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Physical Activity, Measures of Obesity, and Cardiometabolic Risk: The Multi-Ethnic Study of Atherosclerosis (MESA)

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Abstract

Background—The influence of higher physical activity on the relationship between adiposity and cardiometabolic risk is not completely understood.

Methods—Between 2000–2002, data were collected on 6795 Multi-Ethnic Study of Atherosclerosis (MESA) participants. Self-reported intentional physical activity in the lowest quartile (0–105 MET-minutes/week) was categorized as inactive and the upper three quartiles (123–37,260 MET-minutes/week) as active. Associations of body mass index (BMI) and waist circumference categories, stratified by physical activity status (inactive or active) with cardiometabolic risk factors (dyslipidemia, hypertension, upper quartile of homeostasis model assessment of insulin resistance [HOMA-IR] for population, and impaired fasting glucose or diabetes) were assessed using logistic regression analysis adjusting for age, gender, race/ethnicity, and current smoking.

Results—Among obese participants, those who were physically active had reduced odds of insulin resistance (47% lower; $P < .001$) and impaired fasting glucose/diabetes (23% lower; $P = .04$). These associations were weaker for central obesity. However, among participants with a normal waist circumference, those who were inactive were 63% more likely to have insulin resistance (OR [95% CI] 1.63 [1.24–2.15]) compared with the active reference group.

Conclusions—Physical activity was inversely related to the cardiometabolic risk associated with obesity and central obesity.

Keywords

cardiovascular disease; diabetes; ethnicity; obesity

Obesity is associated with a greater risk of cardiovascular disease (CVD) morbidity and mortality.[1] However, this relationship may be mediated by factors on the pathway to obesity.[2] At issue is whether physical activity (PA) or cardiorespiratory fitness (CRF) can partially explain or attenuate the CVD risk associated with obesity.[3] Previous strategies to address the health hazards of obesity, based primarily on weight loss, have thus far been ineffective because long-term maintenance of weight loss remains elusive.[4] Moreover, even intentional weight loss can increase mortality risk.[5] Therefore, if higher levels of PA or CRF can modify CVD risk in obese persons independent of weight loss, this could lead to prevention strategies that have better prospects for long-term maintenance and are potentially more cost effective.

The interrelationships among PA, CRF, adiposity and diverse health outcomes are complex. While CRF is arguably a better measure of assessing habitual PA than self-reported questionnaire data,[6] it requires specialized laboratory equipment and trained personal. Likewise, assessment of PA with doubly labeled water poses similar obstacles. Therefore, for practical reasons and despite their well documented limitations,[7] self-reported PA questionnaires have found wide application in large population studies. However, comparatively few studies have examined the combined associations of self-reported PA and measures of adiposity with cardiometabolic risk. In addition, the study populations have been comprised primarily of white men and women.[8]

Recently, a metabolically benign form of obesity has been identified and characterized.[9] Definitions for this “healthy” obese phenotype vary, but typically include the absence of five common risk factors: low high density lipoprotein (HDL)-cholesterol, high triglycerides, hypertension (HTN), insulin resistance (IR), and impaired fasting glucose (IFG) or type 2 diabetes mellitus (DM).[10] Therefore, for comparison purposes it is useful to examine associations of PA and adiposity with these cardiometabolic risk factors.

In a previous Multi-Ethnic Study of Atherosclerosis (MESA) report,[11] obese participants in all racial/ethnic and sex groups were more likely to have HTN, low HDL-cholesterol, and IFG compared with normal weight participants. However, PA was not included in these analyses. In the current study, observations were extended on the association of obesity to cardiometabolic risk by investigating the influence of PA as an effect modifier. Specifically, the purpose of this study was to quantify the relative importance of two standard measures of adiposity (body mass index [BMI] and waist circumference [WC]) and PA (measured by questionnaire) as indicators of cardiometabolic risk and whether this differs by race/ethnicity and sex in participants from the MESA baseline examination (2000–2002).

Methods

Study Population

The MESA is a community-based cohort study designed primarily to investigate prevalence, correlates, and progression of subclinical CVD. Details about study objectives and design have been published elsewhere.[12] Briefly, participants in MESA were aged 45 to 84 years, represented 4 racial/ethnic categories (white, Chinese, African American, and Hispanic) from Baltimore, MD, Chicago, IL, Forsyth County, NC, Los Angeles, CA, New York, NY, and St Paul, MN, and were free of clinical cardiovascular disease at entry. The study was approved by the Institutional Review Boards of the participating institutions, and all participants gave informed consent. All variables were collected during the baseline visit conducted from July 2000 to August 2002.

