



Secular trends of population-attributable fractions of obesity for hypertension among US population by sex and race/ethnicity: Analysis from NHANES 1999–2018

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ABSTRACT

Obesity is a major risk factor of hypertension, therefore quantifying the contribution of obesity to hypertension is necessary. The current study aimed to investigate the changes in population-attributable fractions (PAFs) of hypertension associated with general obesity and abdominal obesity over the recent 2 decades among the US population, as well as important sub-populations. This report was performed based on national-level cross-sectional data for 46,535 adults aged 18 years and older and 20,745 children aged 8–17 from the US National Health and Nutrition Examination Survey 1999–2018. The PAFs of hypertension due to general obesity and abdominal obesity were calculated by sex, race/ethnicity, and survey year. The linear regression analysis was used to evaluate the secular trends of PAFs over the years. The prevalence of general obesity and abdominal obesity presented significantly increasing trends during the past 2 decades in the US. The PAFs of hypertension due to general obesity increased steadily from 11.9 % to 15.1 % in women with a slope of 0.38 % (95 % CI: 0.31 – 0.45 %) and from 8.4 % to 13.4 % in men with a slope of 0.46 % (95 % CI: 0.36 – 0.56 %). Similar increasing trends were also observed for the PAFs due to abdominal obesity in both women and men. Additionally, there were significantly different trends of PAFs in various races/ethnicities. Over the past 2 decades, the contributions of obesity to hypertension were gradually rising among US population, which emphasizes the importance of controlling weight to further reduce the burden of hypertension.

1. Introduction

Hypertension affects millions of people worldwide in terms of the risk of cardiovascular disease (CVD), and the situation is getting worse. In 2015, it was the number one reason for disability-adjusted life years globally (Faulkner and Belin de Chantemele, 2018). Blood pressure is affected by genetics, metabolic, nutritional, environmental, and behavioral factors throughout the life course (Do et al., 2015). Among these factors, obesity was a relatively important one (Landsberg et al., 2013). Previous studies found a strong association between body weight and hypertension (Litwin and Kulaga, 2021), the magnitude of this association increased over time (Ryu et al., 2019). Obesity is an “epidemic” that has plagued many parts of the industrialized world for a long time (Flegal et al., 2012). A comprehensive understanding of

secular trends in the contribution of obesity to hypertension is useful for perspectives of health policy, resource allocation, and disease prevention. Several studies have examined the prevalence and trends of obesity and hypertension (Flegal et al., 2010; Guo et al., 2012), but none have examined the burden of hypertension due to obesity using nationally representative data across time series.

Population-attributable fractions (PAFs) reflect the reduction of the disease when a specific risk factor is eliminated (Rockhill et al., 1998). In terms of a public health perspective, the PAF aids in prioritizing health budgets and the distribution of resources depending on the proportion of outcomes attributed to a particular exposure. The purpose of the current analysis was to determine the PAFs of hypertension due to general obesity and abdominal obesity among the US population from 1999 to 2000 through 2017–2018. Changes and differences in secular trends of

Abbreviations: BMI, Body mass index; CVD, cardiovascular disease; DBP, diastolic blood pressure; NHANES, National Health and Nutrition Examination Survey; PAF, population-attributable fraction; SBP, systolic blood pressure; WC, waist circumference.

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the PAFs among important subgroups from 1999 to 2000 through 2017–2018 were examined as secondary objectives. To accomplish these goals, data from 10 cycles of the US National Health and Nutrition Examination Survey (NHANES) were analyzed.

2. Material and methods

2.1. Study population

The NHANES is an ongoing cross-sectional, nationally representative survey of the US civilian population conducted by the National Center for Health Statistics of the Centers for Disease Control. It employs a complex, multistage sampling design to select a nationally representative sample of about 10,000 individuals in each survey cycle. A detailed description of the NHANES database is publicly available (<https://www.cdc.gov/nchs/nhanes.htm>). The NHANES protocol was approved by the National Center for Health Statistics Institutional Review Board and written informed consent was obtained (Zipf et al., 2013). Our study followed the Strengthening the Reporting of Observational Studies in Epidemiology reporting guideline (Vandenbroucke et al., 2007). The Institutional Review Board was not required as the data for this study were secondary, as such no human subjects were involved.

The present study included 101,316 participants from ten consecutive survey cycles (NHANES 1999–2000 to 2017–2018). Participants were excluded for being pregnant at examination or uncertain of the pregnancy status ($n = 3376$), aged younger than 8 for lacking data on blood pressure measurement ($n = 20,842$). For adults, due to the small sample size of other races/ethnicities, we only focused on the participants of Mexican American, non-Hispanic white, and non-Hispanic black in the present study. The participants of other races/ethnicities were excluded ($n = 9818$). Finally, a total of 46,535 eligible adults and 20,745 children were included in the analysis.

2.2. Data collection

Demographic characteristics, lifestyle factors, and current taking medicine were administered in the survey by trained interviewers using questionnaires. The demographic characteristics included sex (men, women), age, ethnicity (Mexican American, non-Hispanic white, and non-Hispanic black), education (lower than high school, high school or above), and annual household income (low income $< \$45,000$, high income $\geq \$45,000$). The number of hours of sleep duration was collected by using a questionnaire. Physical activity was estimated using the form of the Global Physical Activity Questionnaire by asking questions about the intensity, duration, and frequency of physical activity. There were different types of physical activity assessment tools used in NHANES 1999–2000 to 2005–2006 and NHANES 2007–2008 to 2017–2018. In NHANES 1999–2000 to 2005–2006, the duration of the physical activity was not ascertained, each kind of physical activity was assigned an intensity value (metabolic equivalent tasks) that represents the ratio of the energy expenditure of the activity to the basal metabolic rate. In NHANES 2007–2008 to 2017–2018, total metabolic equivalent minutes per week were calculated as the measurement of physical activity level for the participants. A higher level of physical activity was defined as having a higher metabolic equivalent per week than the median levels of the metabolic equivalent per week by investigation cycles. All NHANES examinees were eligible for two 24-hour dietary recall interviews. The first dietary recall interview was collected in-person in the Mobile Examination Center, and the second interview was collected by telephone 3–10 days later. The intakes of sodium, energy, and sugar were calculated based on answers provided by respondents (<https://www.ars.usda.gov/ba/bhnrc/fsrg>). The information on currently taking prescribed medicine for treating hypertension was investigated in the survey. The diagnosis history of cardiovascular diseases (congestive heart failure, coronary heart disease, heart attack, and stroke) and hypertension were also collected from respondents.

