The Association of Multiple Anthropometrics of Overweight and Obesity with Incident Heart Failure: The Atherosclerosis Risk in Communities (ARIC) Study

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Abstract

Background—The association of central adiposity with incident heart failure (HF) has yet to be studied in a large population-based study.

Methods and Results—The Atherosclerosis Risk in Communities (ARIC) study is an ongoing bi-racial population-based cohort of those aged 45–65 years from 4 U.S. communities with 16 years median follow-up for incident, hospitalized or fatal HF. Waist-hip ratio (WHR), waist circumference and body mass index (BMI) were measured baseline (1987–1989). After exclusions the sample size was 14,641. BMI was categorized as BMI < 25, BMI 25–29.9, and BMI ≥ 30 (kg/m^2). Waist circumference and WHR were divided into gender-specific tertiles. A first occurrence of ICD-9-CM codes of HF, either hospital discharge (428.0–428.9, N=1,451), or on a death certificate (428.0–428.9 or I50.0–I50.9, N=77) was considered an HF event. Cox models were adjusted for alcohol use, smoking, age, center, and educational level. The adjusted hazard ratios (HR) for the highest category (obese) compared to the lowest were well above 1.0 for all three anthropometric measures (HR for 3rd vs. 1st tertile of WHR: 2.27 (1.71, 3.02), white women; 3.24 (2.25, 4.65); black women; 2.46 (1.95, 3.09), white men; and 2.63 (1.90, 3.65), black men). Hazard ratios for overweight were lower in magnitude suggesting a graded response between body size and HF.

Conclusions—Obesity and overweight, as measured by three different anthropometrics, were associated with incident HF in the ARIC cohort. The current study does not support the superiority of WHR and waist circumference over BMI for the prediction of incident HF.

Keywords

epidemiology; heart failure; obesity

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1. Introduction

Obesity, as measured by body mass index (BMI), has been identified as a risk factor for heart failure (HF) from the Framingham Heart Study[1] and other studies[2, 3]. The increasing prevalence of both obesity and HF in the U.S. make this association an important topic for prevention[4]. Replication of the association of obesity with HF in non-white racial groups has been mentioned as an important area for future research[5].

Furthermore, research on the association of measures of central adiposity with HF has been mentioned as a priority research area[6]. Because diabetes and insulin resistance are risk factors for HF[7, 8], one would expect that a measure of central adiposity, a correlate of impaired insulin sensitivity, would have a stronger association with incident HF than a measure of generalized adiposity. Waist circumference is highly correlated with BMI and is therefore considered a measure of both central and generalized adiposity, whereas waist-hip ratio (WHR) is a measure of central adiposity only[9]. Existing studies imply that waist circumference and WHR are also associated with incident HF[8, 10, 11], however results vary depending on the population under study.

The Atherosclerosis Risk in Communities (ARIC) study is a bi-racial longitudinal cohort study of cardiovascular disease and its risk factors in those aged 45–65 years. It has anthropometric measures of BMI, WHR, and waist circumference from the baseline visit along with 16 years median follow-up for incident HF. In this study, we evaluated the race- and gender-specific associations of overweight and obesity with incident HF. Furthermore, we determined whether a measure of central adiposity (WHR) or a measure of generalized and central adiposity (waist circumference) would be more closely associated with incident HF than BMI.

2. Methods

Study Population

The ARIC cohort (N=15,792) was recruited between 1987–1989 using probability sampling of those aged 45–64 years from the following four US communities: Forsyth County, North Carolina; Jackson, Mississippi; suburbs of Minneapolis, Minnesota; and Washington County, Maryland. The distribution of blacks and whites from each field center was representative of the area (mostly white in Minneapolis and Washington County, 15 % black in Forsyth County), except in Jackson where only blacks were sampled. Response rates at baseline were 46 % in Jackson and between 65–67 % for the other communities. Three subsequent visits were conducted at approximately three-year intervals **(last visit in** 1996–98). The design and rationale of the ARIC study [12] and a comparison of responders and nonresponders [13] have been previously published. The institutional review boards from each site approved the ARIC study. All participants provided written informed consent.

Racial groups not classified as white or black (N = 48), and blacks not from Jackson or Forsyth County (N = 120) were excluded in this study due to their limited numbers. Those with missing anthropometry (N = 33), prevalent HF at baseline (N=751), or missing criteria used to define prevalent HF (N=289) were excluded. Criteria to define prevalent HF were as follows: 1) those answering "yes" to the following question: "Were any of the medications you took during the last two weeks for HF?" (N = 83), or 2) those with stage 3 HF by applying Gothenburg criteria (N = 699)[14]. Gothenburg criteria were defined by self-reported medical history, medication lists and electrocardiography[15]. After these exclusions, the total sample size was 14,641.