Adiposity Measures

Weight was measured using a Detecto Platform Balance Scale (Detecto, Webb City, MO) to the nearest 0.5 kg. Height was measured with an Accu-Hite Measure Device stadiometer (Seca, Hamburg, Germany) to the nearest 0.1 cm. WC was measured at the umbilicus using a Gullick II 150 cm anthropometric steel measuring tape with standard 4-ounce tension (Sammons Preston, Chicago, IL). BMI was calculated as weight (kg) divided by the square of height (m^2). Standard clinical definitions for obesity (BMI ≥ 30 kg/m^2) and central obesity (CO); WC: > 102 cm, men; > 88 cm, women) were used.

Cardiometabolic Risk Factors

Cardiometabolic risk factors included low HDL-cholesterol (< 50 mg/dL), high triglycerides (≥ 200 mg/dL), HTN (systolic blood pressure (BP) ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg or current use of BP medication), IR (upper quartile of homeostasis model assessment of insulin resistance [HOMA-IR] for population), and IFG (≥ 100 mg/dL to < 126 mg/dL) or type 2 DM (≥ 126 mg/dL or current use of insulin or oral glycemic medication). HDL-cholesterol and triglycerides were measured in EDTA-treated plasma on a Roche COBAS FARA centrifugal analyzer (Roche Diagnostics, Indianapolis, IN). BP was measured in the right arm of the participant after 5 minutes in a sitting position with a Dinamap Pro 100 automated oscillometric sphygmomanometer (Critikon, Tampa, FL). The systolic BP and diastolic BP were calculated using the average of the second and third of three measurements. IR was estimated by the HOMA-IR, calculated as insulin (mU/L) × glucose (mmole/L)/22.5.[13] Fasting blood glucose was measured using the Vitros analyzer (Johnson & Johnson Clinical Diagnostics, New Brunswick, NJ) and insulin was determined by the Linco Human Insulin Specific RIA kit (Linco Research, St. Charles, MO).

Self-Reported Physical Activity and Other Covariates

Standardized questionnaires were used to collect information on demographics, PA, education level, smoking, medical history, and medications. The MESA Typical Week Physical Activity Survey (TWPAS), adapted from the Cross-Cultural Activity Participation Study,[14] was designed to identify the time spent in and frequency of various physical activities during a typical week in the past month. The rationale for the selected time frame

was the intention to capture typical activity patterns in one's daily life. The survey has 28 items in categories of household chores, lawn/yard/garden/farm, care children/adults, transportation, walking (not at work), dancing and sport activities, conditioning activities, leisure activities, and occupational and volunteer activities. The survey also inquired about the typical pace at which participants walked in 5 categories ranging from very slow to brisk. Where appropriate, questions differentiated between light-, moderate-, and heavy-intensity activities. Respondents were asked whether they participated in these categories of activity, if yes, they answered questions regarding the average number of days per week and time per day engaged in these activities. Minutes of activity were summed for each discrete activity and multiplied by metabolic equivalent (MET) level.[15] PA was calculated on the basis of duration and intensity of the total intentional exercises (MET-min/wk).

Statistical Analysis

All the analyses for this study were completed in 2012. PA was categorized by quartiles. Sample means and standard errors were computed for the continuous characteristics and proportions were calculated for discrete characteristics by the 4 levels. Log transformation was used for triglycerides and HOMA-IR to achieve better approximation of normality. The estimated means or regression coefficients were back transformed to the original scale and the associated standard errors were obtained using Delta's method. The distribution of pack years of smoking could not be normalized through Box-Cox transformations due to large amount of zeros. Therefore, medians and interquartile ranges were reported and Cuzick's nonparametric test for trend was used. Tests for trend for other characteristics were assessed using linear regression models for continuous characteristics, logistic regression models for binary characteristics, and polytomous regression models for discrete characteristics with more than 2 categories.

