or seasonal variables [36].

A controlled, cross-over trial randomized 20 healthy adults to receive either UPF or MPF diets for 2 weeks immediately followed by the alternate diet for 2 weeks [37]. These meals were designed to be matched for presented calories, energy density, macronutrients, sugar, sodium, and fiber; participants were instructed to consume as much or as little as desired. The participants had higher energy intake, weight gain, and increased fat mass during their UPF diet, whereas they lost weight and fat mass when consuming a MPF diet. Although this small, interventional study provided no information on changes in WC or SAD, it adds plausibility that increases in caloric intake from MPFs could be associated with reduced adiposity and that increases in caloric intake from UPFs could be associated with increased adiposity.

It may be important that we did not find a significant sex difference in the associations between adult UPF or MPF intake and any of the adiposity indicators adopted for our analyses. Three previous reports [5, 12, 38] have found that UPF intake was associated with increased adiposity among women but less so among men. One of these studies was based on NHANES data from 2005 through 2014 [5]. This raises the possibility that a sex difference found earlier in the US has been attenuated during the most recent decade.

Several mechanisms have been suggested to explain the association between UPF intake and increased adiposity [39]. UPFs are typically high in energy density, added sugar, total and saturated fats, and salt and are low in dietary fiber, vitamins, and minerals [3]. The high-intensity flavoring resulting from high levels of fat, salt, sugar, and artificial flavorings makes ultraprocessed products extremely palatable, which may supersede natural satiety mechanisms [40]. Human satiety mechanisms are more sensitive to volume than caloric content, and foods with higher energy density may facilitate excessive energy intakes [41]. Therefore, people may eat more of foods with higher energy density even when they are no longer hungry. In addition, the convenience, omnipresence, affordability, large portion sizes, and persuasive marketing of UPFs may contribute to increased energy intake via poor dietary habits and overeating [42]. Furthermore, an ultraprocessed diet is associated with a greater rate of eating compared with a minimally processed diet [37], resulting in greater energy intakes in a shorter amount of time. It is also possible that artificial sweeteners present in UPFs may contribute to adiposity gains by modulating the gut microbiota [43] and stimulating basal insulin secretion [44]. Finally, as demonstrated by urinary analyses, associations have been found between UPF intake and environmental chemicals from food packaging, such as phthalates and bisphenol, which are associated with increased adiposity [45].

The mechanisms for an inverse effect of MPF intake on adiposity indicators have not been reviewed. This beneficial effect might be explained through greater satiety associated with increased intake of MPFs resulting in less total energy intake or less intake of UPFs, thereby alleviating the direct effects of UPFs on adiposity accumulation. Alternative explanations could become an interesting focus of future research.

Our study is subject to limitations. First, as our analysis was cross-sectional, causal associations between UPF or MPF intake and adiposity indicators could not be determined. Second, the NHANES dietary questionnaires did not consistently determine product brands or characteristics of food acquisition, which could lead to potential misclassification errors. Third, reverse causality could underestimate the association between UPF consumption and adiposity if participants aware of overweight or obesity change their diet and reduce UPF consumption. In addition, although our analyses adjusted for possible confounders, residual confounding cannot completely be ruled out. Finally, our dependence on self-reported dietary recall may introduce inaccurate information such as the underreport of unhealthy food or overreport healthy food intake. Our exclusion of participants with implausible energy intake from these analyses may, to some extent, mitigate biases from misreporting.

Our study adds novel insights to the growing evidence that higher consumption of UPFs is associated with the risk of increased adiposity among adults. The study is the first, to our knowledge, to report associations between adult UPF and MPF intake and abdominal adiposity indicators corrected for height. By reporting specifically on associations with

SADHtR, our study suggests that increased UPF intake may be correlated with an expanded visceral (intra-abdominal) adipose tissue depot, which, in turn, is associated with cardiometabolic risk factors. Our study also adds to the scant previous literature on how MPF intake may be related to reduced adiposity. These findings support public programs to discourage consumption of UPFs and to encourage MPF consumption.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

We would like to thank the staff and participants in NHANES 2011 through 2016 for their hard work and dedication. We also warmly thank our colleagues from Brazil for the development of Nova classification system. 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. Data described in the manuscript, code book, and analytic code will be made available pending email request to corresponding author.

