HHS Public Access

Author manuscript

Arterioscler Thromb Vasc Biol. Author manuscript; available in PMC 2017 June 01.

Published in final edited form as:

Arterioscler Thromb Vasc Biol. 2016 June ; 36(6): 1272–1277. doi:10.1161/ATVBAHA.116.307284.

Walking and calcified atherosclerotic plaque in the coronary arteries: the NHLBI Family Heart Study

Tasnim F. Imran, MD[1], **Yash Patel, MD**[1], **R Curtis Ellison, MD**[2], **J. Jeffrey Carr, MD**[3], **Donna K. Arnett, PhD MSPH**[4], **James S. Pankow, PhD MPH**[5], **Gerardo Heiss, MD PhD**[6], **Steven C. Hunt, PhD**[7], **J. Michael Gaziano, MD MPH**[1], and **Luc Djoussé, MD ScD**[1]

[1]Brigham and Women's Hospital and Boston Veterans Affairs Healthcare System, Harvard Medical School, Boston, MA

[2]Preventive Medicine and Epidemiology, Boston University School of Medicine, Boston, MA

[3]Department of Radiology and Radiological Sciences, Vanderbilt University, Nashville, TN

[4]Department of Epidemiology, University of Alabama at Birmingham, Birmingham, AL

[5]Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, MN

[6]Department of Epidemiology, University of North Carolina, Chapel Hill, NC

[7]Cardiovascular Genetics, Department of Medicine, University of Utah, Salt Lake City, UT

Abstract

Objective—Studies have reported mixed findings on the association between physical activity and subclinical atherosclerosis. We sought to examine whether walking is associated with prevalent coronary artery calcification (CAC) and aortic calcification (AC).

Approach and Results—In a cross-sectional design, we studied 2,971 participants of the NHLBI Family Heart Study without a history of myocardial infarction, coronary artery bypass grafting or percutaneous transluminal angioplasty. A standardized questionnaire was used to ascertain the number of blocks walked daily to compute walking metabolic equivalent hours. CAC was measured by cardiac CT. We defined prevalent CAC and AC using an Agatston score of at least 100 and used generalized estimating equations to calculate adjusted prevalence ratios.

Mean age was 55 years and 60% of participants were women. Compared to the ≤ 3.75 met-hrs/wk group, prevalence ratios for CAC after adjusting for age, sex, race, smoking, alcohol use, total physical activity (excluding walking) and familial clustering were 0.53 95% CI: 0.35–0.79 for >3.75–7.5 met-hrs/wk, 0.72, 95% CI: 0.52–0.99 for >7.5–15 met-hrs/wk, and 0.54, 95% CI: 0.36–0.81 for >15–22.5 met-hrs/wk, (p trend 0.01). The walking-CAC relation remained significant for those with BMI ≥ 25 (p trend 0.02) and persisted with CAC cutoffs of 300, 200, 150, 50, but not 0. When examined as a continuous variable, a J-shaped association between walking and CAC was found. The walking-AC association was not significant.

Corresponding author: Tasnim F. Imran, MD, Brigham & Women's Hospital, Harvard Medical School, 1620 Tremont St, Boston, MA 02120, Ph: 617-525-7591; Fax: 617-525-7739, ; Email: TFImran@partners.org

Disclosure

None

Conclusion—Our findings suggest that walking is associated with lower prevalent coronary artery calcification (but not aortic calcification) in adults without known heart disease.

