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Prospective Association of Occupational and Leisure-Time Physical Activity With Cardiovascular Risk Factors in Early Adulthood: Findings From Pelotas (Brazil) 1982 Birth Cohort

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Background: The benefits of physical activity in health outcomes are well established. However, recent evidence suggests that benefits may differ by domain and population. Thus, we aimed to investigate the prospective association of occupational (OPA) and leisure-time physical activity (LTPA) with cardiovascular risk factors. ***Methods:*** In 1982, the maternity hospitals of Pelotas were visited daily; those live births whose families lived in urban areas were evaluated, and their mothers were later interviewed (n = 5914). In the 2004/5 follow-up (23 y old), both OPA and LTPA were measured in 4295 participants using their respective sections of the International Physical Activity Questionnaire. In the 2012 follow-up (30 y old), the following cardiovascular risk factors were collected: high-density lipoprotein (in milligrams per deciliter), low-density lipoprotein (in milligrams per deciliter), triglycerides (in milligrams per deciliter), glucose (in milligrams per deciliter), and blood pressure (in millimeters of mercury). Multivariable linear regressions were performed to evaluate associations between OPA and LTPA with these specific cardiovascular risk factors. ***Results:*** In total, 3241 participants were analyzed. Our main findings suggest that there was no association between OPA and LTPA with high- and low-density lipoprotein. There were inverse associations between OPA and lower levels of triglycerides among males ($\beta = -0.002$; 95% confidence interval, −0.003 to −0.000) and positive associations between LTPA and higher levels of diastolic blood pressure among females ($\beta = 0.111$; 95% confidence interval, 0.005–0.216). ***Conclusion:*** In conclusion, our findings suggest that there was no association, or association with limited clinical relevance, of OPA and LTPA with cardiovascular risk factors in early adulthood.

Keywords: blood pressure, high-density lipoprotein, low-density lipoprotein, triglycerides

The 2023 heart diseases and stroke statistics of the American Heart Association,[1] as well as the Brazilian cardiovascular statistics (Brazil 2021),[2] demonstrated that cardiovascular diseases (CVDs) are the leading cause of death globally, causing exorbitant health expenditures and decreasing life expectancy and quality of life. A cross-sectional study from the Brazilian National Health Survey conducted in 2013 on a sample of 60,202 adults aged over 18 years demonstrated that self-reported diagnosis of heart disease in Brazil was 4.2%, and this was associated with female sex, hypertension, elevated cholesterol, overweight, and/or obesity.[3]

Although the prevalence of CVD increases significantly with age, it is well documented that middle-aged adults can also be affected by some cardiovascular events.[1,2] This makes the investigation of the modifiable cardiovascular risk factors (eg, blood pressure and cholesterol) and their determinants relevant.

Despite the relatively well-documented physical activity (PA) effect on improvements in cardiovascular health,[4–6] recent studies have suggested that the benefits of PA might depend on the domain in which it is performed.[7–9] Previous studies, restricted to leisure-time PA (LTPA; eg, sports, recreation, and transportation), reported beneficial associations with cardiovascular outcomes and all-cause mortality.[10–13] More recently, a study conducted by Dalene et al (2021) suggested that moderate to high occupational PA (OPA) contributes to longevity in men. However, OPA does not seem to affect longevity in women.[14]

On the other hand, increasing evidence shows that OPA does not seem to improve health.[15–18] Previous evidence documented that high OPA might increase the risk for CVD and mortality outcomes, even after extensive adjustments for other potential confounding factors, including socioeconomic status, LTPA, and other health behaviors.[19,20] A recent umbrella review found that OPA is associated with a reduced risk of some outcomes (eg, some cancers and coronary heart disease), but high OPA was also associated with unfavorable health outcomes for all-cause mortality in men, such as mental health, osteoarthritis, and sleep quality and duration.[21]

This controversial phenomenon, known as the "PA paradox" (OPA has increasing health risks, whereas LTPA reduces such risk), is likely more evident among workers from a lower socioeconomic position with low job resources, low cardiorespiratory fitness, and pre-existing CVD.[20] The literature suggests that although LTPA often includes dynamic movements at conditioning intensity levels, sufficient to improve cardiorespiratory fitness,[22–24] OPA often requires static loading, heavy lifting, and monotonous and awkward working postures (with insufficient recovery time), increasing the cardiovascular risk.[20] Despite this, it may not be clear whether differential associations of OPA and LTPA with health are due to PA characteristics and their biological responses (eg, intensity, frequency, duration, and types of

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employment) or the limited methodology and residual confounding by aspects of socioeconomic demographics.