The physical examinations and laboratory tests in NHANES took place in a mobile examination centre using standardized protocols and calibrated equipment, and details on the data collection are described on the website (<https://www.cdc.gov/nchs/nhanes/analyticguidelines.aspx>). The details of the physical examinations of obesity and blood pressures were described in the [Supplementary file](#). Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. General obesity was defined as a BMI $\geq 30 \text{ kg/m}^2$ for adults and sex- and age-specific BMI ≥ 95 th percentile for children aged 8–17. Abdominal obesity was defined as a waist circumference (WC) $\geq 102 \text{ cm}$ for men and $\geq 88 \text{ cm}$ for women for adults and sex- and age-specific WC ≥ 90 th percentile for children aged 8–17. Blood pressures were measured by a mercury sphygmomanometer using a standardized protocol. Mean systolic blood pressure (SBP) and mean diastolic blood pressure (DBP) were calculated by averaging 3 to 4 measurements. For adults and teenagers aged 13–17 years, hypertension was defined as a mean SBP and/or DBP $\geq 130/80 \text{ mmHg}$ (Whelton et al., 2018). For children aged 8–12 years, hypertension was defined as a mean SBP and/or DBP ≥ 95 th percentile, or SBP and/or DBP $\geq 130/80 \text{ mmHg}$. Participants who were currently taking antihypertensive medications or had been diagnosed with hypertension by a physician were also regarded as hypertensive (Whelton et al., 2018).

2.3. Statistical analysis

All analyses took into account differential probabilities of selection and the complex sample design, and nonresponse and noncoverage by using sample weights. We estimated the weighted means (95 % CIs) for continuous variables and percentages (95 % CIs) for categorical variables of the selected covariates. Standard errors were calculated using Taylor series linearization. A weighted multivariable logistic regression model was performed to evaluate the associations of general obesity and abdominal obesity with hypertension with adjustment of age, educational attainment, current marriage status, household income, smoking behavior, drinking behavior, sleep duration, physical activity level, estimated intakes of sodium, energy, and sugar, HbA1c, low-density lipoprotein cholesterol, triglyceride, and history of cardiovascular diseases (congestive heart failure, coronary heart disease, heart attack, and stroke). The odds ratios (ORs) and 95 % confidence intervals (95 % CIs) were obtained with non-obesity participants as the references. To test the modified effects of sex and race/ethnicity on the associations between obesity and hypertension, interaction tests for those two variables were performed by including the product term (e.g. sex \times general obesity or race/ethnicity \times general obesity) in the models to assess whether the associations of general obesity and abdominal obesity with hypertension vary across different groups. The significance of interactive terms was assessed by the Wald test. The prevalence of hypertension is common in the study population ($>10 \%$), the ORs can no longer approximate the RRs. The more frequent the outcome, the more the ORs overestimate the RRs. Therefore, we used a method to correct the ORs. As suggested by Zhang et al., we used the following formula to obtain corrected RRs: $RR = OR / [(1 - P_0) + (P_0 \times OR)]$. P_0 indicates the prevalence of the outcome of interest in the nonexposed group, that is hypertension prevalence in the non-obesity population in our study. For each wave of a 2-year survey in NHANES from 1999 to 2018, we calculated the weighted prevalence of general obesity and abdominal obesity for different genders, races/ethnicities and survey waves depending on the aims of analyses (Zhang and Yu, 1998). PAF was calculated as follows: $PAF = [\text{prevalence} \times (RR - 1)] / [\text{prevalence} \times (RR - 1) + 1]$ (Ruckinger et al., 2009). Considerable PAF values implied a theoretical causal relationship between general obesity and abdominal obesity with hypertension. The linear regression was used to evaluate the secular trends of PAF over the years with a 2-year survey cycle treated as a continuous variable. The slope across survey years was used as a surrogate indicator of the average increased velocity. The differences in slopes between men and women, as well as different races/

ethnicities, were examined by including the interaction terms (survey year \times sex or survey year \times race/ethnicity) in the regression model. The significance of interactive terms is assessed by the Wald test, and Bonferroni's method is used to adjust the Type-1 error. There are six times comparisons among different ethnic/racial populations, the adjusted *P* value required for significance is $0.05/3 = 0.017$. All the statistical analysis was conducted using the *survey* package in R version 4.0.3 (<https://www.r-project.org>).

3. Results

The characteristics of the adult participants by sex and race/ethnicity are shown in Table 1. Among the 46,535 participants, 49.0 % were women; 10,126 (21.6 %) participants were Mexican Americans, 24,304 (52.2 %) were non-Hispanic whites, and 12,105 (26.0 %) were non-Hispanic blacks. The mean age of the participants was 45.6 (95 % CI: 45.2 – 46.0) years old for men and 48.3 (95 % CI: 47.8 – 48.7) for women. All the characteristics, except for BMI and WC in men, showed significant differences across racial/ethnic adults (all *P* < 0.05). The non-Hispanic black women had the highest prevalence of general obesity (53.3 %) and abdominal obesity (73.3 %), and the highest prevalence of hypertension (55.9 %) than those of Mexican American

and non-Hispanic white women. In men, the highest prevalence of general obesity was observed in Mexican Americans (36.7 %), while the highest prevalence of abdominal obesity was in non-Hispanic whites (46.5 %), and the highest prevalence of hypertension (57.3 %) was in non-Hispanic blacks.

