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Objectively measured physical activity and sedentary-time are associated with arterial stiffness in Brazilian young adults

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1. Introduction

Physical inactivity is a well-established risk factor for non-communicable diseases and premature mortality [1]. It influences cardiovascular risk factors, such as blood pressure, lipid profile and adiposity, and consequently, increases the risk of coronary heart disease [1–3]. In addition, new evidence suggests that the time spent in sedentary activities might be a risk factor for non-communicable diseases, independent of physical activity [4,5].

Non-communicable diseases, particularly cardiovascular diseases are the main causes of death in high- [6] and middle-income countries [7]. The prevalence of cardiovascular risk factors such as diabetes, hypertension and obesity has increased worldwide, and these risk factors are associated with unfavorable changes in lifestyle behaviors such as an unhealthy diet and low levels of physical activity [6,7]. Prevention of adult cardiovascular diseases implies detection and intervention in early life. Atherosclerosis is a chronic
inflammatory disease that has a long asymptomatic phase [8]
beginning in childhood and adolescence, and track into adult-
hood [9,10].

Early detection of subclinical atherosclerosis and arterioscle-
rosis is possible through the evaluation of arterial stiffness, intima-
media thickness and endothelial dysfunction, which can be
measured by noninvasive, reproducible, and inexpensive tech-
niques [11]. Arterial stiffness is associated with traditional cardio-
vascular risk factors, such as diabetes and hypertension [12,13],
and predicts increased risk of cardiovascular events and mortality [14].
Further, increased arterial stiffness in children and adolescents is
associated with obesity and dyslipidemia [15–17].

Arterial stiffness is lower among individuals who regularly
perform aerobic exercise [18,19], and short-term aerobic exercise
training reduces the stiffness in central arteries [19–21], however
this effect cannot be maintained without continued exercise [22].
The association between exercise training and aortic stiffness
observed in clinical studies are reinforced by observations in mu-
rine models suggesting that several genes identified involved in
vasodilation and arterial elasticity are overexpressed by exercise
[23].

Although an association between physical activity and arterial
stiffness has been observed, few studies [24,25] have measured
physical activity using objective methods and it is unknown
whether sedentary time is associated with arterial stiffness inde-
pendent of moderate-to-vigorous physical activity (MVPA) and
other potential confounding factors. We therefore examined the
independent associations between objectively measured physical
activity and sedentary time with pulse wave velocity (PWV) in
Brazilian young adults who have been prospectively followed up
since birth.

2. Methods

2.1. Subjects

In 1982, all hospital deliveries in Pelotas, a southern Brazilian
city, were identified and those live borns (n = 5914) whose families
lived in the urban area of the city were examined, and their
mothers interviewed. These individuals have thereafter been fol-
lowed on several occasions throughout their life-course (at the
mean ages of 1, 2, 4, 13, 15, 18, 19 and 23 years). Further details about
the methods of the cohort are available elsewhere [26–28].
The study was approved by the School of Medicine Ethics Committee
of the Federal University of Pelotas. All participants signed the
informed consent form.

Between June 2012 and February 2013, when participants were
on average 30 years, we tracked the entire cohort using multiple
strategies to locate cohort members. All participants were invited
to visit the research clinic for interviews and a clinical examination.

2.2. Physical activity

Physical activity was measured using the GENEActiv acceler-
ometer (Activinsights Ltd., Kimbolton, UK). The monitor was worn
on the non-dominant wrist. The GENEActiv activity monitor is
waterproof and measures acceleration in three axes (x, y, z) with a
sample frequency of 85.7 Hz. Data are stored directly as sampled
waterproof and measures acceleration in three axes (x, y, z) with a
on the non-dominant wrist. The GENEActiv activity monitor is

2.3. Pulse wave velocity

The carotid-femoral PWV (meters/second) was examined twice
during the clinical visit using a portable ultrasound, Sphygmocor®
(Atcor Medical version 9.0, Sydney, Australia) in the supine position
and measurements were taken in the right side. An electrocardio-
gram was registered at the same time. Duration of the examination
was 10–15 min for each participant. The distance of pulse wave
transit was measured by a flexible tape as the distance from
suprasternal notch to femoral point of application of the tonometer
and the distance from carotid point of tonometer application and
the suprasternal notch. PWV was calculated by the software as the
distance between the measurement sites divided by transit time
delay between femoral and carotid pulse wave. The mean of mea-
surements was used in the data analysis.

