

RESEARCH ARTICLE

On the joint role of non-Hispanic Black race/ethnicity and weight status in predicting postmenopausal weight gain

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Abstract

Objectives

To determine how baseline weight status contributes to differences in postmenopausal weight gain among non-Hispanic Blacks (NHBs) and non-Hispanic Whites (NHWs).

Methods

Data were included from 70,750 NHW and NHB postmenopausal women from the Women's Health Initiative Observational Study (WHI OS). Body Mass Index (BMI) at baseline was used to classify women as having normal weight, overweight, obese class I, obese class II or obese class III. Cox proportional hazards was used to estimate the hazard of a 10% or more increase in weight from baseline.

Results

In both crude and adjusted models, NHBs were more likely to experience $\geq 10\%$ weight gain than NHWs within the same category of baseline weight status. Moreover, NHBs who were normal weight at baseline were most likely to experience $\geq 10\%$ weight gain in both crude and adjusted models. Age-stratified results were consistent with overall findings. In all age categories, NHBs who were normal weight at baseline were most likely to experience $\geq 10\%$ weight gain. Based on the results of adjusted models, the joint influence of NHB race/ethnicity and weight status on risk of postmenopausal weight gain was both sub-additive and sub-multiplicative.

(<https://www.whi.org/page/propose-a-paper>) with an approved manuscript proposal. The authors had no special access privileges to the data.

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Conclusion

NHBs are more likely to experience postmenopausal weight gain than NHWs, and the disparity in risk is most pronounced among those who are normal weight at baseline. To address the disparity in postmenopausal obesity, future studies should focus on identifying and modifying factors that promote weight gain among normal weight NHBs.

Introduction

Non-Hispanic Blacks (NHB) in the US have higher rates of obesity than non-Hispanic Whites (NHW) [1], which is thought to underlie disparities in chronic disease risk [2–4]. Individuals with obesity are at increased risk of cardiometabolic diseases including coronary heart disease [5], stroke [6], and diabetes [7, 8]. In women ages 60 and older, a significantly higher proportion of NHBs have obesity by body mass index (BMI) than NHWs. The most recently available estimates from the National Health and Nutrition Examination Survey show that 57.5% percent of NHBs have obesity, compared with 38.2% of NHWs [9]. Previous studies reported greater risk of weight gain in NHBs compared to NHWs at earlier life stages. In young-, middle-, and older- aged women, NHBs were at greater risk of weight gain than NHWs [10–13], but few studies have examined whether there are differences in the risk of postmenopausal weight gain in NHBs and NHWs. Weight status influences risk of weight gain and significantly higher rates of overweight, obesity and extreme obesity in NHBs compared to NHWs age 60 and older have been previously noted [14]. However, it is unclear how race/ethnicity and weight status jointly influence risk of postmenopausal weight gain in NHBs and NHWs.

This study uses data from the Women's Health Initiative Observational Study (WHI OS) to determine whether there are differences between NHBs and NHWs in the risk of postmenopausal weight gain, and characterize the role of baseline weight status. We compare the risk of $\geq 10\%$ weight gain in NHBs and NHWs overall, examine the interaction of race/ethnicity and baseline weight status, and determine the extent to which differences in risk are explained by differences in baseline weight status with and without adjustment for potential confounders.

Methods

Data and sample

Data were used from the WHI OS, which consists of 93,676 postmenopausal women who were enrolled between September 1993 and December 1998 and followed for up to eight years [15]. The analytic sample was restricted to NHW and NHB women ($n = 85,651$) in order to specifically examine how NHB race/ethnicity and baseline weight status jointly influence risk of postmenopausal weight gain. We excluded those with underweight BMI (< 18.5 kg/m²) at baseline ($n = 955$) in light of the potential for confounding of the relationship between race/ethnicity and weight gain by chronic diseases associated with wasting [16]. We also excluded those who self-identified as diabetic at baseline ($n = 3,291$), and those who reported a history of cancer ($n = 10,655$) thereby resulting in a final analytic sample comprising 70,750 respondents from the WHI OS. Written informed consent was obtained from all respondents. All study procedures were approved by institutional review boards at each of 40 participating clinical centers. A complete list of participating clinical centers is available elsewhere [17]. All data were de-identified before the authors had access to it. This secondary analysis was approved by the Institutional Review Board of Rush University Medical Center.

WHI OS data also included sociodemographic information that was collected at baseline using a standard questionnaire. This information included annual household income (less than \$10,000; \$10,000 to \$19,999; \$20,000 to \$34,999; \$35,000 to \$49,999; \$50,000 to \$74,000; \$75,000 to \$99,999; \$100,000 to \$149,999; and \$150,000 or more), race/ethnicity (American Indian or Alaskan Native, Asian or Pacific Islander, Black or African American, Hispanic/Latino, White [not of Hispanic Origin], or Other), and age (computed from birth date ascertained at screening). Usual alcohol intake was assessed at baseline using a standardized questionnaire. Possible responses ranged from 'non-drinker' to '7 or more drinks per week' (non-drinker, past drinker, <1 drink per month, <1 drink per week, 1–6 drinks per week, or 7 or more drinks per week). Time spent engaging in mild, moderate, and vigorous intensity physical activity was assessed at baseline using a questionnaire which has been described in detail elsewhere [18]. Mild physical activity was defined as walking, while moderate intensity activity was defined as 'not exhausting' and included biking outdoors, using a stationary exercise machine, calisthenics, easy swimming and dancing. Strenuous or hard exercise was defined as activities in which 'You work up a sweat and your heart beats fast' and included activities like aerobics, aerobic dancing, jogging, tennis, and swimming laps. Usual dietary intake during the past year was assessed at baseline and year three of follow-up using a Food Frequency Questionnaire comprising 122 items.

Approach

BMI at baseline, computed using measured height and weight, was used to classify respondents as normal weight (BMI: 18.5–24.9 kg/m²), overweight (BMI: 25.0–29.9 kg/m²), obese class I (BMI: 30.0–34.9 kg/m²), obese class II (BMI: 35.0–39.9 kg/m²), or obese class III (BMI ≥40.0 kg/m²). Weight was subsequently measured only at year one of follow-up, while self-reported 'highest weight since last follow-up' was available at follow-up years one through eight. Measured weight and reported 'highest weight since last follow-up at follow-up year one were found to be highly correlated (Pearson's *r*: 0.97; *p*<0.001). Thus, self-reported 'highest weight since last follow-up' was used to characterize the outcome variable, defined as ≥10% increase in weight from baseline weight.