Anthropometric Measures

Anthropometric indices were measured after an overnight fast with participants in standard scrub attire. Technicians measured height with participants barefoot using a wall mounted ruler.

An anthropometric measuring tape was applied horizontally to measure hip and abdominal girth; participants stood upright with weight evenly distributed between both feet and breathing quietly. Abdominal girth was measured at the level of the umbilicus and hip girth at the level of maximal protrusion of the gluteal muscles. Weight was measured using a scale (Detecto model 437) that was zeroed daily and calibrated quarterly. Body mass index (BMI) was calculated as weight divided by height squared (kilograms/meters^2), whereas WHR was the waist girth divided by the hip girth. Inter-technician reliability coefficients for waist and hip girth and WHR were >0.91[16].

Ascertainment of heart failure events

The following methods were used for ascertainment of HF events: 1) participants were interviewed annually by phone about interim hospitalizations (93–96 % response); 2) local hospitals provided lists of hospital discharges with cardiovascular diagnoses and these were reviewed to identify cohort hospitalizations; and 3) health department death certificate files were continuously surveyed. All discharge diagnosis codes for cohort hospitalizations and underlying or contributory causes of death from death certificates were recorded.

Incident heart failure event criteria

Incident HF was defined as the first occurrence of either: 1) a hospitalization which included an ICD-9-CM (International Classification of Diseases, 9th revision, clinical modification) discharge diagnosis code for HF beginning with `428' (i.e. 428.0 to 428.9) in any position (N = 1,451) or else 2) a death certificate ICD-9 code beginning with `428' (HF) or ICD-10 code `I50' (HF or I50.0 to I50.9) in any position (N = 77). Follow-up time for incident HF events was defined as the time from their baseline examination until the incident event. The date of censoring for those without HF was the first occurrence of either: date of last contact or death, or December 31st, 2004. Secondary analyses inclusive of a broader range of HF ICD-9 codes resulted in only 139 additional events. These codes were as follows: rheumatic heart failure (398.91), hypertensive heart disease (402.01, 402.11, 402.91), cardiomyopathy (425), acute edema of the lung (518.4) and cardiac failure postoperatively (997.1).

Baseline covariate definitions

All covariates were collected from the baseline visit. Race was self-reported; educational level, alcohol use, and smoking status were obtained with interviewer-administered questionnaires. History of myocardial infarction (MI) included self-report of physician-diagnosed MI or silent MI identified by electrocardiography. Past coronary heart disease (CHD) included a history of MI, coronary revascularization or coronary artery bypass surgery.

Hypertension was defined by either a diastolic blood pressure ≥ 90 mm Hg or a systolic blood pressure ≥ 140 mm Hg measured with random-zero mercury manometers, or recent anti-hypertensive medication use. Left ventricular hypertrophy was identified by electrocardiography using Cornell criteria [17]. Diabetes mellitus was defined as either: self-reported physician-diagnosed diabetes, recent diabetes medication use, a blood glucose ≥ 126 mg/dl fasting or ≥ 200 mg/dl non-fasting. Cholesterol measurements were performed after an overnight fast[12].

Statistical Analysis

Pearson correlation coefficients were estimated to determine the correlation between BMI, WHR and waist circumference. Multivariable Cox proportional hazard regression was used to model the association between each anthropometric variable (BMI, WHR and waist circumference) and time to incident HF, after stratification by race and gender. Waist circumference, BMI and WHR were analyzed as both continuous and categorized variables.

BMI was categorized as normal weight (< 25 kg/m^2), overweight ($25 - 29.9$ kg/m^2) and obese (≥ 30 kg/m^2), as represented in the clinical guidelines from the National Institutes of Health[18]. Waist circumference and WHR were categorized into approximate gender-specific tertiles[11]. Furthermore, BMI categories of normal weight, overweight and obese, were stratified by low and high WHR using gender-specific cutpoints from the U.S. Department of Agriculture (WHR cutpoints: women, 0.88; men, 0.95)[19]. **For comparability between sex and race-groups**, HF incidence rates were age-adjusted to the mean age at baseline (54 years) with Poisson models. Log (-log) survival curves and time interaction terms for the main exposures and all covariates were used to **verify** the proportional hazards assumption.

The predictive ability of each anthropometric variable for incident HF was compared using time-dependent receiver operating characteristic (ROC) curves for estimation of area under the curve at 10 years (AUC(10)), as described by Chambless and Diao[20]. We determined the AUC(10) for each anthropometric measure (categorized) based on predicted probabilities estimated from gender-stratified multivariable Cox proportional hazards models. Predicted probabilities at 10 years were divided into deciles and each compared to the lowest group. Goodness of fit was assessed using Hosmer-Lemeshow tests. Furthermore, we adjusted for optimism due to use of the same dataset for determination of AUC(10) for both models being compared[21].

