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

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Abstract

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

Methods—In the Northern Manhattan Study we collected baseline socio-demographics and cardiovascular disease risk factors. The primary exposure was leisure-time physical activity (LTPA) and the outcomes were total, vascular, and non-vascular deaths. 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 and 95% confidence intervals (HR, 95% CI).

Results—A total of 3298 participants (mean age 69, 52% Hispanic, 63% women) were followed over a mean of 11.8 years with 1589 total deaths (641 vascular, 819 non-vascular). Any activity (adjusted HR 0.84, 95%CI:0.75,0.94) was associated with reduced risk of all-cause mortality and non-vascular death, but not vascular death. We found an interaction ($P< 0.05$) of LTPA with BMI<30 for all-cause and vascular mortality. Any LTPA was associated with reduced all-cause

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mortality (adjusted HR 0.77, 95%CI:0.68,0.87) and vascular death (adjusted HR 0.79, 95%CI: 0.65,0.97) only among those with BMI<30.

Conclusion—We found no evidence of an independent survival benefit of LTPA among those with BMI>30. The health benefits of exercise should be considered in the context of obesity.

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 epidemiological studies, though common to most recommendations is a program of regular leisure time physical activity. 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], as well as 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].

Though the benefits of physical activity have been well established, there is 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 leisure time physical activity [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, 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]. In our study we explored the association of physical activity with vascular and non-vascular mortality in an urban dwelling tri-ethnic population. We hypothesized that those who were obese would have a lesser protective effect from physical activity on risk of mortality compared to the non-obese.

Methods

Recruitment of the cohort

The Northern Manhattan Study (NOMAS) is a population-based study designed to evaluate the impact of medical, socio-economic, 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 over the age of 39 years; and (3) resided in Northern Manhattan for ≥3 months 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 body-mass index (BMI) > 30. Hypertension was defined as blood pressure ≥140 mm Hg/90 mm Hg, a physician diagnosis of hypertension, or a patient's self-report. Diabetes mellitus was defined as fasting blood glucose ≥126 mg/dl 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.

Leisure time physical activity (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 two weeks prior to 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 (VO_{2max}) 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 neurological symptoms, interval hospitalizations, medical conditions, or death. Complete loss to follow up is present in <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 vascular death (VaD) and non-vascular death. Vascular death 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 vs. none and 2) quartiles of the MET-score weighted by total activity intensity. Since 40.8% of our cohort 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 and 95% confidence interval (HR, 95% 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 (body mass index, 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 socio-demographic and cardiovascular disease risk factorsran stratified models when the *P* for interaction was < 0.05. Improvement of model fit including the interactions with the 3 categories of MET-score was tested using a chi-squared 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, 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<30. Over a mean 11.8 years of follow up (minimum < 1 year, maximum 20.5 years, interquartile range 8.4 to 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 0.30 (95% CI: 0.28, 0.32) overall, 0.28 (95% CI: 0.26, 0.30) for any LTPA group, and 0.32 (95% CI: 0.30, 0.35) for no LTPA group (*P* for the difference between any LTPA vs. none =0.013).

Physical activity and risk of all-cause mortality, non-vascular and vascular death

Table 2 provides unadjusted and fully adjusted associations of physical activity with mortality. In unadjusted analyses, any physical activity versus none was associated with a lower risk of all-cause, as well as non-vascular, mortality. The associations remained significant after adjusting for baseline demographics and other cardiovascular disease risk factors. We found similar results when LTPA was analyzed as categories of MET-score for all of our outcomes (table 3).