Multiple imputation with chained equations was used to impute missing values in 10 data sets. The choice to impute 10 datasets was made as a conservative application of the approach used by Gao, Wilson and Heggul et al., who reported imputing 20 copies of their data in their 2020 study published in the Journal of the American Medical Association [3]. In sensitivity analyses, we compared our primary results with and without imputation (see S3 Table).

[19]. Analyses were repeated in each data set and estimates were pooled [20]. Following previous works using data from the WHI [21–23], Cox proportional hazards models were used to estimate the relationship between race/ethnicity and a ≥10.0% weight gain from baseline. Respondents' self-reported highest weight since last follow-up was measured at one, three, four, five, six, seven and eight years of follow-up. The proportional hazards assumption was tested using plots of log-log(survival) vs. log(follow-up time), and the proportional hazards assumption was deemed to be satisfied if group-specific plots (e.g., race/ethnicity) were approximately parallel [24]. Overall hazard ratios comparing NHBs to NHWs, and comparing successive categories of weight status to those who were normal weight at baseline, were computed.

To evaluate the interaction of race/ethnicity and weight status on the additive and multiplicative scales, hazard ratios were computed comparing each combination of race/ethnicity and baseline weight status to a common referent group (normal weight NHWs) using an appropriate categorical interaction term. Following Hosmer and Lemeshow (1992), departure from

additive interaction between NHB race/ethnicity and weight status was evaluated using the general formula:

$$(H_{1k}/H_{00}) - (H_{10}/H_{00}) - (H_{0k}/H_{00}) + 1,$$

where k represents category of baseline weight status [25]. Departure from multiplicative interaction was assessed using the following formula:

$$(H_{1k}/H_{00}) / [(H_{10}/H_{00}) * (H_{0k}/H_{00})].$$

If the interaction between race/ethnicity and weight status is additive, then joint impact of these variables on risk of postmenopausal weight gain is equal to the sum of their individual impact. However, if the relationship is multiplicative, then race/ethnicity and weight status jointly influence the risk of postmenopausal weight gain and together have greater influence than the sum of each variable's independent contribution to the overall risk relationship.

Interaction and confounding

Interaction between race/ethnicity (comprising NHWs and NHBs) and weight status at baseline was examined by including an appropriate interaction term and evaluating the resulting Wald test p-value associated with this coefficient. The interaction of race/ethnicity and baseline BMI was significant ($p < 0.001$). Backward selection ($\alpha = 0.10$) was used to identify potential confounders to be included in adjusted models. The fully-saturated model included education level, annual household income, smoking status, alcohol intake, age, total energy intake at baseline and MET-hours of mild, moderate and hard exercise. An equivalent set of potential confounders was obtained using forward selection ($\alpha = 0.10$) when variables were introduced in the opposite order used in the backward selection model. In both approaches, the models were constrained to include a term denoting the interaction of race/ethnicity and baseline weight status. Final adjusted models controlled for education level, annual household income, smoking status, alcohol intake, age, total energy intake at baseline and MET-hours of mild, moderate and hard exercise. All analyses were carried out in Stata (Version 16, Stata Corp, College Station, Texas, USA).

Results

Selected sample characteristics are given in Table 1. The majority of respondents were NHW (91.4%), and normal weight (40.7%) or overweight (34.2%) at baseline, with a mean age of 63.5 years (± 7.3 years). On average, NHB respondents were younger, had greater rates of class I, II, and III obesity at baseline, and were more likely to have an annual household income of less than \$20,000 (27.4%) than NHWs ($p < 0.001$). NHBs were also more than twice as likely as NHWs to report non-drinking status ($p < 0.001$).

Log-log(survival) was plotted against log(follow-up time) by race/ethnicity to determine whether the proportional hazards assumption was met. The plots were approximately parallel, thereby confirming that the proportionality assumption was met.

Unadjusted smoothed hazards by race/ethnicity are given in Fig 1. As shown, NHBs were 1.54 times (95% CI: 1.46, 1.62) more likely to experience $\geq 10\%$ weight gain than NHWs.

Unadjusted smoothed hazards by baseline weight status are presented in S1 Fig. As shown, women with class I obesity (HR: 1.05; 95% CI: 1.00, 1.10) and class II obesity (HR: 1.17; 95% CI: 1.09, 1.26) at baseline were more likely to experience $\geq 10\%$ weight gain than those with normal weight at baseline.

Table 2 shows overall hazard ratios comparing risk of weight gain by weight status to normal weight respondents. In unadjusted overall models, those with class II obesity at baseline

Table 1. Selected characteristics of non-Hispanic White and non-Hispanic Black respondents from the Women’s Health Initiative Observational Study overall, and by race/ethnicity¹.