Fig. 1 shows the secular trends of general obesity, abdominal obesity, and hypertension in women and men. For both women and men, the prevalence of general obesity (women: $\beta = 1.2$ %, 95 %CI = 1.0–1.5 %, *P* < 0.001; men: $\beta = 1.6$ %, 95 %CI = 1.3–2.0 %, *P* < 0.001) and abdominal obesity (women: $\beta = 1.7$ %, 95 %CI = 1.4–2.1 %, *P* < 0.001; men: $\beta = 1.4$ %, 95 %CI = 1.1–1.8 %, *P* < 0.001) were significantly increased during the past two decades. The prevalence of hypertension presented a significantly increased trend during the past two decades in women ($\beta = 0.4$ %, 95 %CI = 0.2–0.6 %, *P* = 0.002), but not in men ($\beta = 0.3$ %, 95 %CI = -0.2–0.8 %, *P* = 0.248). The trends of prevalence for general obesity, abdominal obesity, and hypertension among the three races/ethnicities are presented in Supplementary Fig. 1.

The unadjusted, multivariable-adjusted ORs and transformed RR (95 % CIs) for hypertension in generally and abdominally obese participants compared with non-obese counterparts are shown in Table 2. All the ORs and RRs were significantly associated with hypertension in both women and men by race/ethnicity. There is no interaction effect of sex or race/

Table 1
Baseline characteristics of the adult participants by sex and race/ethnicity, NHANES 1999–2018.