To assess for additive effect measure modification by race, interaction contrast ratios[22] (ICR) with 95 % **CIs** were calculated. A probabilistic sensitivity analysis was performed to assess the impact of systematic error from nondifferential outcome misclassification. A method developed by Lash and Fink[23] was used to incorporate uncertainty from outcome misclassification into traditional regression using Monte Carlo uncertainty analysis. All statistical analyses were performed using SAS software v 9.1 (Cary, NC).

The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

3. Results

Those who developed HF were older at baseline, more likely to be black, male, and less well educated (Table 1). As would be expected, there was a higher percentage of CHD and cardiovascular risk factors among HF events. Waist circumference and WHR cutpoints for men were higher than they were for women (Table 2). The mean value for each anthropometric by anthropometric category was surprisingly similar by gender and across race. For all race and gender groups, age-adjusted HF incidence rates were two-to three-fold higher when comparing the lowest group to the highest for all three measures (Table 2). Across all three anthropometric exposures, HF incidence rates were higher in blacks compared to whites for both men and women. The HF incidence rates in black women compared to white women were nearly two-fold higher for all measures.

Adjusted hazard ratios for the comparison of the highest anthropometric category (obese) to the lowest for all three measures were well above 1.0 for all race and gender groups (Table 3, Figure 1). In fact, when combined across race, the adjusted hazard ratios for HF with obesity were all well above 2.0. Hazard ratios for the middle category (overweight) for all three anthropometric measures tended to be intermediate, thus suggesting a graded response between body size and HF (Table 3). Calculation of interaction contrast ratios and interaction term analysis ($P > 0.05$) did not provide evidence for additive or multiplicative effect measure modification by race. Although we did not find such support for differences by race, for completeness, we present race- and gender-stratified estimates of the association of HF and adiposity.

Multivariable models of each anthropometric measure in continuous form showed positive associations for all race and gender groups (Table 3). For better comparability across measures, a one standard deviation (SD) increment (gender-specific) in each baseline anthropometric was modeled, rather than a one unit change in each measure. After adjustment for confounding factors, women with a 1 SD (0.08) higher WHR were 59 % more likely to develop incident HF over the next 16 years, whereas men with a 1 SD higher WHR (0.05) were 50 % more likely to develop incident HF. Results were similar for BMI and waist circumference. To refute the hypothesis that obesity at baseline was due to fluid retention from HF, we excluded the first three years of follow-up and found little change in the associations examined here (results not shown).

Waist circumference and BMI were highly correlated (r = 0.88), WHR and waist circumference were moderately correlated (r= 0.73), and BMI and WHR were **relatively** poorly correlated (r = 0.43). Stratification of BMI by waist circumference resulted in small sample sizes for certain categories; instead we describe HF incidence rates for BMI categories, stratified by high and low WHR. Age-adjusted HF incidence rates for categories of BMI, stratified by high and low WHR show an increasing trend across these categories, with a particularly high HF incident rate (10.5 per 1,000 person-years) in those with both a high BMI and WHR (Figure 2).

Estimation of AUC(10) from predicted probabilities from multivariable models for each anthropometric were as follows: BMI (0.73 for men, 0.79 for women); WHR (0.74 for men, 0.79 for women); waist circumference (0.73 for men, 0.79 for women); and BMI stratified by high and low WHR (0.74 for men, 0.79 for women). By gender, there were no **pronounced** differences in AUC(10) between the three measures, or when compared to models with cross-classified categories of BMI and WHR (P>0.05 for all comparisons). Results were similar when stratified by race and gender. Of note, in some cases, the Hosmer-Lemeshow tests did not support good model fit.

The sensitivity analysis assessed the effect of outcome misclassification on the multivariable association between obesity (BMI ≥ 30) and HF. Misclassification was assumed nondifferential, meaning it did not depend on values of other variables (namely, BMI) or on errors in measuring them. The selected distributions of the sensitivity parameters for misclassification of HF were initially based on findings from the literature; subsequently, the specified distributions required modification to allow for selection of plausible combinations of the sensitivity parameters (sensitivity: minimum =0.6, mode 1 =0.7, mode 2 =0.85, maximum =1; for specificity, minimum =0.94, mode 1 =0.96, maximum=1)[24]. The sensitivity analysis results show the median odds ratio (OR) was higher and the 2.5[th] and 97.5[th] percentiles much farther apart (OR =4.54, 95 % uncertainty interval =2.93, 14.83) than the results of the conventional analysis (OR =2.89, 95 % CI =2.47, 3.42), which assumes with complete certainty that outcome classification is perfect. As expected, this suggests that the effect of outcome misclassification on our findings, given the chosen distributions of sensitivity and specificity, was to bias them towards the null, and that the assumption of perfect outcome classification in the conventional analysis understates the actual uncertainty about the true value of the OR. In secondary analyses using the expanded definition of HF (inclusive of a broader range of ICD codes), there were no **appreciable** differences in any of our findings (**results** not shown).