The effect of BMI, WC, and PA was examined for the various cardiometabolic risk factors (all in the form of continuous measures). Linear regression models were used while adjusting for age, sex, race/ethnicity, and smoking status. Furthermore, the cardiometabolic risk factors were dichotomized (yes or no) and logistic regression models were fitted to assess the potential modifying effect of PA on the association between adiposity measures and the presence of the cardiometabolic risk factors. Since there is no standardized classification system for active versus inactive, quartiles were used. Self-reported PA in the lowest quartile (0–105 MET-minutes/wk) was categorized as inactive with the upper 3 quartiles (2nd: 123–818 MET-minutes/wk; 3rd: 825–2018 MET-minutes/wk; and 4th: 2025–37,260 MET-minutes/wk) categorized as active. BMI was categorized as normal-weight, overweight and obese, and WC was categorized as normal and centrally-obese. Covariates adjusted for included age, sex, race/ethnicity, and smoking status. Comparisons between various levels of PA and adiposity measures were estimated using linear contrasts. Three-way interactions of sex and race/ethnicity with adiposity measure and PA were also tested. To assess the linearity of the associations, quadratic, cubic terms for adiposity measures were fitted and checked. All analyses were performed using SAS 9.2 (Cary, NC). *P* values $< .05$ were regarded as statistically significant.

Results

Among the 6795 participants, the percentage of participants with HTN, obesity, central obesity, IFG, and type 2 DM was progressively lower with increasing PA quartile (Table 1). In addition, systolic BP, BMI, WC, fasting glucose, serum insulin, and triglycerides trended lower, and HDL-cholesterol and log HOMA-IR trended higher, with increasing PA quartile. No statistically significant trends by PA quartile were observed for age, diastolic BP, total cholesterol, or pack-years of smoking.

BMI and WC were evaluated according to PA status stratified by sex, age, and race/ethnicity (Table 2). Overall, BMI and WC were significantly higher for inactive compared with active men and women across age categories and race/ethnic groups. However, an exception was noted for Chinese Americans where no significant differences by activity status were observed for BMI or WC.

Linear regression analysis was used to evaluate associations of each cardiometabolic risk factor with PA, BMI, and WC adjusting for age, sex, race/ethnicity, and smoking status (Table 3). PA was significantly associated with HDL cholesterol and HOMA IR, BMI was significantly associated with HDL cholesterol, systolic BP and HOMA IR, and WC was significantly associated with HDL cholesterol, triglycerides, systolic BP, diastolic BP, HOMA IR, and glucose.

The association between cardiometabolic risk factors and adiposity measure stratified by PA (active or inactive) using logistic regression adjusting for age, sex, race/ethnicity, and smoking status is presented in Table 4. Compared with the normal and active reference groups, overweight, obese, and centrally obese individuals, both active and inactive, were at increased odds for each cardiometabolic abnormality. Notable differences were observed among obese and physically active participants, who were 47% ($P < .0001$) and 23% ($P = .04$) less likely to have IR and IFG/type 2 DM, respectively, than their physically inactive and obese counterparts. Being physically active also reduced the odds of low-HDL in those who were overweight (23%; $P = .01$) and centrally obese (17%; $P = .04$). Furthermore, inactive participants with normal WCs were 63% more likely to have IR (OR [95% CI] 1.63 [1.24–2.15]) compared with their active counterparts, but they did not differ significantly on any of the other cardiometabolic variables.

Testing of sex and race/ethnicity interactions with adiposity and PA revealed no significant interactions.

Discussion

The principle finding of this investigation was that higher levels of intentional PA were correlated to lower odds of having obesity-related cardiometabolic abnormalities, especially IR. Previous MESA reports found that higher levels of PA were associated with more favorable cardiac structure and function.[16,17] In the current study, observations on the influence of PA on cardiovascular health were extended by examining common clinical markers of cardiometabolic abnormalities according to obesity status. A sedentary lifestyle is a major contributor to obesity and is more common among obese than normal-weight

individuals.[18] Therefore, the association of obesity with increased cardiometabolic risk may be mediated in part by habitual low levels of PA. This hypothesis was tested by examining cross-sectional associations of two standard measures of obesity (BMI and WC), stratified by activity status, with five well-established markers of cardiometabolic risk (low HDL-cholesterol, high triglycerides, HTN, IR, and IFG).