REFERENCES

1. Monteiro CA, Cannon G, Moubarac JC, Levy RB, Louzada MLC, Jaime PC. The UN Decade of Nutrition, the NOVA food classification and the trouble with ultra-processing. Public Health Nutr. 2018;21: 5–17. [PubMed: 28322183]
2. Juul F, Parekh N, Martinez-Steele E, Monteiro CA, Chang VW. Ultra-processed food consumption among US adults from 2001 to 2018. Am J Clin Nutr. 2022;115:211–221. [PubMed: 34647997]
3. Monteiro CA, Cannon G, Levy RB, et al. Ultra-processed foods: what they are and how to identify them. Public Health Nutr. 2019;22:936–941. [PubMed: 30744710]
4. Costa de Miranda R, Rauber F, Levy RB. Impact of ultra-processed food consumption on metabolic health. Curr Opin Lipidol. 2021;32: 24–37. [PubMed: 33315618]
5. Juul F, Martinez-Steele E, Parekh N, Monteiro CA, Chang VW. Ultra-processed food consumption and excess weight among US adults. Br J Nutr. 2018;120:90–100. [PubMed: 29729673]
6. Machado PP, Steele EM, Levy RB, et al. Ultra-processed food consumption and obesity in the Australian adult population. Nutr Diabetes. 2020;10:39. doi:10.1038/s41387-020-00141-0 [PubMed: 33279939]
7. Rauber F, Steele EM, Louzada MLDC, Millett C, Monteiro CA, Levy RB. Ultra-processed food consumption and indicators of obesity in the United Kingdom population (2008-2016). PLoS One. 2020; 15:e0232676. doi:10.1371/journal.pone.0232676 [PubMed: 32357191]
8. 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. [PubMed: 31619309]
9. Rauber F, Chang K, Vamos EP, et al. Ultra-processed food consumption and risk of obesity: a prospective cohort study of UK Biobank. Eur J Nutr. 2021;60:2169–2180. [PubMed: 33070213]
10. Zhang Z, Jackson S, Martinez E, Gillespie C, Yang Q. 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. [PubMed: 33021623]
11. Sandoval-Insausti H, Jiménez-Onsurbe M, Donat-Vargas C, et al. Ultra-processed food consumption is associated with abdominal obesity: A prospective cohort study in older adults. Nutrients. 2020;12:2368. doi:10.3390/nu12082368 [PubMed: 32784758]
12. Sung H, Park JM, Oh SU, Ha K, Joung H. Consumption of ultra-processed foods increases the likelihood of having obesity in Korean women. Nutrients. 2021;13:698. doi:10.3390/nu13020698 [PubMed: 33671557]

13. Li C, Harris M, Tsilimingras D, Liu SZ, Sheng Y, Liu X. Sagittal abdominal diameter and its socioeconomic correlates: perspective of sex differences. BMC Public Health. 2021;21:486. doi:10.1186/s12889-020-09805-z [PubMed: 33706753]

14. Kullberg J, Von Below C, Lönn L, Lind L, Ahlström H, Johansson L. Practical approach for estimation of subcutaneous and visceral adipose tissue. Clin Physiol Funct Imaging. 2007;27:148–153. [PubMed: 17445065]

15. Yim JY, Kim D, Lim SH, et al. Sagittal abdominal diameter is a strong anthropometric measure of visceral adipose tissue in the Asian general population. Diabetes Care. 2010;33:2665–2670. [PubMed: 20843976]

16. Ohrvall M, Berglund L, Vessby B. Sagittal abdominal diameter compared with other anthropometric measurements in relation to cardiovascular risk. Int J Obes Relat Metab Disord. 2000;24:497–501. [PubMed: 10805508]

17. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Obes Rev. 2012;13:275–286. [PubMed: 22106927]

18. Zhang X, Hu EA, Wu H, Malik V, Sun Q. Associations of leg fat accumulation with adiposity-related biological factors and risk of metabolic syndrome. Obesity (Silver Spring). 2013;21:824–830. [PubMed: 23404933]

19. Lotta LA, Gulati P, Day FR, et al. Integrative genomic analysis implicates limited peripheral adipose storage capacity in the pathogenesis of human insulin resistance. Nat Genet. 2017;49:17–26. [PubMed: 27841877]

20. Kahn HS, Cheng YL. Comparison of adiposity indicators associated with fasting-state insulinemia, triglyceridemia, and related risk biomarkers in a nationally representative, adult population. Diabetes Res Clin Pract. 2018;136:7–15. [PubMed: 29183845]

21. Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey. Accessed March 22, 2022. http://www.cdc.gov/nchs/nhanes/index.htm

22. Banna JC, McCroy MA, Fialkowski MK, Boushey C. 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]

23. National Center for Health Statistics. NHANES 2013–2014 data documentation, codebook, and frequencies – body measures (BMX_H). Published October 2015. Accessed March 22, 2022. http://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/BMX_H.htm

24. National Center for Health Statistics. Anthropometry Procedures Manual - National Health and Nutrition Examination Survey (NHANES). Published January 2013. Accessed March 22, 2022. http://www.cdc.gov/nchs/data/nhanes/nhanes_13_14/2013_Anthropometry.pdf

25. Martinez Steele E, Baraldi LG, Louzada ML, Moubarac JC, Mozaffarian D, Monteiro CA. Ultra-processed foods and added sugars in the US diet: evidence from a nationally representative cross-sectional study. BMJ Open. 2016;6:e009892. doi:10.1136/bmjopen-2015-009892

26. US Department of Agriculture, Agricultural Research Service. Food and Nutrient Database for Dietary Studies. Accessed March 22, 2022. https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/food-surveys-research-group/docs/fndds

27. US Department of Agriculture. USDA National Nutrient Database for Standard Reference. https://data.nal.usda.gov/dataset/usda-national-nutrient-database-standard-reference-legacy-release

28. 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. [PubMed: 9187624]

29. 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. [PubMed: 19302405]

30. Lloyd-Jones DM, Hong Y, Labarthe D, et al. American Heart Association Strategic Planning Task Force and Statistics Committee. 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. [PubMed: 20089546]

31. Desquilbet L, Mariotti F. Dose-response analyses using restricted cubic spline functions in public health research. Stat Med. 2010;29:1037–1057. [PubMed: 20087875]