Keywords

subclinical atherosclerosis; physical activity; coronary artery calcification; aortic calcification

INTRODUCTION

Atherosclerosis is a major cause of cardiovascular disease (CVD) in the world. Imaging biomarkers of subclinical atherosclerosis, such as coronary artery calcification (CAC) and aortic calcification (AC), are established predictors of coronary heart disease and cardiovascular events.[1] It is well known that physical activity is integral to the primary prevention of cardiovascular diseases. Physical activity is strongly associated with reduction in blood pressure, improvement in lipid profiles, reduction of cardiac ischemia, improvement in endothelial function and decrease in all-cause and CVD mortality.[2–5]

However, studies have reported mixed findings on the association between physical activity and markers of subclinical atherosclerosis. Though many have suggested an inverse relationship between physical activity and CAC,[6,7] others have found no such association.[8,9] Aortic calcification is also an emerging predictor of cardiac morbidity and mortality and thus may be utilized as another imaging biomarker of atherosclerosis.[10,11] However, no prior studies have evaluated the association between physical activity and AC in a cohort of adult men and women.

Few studies have examined walking as the primary exposure of physical activity in relation to CAC.[12] Walking is a basic measure of physical activity that may be more sustainable than forms of vigorous exercise.[13] Moderate walking has been demonstrated to have similar risk reductions in cardiovascular risk factors and possibly CVD as compared to more strenuous activities.[13] Walking is a primary activity that is in line with the American Heart Association's recommendations for physical activity in adults, and endorsed by the American College of Cardiology Foundation.[4] A better understanding of the relationship between physical activity and subclinical atherosclerosis could lead to implementation of targeted public health interventions for the primary prevention of cardiovascular disease. Therefore, we sought to address whether physical activity, as determined by a walking metabolic index, is associated with prevalent CAC and AC in adult men and women from the NHLBI Family Heart Study.

METHODS

Materials and Methods are available in the online-only Data Supplement.

Briefly, we studied 2,971 participants of the NHLBI Family Heart Study without a history of myocardial infarction, coronary artery bypass grafting or percutaneous transluminal coronary angioplasty in a cross-sectional design. A standardized questionnaire was used to ascertain the number of blocks that each participant walked daily to compute the walking metabolic equivalent hours per week. CAC was measured by cardiac computed tomography

(CT) scans. We defined prevalent CAC and AC using an Agatston score of at least 100 and used generalized estimating equations to calculate adjusted prevalence ratios.

RESULTS

Of the 2,971 participants analyzed, 60.3% were women, and the mean age was 55 ±12.7 years. Table 1 presents lifestyle and clinical characteristics of participant across walking activity categories. The median adjusted walking activity level was 9.33 met-hours/week, (IQR 14.93 met-hours/week). The median CAC score was 0.5 (IQR 61.5), and the median AC score was 93, (IQR 1364). CAC was present in 54%, while AC was present in 65% of participants. CAC and AC were moderately correlated (*Spearman's rho: 0.69, p<0.0001*).

Using prevalence ratios, we found evidence of an inverse association between physical activity and CAC. The >15–22.5 met-hrs/wk group had a 46% lower prevalence of CAC as compared to the reference group (≤ 3.75 met-hrs/wk) in the full model after adjusting for age, sex, race, smoking, alcohol use, total exercise metabolic activity index (excluding walking) and familial clustering. The prevalence ratios for CAC with 95% confidence intervals for each walking activity category are shown in Table 2, *p trend = 0.01* in the fully adjusted model (Table 2). The relationship between walking and AC was significant in the crude model (*p trend = 0.01*), but not in the fully adjusted model (*p trend = 0.75*) (Table 3). However, when we examined walking as a continuous variable (walking metabolic activity), a spline model suggested that there may be a J-shaped association between walking and CAC (suppl Figure 1). Test for linearity using the likelihood ratio test yielded a p-value of 0.01.

In a secondary analysis stratified by body mass index (BMI) (<25 and ≥25 kg/m^2), the walking-CAC association remained statistically significant for those with BMI ≥ 25, *p trend* = 0.02 (Table 4). We did not have adequate number of participants with a BMI < 25 for separate analysis (n=721). In sensitivity analysis, we found similar results across various CAC thresholds. This association between walking and CAC persisted when we repeated the main analyses using CAC cutoffs of 300, 200, 150, 50, but not 0 (suppl Table I). The walking-AC relation was not significant across other thresholds (suppl Table I).

