Joint Associations of Physical Activity and Aerobic Fitness on the Development of Incident Hypertension

Coronary Artery Risk Development in Young Adults

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Abstract—Fitness and physical activity are each inversely associated with the development of hypertension. We tested whether fitness and physical activity were independently associated with the 20-year incidence of hypertension in 4618 men and women. Hypertension was determined in participants who had systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg or who reported antihypertensive medication use. Fitness was estimated based on the duration of a symptom-limited graded exercise treadmill test, and physical activity was self-reported. The incidence rate of hypertension was 13.8 per 1000 person-years (n=1022). Both baseline fitness (hazard ratio: 0.63 [95% CI: 0.56 to 0.70 per SD]; 2.9 minutes) and physical activity (hazard ratio: 0.86 [95% CI: 0.79 to 0.84 per SD]; 297 exercise units) were inversely associated with incident hypertension when included jointly in a model that also adjusted for age, sex, race, baseline smoking status, systolic blood pressure, alcohol intake, high-density lipoprotein cholesterol, dietary fiber, dietary sodium, fasting glucose, and body mass index. The magnitude of association between physical activity and hypertension was strongest among participants in the high fitness (hazard ratio: 0.80 [95% CI: 0.68 to 0.94]) category, whereas the magnitude of association between fitness and hypertension was similar across tertiles of physical activity. The estimated proportion of hypertension cases that could be prevented if participants moved to a higher fitness category (ie, preventive fraction) was 34% and varied by race and sex group. Fitness and physical activity are each associated with incident hypertension, and low fitness may account for a substantial proportion of hypertension incidence. **(*Hypertension*. 2010;56:49-55.)**

Key Words: epidemiology ■ exercise ■ ethnicity ■ risk factors ■ special populations

Aerobic fitness and physical activity are each inversely associated with blood pressure and the development of hypertension.[1,2] Distinguishing between the two is important because physical activity is a behavior, whereas aerobic fitness is a physiological measure that reflects a combination of physical activity behaviors, genetic potential, and functional health of various organ systems. The magnitude of association of low physical activity with incident hypertension and other cardiovascular conditions is smaller than the association of fitness with many of those same cardiovascular diseases.[1–3] These observations may, in part, be related to the measurement error associated with self-reported activity as compared with objective measures of fitness that are determined by graded exercise tests.[4] Alternatively, high levels of activity that do not also raise fitness levels may be insufficient to lower the likelihood of developing hypertension.

The Coronary Artery Risk Development in Young Adults (CARDIA) Study is one of the few longitudinal population studies to jointly measure fitness and physical activity. We tested the independent and joint associations of aerobic fitness and physical activity at baseline as risk factors for the development of hypertension over 20 years in black and white men and women. We hypothesized that fitness and activity would each be inversely associated with hypertension incidence but that the association between fitness and hypertension would be stronger than that of physical activity level and hypertension. In addition, we estimated the proportion of incident hypertension that could be prevented if participants raised their fitness level.

Methods

Study Population

CARDIA is a longitudinal study of the development of cardiovascular disease risk factors over time in 5115 adults initially aged 18 to 30 years in 1985–1986. Black and white women and men were

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sampled from Birmingham, Ala; Chicago, Ill; Minneapolis, Minn; and Oakland, Calif. Participants were re-examined 2, 5, 7, 10, 15, and 20 years after baseline, and retention rates across examinations were 90%, 86%, 81%, 79%, 74%, and 72%, respectively. A total of 4618 participants were included in the present analysis after excluding the following: hypertension at baseline (n=139) or missing information to determine hypertension at baseline (n=57) and missing baseline treadmill test data (n=124) or lost to follow-up after baseline (n=175). Details of study recruitment and design were published previously.[5] All of the participants provided written informed consent at each examination, and institutional review boards from each field center and the coordinating center approved the study annually.