4. Discussion

Generalized obesity and central adiposity, as measured by three different anthropometric measures, were associated with incident HF over16 years median follow-up of the ARIC cohort. The magnitudes and patterns of the associations were similar for all three measures and there was evidence of a graded relation for all race, gender groups. Furthermore, an adverse association existed between adiposity and incident HF even for those who were overweight

compared to normal weight, although **this finding was** less consistent across stratified analyses. Analyses of ROC curves found no **important** differences in the prediction of incident HF for the three measures. This implies that measures of WHR and waist circumference are not superior to BMI in the prediction of HF. This is contrary to our hypothesis that measures of central adiposity will be more closely associated with HF due to their closer association with diabetes, a known HF risk factor. This suggests that beyond the metabolic derangements associated with central adiposity, other mechanisms associated with generalized adiposity, must play a role in mediating the relationship between obesity and HF.

Several previous studies with BMI measures have found similar associations to those observed here. The Framingham Heart Study found that overweight and obesity as measured by BMI were associated with an increased risk for HF over 14 years median follow-up; multivariable adjusted hazard ratios were 1.34 (95 % CI = 1.08, 1.67) and 2.04 (95 % CI =1.59, 2.63), respectively[1]. In these models, the Framingham study adjusted for diseases along the causal pathway between obesity to HF. As they note, adjustment for factors along the causal pathway may underestimate the effect of adiposity with HF. Study differences between the Framingham study and ours include that the outcome of HF from the Framingham study was validated using Framingham criteria. The Framingham Heart Study is a primarily white population from a single community that is approximately a third the size of the ARIC study. Since the ARIC study included blacks and whites from 4 US communities, one might expect to find more heterogeneity in the findings between these two studies. Instead the robustness of these findings supports a lack of differences in this association by race.

The Renfrew-Paisley study, a community-based study from Scotland, observed an association of obesity (as defined by BMI) with HF (HR = 2.16, 95 % CI =1.57, 2.57 for men; HR = 1.37, 95 % CI =1.00, 1.88 for women)[3]. The First National Health and Nutrition Examination Survey (NHANES I) studied the association of excess BMI and incident HF2, using a dichotomous cut-point for BMI (27.8 kg/m^2 in men, 27.3 kg/m^2 in women); their findings were similar to that observed in our overweight group despite the differing cutpoints used.

To date, studies of central obesity and HF have involved less generalizable populations. For example, the Health, Aging and Body Composition Study (Health ABC) is a longitudinal cohort study of those aged 70–79 without CHD, in which multiple metrics of overweight/obesity and body composition were measured10. This study had a limited number of HF events (N = 166), in part due to the exclusion of those with CHD, a common HF precursor25. Despite the small sample size, BMI, waist circumference, and waist/thigh ratio were all positively associated with HF incidence. In contrast, a study of participants with CVD from the Heart Outcomes Prevention Evaluation (HOPE) study did not find an association between obesity and incident HF (N=297 HF events) for BMI or WHR, except there seemed an effect for WHR in women (HR = 2.30, 95 % CI = 1.25, 4.21) 11.

We also describe risk groups using the cross-classification of two anthropometric measures, BMI and WHR. We find that incidence rates increase across categories of BMI stratified by WHR, however the ROC analysis does not support better prediction of incident HF with this cross-classification of BMI and WHR as compared to these measures alone.

Further support for the association of obesity with HF comes from echocardiographic studies. Specifically, results from the Framingham Heart Study found positive correlations between obesity (BMI > 30 kg/m^2) and echocardiographic measures of left ventricular mass, which were also associated with increase in left ventricular internal dimensions, and wall thickness[26]. A small study in normotensive men reported stronger positive correlations of left ventricular mass with WHR and waist circumference as compared to BMI [27]. Recently, McGavock **and others** found that cardiac steatosis as seen with magnetic resonance

spectroscopy occurs with impaired glucose tolerance even before the development of type 2 diabetes mellitus and/or left ventricular dysfunction[28].