Effect modification of increased body mass index on the association of physical activity with the risk of non-vascular and vascular death

We tested for interactions between LTPA and baseline cardiovascular disease risk factors to further investigate for effect modification. There was evidence of an interaction between performing any activity and BMI <30 vs. ≥30 with all-cause mortality (*P* for interaction= 0.01) and VaD (*P* for interaction= 0.02), but not for non-VaD. We found a similar statistical interaction for the categories of the MET-score with BMI <30 vs. ≥30 (chi-squared with 2 degrees of freedom, *P* for interaction=0.04). In summary, compared to none, performing any LTPA was associated with lower risk of all-cause mortality and being in the highest category of the MET-score was associated with lower risk of VaD. These findings however were only seen, among those with a BMI less than 30, and not in those with a BMI ≥ 30 (Table 4). We did not find evidence of an interaction between baseline socio-demographics and cardiovascular disease risk factors with LTPA on mortality risk. We carried out further analyses excluding participants with malignancy (excluding skin cancers, n = 349) and any heart disease (n = 431) and the magnitude of associations in all analyses were unchanged (supplemental tables).

Discussion

In our prospective cohort of elderly, multi-ethnic, urban-dwelling individuals, we found that LTPA was associated with a lower risk of all-cause mortality and non-vascular death, but not with a lower risk of vascular death. Our study further tested the concept of the "metabolically healthy obese" or "fit but fat" [25, 26]. The protective effect of LTPA on all-cause mortality has been well characterized in multiple prospective cohort studies[27], as has the effect on VaD[28].

We, however, found lack of association between LTPA and vascular death, and, moreover, effect modification by obesity, such that protective effect of LPTA on vascular death risk was mitigated in the presence of an elevated BMI. Our results call into question the commonly held perception of "fit but fat", and argue that this group in the population also has a high risk of mortality. Our results should be interpreted with caution though, as they are intended to indicate that those with an elevated BMI should still continue to perform LTPA, as they gain an advantage in all-cause mortality. In a related manner LTPA is insufficient at protecting against vascular death alone, and high risk individuals should have a multi-faceted treatment approach targeted at "life's simple 7's" including consideration of weight, diet, and modification of cardiovascular disease risk factors as a whole [29].

The protective effect of LTPA on VaD among those with lower BMI is likely to be related to two different physiologic processes (independent or through other risk factors) leading to a reduction of myocardial infarction, stroke, and sudden cardiac death. A common finding is that LTPA improves the control of several other modifiable risk factors, such as hypertension[30] and diabetes[31], thereby reducing vascular mortality. This is likely to be an important component of the effect of LTPA, and in recently completed analyses LTPA had a similar protective effect to taking medications for chronic cardiovascular disease risk factors[32]. In our study, however, the protective effect of LTPA on VaD among those with a low BMI remained after adjusting for other cardiovascular disease risk factors. LTPA also

has independent effects from modulation of traditional risk factors, notably by improving inflammatory biomarker profiles and improvement in endothelial function and vascular reactivity[33, 34]. These latter processes may explain, in part, the protective effect of LTPA on non-vascular death and all-cause mortality regardless of BMI. In a recent meta-analysis, LTPA was found to have a potential protective effect on breast and colon cancer, with the effect in part explained by changes in inflammatory and glucose homeostasis pathways[35]. It is unclear however to what degree modulating these pathways with LTPA alters the natural history of malignant cells and the associated immune response, as opposed to protecting against incident cardiovascular disease events in patients with cancer. These mechanistic pathways should be explored in further detail in future studies.

In our study we also found that the survival benefit from LTPA did not extend to those with an elevated BMI. The reasons for this observation may be related to several unique aspects of our study. In the Northern Manhattan Study, we have a high proportion of participants who have not completed high school and are under-insured (Medicaid or no insurance). Both factors were associated with higher vascular disease mortality risk due to psychosocial stressors or impact of vascular disease risk factors, and these may not be easily overcome by heathy lifestyle such as LTPA[36]. In a prior analysis, we showed that LTPA was protective against markers of subclinical cerebrovascular disease only among those patients who had health insurance which is a proxy for access to care or socio-economic status in our cohort[37]. Our prospective cohort study also has a high proportion of elderly participants, including close to a quarter being over the age of 75 (data not shown), and traditional cardiovascular disease risk factors may not be as strongly associated with outcomes in the geriatric population [38]. Lastly in the elderly misclassification in the cause of death may occur, which in our study would influence the protective effect on VaD versus non-VaD, but not mortality overall.