	Overall	Non-Hispanic White	Non-Hispanic Black	p-value
	<-----N (%)----->			
N	70,750	64,676 (91.4%)	6,074 (8.6%)	
Weight status				
Normal weight (BMI: 18.5–24.9 kg/m ²)	28,773 (40.7%)	27,597 (42.7%)	1,176 (19.4%)	<0.001
Overweight (BMI: 25.0–29.9 kg/m ²)	24,187 (34.2%)	22,110 (34.2%)	2,077 (34.2%)	
Obese class I (BMI: 30.0–34.9 kg/m ²)	10,770 (15.2%)	9,270 (14.3%)	1,500 (24.7%)	
Obese class II (BMI: 35.0–39.9 kg/m ²)	3,867 (5.5%)	3,178 (4.9%)	689 (11.3%)	
Obese class III (BMI ≥40.0 kg/m ²)	2,318 (3.3%)	1,771 (2.7%)	547 (9.0%)	
Missing	835 (1.2%)	750 (1.2%)	85 (1.4%)	
Waist circumference				
<88 cm	47,012 (66.4%)	44,022 (68.1%)	2,990 (49.2%)	<0.001
≥88 cm	23,431 (33.1%)	20,359 (31.5%)	3,072 (50.6%)	
Missing	307 (0.4%)	295 (0.5%)	12 (0.2%)	
Highest education completed				
Less than high school	14,181 (20.0%)	12,599 (19.5%)	1,582 (26.0%)	<0.001
High school diploma or equivalent	25,566 (36.1%)	23,353 (36.1%)	2,213 (36.4%)	
Some college	8,301 (11.7%)	7,772 (12.0%)	529 (8.7%)	
Baccalaureate degree or more	22,142 (31.3%)	20,472 (31.7%)	1,670 (27.5%)	
Missing	560 (0.8%)	480 (0.7%)	80 (1.3%)	
Household income				
Less than \$20,000	9,390 (13.3%)	7,724 (11.9%)	1,666 (27.4%)	<0.001
\$20,000 to \$49,999	28,554 (40.4%)	26,273 (40.6%)	2,281 (37.6%)	
\$50,000 to \$99,999	20,153 (28.5%)	18,839 (29.1%)	1,314 (21.6%)	
\$100,000 or more	7,608 (10.8%)	7,334 (11.3%)	274 (4.5%)	
Missing	5,045 (7.1%)	4,506 (7.0%)	539 (8.9%)	
Smoking status				
Never	34,793 (49.2%)	31,782 (49.1%)	3,011 (49.6%)	<0.001
Former	30,644 (43.3%)	28,364 (43.9%)	2,280 (37.5%)	
Current	4,323 (6.1%)	3,678 (5.7%)	645 (10.6%)	
Missing	990 (1.4%)	852 (1.3%)	138 (2.3%)	
Alcohol use				
Non-drinker	6,713 (9.5%)	5,610 (8.7%)	1,103 (18.2%)	<0.001
Past drinker	12,183 (17.2%)	10,277 (15.9%)	1,906 (31.4%)	
<1 drink per month	8,063 (11.4%)	7,289 (11.3%)	774 (12.7%)	
<1 drink per week	14,518 (20.5%)	13,452 (20.8%)	1,066 (17.6%)	
1 to 6 drinks per week	19,236 (27.2%)	18,393 (28.4%)	843 (13.9%)	
7 or more drinks per week	9,560 (13.5%)	9,280 (14.3%)	280 (4.6%)	
Missing	477 (0.7%)	375 (0.6%)	102 (1.7%)	
Age category				
49–54 years	9,458 (13.4%)	8,335 (12.9%)	1,123 (18.5%)	<0.001
55–59 years	13,402 (18.9%)	12,066 (18.7%)	1,336 (22.0%)	
60–64 years	15,633 (22.1%)	14,140 (21.9%)	1,493 (24.6%)	
65 and older	32,257 (45.6%)	30,135 (46.6%)	2,122 (34.9%)	
Missing	0 (0.0%)	0 (0.0%)	0 (0.0%)	
	<-----Mean ± standard deviation----->			

(Continued)

Table 1. (Continued)

	Overall	Non-Hispanic White	Non-Hispanic Black	p-value
Age	63.5 ± 7.3	63.6 ± 7.3	61.8 ± 7.3	<0.001
Missing	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Physical activity (MET-hours/week)				
Mild exercise	1.4 ± 3.1	1.4 ± 3.2	0.7 ± 2.2	<0.001
Missing	804 (1.1%)	736 (1.1%)	68 (1.1%)	
Moderate exercise	3.4 ± 5.4	3.5 ± 5.5	2.3 ± 4.5	<0.001
Missing	804 (1.1%)	736 (1.1%)	68 (1.1%)	
Hard exercise	4.0 ± 8.5	4.0 ± 8.6	3.4 ± 7.8	<0.001
Missing	804 (1.1%)	736 (1.1%)	68 (1.1%)	
Total daily energy intake	1,555 ± 666	1,559 ± 619	1,503 ± 1,045	<0.001
Missing	64 (0.1%)	56 (0.1%)	8 (0.1%)	

¹ P-values given correspond to a X^2 test for categorical variables, and to a Students t-test for continuous variables (age and physical activity).

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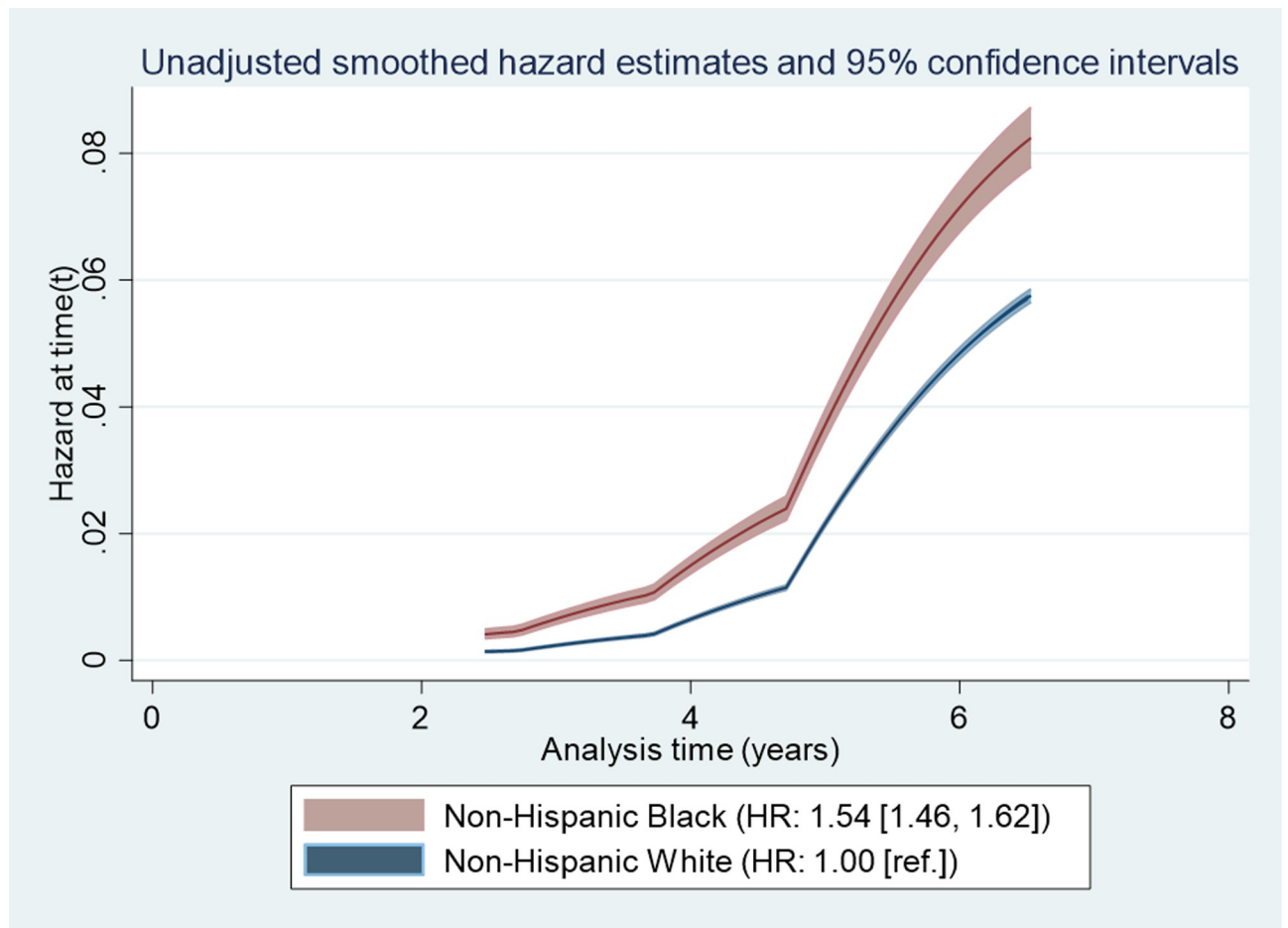


