



Original article

Leisure-time physical activity and mortality in a multiethnic prospective cohort study: the Northern Manhattan Study



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ARTICLE INFO

Article history:

Received 4 December 2014

Accepted 9 April 2015

Available online 18 April 2015

Keywords:

Physical activity

Obesity

Mortality

ABSTRACT

Purpose: To examine whether the survival benefit of exercise is modified by obesity.

Methods: In the Northern Manhattan Study, we collected baseline sociodemographics and cardiovascular disease risk factors. The primary exposure was leisure-time physical activity (LTPA) and the outcomes were total, vascular, and nonvascular deaths (non-VaD). LTPA was defined as any versus none and metabolic equivalent score category (total activity weighted by intensity). We used Cox models to estimate the hazard ratios (HRs) and 95% confidence intervals (CIs).

Results: A total of 3298 participants (mean age 69 years, 52% Hispanic, 63% women) were followed over a mean of 11.8 years with 1589 total deaths (641 vascular, 819 nonvascular). Any activity (adjusted HR: 0.84, 95% CI: 0.75–0.94) was associated with reduced risk of all-cause mortality and non-VaD, but not VaD. We found an interaction ($P < .05$) of LTPA with body mass index (BMI) less than 30 for all-cause and vascular mortality. Any LTPA was associated with reduced all-cause mortality (adjusted HR: 0.77, 95% CI: 0.68–0.87) and VaD (adjusted HR: 0.79, 95% CI: 0.65–0.97) only among those with BMI less than 30.

Conclusions: We found no evidence of an independent survival benefit of LTPA among those with BMI more than 30. The health benefits of exercise should be considered in the context of obesity.

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Introduction

The adoption of a healthy lifestyle is associated with multiple benefits, notably a reduction in incident coronary heart disease and stroke [1,2]. These benefits extend to cardiovascular disease mortality [3], all-cause mortality [4], and cognitive function [5,6], and the effect extends throughout the life span indicating that “it is never too late to start” [7]. The effects on nonvascular mortality, such as cancer, however have not been as well established. The components of a healthy life have been variably delineated by different organizations and epidemiologic studies, although common to most recommendations is a program of regular leisure-time physical activity (LTPA). The benefits of a regular physical activity program influence multiple biological and emotional pathways that

ultimately translate to health benefits across multiple diseases. Physical activity is also an integral component in the lifestyle modifications recommended for the control of cardiovascular disease risk factors [8–10] and prevention of other chronic health conditions [11]. Physical activity also independently influences multiple biological processes, leading to a more beneficial profile in inflammatory and coagulation cascades, as well as improved endothelial cell function [12].

Although the benefits of physical activity have been well established, there are less data on whether the effects could be modified by other known risk factors. In one study, for example, active smokers did not have a protective effect on coronary heart disease from LTPA [13]. In another study, the effect of physical activity on cardiovascular disease was present among individuals with recommended, as well as elevated, body mass indices (BMIs), leading to the concept of the “healthy obese” [14]. This construct, however, has been called into question [15]. These prior studies have rarely included urban dwelling-elderly and Hispanic populations, two segments of the population in whom the obesity epidemic has not abated and physical inactivity is common [16,17].

No potential conflicts of interest relevant to this article were reported.

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<http://dx.doi.org/10.1016/j.annepidem.2015.04.001>

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In our study, we explored the association of physical activity with vascular and nonvascular mortality in an urban dwelling triethnic population. We hypothesized that those who were obese would have a lesser protective effect from physical activity on risk of mortality compared with the nonobese.

Methods

Recruitment of the cohort

The Northern Manhattan Study (NOMAS) is a population-based study designed to evaluate the impact of medical, socioeconomic, and other risk factors on the incidence of vascular disease in a stroke-free cohort. Participants were identified by dual-frame random digit dialing in Northern Manhattan as previously described [18] and were eligible if they met the following criteria: (1) had never been diagnosed with a stroke; (2) were more than the age of 39 years; and (3) resided in Northern Manhattan for 3 months or more in a household with a telephone. The study was approved by the Institutional Review Boards at Columbia University Medical Center and the University of Miami. All participants gave informed consent to participate in the study.