	Total	Mexican American	Non-Hispanic White	Non-Hispanic Black	<i>P</i> value
Women					
Number of participants	22,783	4901	11,852	6030	
Age (years)	48.3 (47.8 – 48.7)	40.6 (39.8–41.3)	49.7 (49.2–50.2)	45.0 (44.4–45.6)	< 0.001
Higher level of education, %	84.0 (83.0 – 85.0)	53.4 (51.2–55.6)	88.4 (87.2–89.5)	77.2 (75.7–78.8)	< 0.001
Former or current smoking, %	41.0 (39.9 – 42.1)	23.7 (21.8–25.7)	44.2 (42.9–45.5)	33.5 (31.7–35.3)	< 0.001
Former or current drinking, %	62.3 (61.7 – 63.0)	48.9 (43.1–54.7)	66.1 (64.6–67.6)	49.1 (48.6–49.7)	< 0.001
Higher level of physical activity, %	38.3 (37.5–39.1)	37.5 (34.7–40.3)	38.8 (37.6–40.1)	35.5 (32.3–38.7)	< 0.001
Sleep duration \geq 8 h, %	30.8 (29.7 – 32.0)	35.1 (33.6–36.6)	31.2 (30.1–32.4)	25.8 (22.5–29.1)	< 0.001
Low annual family income, %	55.9 (54.3 – 57.5)	40.0 (37.0 – 43.1)	60.3 (58.4 – 62.2)	40.1 (37.6 – 42.6)	< 0.001
BMI (kg/m ²)	28.9 (28.8 – 29.2)	30.0 (29.7–30.4)	28.4 (28.2–28.6)	31.8 (31.5 – 32.0)	< 0.001
Waist circumference (cm)	95.8 (95.4 – 96.3)	96.9 (96.1–97.7)	95.0 (94.5–95.5)	100.1 (99.5–100.7)	< 0.001
SBP (mmHg)	121.5 (121.1 – 121.9)	117.7 (117.1–118.4)	121.2 (120.8–121.7)	125.2 (124.5–125.8)	< 0.001
DBP (mmHg)	69.5 (69.2 – 69.9)	68.3 (67.9–68.8)	69.4 (69.0–69.8)	71.1 (70.6–71.7)	< 0.001
HbA1c (%)	5.8 (5.8–5.8)	5.7 (5.6–5.7)	5.5 (5.4–5.5)	5.8 (5.8–5.8)	< 0.001
LDL-cholesterol (mmol/L)	3.0 (2.9 – 3.0)	2.9 (2.8–2.9)	3.0 (2.9 – 3.0)	2.9 (2.8–2.9)	< 0.001
Triglyceride (mmol/L)	1.4 (1.3–1.4)	1.5 (1.4–1.5)	1.4 (1.4–1.5)	1.1 (1.0–1.1)	< 0.001
Mean sodium intake (mg)	2961.1 (2938.5–2983.7)	2997.5 (2985–3009.9)	2960.2 (2927.3–2993.2)	2943.5 (2909.1–2977.9)	< 0.001
Mean energy intake (kcal)	1834.6 (1808.0–1861.2)	1868.7 (1855.4–1882)	1826.7 (1787.6–1865.7)	1860.0 (1833.5–1886.6)	< 0.001
Mean sugar intake (gm)	102.9 (102.0–103.8)	103.7 (100.7–106.7)	101.2 (99.5 – 103.0)	111.7 (110.6–112.8)	< 0.001
Taking antihypertensive medication, %	27.9 (27.0 – 28.7)	17.0 (15.4–18.6)	27.4 (26.4–28.4)	37.1 (35.6–38.6)	< 0.001
Prevalence of general obesity, %	37.6 (36.6 – 38.7)	44.2 (42.0–46.4)	34.1 (32.9–35.4)	53.3 (51.8–54.7)	< 0.001
Prevalence of abdominal obesity, %	63.9 (62.8 – 65.1)	70.4 (68.1–72.6)	61.6 (60.2–63.1)	73.3 (71.9–74.7)	< 0.001
Prevalence of hypertension, %	46.2 (45.2 – 47.2)	32.2 (29.0–35.4)	46.0 (44.6–47.4)	55.9 (55.2–56.6)	< 0.001
Men					
Number of participants	23,752	5225	12,452	6075	
Age (years)	45.6 (45.2 – 46.0)	38.2 (37.6–38.8)	47.0 (46.6–47.5)	42.3 (42.0–43.1)	< 0.001
Higher level of education, %	82.7 (81.7 – 83.7)	49.4 (47.5–51.3)	88.3 (87.1–89.4)	74.5 (72.6–76.4)	< 0.001
Former or current smoking, %	54.8 (52.7 – 55.0)	48.3 (46.6 – 50.0)	55.4 (54.1–56.8)	48.4 (46.7–50.2)	< 0.001
Former or current drinking, %	75.7 (74.6 – 76.8)	75.2 (73.6–76.8)	77.2 (75.6–78.7)	66.7 (66.2–67.1)	< 0.001
Higher level of physical activity, %	45.6 (45.0–46.2)	48.8 (47.4–50.1)	45.4 (44.2–46.6)	44.5 (39.8–49.2)	< 0.001
Sleep duration \geq 8 h, %	25.5 (25.3–25.7)	29.5 (26.6–32.3)	25.4 (25.1–25.7)	22.7 (20.7–24.7)	< 0.001
Low annual family income, %	62.0 (60.4 – 63.6)	41.1 (38.4 – 43.7)	66.9 (65.1 – 68.9)	46.8 (44.3 – 49.2)	< 0.001
BMI (kg/m ²)	28.6 (28.5 – 28.7)	29.0 (28.8–29.3)	28.6 (28.4–28.7)	28.6 (28.4–28.8)	0.184
Waist circumference (cm)	101.1 (100.7 – 101.5)	99.6 (98.9–100.3)	101.9 (101.5–102.3)	97.3 (96.7–97.8)	0.099
SBP (mmHg)	123.9 (123.5 – 124.3)	122.0 (121.4–122.7)	123.6 (123.2 – 124.0)	127.2 (126.6–127.8)	< 0.001
DBP (mmHg)	72.2 (71.8 – 72.5)	70.9 (70.4–71.5)	72.2 (71.8–72.6)	73.3 (72.7–73.9)	< 0.001
HbA1c (%)	5.8 (5.7–5.8)	5.7 (5.6–5.7)	5.5 (5.5–5.5)	5.8 (5.7–5.8)	< 0.001
LDL-cholesterol (mmol/L)	3.0 (2.9 – 3.0)	3.0 (3.0–3.1)	3.0 (2.9 – 3.0)	2.9 (2.8–2.9)	< 0.001
Triglyceride (mmol/L)	1.7 (1.6–1.7)	1.9 (1.9 – 2.0)	1.7 (1.7–1.8)	1.3 (1.3–1.3)	< 0.001
Mean sodium intake (mg)	4164.9 (4155.2–4174.7)	4075.2 (3975.7–4174.7)	4219.9 (4187.6–4252.2)	3877.1 (3816.1–3938.0)	< 0.001
Mean energy intake (kcal)	2606.3 (2587.3–2625.4)	2620.9 (2601.9–2639.9)	2621 (2601.2–2640.9)	2495.8 (2461.7–2529.8)	< 0.001
Mean sugar intake (gm)	136.1 (134.3–137.9)	133.7 (131.3 – 136.0)	136.7 (134.5–138.8)	134.4 (130.3–138.6)	< 0.001
Taking antihypertensive medication, %	23.7 (22.8 – 24.5)	12.6 (11.2 – 14.0)	24.5 (23.4–25.6)	27.6 (26.3–28.8)	< 0.001
Prevalence of general obesity, %	34.1 (33.0 – 35.1)	36.7 (34.8–38.6)	33.6 (32.3–34.9)	34.9 (33.4–36.4)	0.036
Prevalence of abdominal obesity, %	44.5 (43.4 – 45.7)	39.3 (37.3–41.4)	46.5 (45.2–47.9)	36.1 (34.6–37.5)	< 0.001
Prevalence of hypertension, %	51.2 (50.1 – 52.3)	39.2 (36.9–41.4)	51.8 (51.5–52.2)	57.3 (55.9 – 58.7)	< 0.001

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL-cholesterol, low-density lipoprotein-cholesterol.