Fig 1. Unadjusted smooth hazards and 95% confidence intervals (shaded) by race/ethnicity among non-Hispanic White and non-Hispanic Black respondents from the Women’s Health Initiative Observational Study.

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Table 2. Overall and common referent hazard ratios and 95% confidence intervals comparing the hazard for $\geq 10\%$ weight gain by baseline weight status in non-Hispanic Blacks and non-hispanics (n = 70,750)¹⁻³.

	Hazard ratios (95% confidence interval)					p-trend
	Normal weight	Overweight	Obese class I	Obese class II	Obese class III	
Crude models						
Overall	1.00 (ref.)	1.03 (0.99, 1.07)	1.05 (1.00, 1.11)	1.17 (1.09, 1.26)	1.00 (0.90, 1.11)	<0.001
Non-Hispanic White	1.00 (ref.)	1.02 (0.99, 1.06)	1.03 (0.98, 1.09)		0.95 (0.85, 1.05)	0.071
Non-Hispanic Black ⁴	1.77 (1.60, 1.96)	1.49 (1.37, 1.63)	1.47 (1.32, 1.64)	1.72 (1.45, 2.03)	1.43 (1.17, 1.74)	0.063
Non-Hispanic Black ⁵	1.00 (ref.)	0.84 (0.74, 0.95)	0.82 (0.71, 0.94)	0.94 (0.78, 1.14)	0.79 (0.63, 0.97)	0.063
Adjusted models						
Overall	1.00 (ref.)	1.01 (0.97, 1.05)	0.97 (0.92, 1.02)	1.00 (0.93, 1.08)	0.80 (0.72, 0.88)	0.001
Non-Hispanic White	1.00 (ref.)	1.01 (0.97, 1.05)	0.97 (0.92, 1.02)	0.99 (0.91, 1.06)	0.77 (0.70, 0.86)	<0.001
Non-Hispanic Black ⁴	1.45 (1.31, 1.60)	1.21 (1.11, 1.33)	1.15 (1.03, 1.28)	1.26 (1.06, 1.49)	1.02 (0.83, 1.24)	0.005
Non-Hispanic Black ⁵	1.00 (ref.)	0.83 (0.74, 0.95)	0.79 (0.69, 0.91)	0.88 (0.73, 1.07)	0.72 (0.58, 0.90)	0.005
Relative excess hazard, additive interaction ⁷		-0.30	-0.33	-0.18	-0.29	
Relative hazard due to multiplicative interaction ⁸		0.83	0.81	0.86	0.85	

¹ Weight status was defined using baseline body mass index (BMI) as normal weight (BMI: 18.5–24.9 kg/m²), overweight (BMI: 25.0–29.9 kg/m²), obese class I (BMI: 30.0–34.9 kg/m²), obese class II (BMI: 35.0–39.9 kg/m²), or obese class III (BMI ≥ 40.0 kg/m²).

² Adjusted models controlled for education level, annual household income, smoking status, alcohol intake, age, total energy intake at baseline and MET-hours of mild, moderate and hard exercise.

³ P-trend corresponds to a Wald test statistic when a linear term for baseline body weight status was substituted in the model.

⁴ Values shown are relative to the common referent group, normal weight non-Hispanic Whites.

⁵ Values shown are relative to the referent group, normal weight non-Hispanic Blacks.

⁶ Relative excess hazard due to additive interaction is based on adjusted models and defined as the hazard for weight gain in the doubly exposed less the sum of the null value (1) and the risk of weight gain in each singly exposed group. A value less than or greater than 0 would suggest departure from additive interaction.

⁷ The relative hazard due to multiplicative interaction is based on adjusted models and defined as the ratio of the hazard in the doubly exposed to the product of hazards for each singly exposed group. A value less than or greater than 1 would suggest departure from multiplicative interaction.

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were 1.17 (95% CI: 1.09, 1.26) times more likely to experience $\geq 10\%$ weight gain than those who were normal weight at baseline. The overall trend for a linear term for baseline weight status was significant ($p < 0.001$), but the directionality was not consistent across categories of baseline weight status. In adjusted models of the overall relationship between baseline weight status and risk of weight gain, HRs were significantly attenuated and the relationship between class II obesity and risk of weight gain was no longer significant. However, those with class III obesity at baseline were less likely (HR: 0.80; 95% CI: 0.72, 0.88) to than those who were normal weight at baseline. The was evidence of an inverse trend, which was significant ($p = 0.001$).