DISCUSSION

Our study suggests that walking is associated with prevalent CAC (J-shaped association) but not AC in adult men and women who are free of clinically evident cardiovascular disease. This association persists even for those who are overweight and obese (body mass index ≥ 25 kg/m^2) and even after adjusting for other leisure time physical activities. Our study is one of the few studies that evaluated walking as a primary measure of physical activity in relation to CAC.[12] Also, this is the first study to examine the association between physical activity and prevalent AC in a large cohort of adult men and women.

Our results are complementary to and extend the findings from prior reports. In a group of asymptomatic young adults in the Coronary Artery Risk Development in Young Adults (CARDIA) study, odds ratios of having CAC 15 years later were significantly lower for moderately and highly fit participants.[7] In the Multi-Ethnic Study of Atherosclerosis

(MESA) cohort, an inverse association between physical activity and CAC progression and ankle brachial index was noted.[6] Our findings are also in the same direction as those of trials such as the Lifestyle Heart Trial, in which participants who had exercise training with other lifestyle modifications, had a slower rate of atherosclerosis progression, and the Heidelberg Regression Study.[14–17] These studies demonstrated that even patients who already had known coronary artery disease had decreased rate of atherosclerosis progression after physical activity interventions.

However, other studies have found no association between physical activity and CAC.[8,9,18] In the Whitehall II cohort, physical activity was measured using accelerometers, but no association was seen between physical activity and CAC. However the participants were followed for only about seven days, which may not be long enough to evaluate the association.[18] Also, awareness of being recorded may alter participant activity levels and thus introduce bias. In a cross-sectional study of the MESA cohort, no relation between physical activity and CAC was observed.[19] In that study, moderate and vigorous physical activity were combined into one variable, and intentional exercise was evaluated separately. However, the main exposure in our study was a weekly walking metabolic index (walking activity), which mainly consists of low to moderate physical activity. Also, the MESA study differs from that of our study in that it consisted of individuals from at least four different ethnic groups. Thus, the heterogeneity across studies could be due to study design, study population, assessment of physical activity and unaccounted genetic and environmental factors.

When we examined walking as a continuous variable, a nonparametric model suggested a J-shaped association between walking and CAC. Prior studies have described the association between physical activity and CVD outcomes as curvilinear.[20,21] Although less reduction in CVD events was observed at high levels of moderate to vigorous activity, the protective effect for CVD mortality was still present as compared to those who were sedentary.[21] Another plausible explanation for why we might find a J-shaped association lies in the method of measurement of the outcome (CAC). Although the Agatston method is the standard for scoring coronary calcification, some have suggested that other methods of determining coronary calcification should be considered. The Agatston method is upweighted with greater calcium density of the plaque.[22] However, some studies suggest that greater plaque density may actually be associated with stable disease or even decreased risk of coronary heart disease.[22,23]

Although AC has been found to be an independent predictor of cardiovascular events and correlated with CVD risk factors,[10,11,24,25] studies evaluating the association between physical activity and AC are sparse. One prior study that included 276 obese elderly women with peripheral fat distribution found that high intensity physical activity was associated with increased androgens, low interleukin 6 and less AC.[26] Although we hypothesized that physical activity would be inversely associated with both CAC and AC, the relation between physical activity and AC was not significant in our data except at much higher thresholds. There are some considerations regarding why these results may differ. First, current CT imaging techniques do not allow for quantifying medial and intimal calcification separately. Prior studies have demonstrated that medial calcification, which is associated with

conditions of metabolic imbalance such as diabetes and kidney disease, may be commonly present in the abdominal aorta, but is not found in the coronary arteries.[27,28] We adjusted for diabetes, and even after excluding participants with diabetes (6.7% of our population), the effect estimates were not significantly different. Additionally, investigators from the Framingham Heart Study noted that genetic factors play a prominent role in the presence and extent of AC. They found that 49% of the variation in AC in their study population was due to heritable factors.[29] Although we adjusted for familial clustering in our analysis, it is possible that there are genetic and environmental factors that remain unaccounted for and that separate factors may influence calcification at each site.[29] Also, AC is more prevalent than, and may predate the development of CAC.[30] Given our study design, we could not evaluate a temporal relationship. Lastly, since aortic calcification is more prevalent, it is possible that a higher measurement threshold may be more clinically pertinent.