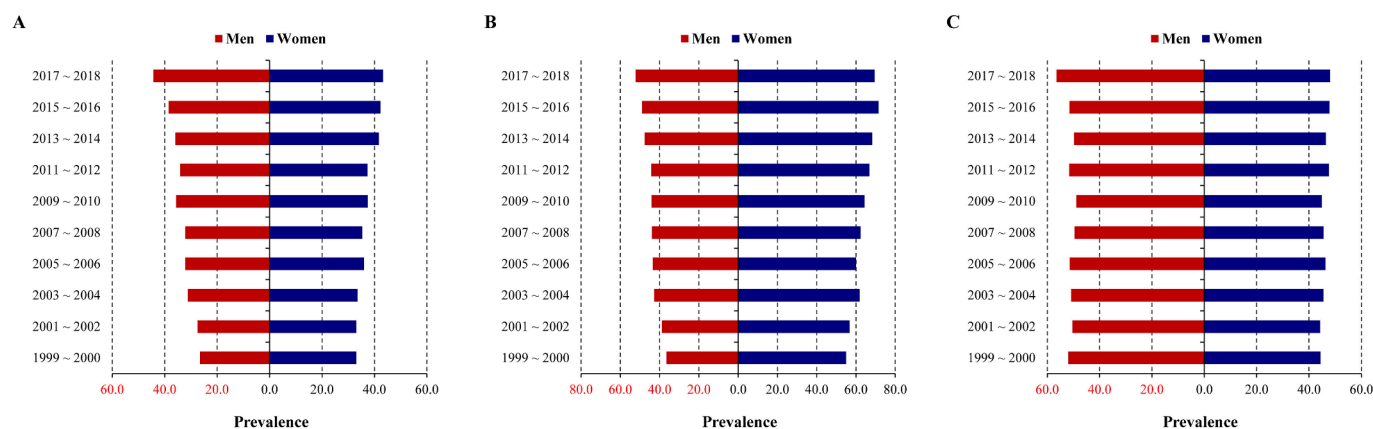


Fig. 1. The secular prevalence of (A) general obesity, (B) abdominal obesity and (C) hypertension in women and men, NHANES 1999–2018.

ethnicity in the associations between general obesity and central obesity with hypertension (all P for interaction > 0.05).

In total, the average PAF of hypertension due to general obesity was significantly higher in women (13.4 %, 95 %CI = 13.0–13.7 %) than those in men (10.7 %, 10.4–10.9 %). The PAFs of hypertension attributed to general obesity increased steadily from 11.9 % to 15.1 % in women from 1999 to 2000 to 2017–2018, with an overall slope of 0.38 % (95 % CI: 0.31–0.45 %, $P < 0.001$) per 2 years (Fig. 2. A). The PAFs of hypertension due to general obesity also increased from 8.4 % to 13.4 % in men, with a slope of 0.46 % (95 % CI: 0.36–0.56 %, $P < 0.001$) per 2 years, with no difference to that in women ($P = 0.225$). The average PAF of hypertension due to abdominal obesity was also significantly higher in women (29.0 %, 95 %CI = 28.7–29.4 %) than that in men (20.2 %, 19.8–20.7 %). The PAFs of hypertension due to abdominal obesity increased from 26.0 % to 30.8 % in women and 17.2 % to 23.0 % in men over the past 2 decades, respectively (Fig. 2. B). There was no significant sex difference in the slopes of increasing PAFs due to abdominal obesity ($\beta = 0.57$ % per 2 years for women vs. $\beta = 0.53$ % per year for men, $P = 0.664$).

Fig. 3 shows the trends of PAFs of hypertension due to general obesity and abdominal obesity from 1999 to 2000 to 2017–2018 by sex and race/ethnicity. All the PAFs increased steadily from 1999 to 2000 to 2017–2018. Significant differences in the trends of increased PAFs among Mexican Americans, non-Hispanic blacks, and non-Hispanic whites were observed. The comparisons for the slopes of the increased PAF curves across races/ethnicities are shown in Table 3. The increased trends of PAFs due to general obesity were significantly higher in Mexican American women ($\beta = 0.64$ %) compared with non-Hispanic white women ($\beta = 0.33$ %) and non-Hispanic black women ($\beta = 0.22$ %) ($P = 0.001$ and $P < 0.001$, respectively) (Fig. 3. A). Similarly, the increased trends of PAFs due to general obesity were also significantly higher in Mexican American men ($\beta = 1.42$ %) compared with that of non-Hispanic white ($\beta = 0.41$ %) and non-Hispanic black ($\beta = 0.27$ %) (both $P = 0.001$) (Fig. 3. B). Slopes of increasing PAFs due to abdominal obesity remained significantly larger among Mexican American women ($\beta = 0.73$ %) compared with those in non-Hispanic black women ($\beta = 0.29$ %) ($P = 0.011$) (Fig. 3. C). The increased trends of PAFs due to abdominal obesity were also significantly higher in Mexican American men ($\beta = 1.35$ %) compared with those of non-Hispanic white ($\beta = 0.49$ %) and non-Hispanic black ($\beta = 0.43$ %) (both $P < 0.001$) (Fig. 3. D).

We also did the same analyses for the children aged 8–17. The secular trends of general obesity, abdominal obesity, and hypertension in children aged 8–17 are shown in Supplementary Fig. 2. For children aged 8–17, general obesity and abdominal obesity were both significantly associated with hypertension in girls and boys (Supplementary Table 1). In total, the PAF of hypertension due to general obesity and abdominal obesity were both higher in boys than in girls (Supplementary Fig. 3). The secular trends of PAFs of hypertension attributed

to general obesity and abdominal obesity were significantly increased for boys, but not for girls.

4. Discussion

The PAF summarized the proportion of disease incidence in the population that was attributed to a risk factor. Periodically calculating PAF could adequately monitor population risk and identify potential benefits of preventive public health initiatives. In the present study, we determined the secular trends in the PAFs of hypertension attributed to general obesity and abdominal obesity in US adults and children from 1999 to 2018, by sex and race/ethnicity. Even though the prevalence of hypertension did not present apparently increased trends, the prevalence of general obesity and abdominal obesity gradually increased during the past two decades in US adults. The PAFs of hypertension attributed to general obesity and abdominal obesity both increased steadily in adults and boys aged 8–17. For adults, there were also racial/ethnic differences in the growth rates of PAFs. Mexican Americans had more rapid growth in PAFs of hypertension due to general obesity and abdominal obesity. Our results emphasize the substantial burden of hypertension that could potentially be eliminated with the optimization of weight and avoidance of obesity across the life course.