Training for PWV assessment was carried out in two days using
volunteers. PWV was calculated by the software as the distance
between the measurement sites divided by transit time delay be-
tween femoral and carotid pulse wave. The software evaluated the
quality of each measurement according to the format of wave and synchronism with wave from electrocardiogram. This protocol followed recommendations from expert consensus document on arterial stiffness and the Research Applications Manual of SphygmoCor [31,32].

2.5. Statistical analysis

Data analysis was carried out using Stata 12.0 (StataCorp, College Station, TX, USA). Description of the sample with complete data of PWV was described in proportion or mean and standard deviation, according to type of variable. Linear regression models were used in the crude and adjusted analyses using all exposures in quartiles to assess possible dose-response. Statistical significance was obtained using two-sided Wald’s tests. Possible confounders were: sex, skin color, family income at birth, socioeconomic status and current smoking. The association of MVPA and sedentary time with PWV was also mutually adjusted between them. We evaluated whether the associations were modified by sex. Mediation analyses for BMI, WC, SBP and DBP were carried out using the g-computation formula [37]. This analysis estimates the direct effect of acceleration, sedentary time and MVPA on PWV at 30 years, and the indirect effect that was mediated through BMI, WC, SBP and DBP, separately. Sex, skin color, family income at birth, socioeconomic status and current smoking were considered as base confounders, whereas daily energy intake was considered as post confounder. Fig. 1 shows the direct acyclic graph of this analysis. G-computation formula adjusts the estimates for base confounders — variables that affect both main exposures and outcome — and post confounders — variables not previously included in the model that can be affected by exposures and related to the mediating variable. In this analysis, sex, skin color, family income at birth, socioeconomic status and current smoking were considered as base confounders. Daily energy intake was considered as post confounder. Total effect (relationship between each exposure and outcome, with and without influence of the mediator), direct effect (relationship between each exposure and outcome without influence of the mediator), indirect effect (relationship between each exposure and outcome with influence of the mediator — captured by the mediator). A p-value of 0.05 was used to assign statistical significance (Fig. 2).

3. Results

At 30 years of age, 3701 individuals were located and examined representing a follow-up rate of 68.1% (following exclusion of 325 known deaths). Due to practical reasons (delay in the arrival of the equipment and tonometer with problem later) PWV was measured in 1576 participants (42.6% of those interviewed) and data on physical activity were available in 2740 participants. In total, 1241 participants provided information on both physical activity and PWV and were included in the present analyses. Table 1 shows that the proportions of females among those who were examined at 30 years and those with complete data were 52% and 49%, respectively (p = 0.02). Individuals with and without complete information from accelerometry and PWV were also statistically different concerning skin color, family income at birth, smoke, sedentary time and waist circumference. Spearman correlation coefficient between MVPA and sedentary time was −0.45 (p < 0.001).

Because there was no evidence of interaction with sex in the

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**Fig. 1.** Direct acyclic graph of the effect of physical activity and sedentary time on pulse wave velocity (PWV).
The association between accelerometry and PWV (p-values for interaction ranging from 0.18 to 0.58), all analyzes were performed sex combined adjusting for sex as a confounder. Fig. 1 shows that PWV among those individuals who spent less than 30 min/day in MVPA was not statistically lower than that observed among those with 0 min/day in MVPA. However, PWV was lower among individuals who achieved the recommendation of ≥30 min/day of MVPA [−0.35 m/s (95%CI: −0.56; −0.14)].