The results of the current study are in general agreement with several recent reviews examining the influence of self-reported PA on cardiometabolic risk factors[3,8,19,20] and also accord with a similar investigation from the ATTICA Study,[21] a cross-sectional health and nutrition survey of over 4000 adults from the province of Attica, Greece. IR and fasting plasma glucose were significantly lower in obese individuals with high levels of PA compared with their inactive counterparts. However, the overweight or obese active subjects had similar levels of IR and glucose as normal-weight inactive individuals, a finding that does not accord with the results of the current study where inactive participants who were normal-weight and had a normal WC were at no greater risk than their active counterparts. This discrepancy may be explained by population factors or differences in the questionnaires that were used to assess or classify PA.

Findings from this multiethnic cohort of middle-aged to older men and women suggest that, with respect to cardiometabolic risk, avoiding obesity may be more important than increasing PA. However, at least three major objections may be urged against this conclusion: 1) higher CRF can eliminate the CVD mortality risk of obesity ("fat and fit" hypothesis), 2) PA may be a less important predictor of health outcomes than CRF, and 3) the validity of self-reported PA questionnaires is suspect. First, the "fat and fit" hypothesis originated from the Cooper Institute in 1999 when the landmark study by Wei et al[22] found that obese men who were fit were no more likely to die of CVD or all-causes than normal-weight and fit men. Another study published that same year found similar results for percentage body fat and central obesity.[23] Though relying only on baseline measures of fitness and adiposity, these studies suggested that higher fitness eliminated the increased mortality risk associated with obesity. More recently, Lee et al[24] examined longitudinal changes in fitness and fatness and mortality and found that preventing the age-associated decline in fitness was more important for longevity than changes in BMI. However, in another study of longitudinal changes in fitness and fatness focusing on CVD risk factors, these same authors[25] reported that the increased risk of developing metabolic syndrome, HTN, and hypercholesterolemia associated with fat gain were not completely eliminated. Nevertheless, taken together, these studies provide compelling evidence that maintaining or improving CRF may be as important as avoiding obesity. Secondly, the different results observed for fitness compared with PA may also be due to inherent differences between these variables.[26,27] Whereas CRF is an attribute that is typically measured by standard maximal exercise testing in 6 to 12 minutes,[28] PA is a temporal behavior that is more difficult to quantify. Thirdly, while self-reported PA questionnaires have been widely employed in epidemiological studies, they have only modest reliability and validity.[7] Furthermore, people tend to over-report their PA levels, especially for overweight individuals.[29] This may lead to underestimation of the effects of PA on cardiometabolic risk factors. In addition, sedentary behavior (eg, time spent reading, sitting, computer, and watching television) appears to be independently associated with unfavorable levels of

inflammation[30] and this variable was not assessed in the current study. Future studies with simultaneous measures of adiposity and objective measures of PA are needed to confirm these findings.

Strengths of this study include: a large community-based sample representing four ethnicities with thorough information about the medical history of each participant; and simultaneous measures of general and central obesity, and PA. However, this study also has limitations: the cross-sectional design prevents causal inferences; since only baseline data on adiposity, PA and other exposures were assessed, how longitudinal changes in any of these variables might have influenced the results cannot be determined; and the use of standard clinical cut-points for WC may not be appropriate for all races/ethnic groups[31] and this may have affected our results.

In conclusion, in this multiethnic cohort of men and women who were free of CVD at baseline, PA was inversely related to cardiometabolic risk factors in obese and centrally obese individuals. However, assessing PA by questionnaire may not capture actual levels of PA. Future studies should focus on the extent to which *changes* in objectively measured PA and body weight can reduce cardiometabolic risk and other health outcomes.

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Table 1

Baseline Characteristics of Participants in the Multi-Ethnic Study of Atherosclerosis by Physical Activity Quartile, 2000–2002[a]