Our study has several limitations. As with any study using self-reported data, the possibility of over-reporting LTPA may lead to misclassification bias. Study participants are likely to over-report, and not under-report, the amount and intensity of activity performed, which would likely lead to an under-estimate of the protective effect of LTPA. We examined one measure of physical activity and BMI at enrollment, rather than longitudinal activity and changes, which may not allow us to capture declines in physical activity associated with frailty and subsequent mortality[39]. The Northern Manhattan Study did not systematically capture non-leisure time physical activity, such as activity with work or commuting, though there is an independent effect of activity performed to improve fitness and health rather than for employment[40]. We also did not capture objective measures of fitness or sedentary behavior using accelerometers or cardiopulmonary exercise testing, which may limit our ability to fully capture the extent of physical activity or the independent effect of total sedentary time. Nonetheless, using a simple self-report questionnaire that mimics questions performed in routine clinical use, we have been able to demonstrate a survival benefit in a sample of older adults. Our study also did not collect important information on other confounders in the association between LTPA and mortality, notably socio-economic status, house-hold income, type of employment, self-classification of race within Hispanics, and insulin resistance among others. In NOMAS the sample size may be insufficient to detect more subtle protective effects from LTPA against VaD, and in other analyses including by

race-ethnicity or other baseline demographics. Lastly, the results of our study may not be generalizable to other populations.

Our study has important strengths, including a sample of older, predominantly Hispanic participants for whom less is known regarding risk of mortality, low loss to follow up, and comprehensive assessment of causes of death.

In conclusion, we found that LTPA was protective against all-cause mortality, while for vascular death the protective effect was present only among those who are not obese. Overall however the protective effects across multiple health domains from LTPA are clear, and all individuals should be encouraged to maintain a lifetime of physical activity across the age spectrum.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Baseline demographics of the Northern Manhattan Study

Socio-demographic characteristics		Entire cohort (n = 3298)	No leisure time physical activity (n = 1345)	Any leisure time physical activity (n = 1953)	p-value difference
		Mean (SD) or n (%)			
Age		69.2 (10.3) years	69.3 (10.3) years	69.3 (10.3) years	0.2
Women		2071 (62.8 %)	897 (66.7%)	1174(60.1%)	0.001
Men		1227 (37.2%)	448 (33.3%)	779 (39.9%)	reference
Race-ethnicity	Hispanic	1728(52.4%)	835 (62.1%)	893 (45.7%)	<0.0001
	Non-Hispanic Black	803 (24.3 %)	269 (20%)	534 (27.3%)	0.3
	Non-Hispanic White	690 (20.9 %)	213 (15.8%)	477 (24.4%)	reference
Less than high school		1786(54.2%)	840 (62.5%)	1007(51.6%)	<0.0001
education					
Medicaid or no insurance		1435 (43.8 %)	687(51.3%)	748 (38.6%)	<0.0001
Medical Comorbidities					
Tobacco use	Never used	1545(46.8%)	624 (46.4%)	921 (47.2%)	reference
	Former smoker	1191 (36.1%)	478 (35.6%)	713 (36.5%)	0.9
	Current user	560(17.0%)	242 (18.0%)	318 (16.3%)	0.2
Alcohol use	Never or heavy	2212 (67.1 %)	976 (72.6%)	1236 (63.3%)	<0.001
	Mild-moderate*	1086(32.7%)	369 (27.4%)	717 (36.7%)	reference
Hypertension		2429 (73.7%)	1017 (75.6%)	1412 (72.3%)	0.03
Diabetes mellitus††		716(21.7%)	324 (24.2%)	392 (20.1%)	0.005
Low-density lipoprotein cholesterol (mg/dl)		129 (36)	129 (36)	129 (36)	0.4
High-density lipoprotein cholesterol (mg/dl)		46(15)	46 (14)	48(15)	0.0002
Body mass-index greater than or equal 30		901 (27.5%)	404 (30.3%)	497 (25.5%)	0.003
Body mass-index less than 30		2379 (72.5%)	929 (69.7%)	1450 (74.5%)	reference