In addition, most of the studies on associations between PA and health outcomes have been designed and conducted in high-income countries. In these countries, the dynamics of work are different, and overall PA is positively associated with socioeconomic status, in opposition to what we observe in middle- and low-income countries where only leisure time is positively associated with socioeconomic status.[25] Finally, studies addressing the PA paradox in early adulthood are scarce.

Considering the approach to PA promotion and surveillance, which account for every movement regardless of context,[10] the importance of assessing PA in different domains, other outcomes, and research settings in low- and middle-income countries seems clear. Therefore, this study was aimed at assessing the prospective association of OPA and LTPA with cardiovascular risk factors among participants of the Pelotas (Brazil) 1982 Birth Cohort in early adulthood.

Methods

Study Design and Participants

In 1982, 7392 births were recorded in the maternity hospitals of Pelotas (Brazil). Briefly, all live births whose families lived in the urban area of the city were eligible for the Pelotas 1982 Birth Cohort Study[26] (n = 5914). Details on the study methodology have been previously published.[26] This study was reported according to Strengthening the Reporting of Observational Studies in Epidemiology (Supplementary Material S1 [available online]).[27] This study was carried out according to the Declaration of Helsinki, and all procedures involving human subjects were approved by the Research Ethics Committee of the College of Medicine, Federal University of Pelotas.

Exposure

In the 2004–5 visits, when participants were 23 years of age, PA was measured using the long version of the International PA Questionnaire.[28] Briefly, this questionnaire evaluates walking as well as moderate and vigorous PA according to frequency (in days) and duration (in minutes) in the following domains: occupational, domestic, leisure time, and commuting. All data were based on self-reports related to the week before the interview. For this study, 2 domains of moderate to vigorous OPA and LTPA were evaluated and presented in hours per week as a unit of analysis to facilitate the interpretation of coefficients in linear regressions.

Outcome Ascertainment

For this study, we considered the following outcomes that were evaluated in the 2012–13 visit, when the participants were 30 years old.

Primary Outcome

Lipid Profile

High-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglycerides were measured using an enzymatic assay (Shenzhen Mindray Bio-Medical Electronics Co, Ltd). Specific sensitivity of the assays can be assessed in previous studies conducted in cohort.[26,29]

Secondary Outcomes

Glucose Profile

Random blood glucose was measured using an automatic enzymatic colorimetric method, BS-380, Mindray (Shenzhen Mindray Bio-Medical Electronics Co, Ltd). As reported in other studies in that cohort, because glucose levels vary according to fasting time, estimates were adjusted for time since the last meal.[26]

Blood Pressure

Diastolic and systolic blood pressure were measured in a sitting position using an automatic digital sphygmomanometer, model Omron HEM 705CPINT. The mean of the 2 measurements was used in the analysis.[26,29]

Confounders

The variables measured at birth and/or 23 years old (2004/5 visit) were considered as potential confounders based on the conceptual directed acyclic graphs (Supplementary Material S2 [available online]). Self-reported skin color was reported at birth (White, Black, Brown, Indigenous, or Asian). The following variables were measured at 23 years: schooling (in complete years); socioeconomic status based on an asset index that was developed by the Brazilian Association of Survey Companies (5 categories were used); family history of CVDs, such as hypertension, stroke, and heart failure (yes and no); smoking (yes and no); alcohol (in the last week); ultraprocessed food consumption based on NOVA Food classification[30] (in kilocalories per day; eg, pizza, cookies, ice cream, shakes, hamburgers, and soft drinks); and PA in a specific domain (when we evaluated associations between OPA and CVD risk factors, we adjusted for LTPA, and, conversely, when we investigated associations between LTPA and CVD risk factors, we adjusted for OPA). Following previous studies available in the literature, our analyses were stratified by sex.[14,31,32]

Statistical Analysis

Descriptive data were expressed as mean and SD or absolute and relative frequencies. Data normality was verified by skewness, kurtosis, and Q–Q plot tests. Multivariable linear regressions were performed to evaluate associations between OPA and LTPA (in hours per week) with specific cardiovascular risk factors (continuous variable), that is, HDL, LDL, triglycerides, glucose, and blood pressure. In these analyses, both triglycerides and glucose were naturally log-transformed due to the skewed distribution and will be presented in the linear regressions as geometric mean.