Physical activity improves blood lipids.[5,31,32] Other potential mechanisms by which physical activity might decrease atherosclerotic disease risk include improvement in blood viscosity, vascular function, platelet activity, blood pressure, triglycerides, and anti-thrombotic effects.[5,32–34] Physical activity has also been found to increase the production of nitric oxide and prostacyclins, which enhances vasodilation, thereby improving endothelial function.[35] It decreases platelet aggregation and adhesion,[36] and enhances fibrinolysis and improves fibrinogen levels.[33] All these effects can slow the progression of atherosclerotic cardiovascular disease.

Our study has limitations. Physical activity was self-reported via standardized questionnaires and interviews, thus the social desirability bias may have impacted reported levels. It has been suggested that combining the duration of walking time with MET levels to produce a met-activity index may not accurately reflect activity level in all situations.[19] For instance, if one walks at a moderate intensity for a long period of time, that may not be equivalent to walking at a more vigorous intensity. Although we adjusted for cardiovascular risk factors based on a priori knowledge, due to the observational nature of the study, unknown confounders may be present and residual confounding cannot be entirely excluded. The cross-sectional design does not allow us to examine changes in physical activity over time. Also, physical activity may be related to other healthy behaviors, and thus may be part of a group of behaviors inversely associated with CAC.

Despite these limitations, our study has several strengths. First, it includes a large sample size, which allows for greater power. Second, our study population included Caucasians and African Americans, men and women, thus increasing generalizability. Third, we incorporated extensive data on lifestyle factors, cardiovascular risk factors and anthropometric measurements into our analysis. Fourth, we used a standardized approach to CAC measurement that has been previously validated.[37] Lastly, our study is one of the few to evaluate walking activity, an essential and sustainable form of physical activity, in relation to CAC and the first to examine the association between walking and aortic calcification in adult and men.

In conclusion, the NHLBI Family Heart Study data show a J-shaped association of walking with coronary artery calcification but not aortic calcification in adult men and women who

are free of cardiovascular disease. This study highlights the need for further studies and trials with a long-term follow-up to more clearly elucidate the association between physical activity and calcified atherosclerotic plaque evolution in the aorta and coronary arteries in individuals without known cardiovascular disease.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We report findings from the National Heart, Lung, and Blood Institute Family Heart Study. The investigators thank the FHS participants and staff for their commitment and valuable contributions.

Sources of Funding

NHLBI FHS was supported by the National Heart, Lung, and Blood Institute cooperative agreement grants U01 HL 67893, U01 HL67894, U01 HL67895, U01 HL67896, U01 HL67897, U01 HL67898, U01 HL67899, U01 HL67900, U01 HL67901, and U01 HL67902.

Abbreviations

CAC	coronary artery calcification
AC	aortic calcification
PA	physical activity
BMI	body mass index
CVD	cardiovascular disease
CARDIA	Coronary Artery Risk Development in Young Adults
MESA	Multi-ethnic Study of Atherosclerosis

References

1. Greenland P, Bonow RO, Brundage BH, et al. ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: a report of the American College of Cardiology Foundation Clinical Expert Consensus Task Force. Circulation. 2007; 115:402–426. [PubMed: 17220398]
2. Blair SN, Kohl HW, Barlow CE, et al. Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men. JAMA. 1995; 273:1093–1098. [PubMed: 7707596]
3. Slattery ML, Jacobs DR. Physical fitness and cardiovascular disease mortality: the US Railroad Study. Am J Epidemiol. 1988; 127:571–580. [PubMed: 3341361]
4. Thompson PD, Buchner D, Pina IL, et al. AHA Scientific Statement. Exercise and Physical Activity in the prevention and treatment of atherosclerotic cardiovascular disease. A statement from the Council on Clinical Cardiology and the Council on Nutrition, Physical Activity and Metabolism. Circulation. 2003; 107:3109–3116. [PubMed: 12821592]
5. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. N Engl J Med. 1988; 319:1173–1179. [PubMed: 3173455]