Measurements

Participants were asked to fast for ≥12 hours before examination and to avoid smoking or engaging in heavy physical activity for ≥2 hours before the examination. Standardized protocols for data collection were used across study centers, and examinations and blood samples were processed at a central laboratory according to standard procedures.[6]

At baseline, aerobic fitness was assessed with a graded, symptom-limited maximal exercise treadmill test using a modified Balke protocol.[7,8] Persons with a history of ischemic or congenital heart disease, on cardiovascular medications other than blood pressure medications, who had a blood pressure >160/90 mm Hg, or who were experiencing a febrile illness were excluded. The protocol included up to nine 2-minute stages of progressively increasing difficulty, and participants were encouraged to exercise as long as possible to maximum exertion. At the end of each stage, heart rate and blood pressure were measured, and during each stage, participants were asked to rate their level of exertion on the Borg scale.[8] We calculated age-predicted maximum heart rate using the Tanaka formula, 208−0.7*age,[9] and carried out secondary analyses restricting our data to the subset of participants who exercised to ≥85% of their age-predicted maximum heart rate.

Fitness was estimated based on the duration of the treadmill test, which is directly correlated with VO_2 max (maximum oxygen uptake). Treadmill duration was classified into sex-specific quintiles and then categorized into 3 levels per the convention in previous reports from CARDIA and the Aerobics Center Longitudinal Study.[1,10] Participants in the lowest 20% (women: 0.9 to <6.5 minutes; men: 1.9 to <10 minutes) were classified as low fitness, participants in the 20th to 60th percentile (women: 6.5 to <8.7 minutes; men: 10.0 to <12.2 minutes) were classified as moderately fit, and those above the 60th percentile (≥8.8 women and ≥12.2 men) were classified as highly fit.

Physical activity was assessed with the CARDIA physical activity questionnaire, an interviewer-administered self-report that measures the frequency of participation in 13 different categories of recreational sports and exercise in the past 12 months. Each activity was scored according to whether it was performed for ≥1 hour during any 1 month during the past year, the number of months it was performed at that level, and the number of months the activity was performed frequently. Physical activity scores were computed by multiplying the frequency of participation by intensity of activity and reporting as "exercise units" (EU). The reliability and validity of the instrument are comparable to other activity questionnaires.[11,12] We categorized activity into sex-specific tertiles (women: 0 to 195, 196 to 401, and 402 to 2126 EU: men: 0 to 345, 346 to 608, and 609 to 1962 EU).

Hypertension

After participants rested in a quiet room for 5 minutes, blood pressure was measured from participants in the seated position 3 times; the last 2 readings were averaged. At baseline and for examinations up through year 15, blood pressure was measured using a random-0 sphygmomanometer. At year 20, an Omron automated blood pressure device was used and calibrated against the random-0 device for comparability (calibrated systolic blood pressure=3.74+0.96*observed Omron systolic blood pressure; calibrated diastolic blood pressure=1.30+0.97*observed Omron systolic blood pressure).[13] Questionnaires were used to assess the use of antihypertensive medications. Prehypertension was determined as systolic blood pressure 120 to 129 mm Hg or diastolic blood pressure from 80 to 89 mm Hg and no use of antihypertension medications.[14] Hypertension was defined if any of the following criteria was met at a single examination: systolic blood pressure >140 mm Hg, a diastolic blood pressure >90 mm Hg on, or use of antihypertensive medications. Incident hypertension was identified if hypertension was identified at any of the follow-up examinations. Follow-up time was calculated as the difference between baseline and the examination when incident hypertension was identified. For participants who did not develop hypertension, follow-up time was censored at the last known follow-up examination. In a secondary analysis, we identified incident hypertension only among those who developed hypertension at ≥2 follow-up examinations.

Statistical Analyses

Baseline characteristics were calculated for the total sample and stratified by race and sex. We calculated the proportion of participants in a race-sex stratified sample who fell into each category of activity and fitness and the rate of diabetes mellitus (number of incident cases/person-time at risk) per 1000 person-years. Next, we tested and confirmed that the proportional hazards assumption was valid using log-log survival plots and tested whether there was statistical interaction of fitness or activity with race or sex (1 degree of freedom tests using continuous measures of fitness and activity). The association of fitness and activity with incident hypertension was determined per SD change from the group mean (ie, standardized coefficients) so that we could compare the relative strength of association between the 2 measures. We further explored the pattern of association of fitness with incident hypertension by activity tertile by computing hazard ratio and 95% CI per SD of treadmill duration within each tertile of activity. Similarly, we tested the association of activity with incident hypertension stratified by fitness category.