In addition to the crude analyses, we ran 3 incremental models considering potential confounders defined according to the conceptual directed acyclic graphs as well as a **model 1** included skin color, educational level, socioeconomic status, and family history of CVD; and **model 2** included variables from model 1 and lifestyle factors, including smoking, dietary intake of alcohol and ultraprocessed food consumption, and PA in the other domain being assessed. The significance level was set at $P < .05$ for all analyses.

Results

At 23 years of age, 4297 individuals were evaluated, which represented a follow-up rate of 77.4% of the original birth cohort. At 30 years of age, 3701 individuals were examined, representing a

follow-up rate of 68.1%. Information on self-reported PA (OPA and LTPA) at 23 years was available for 4295 individuals, information on HDL, LDL, triglycerides, and glucose at 30 years was available for 3541 individuals, and 3620 data points were available on blood pressure. There were 282 and 325 deaths at 23 and 30 years, respectively. The flow diagram detailing the analytical sample is reported in Figure 1.

Table 1 shows data on the biological, socioeconomic, PA, and health characteristics of the participants included in the study. Most of the participants included in the analysis were male (51.3%; n = 3037) in the original sample and female in the 30-year follow-up (51%; n = 1794). Most of the participants included had White skin color (75%; n = 2429). The prevalence of alcohol consumption in the last week and smoking was 65.6% and 24.2%, respectively. About 1893 (59.7%) participants reported a family history of CVD, and 57.5% of participants presented overweight or obesity. The median (interquartile range) of OPA and LTPA (in hours per week) was 0 (0–10.4) and 0.75 (0–4.1), respectively. The mean of HDL and LDL (in milligrams per deciliter) was 58.6 (13.8) and 109.3 (28.9), respectively. The median of triglycerides and glucose (in milligrams per deciliter) was 95 and 86, respectively. Finally, the mean systolic and diastolic blood pressure (in millimeters of mercury) was 121.1 (13.8) and 75.3 (9.3), respectively.

Tables 2 and 3 show the main results of associations of OPA and LTPA, respectively, with cardiovascular risk factors. After adjustments for confounders, our main findings revealed that there was no association of OPA and LTPA with HDL and LDL both in models 2 and 3. Among men, there was an association between OPA and lower levels of triglycerides even after adjustment for confounders (model 2: $\beta = -0.002$; 95% confidence interval, –0.003 to –0.000; $P = .01$; Table 2). No associations were observed between OPA or LTPA and triglycerides among women (in all models). In terms of blood pressure, no associations were identified among men after adjustment in all models. On the other hand, there was an association between LTPA and higher levels of diastolic blood pressure among women even after adjustment for confounders (model 2: $\beta = 0.111$; 95% confidence interval, 0.005–0.216, $P = .04$). Also, we performed additional analyses considering total minutes of OPA and LTPA combined (Supplementary Material S3 [available online]) and OPA and LTPA separately as well as at different intensities (results not presented in tables), but there were no associations between those domains with cardiovascular risk factors.

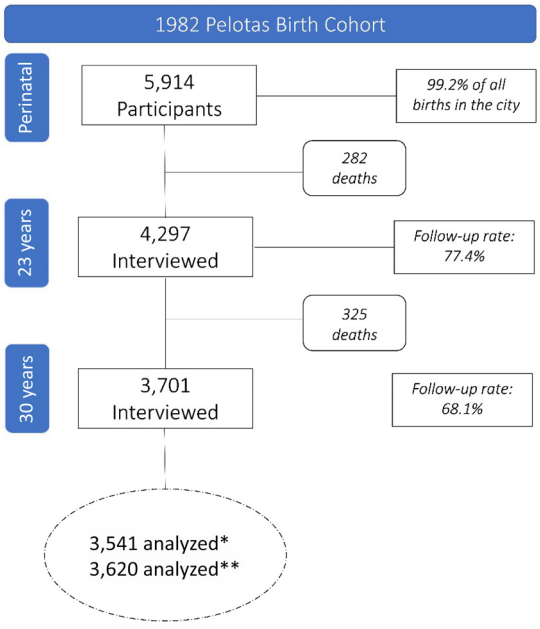

Figure 1 — Flow diagram of study participants included in the study. *Number of participants who had available information on self-reported physical activity at 23 years old and HDL cholesterol, LDL cholesterol, triglycerides, and glucose at 30 years old. **Number of participants who had available information on self-reported physical activity at 23 years old and blood pressure at 30 years old. *Note.* HDL indicates high-density lipoprotein; LDL, low-density lipoprotein.