Acceleration was inversely related to PWV - subjects in the highest quartile of acceleration had on average −0.37 m/s [β = −0.37 m/s (95%CI: −0.56; −0.19)] of PWV than those in the first quartile. Individuals in the third and fourth quartile of time spent in MVPA had similar reductions in PWV. Conversely, participants in the highest quartile of sedentary time had on average 0.36 m/s [β = 0.36 m/s (95%CI: 0.17; 0.52)] of PWV than those in the lowest quartile. Association between MVPA in quartiles and PWV remained after adjustment for sedentary time (p = 0.046), although beta coefficients reduced for −0.18 m/s in the two highest quartiles [β = −0.18 m/s (95%CI: −0.36; −0.01–95%CI: −0.37; 0.01, in third and fourth quartile, respectively)], and 95% confidence interval was not statistically significant in the fourth quartile. On the other hand, PWV was substantially higher in the highest quartile of sedentary time, even after adjustment for MVPA [β = 0.28 m/s (95%CI: 0.09; 0.47)] (Table 2).

Table 3 shows that DBP captured about 46% of the effect of acceleration on PWV. Concerning the association between MVPA and PWV, 44% of this association was explained by WC, whereas DBP captured only a small proportion (27%) of the association between sedentary time and PWV.

4. Discussion

In an analysis involving 1259 young adults from Brazil, we observed that subjects who were more physically active had lower PWV. Subjects who spent more than 30 min/day in MVPA had a lower PWV. In addition, PWV was positively associated with sedentary time. Coefficients from association between MVPA and sedentary time and PWV changed in mutually adjusted analyses and were more consistent for results using sedentary time. WC and DBP were important mediators in the association of MVPA and sedentary time with PWV.

Similarly to our study, an inverse association between physical activity and the central augmentation index has been observed among adults aged 55 years, on average[24]. However, one study carried out among children failed to observe an association between physical activity intensity and stiffness in the carotid arteries[38]. These null findings are possibly related to the small sample studied and the low probability that these children had to develop

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**Fig. 2.** Pulse wave velocity (PWV, m/s) according to time spent in moderate-to-vigorous physical activity in young adults from the 1982 Pelotas Birth Cohort.

**Table 1** Comparison between all participants of the 30 years follow up visit and those with measurements of pulse wave velocity (PWV). The 1982 Pelotas (Brazil) Birth Cohort.

