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Objectively measured physical activity, sedentary time and subclinical vascular disease: Cross-sectional study in older British men

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A B S T R A C T

Low physical activity (PA) and high levels of sedentary time (ST) are associated with higher cardiovascular disease (CVD) risk among older people. However, their independent contribution and importance of duration of PA and ST bouts remain unclear. We investigated associations between objectively measured PA, ST and non-invasive vascular measures, markers of CVD risk.

Cross-sectional study of 1216 men from the British Regional Heart Study, mean age 78.5 years, measured in 2010–2012. Carotid intima thickness (CIMT), distensibility coefficient (DC) and plaque presence were measured using ultrasound; pulse wave velocity (cfPWV) and augmentation index (AIx) using a Vicorder. PA and ST were measured using hip-worn ActiGraph GT3X accelerometers.

After adjusting for covariates, each additional 1000 steps per day was associated with a 0.038 m/s lower cfPWV (95% CI = −0.076, 0.0003), 0.095 10^{-3} kPa^{-1} higher DC (95% CI = 0.006, 0.185), 0.26% lower AIx (95% CI = −0.40, −0.12) and a 0.005 mm lower CIMT (95% CI = −0.008, −0.001). Moderate and vigorous PA (MVPA) was associated with lower AIx and CIMT, light PA (LPA) with lower cfPWV and CIMT and ST with higher cfPWV, AIx and CIMT and lower DC. LPA and ST were highly correlated ($r = -0.62$). The independence of MVPA and ST or MVPA and LPA was inconsistent across vascular measures. Bout lengths for both PA and ST were not associated with vascular measures.

In our cross-sectional study of older men, all PA regardless of intensity or bout duration was beneficially associated with vascular measures, as was lower ST. LPA was particularly relevant for cfPWV and CIMT.

1. Introduction

The role of higher physical activity levels (PA) and lower levels of sedentary time (ST) in reducing the risk of cardiovascular disease (CVD) in middle age is well established (Shiroma and Lee, 2010). Currently, UK physical activity guidelines recommend that older adults accumulate 150 min per week of at least moderate intensity activity in bouts lasting 10 min or more and minimise long periods of sitting (Department of Health, 2011). However, the importance of duration of spells of activity remains uncertain; studies in older adults have not used objectively measured PA to investigate whether bouts lasting 10 min or longer are more beneficial for CVD risk factors than either shorter bouts or total activity. Likewise, there is little information to suggest how often sedentary time should be interrupted, by what level of activity and for how long. In addition, little is known about the independence of PA and ST among older people, who not only have high risk of CVD but also have the highest levels of ST and very low levels of PA with poor adherence to physical activity guidelines (Jefferis et al., 2014). Furthermore, although the evidence base was too sparse to make recommendations about light activity in previous guidelines (Physical Activity Guidelines Advisory Committee, 2008), it may be that light intensity activity also has health benefits in this age-group (de Souto Barreto, 2015).

Non-invasive vascular markers provide valuable proxy indicators of CVD risk, permitting investigation of risk burden and development of subclinical CVD prior to onset of a CVD event. Greater arterial stiffness, measured by carotid femoral pulse wave velocity (cfPWV) (van Sloten et al., 2014), and carotid distensibility coefficient (DC) (van Sloten et al., 2014), wave reflection as indicated by Augmentation Index (AIx) (Wang et al., 2010), increased arterial wall thickening assessed by

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carotid intima-media thickness (CIMT) (van den Oord et al., 2013), and presence of carotid plaque (Inaba et al., 2012) are all associated with onset of CVD events. However, to date, very few studies have investigated objectively measured PA in relation to non-invasive vascular measures; the few that have are in young or middle-aged adults (Gomez-Marcos et al., 2014; Huynh et al., 2015; Kozakova et al., 2010; Andersson et al., 2015), smaller samples (Gomez-Marcos et al., 2014; Kozakova et al., 2010) or have evaluated a much narrower range of outcome measures (Kozakova et al., 2010; Laursen et al., 2015).

We therefore investigated associations between objectively measured physical activity of different intensities, sedentary time and a range of vascular measures, including cfPWV, DC, AIx, CIMT and presence of plaque, using a large sample of community-dwelling older men. We investigated whether (i) the intensity of activity was related to vascular measures, hypothesising adverse associations for sedentary time, and increasing benefits from light intensity activity upwards and (ii) whether time accumulated in bouts of PA or ST was related to vascular measures in a dose-dependent manner, hypothesising greater benefits from longer bouts of PA or increased risks from longer bouts of ST.

2. Material and methods

2.1. Sample

The British Regional Heart Study is a prospective, population-based cohort study following up 7735 men (>99% Caucasian) recruited from primary care practices in 24 British towns in 1978–80. In 2010–2012, 3137 surviving men were invited to a physical examination including non-invasive vascular measurements and to wear a physical activity monitor (accelerometer). A total of 1528 individuals accepted and returned an accelerometer with ≥3 days of data; 254 men with preexisting heart attack, heart failure or stroke were excluded. Of the remaining 1274 men, 1213 had data for all other relevant covariates, leaving 1118–1206 men for data analysis, depending on the vascular outcome. The National Research Ethics Service (NRES) Committee London provided ethical approval. Participants provided informed written consent to the investigation, in accordance with the Declaration of Helsinki.