Obesity's role in the development of HF may be through either direct and/or indirect mechanisms. The indirect mechanisms are those in which obesity causes other diseases, such as diabetes[29], hypertension[30] or CHD, which are themselves risk factors for HF[25]. A direct mechanism might be that cardiac adaptation to excess body fat can result in decreased cardiac function[31]. This has been termed obesity cardiomyopathy[32]. A novel hypothesis is that an increase in inflammatory cytokines from excess adipocytes may increase risk of HF[32, 33]. Also, several mechanisms from animal models have been proposed for the cardiotoxic effect of fat cells[34]. However, true obesity cardiomyopathy is uncommon, and usually occurs in cases of extreme obesity (BMI ≥ 40 kg/m^2) of greater than 10 years duration.

The main limitation of this study is our definition of HF. We included hospitalized and fatal HF, as we did not have data on outpatient HF; however, community surveillance reports have indicated that 74 % of outpatient HF cases are hospitalized within 1.7 years[35]. To address this limitation, we performed a sensitivity analysis to explore the effect of nondifferential outcome misclassification on our findings. As would be expected with outcome misclassification, we found it should bias our findings toward the null and increase our uncertainty about the true magnitude of the association.

This study is relevant and important to the understanding of the etiology of HF for three reasons. First, the ARIC study is the largest population-based cohort study to evaluate the association between WHR and waist circumference and incident HF. Furthermore, this is a well characterized cohort with a long period of follow-up for which standardized methodology was used. Because it is a large biracial study, we were able to describe this association stratified by race and gender.

In conclusion, we observed that obesity was associated with incident HF and there was a graded relation with body size. This association did not vary by race or gender. Selecting the best anthropometric for the prediction of HF could have implications for the screening and prevention of HF. The current study does not support the superiority of WHR or waist circumference over BMI for the prediction of HF.

> This is an important contribution to the literature on heart failure from a large population-based bi-ethnic cohort study with a median of 16 years of follow-up for incident heart failure. This paper adds to the existing literature by providing estimates for the association of adiposity, as measured by three different anthropometric measures (body mass index, waist hip ratio and waist circumference) with incident heart failure for African-Americans as well as Whites. In most clinical settings, body mass index (BMI) is the sole adiposity metric that is measured. This study evaluated whether these other measures were superior to BMI for the determination of HF risk. In fact, our findings do not support waist circumference, waist-hip ratio, or even further stratification of BMI into high and low categories of waist hip ratio, as superior to BMI for the purpose of determining HF risk. Therefore, the potential clinical impact is that measurement of waist circumference or waist hip ratio is not necessary to improve determination of HF risk. This was true for both races (Whites and African-Americans) and across gender.

Acknowledgments

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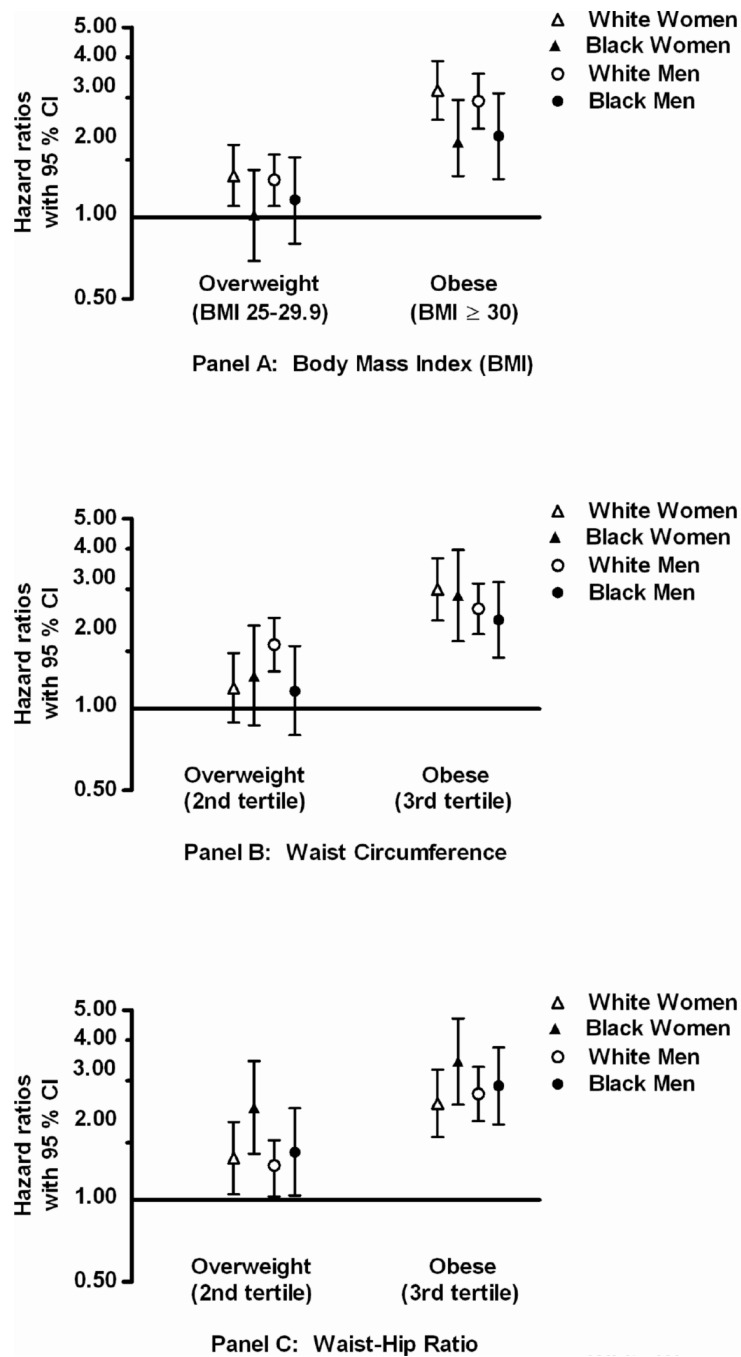
Figure 1.
Graphical representation of race- and gender-stratified adjusted hazard ratios (95 % CI), for overweight and obese as compared to referent* as measured by BMI (Panel A), waist circumference (Panel B), and waist-hip ratio (Panel C), ARIC 1987-2004
*Referent categories were as follows: BMI, < 25; WHR, 1st tertile WHR; and waist circumference, 1st tertile for waist circumference