<table>
<thead>
<tr>
<th></th>
<th>Cohort members followed-up in 2012/3</th>
<th>Participants with complete information</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>1787</td>
<td>48.3</td>
<td>633</td>
</tr>
<tr>
<td>Females</td>
<td>1914</td>
<td>51.7</td>
<td>608</td>
</tr>
<tr>
<td>Skin color</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>2817</td>
<td>76.1</td>
<td>919</td>
</tr>
<tr>
<td>Non-white</td>
<td>884</td>
<td>23.9</td>
<td>322</td>
</tr>
<tr>
<td>Family income at birth (minimal wages)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>730</td>
<td>19.8</td>
<td>252</td>
</tr>
<tr>
<td>1.1-3</td>
<td>1816</td>
<td>49.3</td>
<td>642</td>
</tr>
<tr>
<td>3.1-6</td>
<td>721</td>
<td>19.6</td>
<td>223</td>
</tr>
<tr>
<td>6.1-10</td>
<td>222</td>
<td>6.0</td>
<td>70</td>
</tr>
<tr>
<td>&gt;10</td>
<td>195</td>
<td>5.3</td>
<td>49</td>
</tr>
<tr>
<td>Current smoker</td>
<td>854</td>
<td>23.5</td>
<td>332</td>
</tr>
<tr>
<td>Overweight/Obese</td>
<td>2042</td>
<td>56.6</td>
<td>715</td>
</tr>
<tr>
<td>Sedentary time (min/day)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st quartile (317.8–623.9)</td>
<td></td>
<td></td>
<td>685</td>
</tr>
<tr>
<td>2nd (624.0–684.8)</td>
<td>687</td>
<td>25.1</td>
<td>301</td>
</tr>
<tr>
<td>3rd (684.8–739.7)</td>
<td>688</td>
<td>25.1</td>
<td>310</td>
</tr>
<tr>
<td>4th quartile (739.8–952.1)</td>
<td></td>
<td></td>
<td>678</td>
</tr>
<tr>
<td>MVPA (min/day)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st quartile (0–5.3)</td>
<td>685</td>
<td>25.0</td>
<td>300</td>
</tr>
<tr>
<td>2nd (5.3–16.3)</td>
<td>683</td>
<td>25.0</td>
<td>287</td>
</tr>
<tr>
<td>3rd (16.3–34.6)</td>
<td>681</td>
<td>25.0</td>
<td>321</td>
</tr>
<tr>
<td>4th quartile (34.6–379.6)</td>
<td></td>
<td></td>
<td>681</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>[n - mean(sd)]</td>
<td></td>
<td>3567</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg) [n - mean(sd)]</td>
<td></td>
<td></td>
<td>3592</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg) [n - mean(sd)]</td>
<td></td>
<td></td>
<td>3592</td>
</tr>
<tr>
<td>Daily energy intake (kcal) [n - mean(sd)]</td>
<td></td>
<td></td>
<td>3535</td>
</tr>
</tbody>
</table>

MVPA – Moderate-to-vigorous physical activity.
any arterial derangement at this age [38]. Vascular changes associated with the aging process are progressive, involve hypertrophy and hyperplasia of smooth muscle cells within the vascular tree, coupled with the modification of matrix proteins, and may occur before hypertension begins [39]. The continuous deposition of a variety of proteins, including collagen, coupled with progressive loss of the elastic matrix will result in arterial stiffening [40]. In normotensive volunteers, blood pressure increases slowly from young to older ages, resulting in age-associated increases of arterial stiffness [41]. These information support the idea that, although vascular changes begin early in life — what was seen in this study, it is expected that they could be incipient at that age. Our study draws attention to the influence of an active behavior on vascular stiffness in the first decades of life.

Recently, sedentary behavior has been recognized as a risk factor for obesity, diabetes and cardiovascular diseases, independent of MVPA [42]. Sedentary behavior is a different construct than physical inactivity, with both behaviors having different determinants [43]. Interestingly, in the present study both behaviors were shown to be associated with PWV: sedentary time was positively related to PWV and MVPA was inversely associated with PWV. Given the trend towards increased sedentary behaviors in children and adolescents [44], our findings among young adults suggest that more focus should be directed at increasing the volume of MVPA alongside decreasing sedentary behavior. A minimum of 30 min/day in MVPA resulted in improvements in arterial distensibility of our population.

There is debate in the literature whether the health effects of sedentary time are really independent of those of physical activity [45]. In order to deal with this issue, we present results adjusting one for each other, though beta coefficients were slightly higher for sedentary time.

Training positively affect aortic PWV [41,46] and central arterial distensibility [19]. This effect is due to qualitative structural