	Physical activity MET-min/wk Quartiles (range)				
	Quartile 1 n = 1718 (0–105)	**Quartile 2 n = 1674 (123–818)**	**Quartile 3 n = 1704 (825–2018)**	**Quartile 4 n = 1699 (2025–37,260)**	***P* for trend[b]**
Age, years	62 ± 0.3	62 ± 0.3	62 ± 0.3	62 ± 0.3	.81
Male, %	44	41	49	56	< .0001
Race/ethnicity, %					
White	29	39	42	43	< .0001
Chinese	13	13	12	9	
Black	28	26	27	29	
Hispanic	29	22	19	18	
Physical activity, MET-min/wk	8 ± 39	476 ± 39	1345 ± 39	4384 ± 39	< .0001
Systolic BP, mm Hg	128 ± 0.5	127 ± 0.5	126 ± 0.5	125 ± 0.5	.0007
Diastolic BP, mm Hg	72 ± 0.3	72 ± 0.3	72 ± 0.3	72 ± 0.3	.3
Hypertension, %	48	45	45	41	.001
BMI, kg/m^2	29 ± 0.1	29 ± 0.1	28 ± 0.1	28 ± 0.1	< .0001
Obesity, %[c]	40	34	29	26	< .0001
Waist circumference, cm	101 ± 0.4	98 ± 0.4	97 ± 0.4	96 ± 0.4	< .0001
Central obesity, %[d]	61	58	52	46	< .0001
Fasting glucose, mg/dL	101 ± 0.7	97 ± 0.7	96 ± 0.7	96 ± 0.7	< .0001
Impaired fasting glucose, %	15	15	13	12	< .0001
Diabetes, %	16	13	11	11	< .0001
HOMA-IR[e]	1.52 ± 0.03	1.34 ± 0.02	1.21 ± 0.02	1.12 ± 0.02	< .0001
Serum insulin, umol/L	8 ± 0.1	7 ± 0.1	6 ± 0.1	6 ± 0.1	< .0001
Triglycerides, mg/dL	121 ± 2	117 ± 2	109 ± 1	107 ± 1	< .0001
Total cholesterol, mg/dL	195 ± 0.9	195 ± 0.9	193 ± 0.9	194 ± 0.9	.35
HDL-cholesterol, mg/dL	50 ± 0.4	51 ± 0.4	53 ± 0.4	53 ± 0.4	< .0001
Pack-years of smoking[f]	0 (18)	0 (14)	0 (14)	0 (15)	.004

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; MET, metabolic equivalent; BP, blood pressure.

[a] Data are mean (± standard error) unless otherwise indicated.

[b] Tests for trend were assessed using linear regression models for continuous characteristics and using logistic regression models for binary characteristics and polytomous regression models for discrete characteristics with more than 2 categories.

[c] BMI ≥ 30.0 kg/m^2.

[d] Waist circumference > 102 cm (men) and > 88 cm (women).

[e] Homeostasis model assessment of insulin resistance (glucose [mmol/L] × insulin [mU/L]/22.5).

[f] Median (interquartile ranges).

Table 2

Adiposity Measures for Active Versus Inactive[a] Subjects Stratified by Sex, Age, Race/Ethnicity; the Multi-Ethnic Study of Atherosclerosis, 2000–2002

	Active	Inactive	*P*-value
BMI, kg/m^2 (± SE)			
Sex			
Male	27.7 ± 0.09	28.6 ± 0.16	< .0001
Female	28.3 ± 0.12	29.9 ± 0.20	< .0001
Age			
< 55	28.6 ± 0.15	29.3 ± 0.26	.02
55–64	28.4 ± 0.15	29.9 ± 0.25	< .0001
65–74	27.7 ± 0.13	29.5 ± 0.23	< .0001
≥ 75	26.8 ± 0.17	27.9 ± 0.29	.002
Race/ethnicity			
White	27.4 ± 0.11	29.1 ± 0.22	< .0001
Chinese American	24.0 ± 0.14	24.0 ± 0.22	.97
African American	29.8 ± 0.16	31.2 ± 0.26	< .0001
Hispanic	29.1 ± 0.16	30.0 ± 0.23	.001
Waist circumference, cm (± SE)			
Sex			
Male	98.7 ± 0.25	101.3 ± 0.45	< .0001
Female	96.0 ± 0.31	100.2 ± 0.51	< .0001
Age			
< 55	96.2 ± 0.40	98.8 ± 0.69	.0008
55–64	97.8 ± 0.39	101.2 ± 0.65	< .0001
65–74	98.0 ± 0.36	102.4 ± 0.62	< .0001
≥ 75	97.1 ± 0.48	100.1 ± 0.83	.002
Race/ethnicity			
White	96.9 ± 0.31	102.3 ± 0.64	< .0001
Chinese American	87.4 ± 0.41	86.4 ± 0.66	.18
African American	100.1 ± 0.39	104.2 ± 0.66	< .0001
Hispanic	99.9 ± 0.41	102.1 ± 0.58	.002

[a]Self-reported physical activity in the lowest quartile (0–105 MET-minutes/wk) was categorized as inactive with the upper three quartiles (123–37,260 MET-minutes/wk) categorized as active.