32. Neeland IJ, Ross R, Després JP, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. Lancet Diabetes Endocrinol. 2019;7:715–725. [PubMed: 31301983]
33. Stefan N. Causes, consequences, and treatment of metabolically unhealthy fat distribution. Lancet Diabetes Endocrinol. 2020;8:616–627. [PubMed: 32559477]
34. Neri D, Steele EM, Khandpur N, Levy R. Associations between ultra-processed foods consumption and indicators of adiposity in US adolescents: cross-sectional analysis of the 2011-2016 National Health and Nutrition Examination Survey. J Acad Nutri Diet. 2022;122:1474–1487.e2.doi:10.1016/j.jand.2022.01.005
35. Juul F, Vaidean G, Lin Y, Deierlein AL, Parekh N. Ultra-processed foods and incident cardiovascular disease in the Framingham Offspring Study. J Am Coll Cardiol. 2021;77:1520–1531. [PubMed: 33766258]
36. Salomé M, Arrazat L, Wang J, et al. Contrary to ultra-processed foods, the consumption of unprocessed or minimally processed foods is associated with favorable patterns of protein intake, diet quality and lower cardiometabolic risk in French adults (INCA3). Eur J Nutr. 2021;60:4055–4067. [PubMed: 33966096]
37. 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.
38. da Costa Louzada ML, Baraldi LG, Steele EM, et al. Consumption of ultra-processed foods and obesity in Brazilian adolescents and adults. Prev Med. 2015;81:9–15. [PubMed: 26231112]
39. Juul F, Vaidean G, Parekh N. Ultra-processed foods and cardiovascular diseases: Potential mechanisms of action. Adv Nutr. 2021;12:1673–1680. [PubMed: 33942057]
40. Ludwig DS. Technology, diet, and the burden of chronic disease. JAMA. 2011;305:1352–1353. [PubMed: 21467290]
41. Rolls BJ. Dietary energy density: applying behavioural science to weight management. Nutr Bull. 2017;42:246–253. [PubMed: 29151813]
42. Moubarac JC, Martins AP, Claro RM, Levy RB, Cannon G, Monteiro CA. Consumption of ultra-processed foods and likely impact on human health: evidence from Canada. Public Health Nutr. 2013;16:2240–2248. [PubMed: 23171687]
43. Suez J, Korem T, Zeevi D, Zilberman-Schapira G, Thaiss CA, Maza O, et al. Artificial sweeteners induce glucose intolerance by altering the gut microbiota. Nature. 2014;514:181–186. [PubMed: 25231862]
44. Corkey B Diabetes: Have we got it all wrong? Insulin hypersecretion and food additives: cause of obesity and diabetes? Diabetes Care. 2012;35:2432–2437. [PubMed: 23173132]
45. Martínez Steele E, Khandpur N, da Costa Louzada ML, Monteiro CA. Association between dietary contribution of ultra-processed foods and urinary concentrations of phthalates and bisphenol in a nationally representative sample of the US population aged 6 years and older. PLoS One. 2020;15:e0236738. doi:10.1371/journal.pone.0236738 [PubMed: 32735599]

Author Manuscript

Author Manuscript

Author Manuscript

Study Importance

What is already known?

- US adults consumed on average more than half of their energy from ultraprocessed foods (UPFs) and about 30% from unprocessed or minimally processed foods (MPFs).
- High UPF intake among adults is associated with increased BMI, obesity, and waist circumference.

What does this study add?

- Higher intake of UPFs is associated with greater BMI, waist circumference/height ratio (WHtR) and supine sagittal abdominal diameter/height ratio (SADHtR).
- Increased energy intake from MPF is inversely associated with BMI, WHtR, and SADHtR.
- Increased UPF intake is correlated with an expanded visceral adipose tissue depot, which in turn is associated with cardiometabolic risk factors.
- Our study also adds to the scant literature regarding MPF intake association with reduced adiposity.

How might these results change the direction of research or the focus of clinical practice?

- Our findings support public programs and clinical practice to discourage consumption of UPFs and to encourage MPF consumption.