Discussion

To the best of our knowledge, this is the first study that investigated the prospective association of OPA and LTPA with cardiovascular risk factors in early adulthood in a population-based birth cohort from a middle-income country. The main findings suggested that there was no association of OPA and LTPA with most cardiovascular risk factors apart from (1) inverse associations of OPA with triglycerides in men, (2) positive associations of LTPA with HDL cholesterol in women, and (3) positive association of LTPA with higher levels of diastolic blood pressure among women. The "PA paradox" that has been frequently observed in many studies in the upper middle- and high-income countries (mainly in middle and later adulthood) was not observed in our Brazilian birth cohort addressing young adults.

Pieces of evidence reaffirm that all adults should regularly undertake PA and that some PA is better than none.[10] According to the current guidelines (eg, World Health Organization), PA can be undertaken in any domain, such as leisure time-, occupation-, domestic-, and/or transport-related activities.[10] In terms of PA promotion, indistinct domains, feasibility, and lack of meaning of occupation/domestic PA promotion have been previously discussed considering the social determinants of health as well as considering middle- and low-income settings with specific dynamics of work and limited basic life conditions.[25] However, the traditional approach of PA promotion in all domains might be also discussed with the current evidence about the PA paradox.

For example, although LTPA often includes dynamic movements, providing a considerable increase in cardiorespiratory fitness level (mainly moderate to vigorous intensities and medium and high volume and in untrained participants),[22–24] some kinds of OPA require static loading, heavy lifting, and monotonous and awkward working postures,[20] suggesting a potential mechanism to increase cardiovascular risk. Briefly, according to a recent editorial on the PA paradox, potential mechanisms describing differential health effects of OPA and LTPA are: (1) OPA is of too low intensity or too long duration for maintaining or improving cardiorespiratory fitness[33]; (2) OPA elevates 24-hour heart rate, as well as 24-hour blood pressure, mainly because it often includes heavy lifting or static postures[34]; (3) OPA often comes with insufficient recovery time[35]; and (4) OPA may be associated with levels of inflammation.[36]

Philosophically, the PA paradox is, still, a paradox. As in our study, associations between OPA and health outcomes are mixed (with either positive, negative, or no associations found).[9,14,21,37,38] This is in contrast to the consistently reported beneficial health effects of LTPA. For instance, a meta-analysis (2011) of 82,412 participants from 6 cohort studies pointed out a protective effect of OPA.[37] Similar findings were observed in a large prospective cohort study that suggested that moderate to high OPA contributes

Table 1 Biological, Socioeconomic, Physical Activity, and Health Characteristics of the Participants Included in the Study—Data From 1982 Pelotas Birth Cohort

Variables	N	%	Mean (SD)	Median and IQ range
Gender				
Male	1747	49.3	–	
Female	1794	50.6	–	
Skin color				
White	2429	74.9	–	
Black	518	15.9	–	
Brown/Indigenous/Asian	236	7.4	–	
Yellow	60	1.8	–	
Income, quintiles				
1, richest	775	23.3	–	
2	704	21.2	–	
3	867	26.1	–	
4	315	9.5	–	
5, poorest	652	19.7	–	
Years of schooling			11.3 (4.1)	
Family history of cardiovascular disease				
Yes	1.893	59.7	–	
Alcohol, last wk				
Yes	2.098	65.6	–	
Smoking				
Yes	787	24.2	–	
BMI				
Underweight, <18.5	71	2.0		
Normal, 18.5–24.9	1.428	40.4		
Overweight, 25.0–29.9	1.224	34.6		
Obesity, 30.0–34.0	811	22.9		
Exposure at 23 y				
OPA, h/wk	3.241	–	10.5 (21.8)	0 (0–10.4)
OPA, min/wk	3.241	–	635.0 (1309.0)	0 (0–540)
LTPA, h/wk	3.241	–	3.7 (6.8)	45 (0–4.1)
LTPA, min/wk	3.241	–	226.4 (411.9)	0 (0–250)
People who reported OPA ≥ 1 min/wk	1421	43.8		
People who reported LTPA ≥ 1 min/wk	1725	53.1		
Outcome at 30 y				
HDL cholesterol, mg/dL	3.541	–	58.6 (13.8)	
LDL cholesterol, mg/dL	3.541	–	109.3 (28.9)	
Triglycerides, mg/dL	3541	–		95 (67–42)
Glucose, mg/dL	3541	–		86 (79–95)
Systolic blood pressure, mm Hg	3620	–	121.1 (13.8)	
Diastolic blood pressure, mm Hg	3620	–	75.3 (9.3)	