Table 3

Multivariate Linear Regression Analysis of Cardiometabolic Risk Factors on Exposure Measure (Physical Activity, Body Mass Index, and Waist Circumference); the Multi-Ethnic Study of Atherosclerosis, 2000–2002

	Regression coefficient ± Standard Error[a]					
	HDL-Chol	**TG**	**SBP**	**DBP**	**HOMA-IR**	**Glucose**
Physical activity, MET-min/wk	0.33 ± 0.16[b]	0.99 ± 0.01	−0.23 ± 0.24	−0.01 ± 0.12	0.97 ± 0.01[c]	−0.13 ± 0.36
BMI, kg/m^2	−1.34 ± 0.35[c]	0.99 ± 0.01	2.67 ± 0.52[c]	0.21 ± 0.26	1.18 ± 0.02[c]	1.20 ± 0.78
Waist circumference, cm	−3.13 ± 0.34[c]	1.15 ± 0.01[c]	1.09 ± 0.51[b]	0.60 ± 0.25[b]	1.31 ± 0.02[c]	5.07 ± 0.76[c]

Abbreviations: DBP, diastolic blood pressure; BMI, body mass indes; HDL-Chol, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; MET, metabolic equivalent; SBP, systolic blood pressure; TG, triglycerides.

[a] Models adjusted for age, gender, race, and smoking status.

[b] $P < .05$.

[c] $P < .001$.

Table 4

Multivariate Logistic Regression[a] of Cardiometabolic Abnormality on Adiposity Measure[b] Stratified by Physical Activity Level[c]; the Multi-Ethnic Study of Atherosclerosis, 2000–2002

	Low-HDL	High-TG	HTN	HOMA-IR	IFG
BMI					
Normal-weight active	1.0	1.0	1.0	1.0	1.0
Inactive	1.26 (0.96–1.65)	1.39 (0.94–2.06)	1.05 (0.80–1.38)	1.05 (0.67–1.65)	1.03 (0.73–1.44)
Overweight active	1.90 (1.61–2.24)[d]	2.04 (1.59–2.62)	1.54 (1.31–1.81)	3.19 (2.45–4.16)	2.05 (1.68–2.50)
Inactive	2.47 (1.98–3.08)	2.02 (1.48–2.75)	1.51 (1.21–1.88)	3.81 (2.82–5.16)	2.10 (1.63–2.70)
Obese active	2.94 (2.36–3.65)	2.51 (1.85–3.41)	2.19 (1.77–2.72)	7.60 (5.63–10.24)[d]	3.41 (2.66–4.37)[d]
Inactive	3.04 (2.34–3.94)	2.73 (1.94–3.84)	2.54 (1.97–3.28)	14.25 (10.29–19.73)	4.44 (3.35–5.87)
WC					
Normal WC active	1.0	1.0	1.0	1.0	1.0
Inactive	1.14 (0.91–1.43)	1.10 (0.81–1.48)	1.01 (0.80–1.25)	1.63 (1.24–2.15)[d]	1.01 (0.79–1.28)
Central obese active	1.56 (1.31–1.85)[d]	1.57 (1.26–1.97)	1.29 (1.10–1.52)	2.41 (1.96–2.97)	1.36 (1.12–1.64)
Inactive	1.87 (1.51–2.31)	1.87 (1.42–2.46)	1.44 (1.17–1.77)	2.62 (2.01–3.41)	1.45 (1.15–1.83)

Abbreviations: BMI, body mass index; HTN, hypertension; HDL-Chol, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; IFG, impaired fasting glucose; TG, triglycerides; WC, waist circumference.

[a] Odds ratio (95% confidence interval) adjusting for age, sex, race/ethnicity, and smoking status with Normal-weight Active and Normal WC Active as reference groups.

[b] Normal-weight = BMI, 18.5–24.9 kg/m^2; Overweight = BMI, 25.0–29.9 kg/m^2; Obese = BMI, ≥ 30.0 kg/m^2; Normal WC ≤ 102 cm, men; ≤ 88 cm, women; Central obese > 102 cm, men; > 88 cm, women.

[c] Inactive = lowest quartile of MET-min/wk for population; active = higher three-fourths of MET-min/wk for population.

[d] $P < .05$ for active versus inactive within BMI or WC strata.