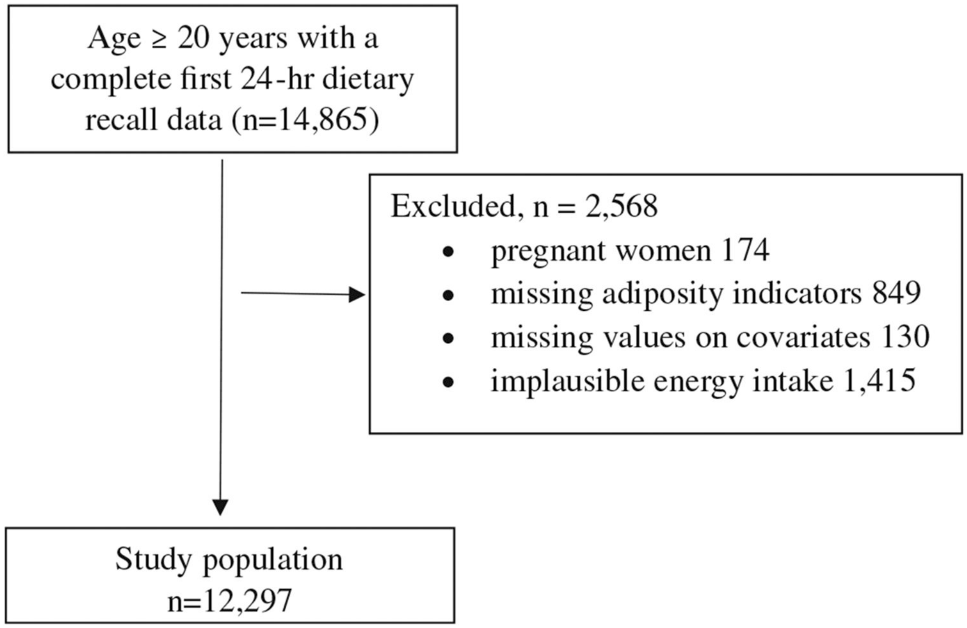

FIGURE 1.

Selection of study population for NHANES 2011 to 2016. NHANES, National Health and Nutrition Examination Survey

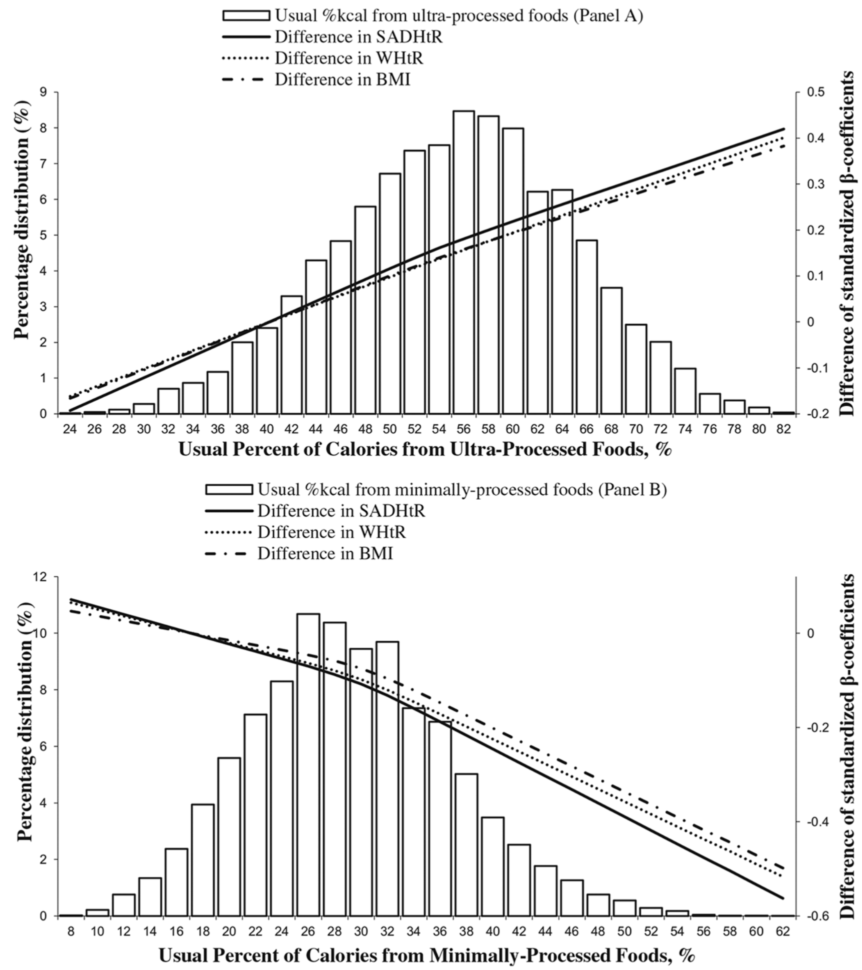

FIGURE 2.