Almost 20 years ago, the Framingham study found that every 10 percent increase in body weight could increase the systolic BP level by about 5.6 mm Hg (Okosun et al., 1999). Since 2001, several guidelines have been issued by American scientific societies and government agencies to provide evidence-based recommendations for cardiometabolic risk factor identification and management (Expert Panel on Detection, 2001; Alberti et al., 2009; Arnett et al., 2019). A previous study using data from NHANES 1999–2000 to 2009–2010 found that the prevalence of hypertension was stable from 1999 to 2010, and the rates of awareness and control were greatly improved (from 63.8 % to 74.0 %; from 27.5 % to 46.5 %) (Guo et al., 2012). Increasing trends in hypertension awareness, treatment, and control might suggest that campaigns of Healthy People 2010 to increase hypertension awareness and treatment had been successful (Olives et al., 2013). However, based on our findings, the prevalence of hypertension in US adults remained at a high level. According to the updated guidelines of hypertension definition (Whelton et al., 2018), the prevalence of hypertension in the US was higher than 40 % for the last 2 decades. The high level of hypertension prevalence suggests that efforts to decrease the prevalence of hypertension are still needed. Our findings indicated that, theoretically, 13.4 % of the prevalence of hypertension in women and 10.7 % in men over 20 years old were attributable to general obesity. The PAFs were even higher and gradually increased for abdominal obesity. Therefore, reducing obesity should be continuously prioritized, especially for women. By quantifying the substantial and rising absolute burden of obesity on hypertension among distinct sex-race/ethnicity subgroups

Table 2
The associations between general obesity and abdominal obesity with hypertension in adults by sex and race/ethnicity, NHANES 1999–2018.

	Unadjusted OR (95 % CI)	Adjusted OR (95 % CI)	Adjusted RR (95 % CI)
The associations between general obesity and hypertension			
Women			
All races	2.48 (2.40–2.56)	1.85 (1.69–2.02)	1.41 (1.35–1.47)
Mexican	2.44 (2.35–2.52)	1.82 (1.35–2.46)	1.53 (1.25–1.84)
American			
Non-Hispanic	2.55 (2.53–2.57)	1.74 (1.57–1.92)	1.37 (1.30–1.43)
White			
Non-Hispanic	2.24 (1.94–2.59)	1.89 (1.85–1.94)	1.35 (1.34–1.36)
Black			
Men			
All races	2.39 (2.34–2.44)	1.84 (1.80–1.88)	1.35 (1.34–1.36)
Mexican	2.47 (2.29–2.66)	2.04 (1.76–2.35)	1.56 (1.45–1.65)
American			
Non-Hispanic	2.46 (2.38–2.54)	1.78 (1.66–1.91)	1.33 (1.29–1.36)
White			
Non-Hispanic	2.17 (2.15–2.20)	1.62 (1.31–2.00)	1.24 (1.13–1.33)
Black			
The association between abdominal obesity and hypertension			
Women			
All races	3.45 (3.35–3.55)	2.11 (1.91–2.33)	1.64 (1.54–1.73)
Mexican	3.36 (2.64–4.29)	1.90 (1.01–3.60)	1.67 (1.01–2.59)
American			
Non-Hispanic	3.52 (3.47–3.57)	2.00 (1.75–2.29)	1.59 (1.46–1.71)
White			
Non-Hispanic	3.40 (3.03–3.82)	2.32 (1.93–2.78)	1.62 (1.48–1.75)
Black			
Men			
All races	3.09 (3.04–3.13)	2.43 (2.38–2.49)	1.57 (1.56–1.59)
Mexican	2.72 (2.61–2.83)	2.67 (1.78–4.04)	1.70 (1.39–1.98)
American			
Non-Hispanic	3.22 (3.20–3.25)	2.40 (2.03–2.84)	1.58 (1.47–1.69)
White			
Non-Hispanic	2.90 (2.85–2.95)	2.19 (1.74–2.75)	1.40 (1.29–1.51)
Black			

OR, odds ratio; RR, risk ratio. The covariates in the adjusted model included age, educational attainment, current marriage status, household income, smoking behavior, drinking behavior, sleep duration, physical activity level, estimated intakes of sodium, energy, and sugar, HbA1c, low-density lipoprotein cholesterol, triglyceride, and history of cardiovascular diseases (congestive heart failure, coronary heart disease, heart attack, and stroke).

There is no interaction effect of gender in the associations between general obesity and central obesity with hypertension (both $P_{\text{for interaction}} > 0.05$).

There is no interaction effect of race/ethnicity in the associations between general obesity and central obesity with hypertension in women or men (all $P_{\text{for interaction}} > 0.05$).

The reference groups were the subjects without general obesity or abdominal obesity in each group.

All the P values < 0.001 for the ORs and RRs.

using the PAF, it might also be useful concerning other countries with similar rising obesity burdens. Similarly, another study also found that the PAF of hypertension due to overweight or obesity increased from 27.1 % in 1991 to 44.6 % in 2011 in China (Gou and Wu, 2021). Given the morbidity and mortality associated with hypertension (Aune et al., 2021), it is important to control obesity to reduce the prevalence of hypertension, thereby reducing the burden of hypertension-induced CVD.

As shown in our study, even though there was no sex difference in the rising temporal trends in PAFs for hypertension attributed to obesity, the PAFs remained higher in women compared with men. A previous study compared the sex differences in the temporal trends in the PAF for CVD using data from the Atherosclerosis Risk in Communities Study (Cheng et al., 2014). They also found that the combined contribution of all traditional risk factors, including hypertension, diabetes, obesity, hypercholesterolemia, and smoking, was substantially higher in women

compared with men. It's still unknown the reasons for the persistent sex differences in attributable hypertension and CVD risks. Possible explanations include the fact that women tend to cluster more traditional risk factors than men (Ford et al., 2002), or there are unidentified hormonal or non-hormonal biological variations (Stock and Redberg, 2012). Men may also have more unexplained risks, which may be explained by non-traditional risk factors.