We carried out a series of secondary analyses to test whether or findings persisted. Because of a relatively large number of missing data for fasting insulin we substituted fasting insulin for glucose in a secondary analysis; similarly, we included parental history of hypertension in a secondary analysis, because 1413 did not respond to that question. Next, we defined incident hypertension if it was identified at ≥2 examinations. (We used logistic regression analysis for this secondary analysis because there was no reliable way to define follow-up time.) Finally, we tested our associations in subsets of the cohort who exercised to ≥85% of age-predicted maximum (n=4029) and did not experience any hospitalized cardiovascular disease events during follow-up (n=4320).

To estimate the proportion of potential cases of hypertension that could be prevented if participants moved from low or moderate fitness to high fitness, we calculated the preventive fraction. Because of marked differences in the incidence of hypertension by race and sex group, we estimated the preventive fraction separately by race and sex group. We used the following formula: 1−relative risk, where the relative risk reflects the multivariable adjusted relative risk with an increase from the nominal low to moderate to high fitness or activity categories. When the inverse association was not statistically significant, we did not calculate the preventive fraction. The preventive fractions for activity and fitness cannot be compared to one another because they are categorically determined using different criteria. Statistical significance was determined at $P<0.05$. All of the analyses were conducted using SAS version 9.2 (SAS Institute).

Results

Baseline characteristics are presented in Table 1. Blood pressure, treadmill test duration, and activity levels varied

Table 1. Baseline Characteristics of Study Population by Sex and Ethnicity

		Women			Men		
Baseline Characteristics	Total	Black	White	*P*	Black	White	*P*
N	4618	1309	1222		1004	1083	
Person-years of observation	16.0 (5.9)	15.5 (6.0)	17.4 (5.1)		14.4 (6.3)	16.7 (5.6)	
Age, y	24.8 (3.7)	24.3 (3.9)	25.4 (3.4)	<0.01	24.1 (3.8)	25.4 (3.4)	<0.01
Cigarette smoking, %							
Current	29.8	30.5	26.3	0.02	36.9	26.2	0.03
Former	12.8	15.4	19.3	<0.01	8.7	7.6	0.26
Family history of hypertension, % yes*	56.2	65.1	51.9	<0.01	60.8	47.3	<0.01
Alcohol intake, drinks per week	4.8 (8.8)	2.0 (4.8)	3.6 (6.0)	<0.01	7.2 (12.3)	7.5 (10.0)	0.49
Fiber, g	5.6 (3.2)	4.8 (3.0)	5.3 (3.0)	<0.01	6.0 (3.5)	6.5 (3.2)	<0.01
Sodium, mg	4172.0 (2118.1)	3556 (1789.4)	3239.5 (1326.4)	<0.01	5298.2 (2487.1)	4956 (2102.8)	<0.01
Calcium, mg	1229.1 (744.2)	977.6 (607.4)	1077.3 (557.9)	<0.01	1416.6 (768.5)	1532 (889.4)	<0.01
Potassium, mg	3664.7 (1728.8)	3061.4 (1533.3)	3237.7 (1330.5)	<0.01	4206.1 (1868.1)	4381.1 (1809)	0.03
Magnesium, mg	370.8 (184.9)	293 (155.6)	318.9 (130.7)	<0.01	432.1 (196.5)	467.2 (196.2)	<0.01
BMI, kg/m^2	24.4 (4.9)	25.7 (6.3)	23.0 (4.3)	<0.01	24.5 (4.2)	24.2 (3.5)	0.10
Waist circumference, cm	77.4 (11.0)	76.0 (12.5)	71.7 (9.2)	<0.01	80.4 (9.7)	82.7 (8.6)	<0.01
Systolic blood pressure, mm Hg	109.8 (10.2)	107.5 (9.1)	104.4 (8.8)	<0.01	114.9 (9.6)	113.8 (9.6)	0.01
Diastolic blood pressure, mm Hg	68.1 (9.1)	66.9 (9.0)	65.9 (8.0)	<0.01	69.9 (9.7)	70.3 (8.8)	0.29
Prehypertension, %	22	14	8	<0.01	37.4	33.2	<0.01
Treadmill test duration, min	9.8 (2.9)	7.3 (1.9)	9.3 (2.1)	<0.01	11.2 (2.3)	12.2 (2.2)	<0.01
Fasting plasma glucose, mg/dL	4.52 (0.73)	4.39 (0.68)	4.46 (0.66)	0.02	4.56 (0.95)	4.66 (0.59)	0.16
Fasting insulin, μU/mL†	11.43 (5.8)	13.28 (7.0)	10.32 (4.6)	<0.01	11.82 (6.6)	10.35 (3.9	<0.01
High-density lipoprotein cholesterol, mg/dL	53.1 (13)	55.1 (12.6)	56.1 (13.0)	0.05	53.3 (13.3)	47.0 (11.0)	<0.01
Physical activity, EU‡	363 (378)	228 (291)	354 (332)	<0.01	472 (437)	464 (388)	0.20