Abbreviations: BMI, body mass index; IQ, interquartile; HDL indicates high-density lipoprotein; LDL, low-density lipoprotein; LTPA, leisure-time physical activity; OPA, occupational physical activity.

to longevity in men but does not seem to affect longevity in women.[14]

On the other hand, a recent meta-analysis with 193,696 participants from 17 cohort studies suggested that high levels of OPA were associated with a higher risk of all-cause mortality among male workers.[38] Finally, a recent umbrella review of 17 systematic reviews and 23 unique health outcomes found benefits of high versus low OPA for cancer, ischemic stroke, coronary heart disease, and mental health. However, high OPA was associated with unfavorable health outcomes for all-cause mortality in men, mental ill health, osteoarthritis, and sleep quality and duration.[21]

Indeed, most of the aforementioned studies were designed and conducted for the middle adulthood and older population. As reported in our results, we did not find associations between OPA, LTPA, and cardiovascular risk factors, except for a few associations with limited clinical relevance. We found both negative

Table 2 Association Between Occupational Physical Activity at 23 years and Cardiovascular Risk Factors at 30 years in 1982 Pelotas Birth Cohort

	Occupational physical activity, h/wk					
	Male			Female		
Variables	N	β (95% CI)	*P* value	N	β (95% CI)	*P* value
HDL cholesterol, mmol/L						
Crude model	1608	–0.002 (–0.025 to 0.019)	.80	1633	–0.057 (–0.114 to –0.000)	**.04**
Model 1[a]	1070	0.002 (–0.028 to 0.033)	.85	1386	–0.045 (–0.105 to 0.014)	.13
Model 2[b]	1039	0.000 (–0.003 to 0.031)	.97	1366	–0.043 (–0.103 to 0.016)	.15
LDL cholesterol, mmol/L						
Crude model	1608	–0.017 (–0.071 to 0.037)	.53	1633	0.046 (–0.071 to 0.164)	.43
Model 1[a]	1070	–0.055 (–0.128 to 0.017)	.13	1386	0.004 (–0.125 to 0.135)	.94
Model 2[b]	1039	–0.049 (–0.012 to 0.024)	.18	1366	0.015 (–0.147 to 0.116)	.82
Triglycerides, mmol/L						
Crude model	1608	–0.002 (–0.003 to –0.001)	**.01**	1633	0.000 (–0.001 to 0.002)	.47
Model 1[a]	1070	–0.002 (–0.003 to –0.001)	**.01**	1386	0.000 (–0.001 to 0.002)	.45
Model 2[b]	1439	–0.002 (–0.003 to –0.000)	**.01**	1366	0.000 (–0.001 to 0.002)	.59
Glucose, mmol/L						
Crude model	1608	0.000 (–0.000 to 0.000)	.54	1633	–0.000 (–0.001 to 0.000)	.36
Model 1[a]	1070	–0.000 (–0.000 to 0.000)	.63	1386	–0.000 (–0.001 to 0.000)	.39
Model 2[b]	1039	–0.000 (–0.000 to 0.000)	.65	1366	–0.000 (–0.000 to 0.000)	.48
Systolic blood pressure, mm Hg						
Crude model	1629	0.015 (–0.007 to 0.037)	.18	1681	–0.038 (–0.086 to 0.010)	.12
Model 1[a]	1086	0.015 (–0.015 to 0.046)	.32	1432	–0.041 (–0.094 to 0.012)	.13
Model 2[b]	1053	.019 (–0.011 to 0.050)	.22	1409	–0.036 (–0.090 to 0.016)	.17
Diastolic blood pressure, mm Hg						
Crude model	1629	–0.005 (–0.022 to 0.011)	.52	1681	–0.037 (–0.074 to 0.000)	**.05**
Model 1[a]	1086	–0.006 (–0.030 to 0.016)	.58	1432	–0.039 (–0.080 to 0.001)	.06
Model 2[b]	1053	–0.005 (–0.029 to 0.018)	.09	1409	–0.035 (–0.075 to 0.005)	.09