Table 2 also shows the results of common referent models comparing risk of weight gain in NHBs and NHWs to that of NHWs with normal weight at baseline. In unadjusted models, NHWs with class II obesity were more likely (HR: 1.13; 95% CI: 1.04, 1.22) to experience $\geq 10\%$ weight gain. No other significant relationships were observed and the trend was not significant ($p = 0.071$). In adjusted models, the relationship between class II obesity and risk of weight gain in NHWs was no longer significant. The was evidence of an inverse trend, which was significant ($p < 0.001$). However, NHWs with class III obesity at baseline were less likely (HR: 0.77; 95% CI: 0.70, 0.86) to experience $\geq 10\%$ weight gain. In unadjusted models, NHBs who were normal weight (HR: 1.77; 95% CI: 1.60, 1.96), overweight (HR: 1.49; 95% CI: 1.37, 1.63), class I obesity (HR: 1.47; 95% CI: 1.32, 1.64), class II obesity (HR: 1.72; 95% CI: 1.45, 2.03) and class III obesity (HR: 1.43; 95% CI: 1.17, 1.74) were more likely to experience $\geq 10\%$

weight gain than normal weight NHBs. These relationships were attenuated in adjusted models. In unadjusted models, the trend was not significant in NHBs ($p = 0.063$). NHW with normal weight (HR: 1.45; 95% CI: 1.31, 1.60), overweight (HR: 1.21; 95% CI: 1.11, 1.33), class I obesity (HR: 1.15; 95% CI: 1.03, 1.28) and class II obesity (HR: 1.26; 95% CI: 1.06, 1.49) were more likely to experience $\geq 10\%$ weight gain than NHWs with normal weight at baseline. The relationship between class III obesity and risk of weight gain in NHBs was no longer significant in adjusted models. There was evidence of an inverse trend, which was significant ($p = 0.005$).

Also presented in [Table 2](#) are within strata HRs comparing risk of weight gain in NHBs to those who were normal weight at baseline. HRs in unadjusted and adjusted models were similar, but there was no significant trend in unadjusted models. In adjusted models, NHBs with overweight (HR: 0.83; 95% CI: 0.74, 0.95), class I obesity (HR: 0.79; 95% CI: 0.69, 0.91) and class III obesity (HR: 0.72; 95% CI: 0.58, 0.90) were less likely to experience $\geq 10\%$ weight gain than NHBs who were normal weight at baseline. The trend was significant ($p = 0.005$), but the directionality of the trend was not clear. In both crude and adjusted models, there was no significant relationship observed for NHBs with class II obesity at baseline.

The relative excess risk due to additive interaction, and the proportion of risk due to multiplicative interaction are also presented in [Table 2](#). As shown, the interaction of NHB race/ethnicity and weight status at baseline was less than additive and less than multiplicative.

Findings stratified by age group (49 to 54, 55 to 59, 60 to 64 and 65 and older) are presented in [S1 Table](#). As in unstratified models, NHBs were more likely to experience $\geq 10\%$ weight gain than NHWs in the same category of baseline weight status. Results were similar in unadjusted and adjusted models. Findings from age-stratified overall models and stratum-specific models comparing the risk of weight gain in NHBs to those who were normal weight at baseline are presented in [S2 Table](#). Results of these models were also consistent with those of models not stratified by age.

Discussion

NHB postmenopausal women were more likely to experience $\geq 10\%$ weight gain than NHWs, which is consistent with findings from previous studies [[10–13](#)]. While this was true across all categories of baseline weight status, the difference in risk was most prominent in women with normal weight at baseline, with NHBs being more than 50% more likely to experience $\geq 10\%$ weight gain than NHWs. This finding suggests that efforts to reducing the disparity in the prevalence of postmenopausal obesity among NHBs and NHWs should focus on preventing excess weight gain in NHBs with normal weight. Moreover, NHB postmenopausal women in our study had greater rates of class I, II and III obesity than NHWs, thereby suggesting that racial/ethnic divergence in the prevalence of obesity in NHBs and NHWs may have begun prior to study enrollment. A number of prior studies have also reported that in young-, middle-, and older- aged women, NHBs were at greater risk of weight gain than NHWs [[10–13](#)]. Moreover, NHB women enter middle age at a higher BMI, and gain less weight thereafter, than NHW women [[12](#)]. Accordingly, interventions to prevent weight gain at earlier ages would be instrumental to reducing racial/ethnic disparities in the prevalence of postmenopausal obesity.

Like others [[26](#)], we found that risk of weight gain was lower in women with obesity at baseline than those with normal weight. This was true in both NHBs and NHWs. With regard to the joint influence of NHB race/ethnicity and weight status at baseline on risk of postmenopausal weight gain, we found that overall, the relationship was both sub-additive and sub-multiplicative. Moreover, the overall greater risk of weight gain in NHBs vs. NHWs was due to a sharply higher risk of weight gain in normal weight NHBs. However, the risk of weight gain

was higher in NHBs across all categories of baseline weight status. This would suggest that the overall higher risk of weight gain in NHBs was not due to differences in weight status alone, but rather due to other biological [27, 28], social [10, 13, 29], or environmental factors [30, 31]. Biological differences may include differences in energy expenditure and fatty acid metabolism that promote excess weight gain in NHBs relative to NHWs [32–34]. Sociocultural differences between NHBs and NHWs may also contribute to the disparity in weight status. These include sociocultural differences in perceived weight status—it has been established that there are racial/ethnic differences in perceived weight status in NHB and NHW women. NHBs are more likely to perceive themselves as having a healthy body weight, even at higher BMIs, compared with NHWs [35], and this could contribute to higher rates of obesity among NHB women. Racial/ethnic differences in other sociocultural factors may also play a role. NHB women are more likely than white women to experience lower socioeconomic positioning, thereby increasing their likelihood of weight gain throughout the lifespan [11]. Other sociocultural differences in education level [36], annual household income [37], and physical activity level [38], have also been noted and may also contribute to the disparity in obesity prevalence among postmenopausal NHB and NHW women, along with environmental factors. NHBs are more likely to reside in neighborhoods with limited access to healthy foods [39], and limited neighborhood walkability [40], both of which are associated with increased risk of weight gain. Lastly, it has been shown that differences in weight gain throughout the life-cycle may also contribute to racial/ethnic disparity in obesity among postmenopausal women. NHB women experience greater weight gain than NHWs in early [41], middle [11, 42], and older adulthood [43], thereby placing them at greater risk of obesity than NHWs at virtually every age. This is consistent with the findings of our study. We found that postmenopausal NHBs had markedly higher rates of class I obesity, and more than twice the rates of class II and class III obesity, as NHWs. Recent evidence from the US nationally representative National Health and Nutrition Examination Survey (NHANES), which showed that in middle-(40–59 y) and older-(60+ y) aged women, the prevalence of obesity was an average of 20 percentage points higher in NHBs compared to NHWs (1), also supports this result. Taken together, these findings suggest that much of the divergence in the prevalence of obesity in NHW and NHB postmenopausal women occurs prior to age 40. Accordingly, efforts to reduce racial/ethnic disparities in obesity will require a focus on preventing excess weight gain in NHB women at earlier life stages, and particularly in those younger than age 40.