Values presented as mean (SD) unless otherwise noted.
*Data were available only in a subset of 3205 participants.
†Data were available only in a subset of 3576 participants.
‡Data show the median and interquartile range median and interquartile range.

across race and sex groups. Black women had higher systolic and diastolic blood pressures than white women, whereas only systolic blood pressure was significantly higher in black versus white men. Activity and fitness were lower in black and white women as compared with men. The Figure shows the proportion of study participants with a given level of fitness within activity categories. The proportion of participants with low fitness was smaller with higher activity levels among all of the participants; the inverse was true for high fitness. These patterns were more pronounced in white participants than black participants, and a greater proportion of white participants had high fitness as compared with black participants.

Over 20 years, 1022 participants developed hypertension at a rate of 13.8 per 1000 person-years. One third (n=340) of participants with incident hypertension had high blood pressure but did not use medications; 451 participants were identified based on antihypertension medications but blood pressure was not elevated (44.1%), and 231 participants had a combination of high blood pressure and medications (22.6%). The rate of hypertension was 20.7, 19.6, 10.6, and 6.1 in black women, black men, white men, and white women, respectively. Across race-sex groups, there was an inverse, graded association between fitness category and the development of hypertension (Table 2). The incidence of hypertension was inversely associated with fitness category in all of the race and sex groups, although the absolute rates varied by sex (Table 3). By contrast, patterns of association between activity and incident hypertension were less pronounced.

Neither race nor sex modified the association between fitness and hypertension. Treadmill duration (per 2.9 minutes longer) and activity score (per 297 EU higher) were each inversely associated with incident hypertension (Table 2). The magnitude of association of both fitness and activity with incident hypertension was modestly attenuated but remained statistically significant following additional adjustment for covariates. There was no evidence of statistical interaction between activity and fitness in association with the development of hypertension (χ^2=1.15; P=0.28). When both were included in the same model, fitness remained significantly and independently associated with a lower likelihood of developing hypertension, whereas activity attenuated to nonsignificance. Our find-