Abbreviations: CI, confidence interval; HDL, high-density lipoprotein; LDL, low-density lipoprotein. Note: Analyses were adjusted according to the following models. [a]Model 1 (adjusted for skin color, educational level, socioeconomic status, and family history of cardiovascular disease). [b]Model 2 (model 1 + lifestyle factors, including smoking, occupational physical activity, and dietary intake of alcohol and ultraprocessed food consumption).

associations in triglycerides in men, providing a reduction of –0.001 mg/dL for each 1 hour of OPA, and positive associations in women, providing an increase of 0.112 mm Hg for each 1 hour of LTPA. Although we have observed statistically significant differences (eg, *P* value < .05), there is no clinical relevance in these associations. In this situation, for example, the literature suggests a minimal clinically important difference in blood pressure in terms of 2, 5, and 10 mm Hg, providing a reduction in cardiovascular events.[39–41]

As reported in Table 1, the majority of the participants were healthy according to the values of cardiovascular risk factors, aside from the large prevalence of overweight and obesity (57.5%). In this scenario, it is well documented that unfavorable health outcomes, especially changes in cardiovascular risk factors and incidence of CVD, such as heart failure, stroke, acute myocardial infarction, and cardiovascular death, may occur more in middle adulthood and older populations than young adults.[42,43] Thus, we believe that the absence of association of both OPA and LTPA may be explained by the relatively young age of our participants.

In addition to the age of our participants, there are some limitations to be considered in our study. PA measurement errors must be present due to self-report characteristics. In addition, according to the "Lessons Learned After 10 Years of IPAQ Use in Brazil and Colombia," self-reported OPA presents an even stronger challenge as it has considerable variation from day to day and even within the same day in terms of types, intensity, bout lengths, and periods of rest.[44]

Of note, in the 1982 Pelotas Birth Cohort, an 8-year difference between exposure data (PA) and outcomes (cardiovascular risk factors) may also attenuate the magnitude of associations. In that context, a recent study conducted by Strain et al[45] with data from the UK Biobank demonstrated that potential biases can be introduced by using very long follow-up periods, attenuating the protective inverse association between PA behavior and health outcomes. In contrast, shorter follow-up time (<4 y) can provide stronger associations between PA and health outcomes (eg, all-cause mortality and CVD). However, as the authors mentioned, shorter follow-up periods have a major limitation in terms of reverse causality. Nonetheless, we believe that our findings have a greater relationship with the age of the participants than the follow-up time.

Finally, in terms of limitations, we point out that blood samples were not collected during the fasting period. Concerning cholesterol measures, it has been reported that nonfasting blood collection is better for estimating individual cardiovascular risk.[46] On the other hand, for blood glucose, the ideal scenario would be blood sample collection with fasting blood. To minimize this

Table 3 Association Between Leisure-Time Physical Activity at 23 years and Cardiovascular Risk Factors at 30 years in 1982 Pelotas Birth Cohort