A recent study found that Black adults consistently experienced the highest rates of hypertension, and significant increases were observed among Mexican American adults (Aggarwal et al., 2023). In the present study, we observed a faster-increased rate of PAF of hypertension due to obesity in the Mexican American population than those in non-Hispanic whites and non-Hispanic blacks during the past two decades. Some studies also reported that Mexican American men had a higher PAF of type 2 diabetes due to obesity than non-Hispanic white and non-Hispanic black men (Cameron et al., 2021; Quinones et al., 2013). These findings likely reflected the high and rising burden of obesity among Mexican Americans, as well as large increases in the consumption of ultraprocessed foods (Rehm et al., 2016), and socioeconomic factors, including barriers in access to insurance and healthy foods. Differences in the secular trends of the population burden of obesity on hypertension among sex-race/ethnicity subgroups may be related to the differences in the development stage of the obesity pandemic and the incidence of hypertension among those with obesity. It is well known that regional fat distribution differs between multiple racial/ethnic population (Lear et al., 2007). Besides, these disparities are likely a result of the complex interplay of social determinants of health, including socioeconomic, environmental, and healthcare-related factors. Our study also indicates that a more in-depth exploration of racial/ethnic and sex differences might facilitate identifying optimal approaches for the prevention and treatment of hypertension, especially in a society with ethnic diversity.

The strength of our study was that the data were collected from serial surveys over 18 years. The physical examinations, including the measurement of height, weight, WC, and blood pressure, were measured using standardized methods in each survey. Our study also had limitations. Firstly, the generalizability of our findings is limited to US adults aged over 18, and the transferability of these findings to other ethnic/racial populations is not clear. However, other countries may draw lessons from the US and take action to lower obesity burdens. Second, the current study was based on cross-sectional surveys. Future studies with long-term follow-up data should be analyzed to identify the magnitude of hypertension due to obesity over time. Third, the studies, conducted in other developing and developed countries, are warranted to further confirm our results in the future. Additionally, other factors might contribute to the high prevalence of hypertension and deserve to be assessed, such as ageing, high salt intake, smoking, alcohol dependence, and lack of physical activity (Muntner et al., 2020).

5. Conclusions

In the past two decades, the contributions of obesity to hypertension have been gradually rising among the US population. Our findings emphasize the need for weight control strategies for hypertension prevention and will contribute to informing other countries facing similar rising obesity burdens.

Author contributions

Z.Z. and Z.R. conceived and designed the study; Z.Z. and C.L. collected and analyzed the data. Z.Z., J.H. and X.Z. interpreted the data. Z.Z. and Z.R. drafted the manuscript. R.D., Z.S. and H.H. revised it. All authors agreed to be accountable for all aspects of the work and approved the final version of the paper. Z.Z. and Z.R. are the guarantors of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the

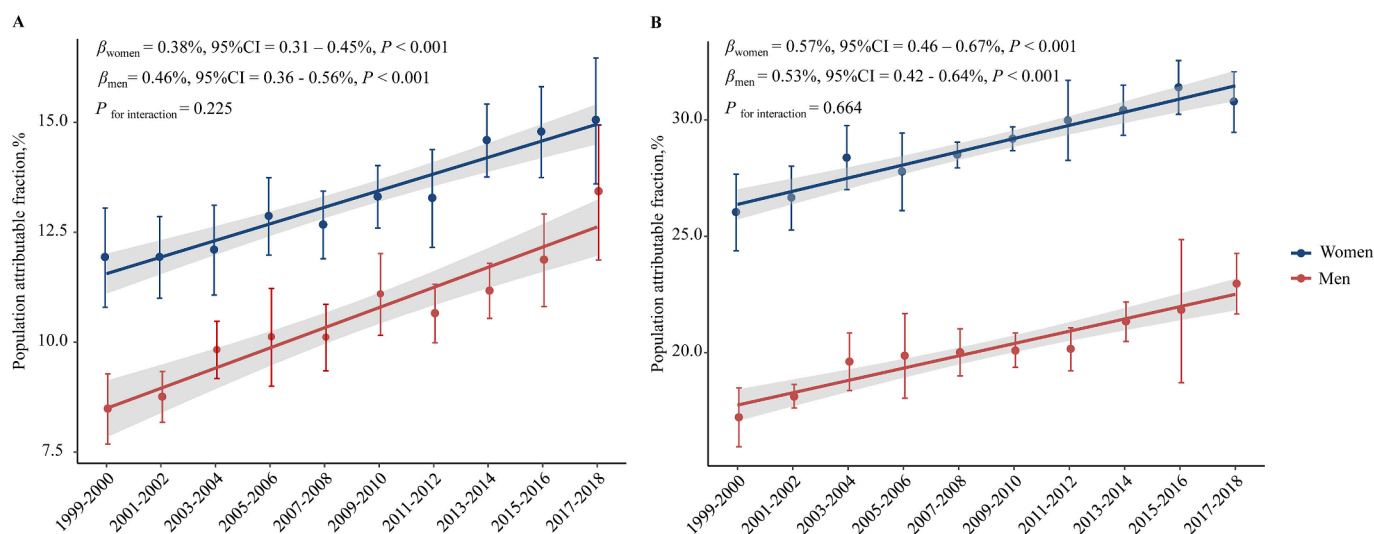


Fig. 2. Secular trends in population-attributable fractions (95% CIs) for hypertension due to (A) general obesity and (B) abdominal obesity in women and men, NHANES 1999–2018.