6. Delaney JA, Jensky NE, Criqui MH, et al. The association between physical activity and both incident coronary artery calcification and ankle brachial index progression: the multi-ethnic study of atherosclerosis. Atherosclerosis. 2013; 230:278–83. [PubMed: 24075757]
7. Lee CD, Jacobs DR Jr, Hankinson A, et al. Cardiorespirtaory fitness and coronary artery calcification in young adults: the CARDIA study. Atherosclerosis. 2009; 203:263–268. [PubMed: 18653190]
8. Folsom AR, Evans GW, Carr JJ, et al. Association of traditional and nontraditional cardiovascular risk factors with coronary artery calcification. Angiology. 2004; 55:613–623. [PubMed: 15547647]
9. Taylor AJ, Watkins T, Bell D, et al. Physical activity and the presence and extent of calcified coronary atherosclerosis. Med Sci Sports Exerc. 2002; 34:228–233. [PubMed: 11828230]
10. Bastos Goncalves F, Voute MT, Hoeks SE, et al. Calcification of the abdominal aorta as an independent predictor of cardiovascular events: a meta-analysis. Heart. 2012; 98:988–994. [PubMed: 22668866]
11. An C, Lee HJ, Lee HS, et al. CT-based abdominal aortic calcification score as a surrogate marker for predicting the presence of asymptomatic coronary artery disease. Eur Radiol. 2014:2491–98. [PubMed: 25030461]
12. Hamer M, Kivimaki M, Lahiri A, et al. Walking speed and subclinical atherosclerosis in healthy older adults: the Whitehall II study. Heart. 2010; 96:380–384. [PubMed: 19955091]
13. Williams PT, Thompson PD. Walking versus running for hypertension, cholesterol, and diabetes mellitus risk reduction. Arterioscler Thromb Vasc Biol. 2013; 33:1085–91. [PubMed: 23559628]
14. Ornish D, Brown SE, Scherwitz LW, et al. Can lifestyle changes reverse coronary heart disease: the Lifestyle Heart Trial. Lancet. 1990; 336:129–133. [PubMed: 1973470]
15. Ornish D, Scherwitz LW, Billings JH, et al. Intensive lifestyle changes for reversal of coronary heart disease. JAMA. 1998; 280:2001–07. [PubMed: 9863851]
16. Schuler G, Hambrecht R, Schlierf G, et al. Regular physical exercise and low-fat diet: effects on progression of coronary artery disease. Circulation. 1992; 86:1–11. [PubMed: 1617762]
17. Niebaurer J, Hambrecht R, Velich T, et al. Attenuated progression of coronary artery disease after 6 years of multifactorial risk intervention: role of physical exercise. Circulation. 1997; 96:2534–2541. [PubMed: 9355890]
18. Hamer M, Venuraju SM, Lahiri A, et al. Objectively assessed physical activity, sedentary time, and coronary artery calcification in healthy older adults. Arterioscler Thromb Vasc Biol. 2012; 32:500–505. [PubMed: 22075247]
19. Bertoni AG, Whitt-Glover MC, Chung H, et al. The association between physical activity and subclinical atherosclerosis: the Multi-Ethnic Study of Atherosclerosis. Am J Epidemiol. 2009; 169:444–454. [PubMed: 19075250]
20. Powell KE, Paluch AE, Blair SN. Physical activity for health: what kind? How much? How intense? On top of what? Annu Rev Public Health. 2011; 32:349–65. [PubMed: 21128761]
21. Eijsvogels TMH, Molossi S, Lee D, et al. Exercise at the extremes: the amount of exercise to reduce cardiovascular events. Journal of Am Coll of Cardiol. 2016; 67(3):316–29. [PubMed: 26796398]
22. Criqui MH, Denenberg JO, Ix JH, et al. Calcium density of coronary artery plaque and risk of incident cardiovascular events. JAMA. 2014; 311(3):271–278. [PubMed: 24247483]
23. Hou ZH, Lu B, Gao Y, et al. Prognostic value of coronary CT angiography and calcium score for major adverse cardiac events in outpatients. JACC Cardiovasc Imaging. 2012; 5(10):990–999. [PubMed: 23058065]
24. Criqui MH, Kamineni A, Allison MA, et al. Risk factor differences for aortic versus coronary calcified atherosclerosis: the multiethnic study of atherosclerosis. Arterioscler Thromb Vasc Biol. 2010; 30:2289–96. [PubMed: 20814018]
25. Criqui MH, Denenberg JO, McClelland RL, et al. Abdominal aortic calcium, coronary artery calcium, and cardiovascular morbidity and mortality in the Multi-Ethnic Study of Atherosclerosis. Arterioscler Thromb Vasc Biol. 2014; 34:1574–9. [PubMed: 24812323]
26. Straub RH, Tanko LB, Christiansen C, et al. Higher physical activity is associated with increased androgens, low interleukin 6 and less aortic calcification in perihperhal obese elderly women. J Endocrinol. 2008; 199:61–8. [PubMed: 18617605]