Distributions of usual percentage of kilocalories from (A) UPFs and (B) MPFs and adjusted differences in three adiposity indicators (40.2% for UPFs and 18.1% for MPFs as reference values), US adults (n = 12,297), NHANES 2011 to 2016. Multivariable linear regression models were used to estimate the adjusted differences in adiposity indicators and corresponding 95% CI and were adjusted by age, sex, race-Hispanic origin, education, smoking status, physical activity, and awareness of diabetes. For each percentage of intake category in the histogram, we provide the adjusted difference in each of the adiposity indicators between that percentage of intake and a reference value (the midpoint of the lowest quartile: 40.2% for UPFs and 18.1% for MPFs). MPF, minimally processed food; NHANES, National Health and Nutrition Examination Survey; SADHtR, supine sagittal abdominal diameter to height ratio; UPF, ultraprocessed food; WC, waist circumference; WHtR, WC to height ratio

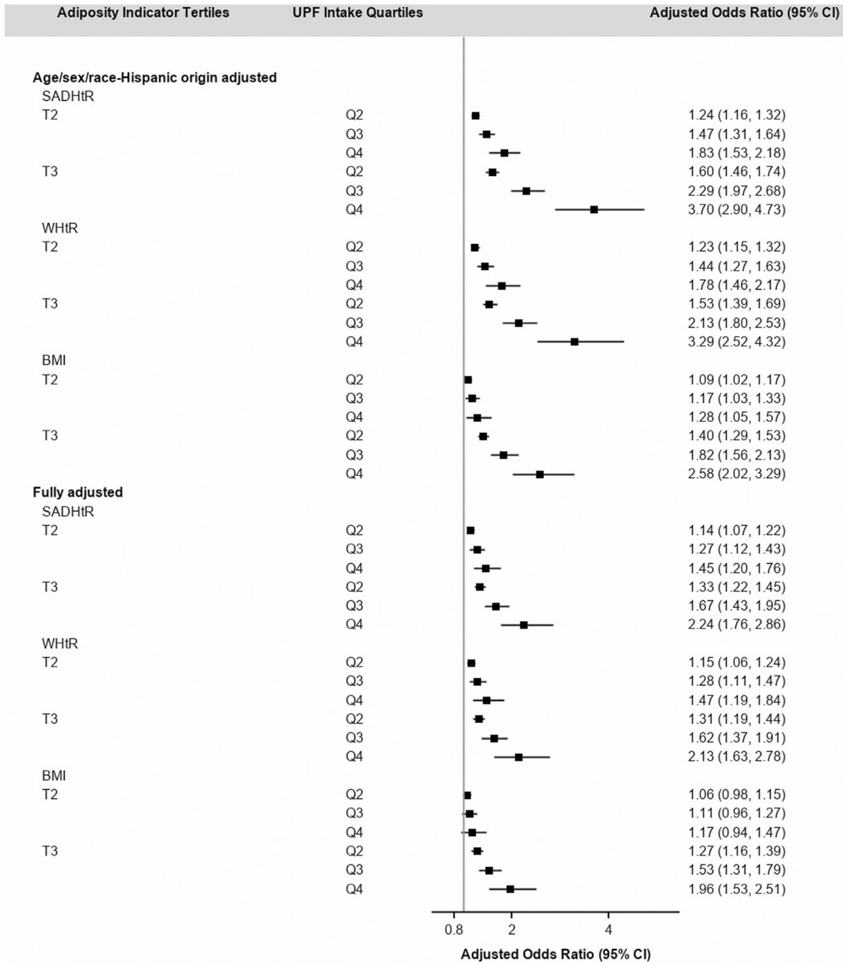

FIGURE 3.