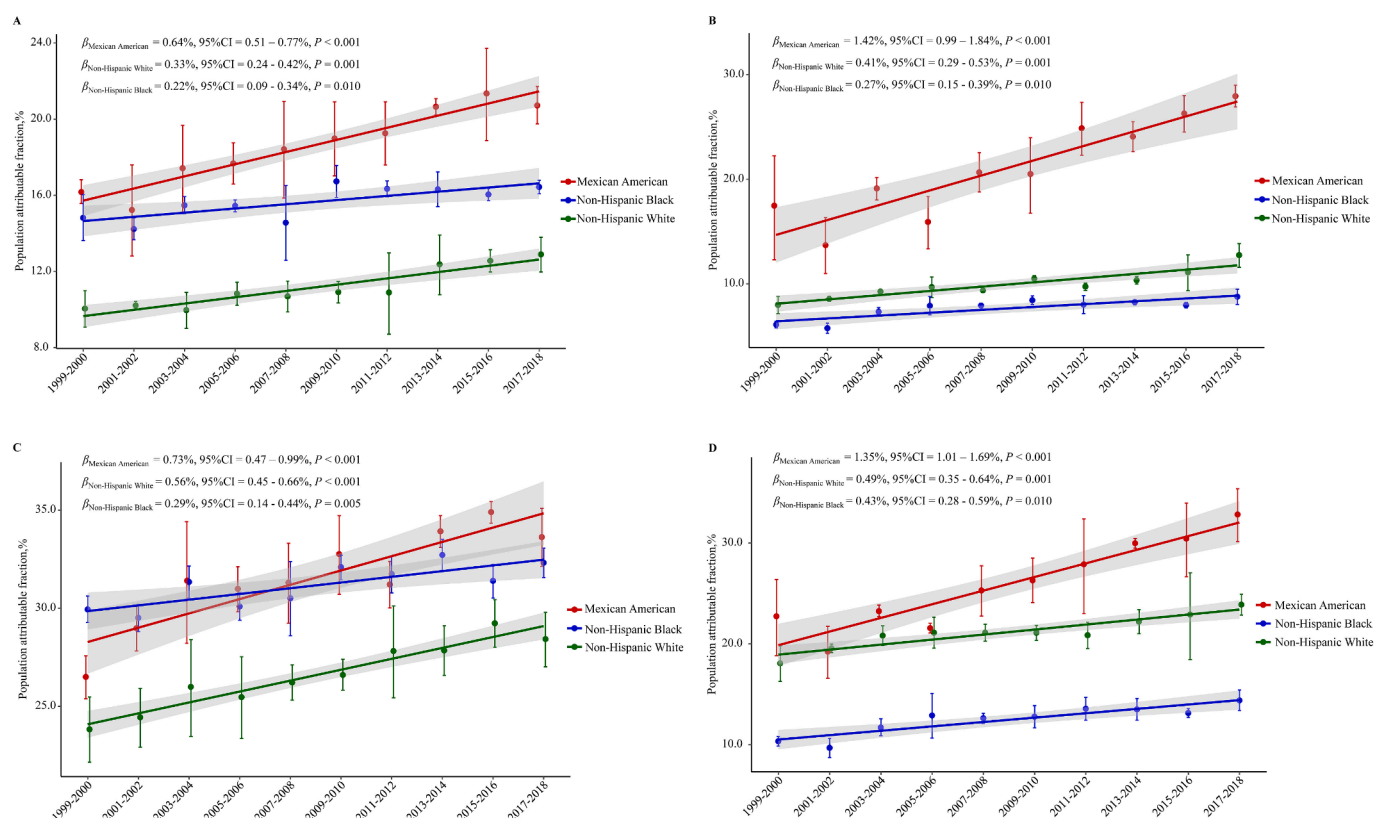


Fig. 3. Secular trends in population-attributable fractions (95% CIs) for hypertension due to general obesity in (A) women and (B) men, and abdominal obesity in (C) women and (D) men by race/ethnicity, NHANES 1999–2018.

data analysis.

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decision to submit the article for publication.

CRediT authorship contribution statement

Zhichao Zhang: Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Chen Li:** Formal analysis, Data curation. **Jiakang Hong:** Formal analysis, Data curation. **Xia Zhou:** Validation. **Rongquan Dai:** Writing – review & editing. **Zhiqiang Shu:** Writing – review & editing. **Hui Han:** Writing – review &

Table 3

P values of the comparisons for the slopes of increased population-attributable fractions of hypertension due to general obesity and abdominal obesity, as measured by regression coefficient (β s), among Mexican American, non-Hispanic white, and non-Hispanic black adults.

	Women	Men
Comparisons for the slopes (β s) of increased PAFs of hypertension due to general obesity		
$\beta_{\text{Mexican American VS. } \beta_{\text{Non-Hispanic white}}$	0.001	<0.001
$\beta_{\text{Mexican American VS. } \beta_{\text{Non-Hispanic black}}$	0.001	0.001
$\beta_{\text{Non-Hispanic white VS. } \beta_{\text{Non-Hispanic black}}$	0.192	0.142
Comparisons for the slopes (β s) of increased PAFs of hypertension due to abdominal obesity		
$\beta_{\text{Mexican American VS. } \beta_{\text{Non-Hispanic white}}$	0.241	<0.001
$\beta_{\text{Mexican American VS. } \beta_{\text{Non-Hispanic black}}$	0.011	<0.001
$\beta_{\text{Non-Hispanic white VS. } \beta_{\text{Non-Hispanic black}}$	0.013	0.569

Bonferroni’s method is used to adjust the Type-1 error. There are six times comparisons among different ethnic/racial adults, the adjusted P value required for significance is 0.05/3 = 0.017.

editing. **Zudong Ren:** Writing – review & editing, Writing – original draft, Supervision, Funding acquisition, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

This study uses data from the US National Health and Nutrition Examination Survey, which is an open database. The datasets used or analyzed during the current study are available from the URL (<https://www.cdc.gov/nchs/nhanes>) or the corresponding author upon reasonable request.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.pmedr.2024.102719>.

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