**Table 2**

<table>
<thead>
<tr>
<th>Variables included in the analysis as post confounder: daily energy intake.</th>
<th>N</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β (95% CI)</td>
<td>p</td>
<td>β (95% CI)</td>
</tr>
<tr>
<td><strong>Acc (mg)</strong></td>
<td>1241</td>
<td>0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>1st quartile</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>2nd</td>
<td>–0.03 (–0.20; 0.15)</td>
<td>–0.06 (–0.24; 0.13)</td>
<td>–</td>
</tr>
<tr>
<td>3rd</td>
<td>–0.21 (–0.39; –0.04)</td>
<td>–0.24 (–0.42; –0.06)</td>
<td>–</td>
</tr>
<tr>
<td>4th quartile</td>
<td>–0.30 (–0.47; –0.12)</td>
<td>–0.37 (–0.56; –0.19)</td>
<td>–</td>
</tr>
<tr>
<td><strong>MVPA (minutes)</strong></td>
<td>1238</td>
<td>0.004</td>
<td>0.001</td>
</tr>
<tr>
<td>1st quartile</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>2nd</td>
<td>0.00 (–0.19; 0.17)</td>
<td>–0.02 (–0.20; 0.15)</td>
<td>0.02 (–0.16; 0.20)</td>
</tr>
<tr>
<td>3rd</td>
<td>–0.22 (–0.39; –0.05)</td>
<td>–0.25 (–0.42; –0.07)</td>
<td>–0.18 (–0.36; 0.01)</td>
</tr>
<tr>
<td>4th quartile</td>
<td>–0.24 (–0.41; –0.07)</td>
<td>–0.29 (–0.46; –0.11)</td>
<td>–0.18 (–0.37; 0.01)</td>
</tr>
<tr>
<td><strong>Sedentary time (minutes)</strong></td>
<td>1259</td>
<td>0.002</td>
<td>0.001</td>
</tr>
<tr>
<td>1st quartile</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>2nd</td>
<td>0.07 (–0.10; 0.23)</td>
<td>0.09 (–0.08; 0.26)</td>
<td>0.06 (–0.12; 0.23)</td>
</tr>
<tr>
<td>3rd</td>
<td>0.14 (–0.02; 0.31)</td>
<td>0.16 (0.01; 0.33)</td>
<td>0.12 (–0.06; 0.29)</td>
</tr>
<tr>
<td>4th quartile</td>
<td>0.32 (0.15; 0.49)</td>
<td>0.35 (0.17; 0.52)</td>
<td>0.28 (0.09; 0.47)</td>
</tr>
</tbody>
</table>

Acc — Acceleration by raw data accelerometer; MVPA — Moderate-to-Vigorous Physical Activity. Model 1: Adjustment for sex, skin color, family income at birth, National Economic Indicator score and smoking. Model 2: Adjustment for Model 1 + MVPA/sedentary time.

P-values tested the heterogeneity between groups.

Table 3

<table>
<thead>
<tr>
<th>Pulse wave velocity (PWV, m/s)</th>
<th>Total effect β (95% CI)</th>
<th>Direct effect β (95% CI)</th>
<th>Indirect effect β (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acc — mg (quartile)</strong></td>
<td>BMI</td>
<td>–0.13 (–0.19; –0.07)</td>
<td>–0.09 (–0.15; –0.03)</td>
</tr>
<tr>
<td></td>
<td>WC</td>
<td>–0.13 (–0.19; –0.07)</td>
<td>–0.09 (–0.15; –0.03)</td>
</tr>
<tr>
<td></td>
<td>SBP</td>
<td>–0.13 (–0.19; –0.07)</td>
<td>–0.10 (–0.17; –0.04)</td>
</tr>
<tr>
<td></td>
<td>DBP</td>
<td>–0.13 (–0.19; –0.07)</td>
<td>–0.07 (–0.13; –0.01)</td>
</tr>
<tr>
<td><strong>MVPA — minutes (quartile)</strong></td>
<td>BMI</td>
<td>–0.09 (–0.15; –0.03)</td>
<td>–0.08 (–0.14; –0.02)</td>
</tr>
<tr>
<td></td>
<td>WC</td>
<td>–0.09 (–0.15; –0.03)</td>
<td>–0.05 (–0.11; 0.02)</td>
</tr>
<tr>
<td></td>
<td>SBP</td>
<td>–0.09 (–0.15; –0.03)</td>
<td>–0.07 (–0.13; –0.01)</td>
</tr>
<tr>
<td></td>
<td>DBP</td>
<td>–0.09 (–0.15; –0.03)</td>
<td>–0.06 (–0.12; 0.00)</td>
</tr>
<tr>
<td><strong>Sedentary time — minutes (quartile)</strong></td>
<td>BMI</td>
<td>0.11 (0.05; 0.17)</td>
<td>0.10 (0.04; 0.16)</td>
</tr>
<tr>
<td></td>
<td>WC</td>
<td>0.11 (0.05; 0.17)</td>
<td>0.10 (0.04; 0.16)</td>
</tr>
<tr>
<td></td>
<td>SBP</td>
<td>0.11 (0.05; 0.17)</td>
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</tr>
<tr>
<td></td>
<td>DBP</td>
<td>0.11 (0.05; 0.17)</td>
<td>0.08 (0.02; 0.14)</td>
</tr>
</tbody>
</table>