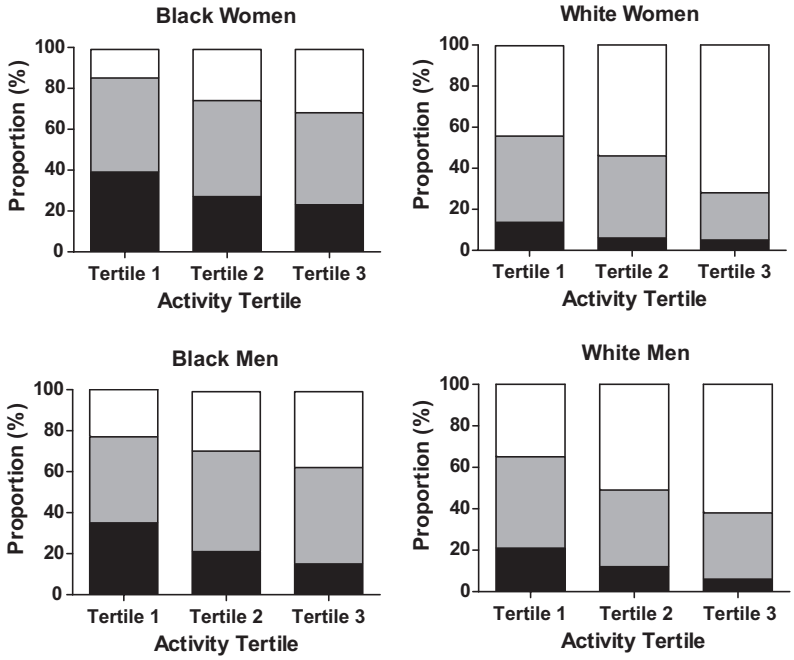

Figure. Proportion of participants by sex-specific fitness categories and activity tertiles by race-sex. Black bars indicate low 20th percentile (women: 0.9 to <7.0 minutes; men: 3.0 to <10.2 minutes); gray bars, moderate 21st to 59th percentile (women: 7.0 to <9.0 minutes; men: 10.2 to <12.8 minutes); white bars, high ≥60th percentile fitness. Activity tertiles (exercise units): low, 0 to 195 (women) and 0 to 345 (men); moderate, 196 to 401 (women) and 346 to 608 (men); high, 402 to 2126 (women) and 609 to 1962 (men).

ings were robust, because they persisted with adjustment for family history and were present in the subset of participants who exercised to ≥85% of their age-predicted maximum heart rate, participants who did not experience hospitalized cardiovascular disease during follow-up, and when incident hypertension was determined among those participants who had it at >1 follow-up examination.

Fitness was significantly and inversely associated with the development of hypertension within each tertile of physical activity. Despite the absence of an interaction between activity and fitness, activity was only significantly inversely associated with the development of hypertension in the highest category of fitness. There was no difference when fasting insulin was used in place of fasting glucose or waist circumference was used in place of body mass index (BMI).

The HR for developing hypertension ranged from 0.54 to 0.79 per 1 category of higher fitness measure across race and sex groups (Table 3). The proportion of hypertension that could theoretically be prevented was highest in white men and women, but all of the race-sex groups demonstrated the potential for a lower hazard with higher fitness. Although

Table 2. Hazard Ratios (95% CIs) of the Association of Fitness or Activity With Incident Hypertension in the Full Sample and Within Activity and Fitness Categories

			Model 1*			Model 2			Model 3		
Baseline Fitness	N	No. of Events	HR	95% CI	*P*	HR	95% CI	*P*	HR	95% CI	*P*
Fitness, per 2.9 min	4618	1022	0.52	0.48 to 0.57	<0.01	0.63	0.56 to 0.70	<0.01	0.64	0.57 to 0.72	<0.01
Association of fitness with hypertension within activity tertiles											
Low activity	1537	430	0.51	0.44 to 0.59	<0.01	0.64	0.54 to 0.77	<0.01			
Moderate activity	1541	306	0.57	0.49 to 0.68	<0.01	0.71	0.57 to 0.88	<0.01			
High activity	1539	286	0.50	0.43 to 0.59	<0.01	0.58	0.47 to 0.70	<0.01			
Activity, per 297 EU	4617	1022	0.83	0.77 to 0.90	<0.01	0.86	0.79 to 0.94	<0.01	0.93	0.86 to 1.01	0.10
Association of activity with hypertension within fitness categories											
Low fitness	886	348	0.98	0.84 to 1.15	0.82	0.86	0.72 to 1.03	0.10			
Moderate fitness	1882	450	0.98	0.88 to 1.09	0.71	1.01	0.90 to 1.14	0.86			
High fitness	1850	224	0.82	0.71 to 0.95	0.01	0.80	0.68 to 0.94	0.01			

Model 1 was adjusted for age, sex, and race. Model 2 was adjusted for model 1+smoking status (current vs former or never), baseline systolic blood pressure, baseline BMI, drinks per week of alcohol, high-density lipoprotein cholesterol, dietary fiber (grams), dietary sodium (milligrams), and baseline fasting glucose. Model 3 is the same as model 2 with activity and fitness both in the model.