Associations of UPF intake quartiles 2, 3, and 4 with the adjusted OR (95% CI) for the second and third tertiles of adiposity indicators SADHtR, WHtR, and BMI. Base and fully adjusted models are shown. Adjusted for age as categorical variable, sex, race-Hispanic origin, education, smoking status, physical activity, and awareness of diabetes. NHANES, National Health and Nutrition Examination Survey; OR, odds ratio; SADHtR, supine sagittal abdominal diameter to height ratio; T2, second tertile; T3, third tertile; UPF, ultraprocessed food; WC, waist circumference; WHtR, WC to height ratio

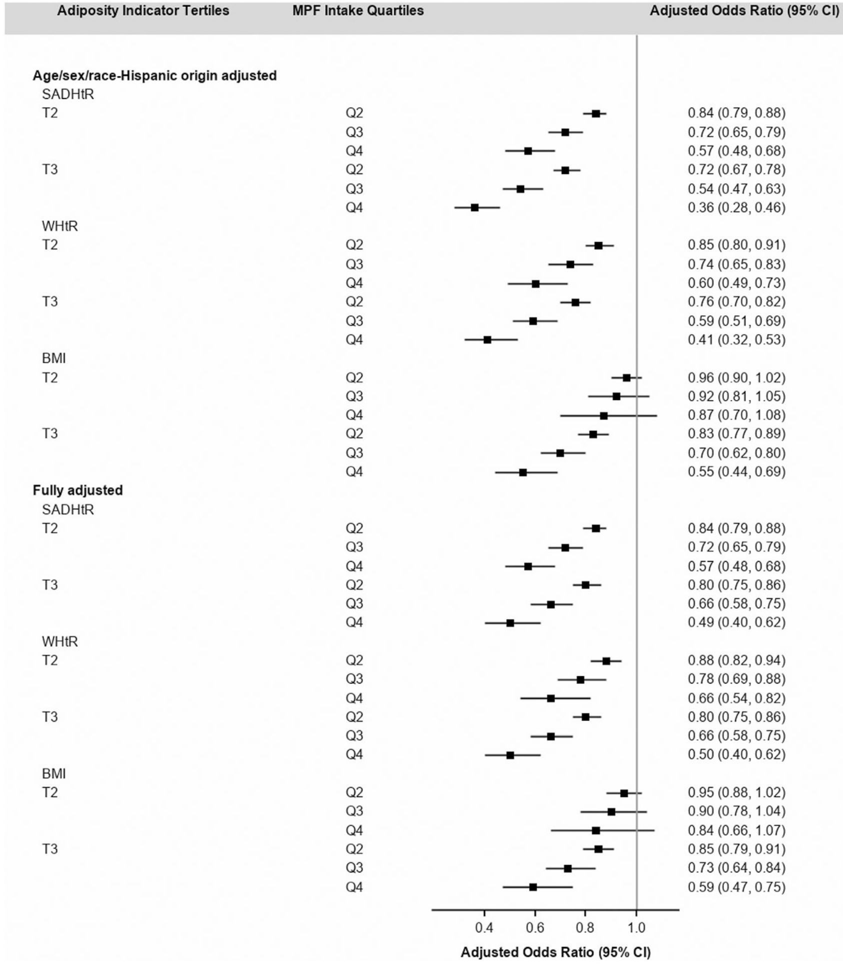

FIGURE 4.

Associations of MPF intake quartiles 2, 3, and 4 with the adjusted OR (95% CI) for the second and third tertiles of adiposity indicators SADHtR, WHtR, and BMI. Base and fully adjusted models are shown. Adjusted for age as categorical variable, sex, race-Hispanic origin, education, smoking status, physical activity, and awareness of diabetes. MPF, minimally processed food; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio; SADHtR, supine sagittal abdominal diameter to height ratio; T2, second tertile; T3, third tertile; WC, waist circumference; WHtR, WC to height ratio

TABLE 1

Weighted means (SE) for anthropometry and age and percentage distributions (SE) for demographic and clinical characteristics by sex (NHANES 2011 through 2016)