Figure 2.
Age-adjusted (to mean age at baseline, 54 years) heart failure incidence rates (per 1,000) person-years by categories of body mass index (BMI), stratified by waist-hip ratio (WHR), (0.88 for women, 0.95 for men), ARIC 1987–2004

Table 1

Characteristics at baseline (1987–1989) of those who did or did not develop heart failure, ARIC

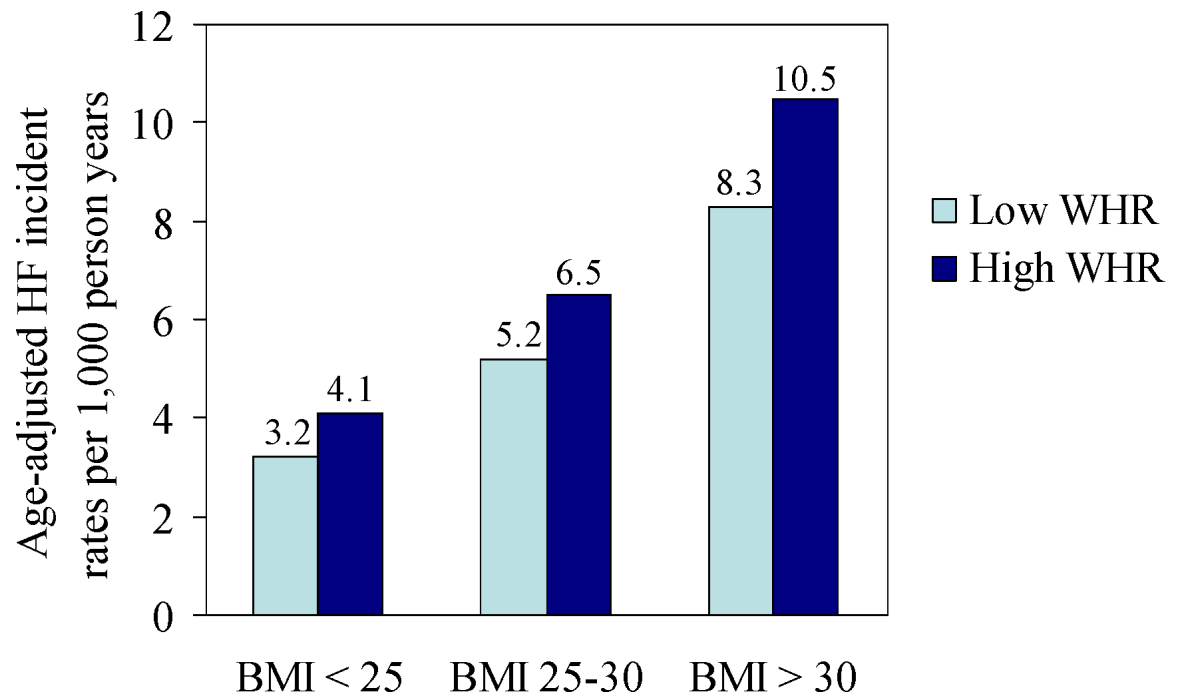| Characteristics	Incident Heart Failure (N = 1,528)	Non-cases (N = 13,113)
Age, mean (SD), years	56.8 (5.4)	53.8 (5.7)
Men, %	54	44
Black, %	34	25
Center, %		
Jackson, MS	31	22
Forsyth County, NC	24	26
Minneapolis, MN	17	27
Washington County, MD	28	25
Educational level, %		
Less than high school	40	21
High school graduate	30	33
Greater than high school	30	46
Smoking status, %		
Never smoker	30	43
Former smoker	33	32
Current smoker	37	25
Alcohol use, %		
Never drinker	27	25
Former drinker	28	17
Current drinker	45	58
Diabetes, %	31	9
Hypertension, %	54	30
Left ventricular hypertrophy by ECG, %	7	2
History of coronary heart disease, %	14	3
History of myocardial infarction, %	13	2
Plasma lipids, mean (SD), mmol/L		
Total cholesterol	5.70 (1.17)	5.55 (1.07)
Low density lipoprotein cholesterol, (LDL-C)	3.71 (1.06)	3.55 (1.01)
High density lipoprotein cholesterol, (HDL-C)	1.20 (0.41)	1.36 (0.44)
Triglycerides	1.79 (1.34)	1.43 (0.95)
Anthropometric variables, mean (SD)		
Body mass index (BMI), kg/m^2	29.7 (6.2)	27.3 (5.1)
Waist circumference, cm	103.4 (15.0)	96.0 (13.3)
Waist-hip ratio (WHR)	0.96 (0.07)	0.92 (0.08)