There are a number of important limitations to our study that bear mentioning here. Foremost, the study sample may not have been representative of the current population of US postmenopausal women. Notably, women in the WHI OS were ages 49 to 81 years upon enrollment, which completed in 1998, and study participants were recruited from 40 clinical centers across 24 US states and the District of Columbia, which may have limited the representativeness of the sample. A further limitation is the potential for misreporting of dietary intake due by measuring dietary intake using an FFQ, which may be more prone to misreporting than other self-report measures such as a 24-hour recall or food records [44]. Common sources of error include the comprehensiveness of the food list and the length of time over which respondents are asked to recall their diet, and the total time it takes to complete the questionnaire [45, 46]. Nonetheless the WHI FFQ has been validated in a number of studies, which have shown the WHI FFQ to have acceptable correlations with other common measures of diet [47]. Moreover, as the current study was interested in capturing ‘usual’ dietary intake, the FFQ is more aptly suited than measures designed to measure diet over short periods.

To characterize the primary outcome self-reported weight was used, which may be prone to both intentional and unintentional misreporting [48, 49]. Moreover, weight was measured only at baseline, while self-reported ‘highest weight since last follow-up’ was ascertained at all

subsequent years of follow-up starting with year one. Accordingly, the self-report measure was used in conjunction with measured weight (at baseline) to compute percent change in weight from baseline. Nonetheless, we found measured weight at baseline to have acceptable correlation with highest reported weight in the time since last follow-up at year one. Finally, it should be noted that the average age of enrollment into the WHI OS was 63.7 years, whereas it has been previously reported that the median age of menopause among US women is 52.6 years [50]. Accordingly, for some women in this study, the period immediately after the onset of menopause may not have been captured, which limits our ability to draw conclusions about weight gain during the period shortly after menopause. Lastly, it is a limitation of this study that the outcome was defined as a relative measure ($\geq 10\%$). This definition of significant weight gain corresponds to the widely-used definition of significant weight loss ($\geq 10\%$) as defined by Wing and Hill of the National Weight Loss Consortium [51]. Use of a relative measure of weight loss meant that women who were heavier at baseline would need to gain more weight than those who weighed less in order to achieve a 10% weight gain. Nonetheless, in sensitivity analysis using ≥ 10 pound weight gain as the primary outcome, the results were similar to those presented from the primary analysis (see S4 Table).

Conclusion

This study provides several important contributions to the literature. First, there are few studies that explore differences in the risk of postmenopausal weight gain in NHBs and NHWs. Consistent with prior studies we found that, overall, NHB women were more likely to experience $\geq 10\%$ weight gain than NHW women. This was true in every category of baseline weight status, and the difference in risk was especially pronounced in women with normal weight at baseline. This finding suggests that efforts to prevent postmenopausal weight gain in NHBs would be best directed toward those who are normal weight. Furthermore, we found that NHB postmenopausal women in our sample had significantly higher rates of class I, II, and III obesity than NHWs at baseline, suggesting that divergence in the prevalence of obesity begins prior to menopause. Future studies seeking to address the disparity in postmenopausal obesity should focus on preventing weight gain in NHBs prior to menopause. Reducing the rates of obesity in NHB postmenopausal women could help to reduce racial/ethnic disparities in risk of obesity-related chronic diseases in NHBs and NHWs.

Supporting information

S1 Fig. Unadjusted smooth hazards by weight status among non-Hispanic White and non-Hispanic Black respondents from the Women's Health Initiative Observational Study.
(TIF)

S1 Table. Common referent hazard ratios and 95% confidence intervals comparing the hazard of a $\geq 10\%$ weight gain by baseline weight status in non-Hispanic Blacks and non-Hispanic Whites stratified by age (n = 70,750)¹⁻³.
(DOCX)

S2 Table. Overall and stratum-specific hazard ratios and 95% confidence intervals comparing the hazard for $\geq 10\%$ weight gain by baseline weight status in non-Hispanic Blacks and non-hispanics stratified by age (n = 70,750)¹⁻³.
(DOCX)

S3 Table. Overall and common referent hazard ratios and 95% confidence intervals comparing the hazard for $\geq 10\%$ weight gain by baseline weight status in non-Hispanic Blacks

and non-hispanics using complete-case analysis¹⁻³.
(DOCX)

S4 Table. Overall and common referent hazard ratios and 95% confidence intervals comparing the hazard for ≥ 10 pound weight gain by baseline weight status in non-Hispanic Blacks and non-hispanics¹⁻³.

(DOCX)

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References

1. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. *Jama*. 2014; 311(8):806–14. <https://doi.org/10.1001/jama.2014.732> PMID: 24570244
2. Brancati FL, Kao WL, Folsom AR, Watson RL, Szklo M. Incident type 2 diabetes mellitus in African American and white adults: the Atherosclerosis Risk in Communities Study. *Jama*. 2000; 283(17):2253–9. <https://doi.org/10.1001/jama.283.17.2253> PMID: 10807384
3. DeSantis CE, Fedewa SA, Goding Sauer A, Kramer JL, Smith RA, Jemal A. Breast cancer statistics, 2015: Convergence of incidence rates between black and white women. *CA: A Cancer Journal for Clinicians*. 2015:n/a-n/a. <https://doi.org/10.3322/caac.21320> PMID: 26513636
4. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005; 111(10):1233–41. <https://doi.org/10.1161/01.CIR.0000158136.76824.04> PMID: 15769763
5. Bogers RP, Bemelmans WJ, Hoogenveen RT, Boshuizen HC, Woodward M, Knekt P, et al. Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: a meta-analysis of 21 cohort studies including more than 300 000 persons. *Archives of internal medicine*. 2007; 167(16):1720–8. <https://doi.org/10.1001/archinte.167.16.1720> PMID: 17846390
6. Lu Y, Hajifathalian K, Ezzati M, Woodward M, Rimm EB, Danaei G. Metabolic mediators of the effects of body-mass index, overweight, and obesity on coronary heart disease and stroke: a pooled analysis of 97 prospective cohorts with 1·8 million participants. *Lancet (London, England)*. 2013; 383(9921):970–83.
7. Kivimäki M, Kuosma E, Ferrie JE, Luukkonen R, Nyberg ST, Alfredsson L, et al. Overweight, obesity, and risk of cardiometabolic multimorbidity: pooled analysis of individual-level data for 120 813 adults from 16 cohort studies from the USA and Europe. *The Lancet Public Health*. 2017; 2(6):e277–e85. [https://doi.org/10.1016/S2468-2667\(17\)30074-9](https://doi.org/10.1016/S2468-2667(17)30074-9) PMID: 28626830
8. Abdullah A, Peeters A, de Courten M, Stoelwinder J. The magnitude of association between overweight and obesity and the risk of diabetes: a meta-analysis of prospective cohort studies. *Diabetes research and clinical practice*. 2010; 89(3):309–19. <https://doi.org/10.1016/j.diabres.2010.04.012> PMID: 20493574
9. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in Obesity Among Adults in the United States, 2005 to 2014. *Jama*. 2016; 315(21):2284–91. <https://doi.org/10.1001/jama.2016.6458> PMID: 27272580