*Data show the fitness and physical activity per SD higher than the mean.

Table 3. Incident Rates, Hazard Ratios (95% CIs), and Preventive Fraction for the Association of Activity and Fitness With Incident Hypertension

	Fitness†						Activity†					
	Rate per 1000 Person-Years			Hazard Ratio per 1 Category Higher			Rate per 1000 Person-Years			Hazard Ratio per 1 Category Higher		
Study Sample	Low	Moderate	High	HR‡	95% CI	PF*	Low	Moderate	High	HR‡	(95% CI)	PF*
Total population	7.1	15.1	27.6	0.66	0.59 to 0.74	0.34	11.4	12.3	18	0.88	0.81 to 0.96	0.12
Black women	12	19.1	30	0.72	0.60 to 0.86	0.28	20.5	18.4	22.5	1.01	0.88 to 1.16	
White women	3.5	9.3	15.1	0.63	0.46 to 0.87	0.37	5.4	5.6	8.4	0.91	0.71 to 1.15	
Black men	14	18.4	29.7	0.79	0.65 to 0.97	0.21	15.9	17	25.8	0.75	0.63 to 0.89	0.25
White men	6.4	12	25.8	0.54	0.42 to 0.68	0.46	9.5	10.1	12.4	0.89	0.73 to 1.09	

*PF indicates preventive fraction, the proportion of hypertension incidence that can theoretically be prevented if participants move 1 category higher fitness (low to moderate or moderate to high). Calculated as 1−relative risk after adjustment for age, race, sex, smoking status, baseline systolic blood pressure, drinks per week of alcohol, high-density lipoprotein cholesterol, dietary fiber (grams), dietary sodium (milligrams) baseline fasting glucose, and baseline BMI. The PF is not calculated when the hazard ratios are not statistically significant.

†Fitness categories were as follows: low, <20th percentile (women: 0.9 to <7.0 minutes; men: 3.0 to <10.2 minutes); moderate, 21st to 59th percentile (women: 7.0 to <9.0 minutes; men: 10.2 to <12.8 minutes); and high, >60th percentile. Activity tertiles (EU) were as follows: low, 0 to 195 (women) and 0 to 345 (men); moderate, 196 to 401 (women) and 346 to 608 (men); and high, 402 to 2126 (women) and 609 to 1962 (men).

‡Hazard ratio and 95% CI were adjusted for age, race, sex, smoking status, baseline systolic blood pressure, drinks per week of alcohol, high-density lipoprotein cholesterol, dietary fiber (grams), dietary sodium (milligrams) baseline fasting glucose, and baseline BMI.

baseline activity category was inversely associated with incident hypertension, the HR was only statistically significant in black men.

Discussion

Findings from our longitudinal study demonstrate that both fitness and activity are inversely associated with the development of hypertension over 20 years. As estimated by the preventive fraction, achieving higher fitness by category could theoretically "prevent" more cases of incident hypertension. Clinical trials indicate that increasing physical activity is an important hypertension prevention behavior[15,16]; however, it is possible that the behavior is only protective if activity levels are adequate to improve fitness. Activity was only significantly inversely associated with the development of hypertension among participants in the highest fitness category, although that heterogeneity was not statistically significant (ie, no interaction). In a previous CARDIA Study, Parker et al[2] showed that young adults who were the most physically active had a decreased risk of developing hypertension and that the risk was attenuated after adjustment for fasting insulin level and waist circumference. Their findings suggest that some of the association of activity with incident hypertension was mediated by obesity and the metabolic syndrome. Given that regular physical activity can help with weight loss, which can help modify risk for hypertension, the study by Parker et al[2] adds further evidence that the greatest benefit is observed when activity levels are sufficient to maintain a normal weight and prevent insulin resistance.