	Overall (n = 12,297)	**Male (n = 5,923)**	**Female (n = 6,374)**	***p* value**
Anthropometry				
SAD, cm	22.6 (0.10)	23.3 (0.11)	22.0 (0.10)	<0.001
WC, cm	99.2 (0.33)	101.7 (0.39)	97.0 (0.37)	<0.001
Height, cm	168.4 (0.17)	175.7 (0.18)	161.8 (0.22)	<0.001
Weight, kg	82.3 (0.38)	88.8 (0.47)	76.2 (0.42)	<0.001
SADHtR	0.1346 (0.0006)	0.1330 (0.0006)	0.1361 (0.0007)	<0.001
WHtR	0.5903 (0.0021)	0.5794 (0.0022)	0.6004 (0.0025)	<0.001
BMI, kg/m^2	28.9 (0.14)	28.7 (0.14)	29.1 (0.16)	0.023
Age, y	47.8 (0.40)	46.9 (0.43)	48.6 (0.40)	<0.001
Race-Hispanic origin, %				
Non-Hispanic White	66.2 (2.04)	66.5 (2.07)	66.0 (2.11)	0.605
Non-Hispanic Black	10.6 (1.08)	10.0 (1.00)	11.2 (1.19)	0.013
Non-Hispanic Asian	5.7 (0.62)	5.6 (0.64)	5.8 (0.63)	0.592
Hispanic	14.5 (1.40)	14.8 (1.42)	14.3 (1.45)	0.391
Other	3.0 (0.29)	3.1 (0.38)	2.8 (0.35)	0.521
Education, %				
Below high school completion	14.0 (0.98)	14.7 (1.11)	13.4 (0.98)	0.062
High school graduate	20.3 (0.75)	21.4 (0.96)	19.3 (0.88)	0.059
College or above	65.7 (1.44)	63.9 (1.65)	67.3 (1.49)	0.009
Smoking status, %				
Current	18.7 (0.68)	20.5 (0.83)	17.0 (0.90)	0.002
Former	2.1 (0.23)	2.9 (0.33)	1.4 (0.23)	<0.001
Never	79.2 (0.70)	76.6 (0.84)	81.6 (0.92)	<0.001
Physical activity, %				
Poor	43.3 (1.13)	41.4 (1.35)	45.0 (1.30)	0.015
Intermediate	16.3 (0.50)	14.8 (0.67)	17.6 (0.68)	0.004
Ideal	40.5 (1.00)	43.7 (1.07)	37.4 (1.33)	<0.001

	Overall (n = 12,297)	**Male (n = 5,923)**	**Female (n = 6,374)**	***p* value**
Awareness of diagnosed diabetes, %				
Yes	10.0 (0.40)	10.9 (0.49)	9.2 (0.48)	0.019
No	90.0 (0.40)	90.1 (0.49)	90.8 (0.48)	

Abbreviations: NHANES, National Health and Nutrition Examination Survey; SAD, supine sagittal abdominal diameter; SADHtR, supine sagittal abdominal diameter to height ratio; WC, waist circumference; WHtR, WC to height ratio.

TABLE 2

Weighted distributions, by sex, of anthropometric dimensions and adiposity indicators among US adults, NHANES 2011 through 2016 (n = 12,297)

			Tertile cut points	
	Mean	**Median**	**P33.3**	**P66.7**
Male (n = 5,923)				
SAD, cm	23.3	22.8	21.1	24.7
WC, cm	101.7	100.5	94.5	106.9
Height, cm	175.7	175.5	172.4	178.8
Weight, kg	88.8	85.9	78.7	94.0
SADHtR	0.1330	0.1303	0.1204	0.1414
WHtR	0.5794	0.5720	0.5372	0.6103
BMI, kg/m^2	28.7	27.7	25.8	30.2
Female (n = 6,374)				
SAD, cm	22.0	21.3	19.4	23.6
WC, cm	97.0	95.1	88.4	102.4
Height, cm	161.8	161.9	158.9	164.9
Weight, kg	76.2	72.6	65.3	80.9
SADHtR	0.1361	0.1326	0.1203	0.1467
WHtR	0.6004	0.5885	0.5464	0.6368
BMI, kg/m^2	29.1	27.7	25.0	31.0

Abbreviations: NHANES, National Health and Nutrition Examination Survey; SAD, supine sagittal abdominal diameter; SADHtR, supine sagittal abdominal diameter to height ratio; WC, waist circumference; WHtR, WC to height ratio.