27. Doherty TM, Fitzpatrick LA, Inoue D, et al. Molecular, endocrine, and genetic mechanisms of arterial calcification. Endocr Rev. 2004; 25:629–72. [PubMed: 15294885]
28. Hruby A, O'Donnell CJ, Jacques PF, et al. Magnesium intake is inversely associated with coronary artery calcification: the Framingham Heart Study. J Am Coll Cardiol Img. 2014; 7:59–69.
29. O'Donnell CJ, Chazaro I, Wilson PWF, et al. Evidence for heritability of abdominal aortic calcific deposits in the Framingham Heart Study. Circulation. 2002; 106:337–341. [PubMed: 12119250]
30. Odink AE, van der Lugt A, Hofman A, et al. Association between calcification in the coronary arteries, aortic arch and carotid arteries: the Rotterdam Study. Atherosclerosis. 2007; 193:408–13. [PubMed: 16919637]
31. Stefanick ML, Mackey S, Sheehan M, et al. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. N Engl J Med. 1998; 339:12–20. [PubMed: 9647874]
32. Wang JS, Jen CJ, Kung HC, et al. Different effects of strenuous exercise and moderate exercise on platelet function in men. Circulation. 1994; 90:2877–2885. [PubMed: 7994833]
33. Ernst E. Regular exercise reduces fibrinogen levels: a review of longitudinal studies. Br J Sports Med. 1993; 27:175–176. [PubMed: 8242274]
34. Wolfgang K, Sund M, Döring A, et al. Leisure-time physical activity but not work-related physical activity is associated with decreased plasma viscosity: results from a large population sample. Circulation. 1997; 95:335–341. [PubMed: 9008446]
35. Sherman DL. Exercise and endothelial function. Coronary Artery Dis. 2000; 11:117–122.
36. Niebauer J, Cooke JP. Cardiovascular effects of exercise: role of endothelial shear stress. J Am Coll Cardiol. 1996; 28:1652–1660. [PubMed: 8962548]
37. Carr JJ, Nelson JC, Wong ND, et al. Calcified coronary artery plaque measurement with cardiac CT in population-based studies: standardized protocol of Multi-Ethnic Study of Atherosclerosis (MESA) and Coronary Artery Risk Development in Young Adults (CARDIA) study. Radiology. 2005; 234:35–43. [PubMed: 15618373]