Table 2

Association of leisure-time physical activity (any activity versus none) with all-cause mortality (n = 1589), non-vascular death (n = 819), and vascular death (n = 641) in the Northern Manhattan Study†

	All-cause mortality: Hazard ratio and 95% confidence interval	Non-vascular death: Hazard ratio and 95% confidence interval	Vascular death: Hazard ratio and 95% confidence interval
Any leisure-time physical activity versus none (univariate)	0.87 (0.79-0.96)	0.87 (0.79-0.96)	0.89 (0.77-1.05)
Any leisure-time physical activity versus none (multi-variable) *	0.84 (0.75-0.94)	0.84 (0.75-0.94)	0.90 (0.76-1.08)

† 129 deaths with insufficient documentation to classify the cause of death

* Adjusted for age, race-ethnicity, high school education, health insurance, moderate alcohol use, tobacco use, hypertension, diabetes, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, body mass index any heart disease.

Table 3

Association of leisure-time physical activity (MET-score) with all-cause mortality, non-vascular death, and vascular death in the Northern Manhattan Study.

	All-cause mortality Hazard ratio and 95% confidence interval	**Non-vascular death Hazard ratio and 95% confidence interval**	**Vascular death Hazard ratio and 95% confidence interval**
Third quartile of MET-score versus no activity (univariate)	0.87 (0.78-0.97)	0.84 (0.71-0.98)	0.89 (0.75-1.07)
Fourth quartile of MET-score versus no activity (univariate)	0.87 (0.77-0.99)	0.86 (0.73-1.03)	0.91 (0.74-1.11)
Third quartile of MET-score (range) versus no activity *¥	0.87 (0.77-0.98)	0.83 (0.70-0.98)	0.92(0.75-1.11)
Fourth quartile of MET-score (range) versus no activity *¥	0.79 (0.69-0.91)	0.75 (0.62-0.91)	0.89 (0.71-1.10)

*Adjusted for age, race-ethnicity, high school education, health insurance, moderate alcohol use, tobacco use, hypertension, diabetes, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, body mass index, any heart disease.

¥p-value for the improvement of fit with chi-squared test with 2 degrees of freedom=0.003 for all-cause mortality, =0.01 for non-vascular death and =0.51 for vascular death.

Table 4

Association of leisure-time physical activity (any versus none) with all-cause mortality, non-vascular death, and vascular death in the Northern Manhattan Study based on body-mass index.

		All-cause mortality: ‡ Adjusted Hazard ratio and 95% confidence interval	Non-vascular death: Adjusted Hazard ratio and 95% confidence interval	Vascular death: ‡ Adjusted Hazard ratio and 95% confidence interval
Body-mass index less than 30	Any leisure-time physical activity versus none *	0.77 (0.68-0.88)	0.76 (0.64-0.91)	0.80 (0.65-0.97)
	Third quartile of MET-score versus no activity *	0.81 (0.71-0.93)	0.79 (0.65-0.96)	0.82 (0.66-1.04)
	Fourth quartile of MET-score versus no activity *	0.70 (0.59-0.83)	0.73 (0.59-0.90)	0.75 (0.59-0.97)
Body-mass index greater than or equal to 30	Any leisure-time physical activity versus none *	1.07 (0.86-1.34)	0.93 (0.68-1.28)	1.27 (0.91-1.79)
	Third quartile of MET-score versus no activity *	1.08 (0.84-1.38)	0.98 (0.69-1.39)	1.19 (0.81-1.74)
	Fourth quartile of MET-score versus no activity *	1.06 (0.79-1.43)	0.84 (0.54-1.31)	1.43 (0.93-2.20)

*Adjusted for age, race-ethnicity, high school education, health insurance, moderate alcohol use, tobacco use, hypertension, diabetes, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, any heart disease.

‡p for interactions between LPTA and BMI<30 vs ≥30 were < 0.05.