Table 2

Number of incident heart failure events, person-years of follow-up and age-adjusted* incidence rates (IR) for heart failure by category of each anthropometric (BMI, waist circumference, and waist hip ratio), stratified by race and gender, ARIC, 1987–2004

	White Women (N = 5,659)				Black Women (N = 2,350)			
	Mean Value	Total person-years, N (%)	Heart failure Events (N)	Age-adjusted* heart failure IR (95 % CI) per 1,000 person years	Mean Value	Total person-years, N (%)	Heart failure Events (N)	Age-adjusted* heart failure IR (95 % CI) per 1,000 person years
BMI, kg/m²								
<25	22.2	41,935 (48)	119	2.40 (1.98, 2.92)	22.7	6,074 (18)	40	5.21 (3.83, 7.08)
25–30	27.2	26,734 (31)	112	4.17 (3.69, 4.72)	27.5	12,058 (35)	82	7.69 (6.55, 9.04)
≥ 30	34.4	18,738 (21)	163	7.25 (6.10, 8.61)	35.8	15,960 (47)	192	11.37 (9.66, 13.39)
Waist circumference, cm								
1st Tertile, < 87	78.9	33,743 (39)	84	2.07 (1.64, 2.62)	79.4	7,164 (21)	32	4.15 (2.97, 5.80)
2nd Tertile, 87 < 100	92.6	29,767 (34)	101	3.79 (3.30, 4.34)	93.2	11,281 (33)	74	7.16 (6.02, 8.52)
3rd Tertile, ≥ 100	111.2	23,899 (27)	209	6.92 (5.83, 8.21)	113.4	15,647 (46)	208	12.37 (10.51, 14.55)
Waist hip ratio								
1st Tertile, < 0.86	0.80	32,321 (37)	69	2.20 (1.81, 2.67)	0.80	10,749 (32)	37	4.29 (3.20, 5.76)
2nd Tertile, 0.86<0.93	0.89	27,416 (31)	109	3.73 (3.33, 4.18)	0.89	9,880 (29)	85	7.59 (6.49, 8.89)
3rd Tertile, ≥ 0.93	0.98	27,671 (32)	216	6.33 (5.51, 7.29)	0.98	13,463 (39)	192	13.44 (11.39, 15.85)
BMI, kg/m²								
<25	23.1	20,655 (28)	123	4.06 (3.45, 4.78)	22.4	6,070 (30)	51	6.95 (5.45, 8.87)
25–30	27.3	38,677 (52)	278	6.36 (5.77, 7.01)	27.4	8,885 (43)	78	9.81 (8.54, 11.26)
≥ 30	33.0	15,759 (21)	207	9.96 (8.69, 11.41)	33.6	5,530 (27)	83	13.83 (11.34, 16.88)
Waist Circumference, cm								
1st Tertile, < 95	88.9	24,832 (33)	122	4.19 (3.36, 4.94)	86.2	9,440 (46)	71	6.84 (5.39, 8.67)
2nd Tertile, 95–103	98.4	25,009 (33)	204	6.17 (5.56, 6.83)	98.5	5,372 (26)	48	9.81 (8.44, 11.41)