Highlights

- The NHLBI's Family Heart Study data demonstrate that walking is associated with coronary artery calcification (J-shaped association), but not aortic calcification, in adult men and women without known coronary heart disease. This association persists even among those who are overweight or obese.
- This is one of the few studies to evaluate walking activity, an essential and sustainable form of physical activity, in relation to subclinical atherosclerosis and the first to examine the association between walking and aortic calcification in adult and men.
- This study highlights the importance of moderate activity as a key component in the primary prevention of cardiovascular disease.
- Further studies with a long-term follow-up are needed to more clearly elucidate the association between physical activity and calcified atherosclerotic plaque evolution in the aorta and coronary arteries over time.

Table 1

Participant characteristics in categories of walking metabolic activity (n=2,971)

Variable	≤ 3.75 met-hr/wk (n=829)	>3.75–7.5 met-hrs/wk (n=423)	>7.5–15 met-hrs/wk (n=873)	>15–22.5 met-hrs/wk (n=450)	>22.5 met-hrs/wk (n=392)
Age, years	56.8±12.8	55.2±13.5	54.2±12.5	53.6±12.5	54.2±11.8
Women (n, %)	550 (66.3%)	257 (60.8%)	492 (56.4%)	243 (54.0%)	243 (62.0%)
Race (n, %)					
White	576 (69.7%)	362 (86.0%)	725 (83.9%)	390 (86.9%)	340 (87.2%)
Black	250 (30.3%)	59 (14.0%)	139 (16.1%)	59 (13.1%)	50 (12.8%)
Marital status					
Never married	77 (9.3%)	28 (6.6%)	47 (5.4%)	29 (6.4%)	23 (5.9%)
Married	553 (66.7%)	296 (70.0%)	671 (76.9%)	361 (80.2%)	308 (78.6%)
Separated/divorced/widowed	199 (24.0%)	99 (23.4%)	155 (17.8%)	60 (13.3%)	61 (15.6%)
Total family Income					
<$25,000	226 (27.8%)	85 (20.6%)	126 (14.8%)	54 (12.3%)	47 (12.2%)
≥$25,000 – <$75,000	409 (50.4%)	215 (52.1%)	456 (53.5%)	233 (53.2%)	204 (53.0%)
≥$75,000	177 (21.8%)	113 (27.4%)	271 (31.8%)	151 (34.5%)	134 (34.8%)
BMI, kg/m^2	31.2±7.0	29.6±6.1	28.9±5.8	28.7±5.8	27.9±5.5
Hypertension (n, %)	473 (57.1%)	170 (40.2%)	334 (38.2%)	150 (33.3%)	129 (32.9%)
SBP (mmHg)	126.6±23.5	122.4±19.0	120.9±20.7	119.0±17.8	118.9±19.8
DBP (mmHg)	72.1±11.0	70.7±10.1	70.9±10.1	71.0±10.1	70.1±10.9
Diabetes Mellitus (n, %)	138 (16.7%)	38 (9.0%)	97 (11.1%)	35 (7.8%)	35 (8.9%)
Current Smokers (n, %)	111 (13.4%)	49 (11.6%)	110 (12.6%)	46 (10.2%)	46 (11.7%)
Pack years smoked	9.1±19.6	8.4±18.1	9.0±17.7	9.0±18.2	8.3±15.8
Triglycerides (mg/dL)	141.5±90.9	142.8±94.4	133.4±83.9	132.9±88.4	124.8±94.2

Variable	≤ 3.75 met-hr/wk (n=829)	>3.75–7.5 met-hrs/wk (n=423)	>7.5–15 met-hrs/wk (n=873)	>15–22.5 met-hrs/wk (n=450)	>22.5 met-hrs/wk (n=392)
LDL Cholesterol (mg/dL)	112.6±33.8	114.7±34.1	114.0±35.2	116.0±32.6	111.9±32.0
Average hours of TV/computer per day	3.3±2.5	2.7±2.0	2.6±1.7	2.4±1.7	2.2±1.4
Alcoholic drinks per week, #	2.6±6.6	3.7±7.7	3.8±6.8	4.0±8.3	5.3±13.2

BMI = body mass index, LDL = low density lipoprotein

Characteristics are shown as follows: mean ± standard deviation for continuous variables and frequencies (percentages) for categorical variables.