10. Ailshire JA, House JS. The unequal burden of weight gain: an intersectional approach to understanding social disparities in BMI trajectories from 1986 to 2001/2002. *Social forces*. 2012;:sor001.
11. Baltus PT, Lynch JW, Everson-Rose S, Raghunathan TE, Kaplan GA. Race/ethnicity, life-course socio-economic position, and body weight trajectories over 34 years: the Alameda County Study. *American Journal of Public Health*. 2005; 95(9):1595–601. <https://doi.org/10.2105/AJPH.2004.046292> PMID: 16051936
12. Botosaneanu A, Liang J. Social stratification of body weight trajectory in middle-age and older Americans: results from a 14-year longitudinal study. *Journal of aging and health*. 2010;0898264310385930. <https://doi.org/10.1177/0898264310385930> PMID: 21068396
13. Clarke P, O'Malley PM, Johnston LD, Schulenberg JE. Social disparities in BMI trajectories across adulthood by gender, race/ethnicity and lifetime socio-economic position: 1986–2004. *International Journal of Epidemiology*. 2009; 38(2):499–509. <https://doi.org/10.1093/ije/dyn214> PMID: 18835869
14. Wang Y, Beydoun MA. The Obesity Epidemic in the United States—Gender, Age, Socioeconomic, Racial/Ethnic, and Geographic Characteristics: A Systematic Review and Meta-Regression Analysis. *Epidemiologic Reviews*. 2007; 29(1):6–28.
15. Langer RD, White E, Lewis CE, Kotchen JM, Hendrix SL, Trevisan M. The Women's Health Initiative Observational Study: baseline characteristics of participants and reliability of baseline measures. *Annals of epidemiology*. 2003; 13(9):S107–S21. [https://doi.org/10.1016/s1047-2797\(03\)00047-4](https://doi.org/10.1016/s1047-2797(03)00047-4) PMID: 14575943
16. Muscaritoli M, Anker S, Argiles J, Aversa Z, Bauer J, Biolo G, et al. Consensus definition of sarcopenia, cachexia and pre-cachexia: joint document elaborated by Special Interest Groups (SIG)“cachexia-anorexia in chronic wasting diseases” and “nutrition in geriatrics”. *Clinical nutrition*. 2010; 29(2):154–9. <https://doi.org/10.1016/j.clnu.2009.12.004> PMID: 20060626
17. The Women's Health Initiative Study Group. Design of the Women's Health Initiative Clinical Trial and Observational Study. *Controlled Clinical Trials*. 1998; 19(1):61–109. [https://doi.org/10.1016/s0197-2456\(97\)00078-0](https://doi.org/10.1016/s0197-2456(97)00078-0) PMID: 9492970
18. Meyer A-M, Evenson KR, Morimoto L, Siscovick D, White E. Test-Retest Reliability of the WHI Physical Activity Questionnaire. *Medicine and science in sports and exercise*. 2009; 41(3):530–8. <https://doi.org/10.1249/MSS.0b013e31818ace55> PMID: 19204598
19. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. *Statistics in medicine*. 2011; 30(4):377–99. <https://doi.org/10.1002/sim.4067> PMID: 21225900
20. Rubin DB. *Multiple imputation for nonresponse in surveys*: John Wiley & Sons; 2004.
21. Howard BV, Manson JE, Stefanick ML, Beresford SA, Frank G, Jones B, et al. Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial. *Jama*. 2006; 295(1):39–49. <https://doi.org/10.1001/jama.295.1.39> PMID: 16391215
22. Howard BV, Van Horn L, Hsia J, Manson JE, Stefanick ML, Wassertheil-Smoller S, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *Jama*. 2006; 295(6):655–66. <https://doi.org/10.1001/jama.295.6.655> PMID: 16467234
23. Belin RJ, Greenland P, Allison M, Martin L, Shikany JM, Larson J, et al. Diet quality and the risk of cardiovascular disease: the Women's Health Initiative (WHI). *The American journal of clinical nutrition*. 2011;ajcn. 011221.
24. Hess KR. Graphical methods for assessing violations of the proportional hazards assumption in Cox regression. *Statistics in medicine*. 1995; 14(15):1707–23. <https://doi.org/10.1002/sim.4780141510> PMID: 7481205
25. Hosmer DW, Lemeshow S. Confidence interval estimation of interaction. *Epidemiology*. 1992; 3(5):452–6. <https://doi.org/10.1097/00001648-199209000-00012> PMID: 1391139
26. Brown WJ, Williams L, Ford JH, Ball K, Dobson AJ. Identifying the energy gap: magnitude and determinants of 5-year weight gain in midage women. *Obesity research*. 2005; 13(8):1431–41. <https://doi.org/10.1038/oby.2005.173> PMID: 16129726
27. Locke AE, Kahali B, Berndt SI, Justice AE, Pers TH, Day FR, et al. Genetic studies of body mass index yield new insights for obesity biology. *Nature*. 2015; 518(7538):197–206. <https://doi.org/10.1038/nature14177> PMID: 25673413
28. Berman DM, Rodrigues LM, Nicklas BJ, Ryan AS, Dennis KE, Goldberg AP. Racial Disparities in Metabolism, Central Obesity, and Sex Hormone-Binding Globulin in Postmenopausal Women 1. *The Journal of Clinical Endocrinology & Metabolism*. 2001; 86(1):97–103.
29. Kahn HS, Williamson DF, Stevens JA. Race and weight change in US women: the roles of socioeconomic and marital status. *American Journal of Public Health*. 1991; 81(3):319–23. <https://doi.org/10.2105/ajph.81.3.319> PMID: 2036117
30. Larson NI, Story MT, Nelson MC. Neighborhood environments: disparities in access to healthy foods in the US. *American journal of preventive medicine*. 2009; 36(1):74–81. e10.