Assessments at enrollment of the cohort

Baseline status and risk factors were collected through interviews of participants by trained bilingual research assistants. Physical examinations and in-person measurements were carried out by study physicians; fasting blood specimen phlebotomy was performed by study nurses. Race-ethnicity was determined by self-identification in response to a questionnaire modeled after the 2000 U.S. Census. Education was classified as completing high school versus not. Standardized questions were adapted from the Behavioral Risk Factor Surveillance System regarding the following conditions: hypertension, diabetes, and cigarette smoking. Standard techniques were used to measure blood pressure, height, weight, and fasting glucose and lipid panels as previously described [19]. Obesity was defined as a BMI more than 30. Hypertension was defined as blood pressure 140 mm Hg/90 mm Hg or more, a physician diagnosis of hypertension or a patient's self-report. Diabetes mellitus was defined as fasting blood glucose 126 mg/dL or more or the patient's self-report. Fasting blood samples were obtained and lipid profile was measured as previously described. Alcohol intake was ascertained with the use of previously validated questionnaires.

LTPA was measured using an in-person questionnaire adapted from the National Health Interview Survey of the National Center for Health Statistics [20]; it records the duration and frequency of various leisure-time activities for the 2 weeks before the interview. Participants who reported no physical activity were coded as inactive. For each activity, we obtained duration and frequency and if duration of activity was less than 10 minutes, it was coded as "no activity". This questionnaire has been previously reported as reliable and valid in this population [18]. This same measure also correlated with BMI, activities of daily living scores, and activity scores on a quality of well-being scale. Objective measures of physical fitness, moreover, as measured by exercise and treadmill testing or maximum oxygen uptake correlate well with physical activity questionnaires [21]. The participants' responses were correlated with compendia of physical activity to categorize the intensity of each activity in metabolic equivalents (MET) [22]. Total activity was summarized via the MET-score, whereby the MET for each individual activity is multiplied by the frequency per week and duration [23].

Follow-up and outcome measures

Participants are followed annually via phone screening to detect any new cardiac or neurologic symptoms, interval hospitalizations, medical conditions, or death. Complete loss to follow-up is present in less than 1% and is not associated with race-ethnicity [24]. Cause of death was ascertained through information gathered from the participant's family, review of medical records, and a copy of the death certificate if available. All-cause mortality was further divided into VaD and non-VaD. VaD included underlying heart disease (myocardial infarction, sudden cardiac death, congestive heart failure, and other cardiac arrhythmias), stroke, and pulmonary emboli.

Statistical analysis

Baseline characteristics were calculated as means for continuous variables and proportions for categorical variables. The 10-year cumulative risks of death were calculated using Kaplan-Meier method. The primary outcome was all-cause mortality, and the secondary outcomes were VaD and non-VaD; if cause of death could not be defined as VaD or non-VaD, participants were excluded from the secondary analyses. The primary exposure of interest was LTPA categorized as (1) any physical inactivity versus none and (2) quartiles of the MET-score weighted by total activity intensity. Because 40.8% of our cohort participants were physically inactive, we categorized the MET-score into three groups: the physically inactive as a reference (40.8%), intermediate level of MET-score (35.8%), and the highest level of MET-score (23.8%). Cox proportional hazard models were fitted to calculate hazard ratios (HRs) and 95% confidence interval (CI) for the association of LTPA with the risk of total mortality, non-VaD, and VaD. The models were unadjusted and fully adjusted for confounders including demographics (age, sex, and education) and cardiovascular disease risk factors (BMI, tobacco use, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, moderate alcohol consumption, hypertension, diabetes, and prior heart disease). We tested for interactions between physical activity with baseline sociodemographic and cardiovascular disease risk factors ran stratified models when the *P* for interaction was less than .05. Improvement of model fit including the interactions with the three categories of MET-score was tested using a χ^2 test with 2 degrees of freedom. The proportionality assumption was examined in all models. All statistical analyses were performed with SAS, version 9.3 (SAS Institute Inc., Cary, NC).

Results

Baseline demographics of our cohort are outlined in Table 1. Briefly, 40.8% of our sample was physically inactive and 52.4% were Hispanics, 62.8% women, and 72.5% had BMI less than 30. Over a mean 11.8 years of follow-up (minimum <1 year, maximum 20.5 years, interquartile range 8.4–15.1 years), there were 1589 deaths of which 641 were VaD, 819 were non-VaD, and 129 with insufficient data to classify. The 10-year cumulative risk probabilities of death were .30 (95% CI: 0.28–0.32) overall, .28 (95% CI: 0.26–0.30) for any LTPA group, and .32 (95% CI: 0.30–0.35) for no LTPA group (*P* for the difference between any LTPA vs. none = .013).

Physical activity and risk of all-cause mortality, non-VaD, and VaD

Table 2 provides unadjusted and fully adjusted associations of physical activity with mortality. In unadjusted analyses, any

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