Acc — Physical activity evaluated by raw data accelerometer; MVPA — Moderate-to-Vigorous Physical Activity; BMI — Body Mass Index; WC — waist circumference; SBP — Systolic Blood Pressure; DBP — Diastolic Blood Pressure.

Variables included in the analysis as base confounders: sex, skin color, family income at birth, National Economic Indicator score and smoking.

Variables included in the analysis as post confounder: daily energy intake.
elements (interstitial collagen of the arterial wall that can react producing advanced glycation end-products) and functional elements (vasoconstrictor tone, endothelial function) that are influenced by physical activity. Aortic PWV modulation by exercise was shown to occur in parallel to changes in plasma concentrations of endothelin-1 (ET-1), independent of blood pressure, suggesting that ET-1 may be involved in the adaptations of arterial stiffness to exercise training [46]. Moreover, exercise can reduce chronic inflammation, improving endothelial function [48]. In addition, sedentary behavior may reduce glucose transporter protein content [49], leading to insulin resistance [50], which is associated with arterial stiffness independent of glucose tolerance status [51]. Waist circumference captured part of the relationship between MVPA and PWV. This can be due to the effect of physical activity on weight loss and in visceral fat [52], which in turn, are positively related to arterial stiffness [53,54].

High levels of sedentary time can coexist with high levels of MVPA [55]. In this case, it may be that sedentary time substitutes light-intensity physical activity [56] that, in turn, has been beneficially associated with health outcomes, such as blood glucose [57]. There is some evidence that not only the decrease in the total amount of physical activity, including light-intensity activity, is a concern for health, but also the sedentary time itself, operating through other mechanisms, such as decreasing lipoprotein lipase, which maintains cardiometabolic homeostasis and regulate lipid concentrations [58].

Possible limitations of our study include losses to follow up, particularly because due to operational reasons, we were unable to measure PWV and physical activity from accelerometry in all those followed up at 30 years. However, since this is an association study, these losses do not affect the relevance of our findings. In addition, though with statistical significance, the differences found showed very similar distribution of the individuals in the variables investigated. Moreover, the amount of days/time measure was variable depending on the day of the week the person was included. In addition, some had more weekdays, others less. Since the amount of activity varies between weekdays and weekend, this could have influenced the results. Another issue to be considered is that since only a couple of days/hours were collected, this may not represent routine life. Finally, since the changes in PWV are a chronic process, and the amount of activity may change over the years, it may well happen that the PWV measured today reflects the history of physical activity over the last years, which may not be related to today’s activity.

The most important strengths of the current study are the large and representative sample of young adults who had their levels of MVPA and sedentary time objectively evaluated by the use of accelerometer. In addition, early vascular disease was objectively assessed. Moreover, the availability of information to perform a mediating analysis helped to elucidate the pathways that connect physical activity and sedentary time to PWV.

In conclusion, the findings showed an inverse association between objectively measured physical activity and PWV, as well as a positive association between sedentary time and PWV. Such associations were only partially mediated by WC and DBP. Reducing sitting time and promoting physical activity is essential to help prevent the incidence of cardiovascular diseases across the lifespan.

Conflict of interest
None declared.

References