31. Taveras EM, Gillman MW, Kleinman K, Rich-Edwards JW, Rifas-Shiman SL. Racial/ethnic differences in early-life risk factors for childhood obesity. *Pediatrics*. 2010; 125(4):686–95. <https://doi.org/10.1542/peds.2009-2100> PMID: 20194284
32. Bower JF, Davis JM, Hao E, Barakat HA. Differences in transport of fatty acids and expression of fatty acid transporting proteins in adipose tissue of obese black and white women. *American Journal of Physiology-Endocrinology and Metabolism*. 2006; 290(1):E87–E91. <https://doi.org/10.1152/ajpendo.00194.2005> PMID: 16339926
33. Barakat H, Davis J, Lang D, Mustafa SJ, McConaughy MM. Differences in the Expression of the Adenosine A1 Receptor in Adipose Tissue of Obese Black and White Women. *The Journal of Clinical Endocrinology & Metabolism*. 2006; 91(5):1882–6. <https://doi.org/10.1210/jc.2005-2109> PMID: 16507638
34. Luke A, Dugas L, Kramer H. Ethnicity, energy expenditure and obesity: are the observed black/white differences meaningful? *Current Opinion in Endocrinology, Diabetes and Obesity*. 2007; 14(5):370–3.
35. Bennett GG, Wolin KY. Satisfied or unaware? Racial differences in perceived weight status. *International Journal of Behavioral Nutrition and Physical Activity*. 2006; 3(1):40.
36. Burke GL, Bild DE, Hilner JE, Folsom AR, Wagenknecht LE, Sidney S. Differences in weight gain in relation to race, gender, age and education in young adults: the CARDIA study. *Ethnicity & Health*. 1996; 1(4):327–35. <https://doi.org/10.1080/13557858.1996.9961802> PMID: 9395577
37. Ball K, Crawford D. Socioeconomic status and weight change in adults: a review. *Social Science & Medicine*. 2005; 60(9):1987–2010. <https://doi.org/10.1016/j.socscimed.2004.08.056> PMID: 15743649
38. Jakicic JM, Powell KE, Campbell WW, Di Pietro L, Pate RR, Pescatello LS, et al. Physical activity and the prevention of weight gain in adults: a systematic review. *Medicine & Science in Sports & Exercise*. 2019; 51(6):1262–9. <https://doi.org/10.1249/MSS.0000000000001938> PMID: 31095083
39. Walker RE, Keane CR, Burke JG. Disparities and access to healthy food in the United States: A review of food deserts literature. *Health & place*. 2010; 16(5):876–84. <https://doi.org/10.1016/j.healthplace.2010.04.013> PMID: 20462784
40. Lim S, Harris TG. Neighborhood contributions to racial and ethnic disparities in obesity among New York City adults. *Am J Public Health*. 2015; 105(1):159–65. <https://doi.org/10.2105/AJPH.2013.301782> PMID: 24625176
41. Lewis CE, Smith DE, Wallace DD, Williams OD, Bild DE, Jacobs DR Jr. Seven-year trends in body weight and associations with lifestyle and behavioral characteristics in black and white young adults: the CARDIA study. *Am J Public Health*. 1997; 87(4):635–42. <https://doi.org/10.2105/ajph.87.4.635> PMID: 9146444
42. Insaf TZ, Shaw BA, Yucel RM, Chasan-Taber L, Strogatz DS. Lifecourse Socioeconomic Position and Racial Disparities in BMI Trajectories Among Black and White Women: Exploring Cohort Effects in the Americans' Changing Lives Study. *Journal of racial and ethnic health disparities*. 2014; 1(4):309–18. <https://doi.org/10.1007/s40615-014-0038-y> PMID: 25506543
43. He XZ, Meng H. Changes in weight among US adults aged 70 and over, 1993 to 2002. *Preventive medicine*. 2008; 47(5):489–93. <https://doi.org/10.1016/j.yjmed.2008.06.022> PMID: 18671999
44. Subar AF, Kipnis V, Troiano RP, Midthune D, Schoeller DA, Bingham S, et al. Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: the OPEN study. *American journal of epidemiology*. 2003; 158(1):1–13. <https://doi.org/10.1093/aje/kwg092> PMID: 12835280
45. Willett WC. Recall of remote diet. *Nutritional Epidemiology*. Third ed. New York, NY: Oxford University Press; 2013. p. 142–9.
46. Willett WC. Food Frequency Methods. *Nutritional Epidemiology*. Third ed. New York, NY: Oxford University Press; 2013. p. 142–9.
47. Patterson RE, Kristal AR, Tinker LF, Carter RA, Bolton MP, Agurs-Collins T. Measurement characteristics of the Women's Health Initiative food frequency questionnaire. *Annals of epidemiology*. 1999; 9(3):178–87. [https://doi.org/10.1016/s1047-2797\(98\)00055-6](https://doi.org/10.1016/s1047-2797(98)00055-6) PMID: 10192650
48. Palta M, Prineas RJ, Berman R, Hannan P. Comparison of self-reported and measured height and weight. *American journal of epidemiology*. 1982; 115(2):223–30. <https://doi.org/10.1093/oxfordjournals.aje.a113294> PMID: 7058781
49. Rowland ML. Self-reported weight and height. *The American journal of clinical nutrition*. 1990; 52(6):1125–33. <https://doi.org/10.1093/ajcn/52.6.1125> PMID: 2239790
50. Reynolds RF, Obermeyer CM. Age at natural menopause in Spain and the United States: Results from the DAMES project. *American Journal of Human Biology*. 2005; 17(3):331–40. <https://doi.org/10.1002/ajhb.20121> PMID: 15849704
51. Wing RR, Phelan S. Long-term weight loss maintenance—. *The American journal of clinical nutrition*. 2005; 82(1):222S–5S.