Table 2

Prevalence ratios and 95% confidence intervals of CAC based on walking metabolic activity in 2,971 participants from the NHLBI Family Heart Study

	Cases/n	*Crude	Age and sex adjusted	Full model
≤ 3.75 met-hrs/wk	206/829	1.0	1.0	1.0
>3.75–7.5 met-hrs/wk	82/423	0.73 (0.55–0.97)	0.70 (0.50–0.98)	0.53 (0.35–0.79)
>7.5–15 met-hrs/wk	176/873	0.76 (0.60–0.97)	0.81 (0.62–1.06)	0.72 (0.52–0.99)
>15–22.5 met-hrs/wk	83/450	0.68 (0.52–0.91)	0.72 (0.51–1.01)	0.54 (0.36–0.81)
>22.5 met-hrs/wk	79/392	0.76 (0.57–1.02)	0.92 (0.64–1.31)	0.76 (0.50–1.15)
p for trend		0.02	0.32	0.01

*The crude model adjusted for familial clustering. The second model adjusted for age, sex and familial clustering. The full model adjusted for age, sex, race, smoking, alcohol use, total exercise metabolic activity index (excluding walking), and familial clustering.

CAC = coronary artery calcification, defined as CAC>100

Table 3

Prevalence ratios and 95% confidence intervals of AC based on walking metabolic activity in 2,976 participants from the NHLBI Family Heart Study

	Cases/n	*Crude	Age and sex adjusted	Full model
≤ 3.75 met-hrs/wk	443/830	1.0	1.0	1.0
>3.75–7.5 met-hrs/wk	215/424	0.91 (0.72, 1.14)	1.04 (0.76, 1.45)	1.11 (0.75–1.64)
>7.5–15 met-hrs/wk	415/876	0.79 (0.64, 0.96)	0.92 (0.72, 1.19)	0.89 (0.66–1.20)
>15–22.5 met-hrs/wk	216/450	0.81 (0.63, 1.03)	1.02 (0.74, 1.40)	1.11 (0.75–1.64)
>22.5 met-hrs/wk	183/392	0.77 (0.60, 0.98)	0.92 (0.67, 1.27)	0.97 (0.66–1.44)
P trend		0.01	0.63	0.75

*The crude model adjusted for familial clustering. The second model adjusted for age, sex and familial clustering. The full model adjusted for age, sex, race, smoking, alcohol use, total exercise metabolic activity (excluding walking), and familial clustering.

AC = aortic calcification, defined as AC>100

Table 4

Subgroup analysis by BMI ≥ 25 kg/m^2 across categories of walking activity for CAC in 2,251 participants

	Cases/n	*Crude	Age and sex adjusted	Full model
≤ 3.75 met-hrs/wk	171/688	1	1	1
>3.75–7.5 met-hrs/wk	66/329	0.76 (0.56–1.04)	0.72 (0.49–1.04)	0.52 (0.34–0.82)
>7.5–15 met-hrs/wk	130/643	0.77 (0.59–1.00)	0.76 (0.56–1.03)	0.64 (0.45–0.92)
>15–22.5 met-hrs/wk	61/326	0.70 (0.51–0.96)	0.68 (0.46–1.01)	0.53 (0.34–0.82)
>22.5 met-hrs/wk	57/260	0.85 (0.60–1.20)	0.95 (0.63–1.45)	0.73 (0.45–1.19)
P trend		0.09	0.30	0.02

*The crude model adjusted for familial clustering. The second model adjusted for age, sex and familial clustering. The full model adjusted for age, sex, race, smoking, alcohol use, total exercise metabolic activity (excluding walking), and familial clustering.