	Leisure-time physical activity, h/wk					
	Male			Female		
Variables	N	β (95% CI)	*P* value	N	β (95% CI)	*P* value
HDL cholesterol, mmol/L						
Crude model	1608	–0.022 (–0.098 to 0.054)	.56	1634	0.330 (0.179 to 0.482)	**.01**
Model 1[a]	1071	–0.004 (–0.095 to 0.087)	.92	1387	0.146 (0.009 to 0.311)	.06
Model 2[b]	1039	–0.011 (–0.105 to 0.081)	.80	1366	0.130 (–0.016 to 0.296)	.11
LDL cholesterol, mmol/L						
Crude model	1609	–0.113 (–0.296 to 0.070)	.22	1634	–0.094 (–0.410 to 0.222)	.56
Model 1[a]	1071	–0.025 (–0.241 to 0.189)	.81	1387	–0.004 (–0.335 to 0.343)	.98
Model 2[b]	1039	–0.034 (–0.252 to 0.183)	.75	1366	–0.143 (–0.051 to 0.301)	.77
Triglycerides, mmol/L						
Crude model	1609	–0.000 (–0.004 to 0.002)	.62	1634	0.001 (–0.003 to 0.006)	.55
Model 1[a]	1071	–0.000 (–0.007 to 0.001)	.19	1387	0.000 (–0.004 to 0.007)	.57
Model 2[b]	1039	0.002 (–0.007 to 0.001)	.22	1366	0.000 (–0.005 to 0.005)	.98
Glucose, mmol/L						
Crude model	1609	0.000 (–0.001 to 0.001)	.75	1634	0.000 (–0.001 to 0.002)	.99
Model 1[a]	1071	0.002 (–0.001 to 0.001)	.99	1387	0.000 (–0.001 to 0.002)	.65
Model 2[b]	1039	0.000 (–0.001 to 0.001)	.91	1366	0.000 (–0.001 to 0.002)	.88
Systolic blood pressure, mm Hg						
Crude model	1630	0.027 (–0.048 to 0.104)	.47	1682	0.080 (–0.049 to 0.210)	.22
Model 1[a]	1087	0.003 (–0.058 to 0.123)	.48	1433	0.141 (–0.003 to 0.280)	**.05**
Model 2[b]	1053	0.001 (–0.082 to 0.100)	.85	1409	0.129 (–0.012 to 0.270)	.07
Diastolic blood pressure, mm Hg						
Crude model	1630	–0.009(–0.067 to 0.048)	.75	1630	0.082 (–0.017 to 0.182)	.10
Model 1[a]	1087	–0.001 (–0.068 to 0.070)	.97	1433	0.132 (0.246 to 0.238)	**.01**
Model 2[b]	1053	–0.007 (–0.078 to 0.063)	.83	1409	0.124 (0.016 to 0.232)	**.02**

Abbreviations: CI, confidence interval; HDL, high-density lipoprotein; LDL, low-density lipoprotein. Note: Analyses were adjusted according to the following models. [a]Model 1 (adjusted for ethnicity, educational level, socioeconomic status, and family history of cardiovascular disease). [b]Model 2 (model 1 + lifestyle factors, including smoking, occupational physical activity, and dietary intake of alcohol and ultraprocessed food consumption).

limitation, we corrected the estimates for the time since the last meal. For blood pressure, we used only a single measurement.

The strengths of our study should be mentioned. As a strength, this is a prospective population-based birth cohort from a middle-income country. Moreover, a good retention rate is noteworthy considering the long follow-up period and other longitudinal studies.[47,48] Second, there is high heterogeneity in terms of socioeconomic status, ranging from low to high income, which we consider important to understand the associations between OPA and LTPA in cardiovascular risk factors. Third, despite some limitations, we used blood samples as a measure of cardiovascular risk factors, including LDL, that have been suggested as a proxy for heart failure and CVD mortality.[42] Finally, a key point was the availability of many variables in our database, generating the possibility of more robust analyses adjusting for different potential confounders.

Conclusions

To conclude, the findings suggest that there was no association, or association with limited clinical relevance, of OPA and LTPA when participants were 23 years old with most of the cardiovascular risk factors when they were 30 years old. Nonetheless, we endorse that future studies in this field should be designed for a better understanding of this important research question, including other settings and other age ranges.