We extend beyond the previous analysis to demonstrate a significant inverse association of fitness, another physiological response of activity, with incident hypertension. With the addition of 5 years of cohort follow-up, 388 additional hypertension events developed, which permitted us to stratify our analysis by activity categories to demonstrate that physical activity is only significantly inversely associated with hypertension when fitness levels are the highest. Activity levels that boost fitness may lead to the most benefit. A related observation is that our findings on the association of fitness or activity with incident hypertension were modestly attenuated when we included BMI in models and waist circumference (data not shown). Both BMI and waist circumference are significantly inversely associated with categories of fitness and activity. It is plausible that the mechanism by which fitness and activity are associated with a lower rate of developing hypertension is through lowered BMI. We observed a similar finding in our study of the association of fitness with incident diabetes mellitus in the CARDIA Study,[17] and patterns of association of fitness with BMI and waist in CARDIA were published previously.[1]

The disparity in hypertension incidence between black and white adults in the CARDIA Study is consistent with patterns in other US samples.[18] In National Health and Nutrition Examination Survey III, the prevalence of hypertension was higher among black as compared with white and Mexican-American adults, and within each group hypertension prevalence was inversely associated with the number of bouts per week of leisure-time physical activity.[19] We observed no difference in the association of activity or fitness with incident hypertension by race group, as evidenced by an absence of statistical interaction. According to our preventive fraction estimates, a smaller proportion of hypertension incidence would be prevented in black as compared with white participants if they were to move into the high fitness category. However, because the incidence of hypertension is 2 to 3 times higher in black men and women as compared with their counterparts, those proportions represent a large number of cases and a substantial reduction in the public health burden of hypertension. Further exploration of factors to explain the ethnic disparity in hypertension is warranted.

Limitations

The relatively stronger association of fitness as compared with activity in relation to incident hypertension may be attributable to underreporting of activities that participants may engage in that are not queried on the survey, as well as nonpurposeful movements. Nonexercise activity thermogenesis is an important source of energy expenditure that can only be measured objectively using accelerometry combined with a posture sensor.[20,21] Unfortunately, no such measurements were collected at the baseline examination of CARDIA. By contrast, treadmill testing is an objective measure of the physiological response to purposeful and, possibly, nonpurposeful activities. Underreporting from these sources may bias the association of activity with incident hypertension toward the null.

The CARDIA physical activity history does not query duration of exercise session, and so neither caloric expenditure nor metabolic equivalents of exercise can be estimated.[12] As such, they lack clinical interpretation. However, in a review of the CARDIA activity history, the instrument was reliable over a 1-month interval and was correlated with estimates of body fatness and treadmill test performance.[11] Previous research in the CARDIA Study demonstrates that the associations of activity with the development of hypertension[2] and metabolic syndrome[22] are in the expected direction.

Although fitness and activity measures were available at multiple examinations over follow-up, the present investigation is restricted to baseline measures of activity and fitness. We chose to use the baseline measure because baseline fitness and activity commonly "track" over time in the CARDIA sample. Furthermore, baseline risk factors are more strongly associated with incident disease outcomes in the CARDIA Study and in other longitudinal studies than are measures that are updated over time.

Despite clinical recommendations to diagnose hypertension based on multiple readings and across many days,[14] we classified participants as having hypertension based on a single elevated blood pressure reading, a definition shared by most epidemiological studies. A single elevated blood pressure reading may be an isolated event that has misclassified participants who were truly normotensive. The resulting misclassification bias has most likely shifted our estimates toward the null. However, a secondary analysis restricting the definition of incidence to hypertension at $\geq$2 examinations demonstrated that our findings were robust.

The preventive fraction is an estimate of the population burden of disease under a hypothetical scenario of risk factor modification. The fractions are appropriate when the hypothesized relationships are causal and when all of the competing causes have been accounted for using multivariable adjustment. In our observational study, it is possible that residual confounders are influencing our hazard ratio estimates and biasing our estimates away from the null.

In conclusion, low fitness demonstrates a more robust association with the development of hypertension than low self-reported physical activity, yet the 2 appear to exert independent effects. Although the population burden of hypertension attributable to low fitness and activity varies across race and sex groups, the contributions are meaningful and suggest a plausible target for intervention to lower hypertension rates in the population.

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Disclosures

None.

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