	White Women (N = 5,659)				Black Women (N = 2,350)			
	Mean Value	Total person-years, N (%)	Heart failure Events (N)	Age-adjusted* heart failure IR (95 % CI) per 1,000 person years	Mean Value	Total person-years, N (%)	Heart failure Events (N)	Age-adjusted* heart failure IR (95 % CI) per 1,000 person years
3rd Tertile, >103	110.4	25,251 (34)	282	9.07 (8.00, 10.28)	111.8	5,673 (28)	93	14.01 (11.34, 17.46)
Waist-Hip Ratio								
1st Tertile, < 0.94	0.91	22,209 (30)	92	3.37 (2.81, 4.05)	0.90	10,749 (52)	69	6.50 (5.09, 8.29)
2nd Tertile, 0.94<0.98	0.96	22,662 (30)	142	5.64 (5.07, 6.28)	0.96	5,112 (25)	53	10.60 (9.07, 12.38)
3rd Tertile, ≥ 0.98	1.02	30,221 (40)	374	9.44 (8.42, 10.59)	1.01	4,625 (23)	90	17.29 (13.77, 21.72)

*
age-adjusted to mean age at baseline, 54 years

Table 3

Multivariable adjusted [*] hazard ratios (with 95 % CI) for incident heart failure by body mass index, waist circumference and waist-hip ratio, stratified by race and gender, ARIC, 1987–2004

	Women				Men		
	White women HR (95 % CI)	Black women HR (95 % CI)	All women HR (95 % CI)	White men HR (95 % CI)	Black men HR (95 % CI)	All men HR (95 % CI)
Body Mass Index						
Overweight vs. normal	1.42 (1.10, 1.85)	1.02 (0.69, 1.49)	1.31 (1.05, 1.62)	1.37 (1.10, 1.70)	1.16 (0.80, 1.66)	1.32 (1.10, 1.59)
Obese vs. normal	2.93 (2.28, 3.75)	1.89 (1.33, 2.70)	2.56 (2.09, 3.14)	2.67 (2.12, 3.37)	1.99 (1.38, 2.86)	2.49 (2.05, 3.02)
Continuous, 1 SD change[†]	1.64 (1.49, 1.82)	1.36 (1.24, 1.50)	1.49 (1.39, 1.59)	1.55 (1.44, 1.67)	1.34 (1.20, 1.49)	1.47 (1.39, 1.57)
Waist Circumference						
2nd Tertile vs. 1st tertile	1.19 (0.89, 1.60)	1.32 (0.87, 2.02)	1.24 (0.98, 1.58)	1.72 (1.37, 2.16)	1.16 (0.80, 1.70)	1.56 (1.29, 1.88)
3rd tertile vs. 1st tertile	2.75 (2.11, 3.58)	2.61 (1.77, 3.83)	2.68 (2.16, 3.33)	2.33 (1.88, 2.89)	2.12 (1.54, 2.92)	2.25 (1.89, 2.69)
Continuous, 1 SD change[†]	1.61 (1.46, 1.77)	1.47 (1.33, 1.63)	1.54 (1.44, 1.66)	1.60 (1.48, 1.73)	1.41 (1.27, 1.56)	1.52 (1.43, 1.62)
Waist-Hip Ratio						
2nd Tertile vs. 1st tertile	1.43 (1.05, 1.94)	2.19 (1.48, 3.25)	1.70 (1.34, 2.16)	1.34 (1.03, 1.75)	1.50 (1.04, 2.18)	1.38 (1.12, 1.71)
3rd tertile vs. 1st tertile	2.27 (1.71, 3.02)	3.24 (2.25, 4.65)	2.63 (2.11, 3.28)	2.46 (1.95, 3.09)	2.63 (1.90, 3.65)	2.50 (2.08, 3.02)
Continuous, 1 SD change[†]	1.56 (1.35, 1.68)	1.67 (1.48, 1.89)	1.59 (1.46, 1.72)	1.50 (1.40, 1.61)	1.51 (1.33, 1.71)	1.50 (1.41, 1.60)

[*] All models adjusted for age, alcohol use, educational level, smoking status, and center. Models not stratified by race were also adjusted for race using a combined race and center variable. Models exclude 243 (1.7%) participants with missing covariates.

[†] 1 standard deviation (SD) change for BMI = 6 kg/m^2, women; 4.2 kg/m^2, men; 1 SD waist circumference = 15.4 cm, women; 10.9 cm for men; 1 SD change for WHR = 0.08 women; 0.05 for men