2.2. Non-invasive vascular measures

Left and right carotid arteries were imaged using a Z.One Ultra ultrasound system (Zonare Medical Systems, Mountain View, CA) with a 5- to 10-mHz linear probe. A cross-sectional sweep from the base of the common carotid artery to the jaw bone and longitudinal images of the common carotid artery approximately 1 cm proximal to the carotid bifurcation were recorded. Ipsilateral brachial blood pressures were taken immediately after each carotid assessment (Omron HEM 907 recorder, mmHg). Peak systolic and end-diastolic common carotid artery diameter and CIMT were measured using Carotid Analyser software (Medical Imaging Applications, Iowa City, IA). From the longitudinal images, a region of interest (5–10 mm) was selected in a plaque free area, at least 1 cm from the bifurcation. CIMT was measured in three end-diastolic images on each side and the mean calculated. DC was calculated as previously described (Dijk, 2005). Mean values of right and left CIMT and DC were used in analyses. Ultrasound images were reviewed offline for presence of plaque, defined as a focal area of intima medial thickening ≥1.2 mm at its thickest point or with ≥50% thickness than the adjacent intima medial thickness.

Carotid femoral pulse wave velocity (cfPWV) was assessed using a Vicorder (Skidmore Medical, Bristol UK), with participants semi-supine. A 2 × 9-cm cuff was positioned around the neck with the bladder over the right carotid pulse, and a Hokanson SC10 cuff around the right thigh. Path length was measured from the sternal notch to the centre of the thigh cuff. The cuffs were simultaneously inflated and traces with a minimum of 3 good quality waveforms recorded. Two cfPWV measurements, within ≤0.5 m/s of each other, were accepted and averaged.

AIx was measured with participants seated using the Vicorder. A Hokanson SC10 cuff was positioned mid upper right arm, inflated to diastolic pressure, and once good quality waveforms were acquired, the signal was saved. Two recordings with both readings of augmentation pressure and AIx within ≤5% of each other were accepted and averaged.

All measures were made by 2 vascular technicians. The coefficients of variation for cfPWV, DC, AIx and CIMT were 4.7%, 12.7%, 14.6% and 7.7%, respectively; the agreement coefficient for presence of plaque was 0.8.

2.3. Physical activity

Men wore the GT3X accelerometer (ActiGraph, Pensacola, Florida) over the right hip for 7 days, during waking hours, removing it for swimming or bathing. Accelerometers were set to record movements on the vertical axis every 5 s, and data were integrated into 60 s epochs. Nonwear time was identified as reported previously (Jefferis et al., 2014) and excluded using the R package “Physical Activity” (Choi et al., 2011). Non-wear time was defined as periods of continuous zeros lasting more than 90 min; within these periods, up to 2 min of non-zero counts were allowed as non-wear time if no activity counts were detected during both the 30 min before and after that interval, to allow for the possibility of artefactual monitor movements (e.g., accidental movement of the monitor while left on a table). Therefore, any non-zero counts except the ≤2 min allowed within a period of zeros were considered as wear time. Valid wear days were defined as ≥600 min wear time, and participants with ≥3 valid days (92% of men who received an accelerometer) were included in analyses. Each minute of activity was categorised using intensity threshold values of counts per minute developed for older adults: <100 for sedentary time (ST) (<1.5 MET), 100–1040 for light activity (LPA) (1.5–3 MET) and >1040 for moderate and vigorous activity (MVPA) (≥3 MET) (Copeland and Esliger, 2009).

2.4. Other measures

Body mass index (BMI, kg/m^2) was calculated from height (Harpenden stadiometer) and weight in light indoor clothing (Tanita body composition analyser (BC-418 or Tanita scales if the participant had a pacemaker or defibrillator). The average of two seated blood pressure readings (Omron HEM-907 recorder, mmHg) were used. Heart rate (HR) was measured by electrocardiogram. Men self-completed a questionnaire including information about: current cigarette smoking, alcohol consumption, living alone, ever receiving a doctor diagnosis of heart attack, heart failure or stroke (with symptoms lasting >24 h), narrowing or hardening of the leg arteries (including claudication) (peripheral arterial disease), diabetes and current use of anti-hypertensive medication. Social class was based on longest held occupation at study entry (1978–80) and categorised as manual and non-manual. Region of residence (1978–80) was grouped into Scotland, North, Midlands and South of England.

3. Statistical methods

Men reporting a clinical diagnosis of heart attack, heart failure or stroke (with symptoms lasting >24 h) were excluded from analyses. Descriptive statistics for demographic characteristics, vascular measures, PA and ST were calculated by quartile of daily minutes of ST and MVPA.

We used regression models to investigate associations between each vascular and PA measure; linear regression models for cfPWV, DC, AIx and CIMT and logistic models for plaque. PA exposures investigated were total activity counts per day, steps per day and minutes per day of ST, LPA and MVPA. The mean difference (or OR) for each outcome