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References

1. Tsao CW, Aday AW, Almarzooq ZI, et al. Heart disease and stroke statistics—2023 update: a report from the American Heart Association. *Circulation*. 2023;147(8):e93–e621. doi:10.1161/CIR.0000000000001123
2. Oliveira GMM, Brant LCC, Polanczyk CA, et al. Cardiovascular statistics—Brazil 2021. *Arq Bras Cardiol*. 2022;118(1):115–373. doi:10.36660/abc.20211012
3. Gonçalves RPF, Haikal DS, Freitas MIF, Machado ÍE, Malta DC. Self-reported medical diagnosis of heart disease and associated risk factors: National health survey. *Rev Bras Epidemiol*. 2019;22(suppl 2):Article 16. doi:10.1590/1980-549720190016.supl.2
4. Dunstan DW, Dogra S, Carter SE, Owen N. Sit less and move more for cardiovascular health: emerging insights and opportunities. *Nat Rev Cardiol*. 2021;18(9):637–648. doi:10.1038/s41569-021-00547-y
5. Shiroma EJ, Lee IM. Physical activity and cardiovascular health: lessons learned from epidemiological studies across age, gender, and race/ethnicity. *Circulation*. 2010;122(7):743–752. doi:10.1161/CIRCULATIONAHA.109.914721
6. Del Pozo Cruz B, Ahmadi MN, Lee IM, Stamatakis E. Prospective associations of daily step counts and intensity with cancer and cardiovascular disease incidence and mortality and all-cause mortality. *JAMA Intern Med*. 2022;182(11):1139–1148. doi:10.1001/jamainternmed.2022.4000
7. Hall C, Heck JE, Sandler DP, Ritz B, Chen H, Krause N. Occupational and leisure-time physical activity differentially predict 6-year incidence of stroke and transient ischemic attack in women. *Scand J Work Environ Health*. 2019;45(3):267–279. doi:10.5271/sjweh.3787
8. Allesøe K, Holtermann A, Aadahl M, Thomsen JF, Hundrup YA, Søgaard K. High occupational physical activity and risk of ischaemic heart disease in women: the interplay with physical activity during leisure time. *Eur J Prev Cardiol*. 2015;22(12):1601–1608. doi:10.1177/2047487314554866
9. Wanner M, Lohse T, Braun J, et al. Occupational physical activity and all-cause and cardiovascular disease mortality: results from two longitudinal studies in Switzerland. *Am J Ind Med*. 2019;62(7):559–567. doi:10.1002/ajim.22975
10. Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. *Br J Sports Med*. 2020;54(24):1451–1462. doi:10.1136/bjsports-2020-102955
11. Ekelund U, Tarp J, Fagerland MW, et al. Joint associations of accelero-meter measured physical activity and sedentary time with all-cause mortality: a harmonised meta-analysis in more than 44 000 middle-aged and older individuals. *Br J Sports Med*. 2020;54(24):1499–1506. doi:10.1136/bjsports-2020-103270
12. Ekelund U, Brown WJ, Steene-Johannessen J, et al. Do the associations of sedentary behaviour with cardiovascular disease mortality and cancer mortality differ by physical activity level? A systematic review and harmonised meta-analysis of data from 850 060 participants. *Br J Sports Med*. 2019;53(14):886–894. doi:10.1136/bjsports-2017-098963
13. Ekelund U, Tarp J, Steene-Johannessen J, et al. Dose-response associations between accelerometry measured physical activity and sedentary time and all cause mortality: systematic review and harmonised meta-analysis. *BMJ*. 2019;366:14570. doi:10.1136/bmj.l4570
14. Dalene KE, Tarp J, Selmer RM, et al. Occupational physical activity and longevity in working men and women in Norway: a prospective cohort study. *Lancet Public Heal*. 2021;6(6):e386–e395. doi:10.1016/S2468-2667(21)00032-3
15. Smith P, Ma H, Glazier RH, Gilbert-Ouimet M, Mustard C. The relationship between occupational standing and sitting and incident heart disease over a 12-year period in Ontario, Canada. *Am J Epidemiol*. 2018;187(1):27–33. doi:10.1093/aje/kwx298
16. Richard A, Martin B, Wanner M, Eichholzer M, Rohrmann S. Effects of leisure-time and occupational physical activity on total mortality risk in NHANES III according to sex, ethnicity, central obesity, and age. *J Phys Act Health*. 2015;12(2):184–192. doi:10.1123/jpah.2013-0198
17. Clays E, Lidegaard M, De Bacquer D, et al. The combined relationship of occupational and leisure-time physical activity with all-cause mortality among men, accounting for physical fitness. *Am J Epidemiol*. 2014;179(5):559–566. doi:10.1093/aje/kwt294
18. Harari G, Green MS, Zelber-Sagi S. Combined association of occupational and leisure-time physical activity with all-cause and coronary heart disease mortality among a cohort of men followed-up for 22 years. *Occup Environ Med*. 2015;72(9):617–624. doi:10.1136/oemed-2014-102613
19. Luo M, Gupta N, Holtermann A, Stamatakis E, Ding D. Revisiting the 'physical activity paradox' in a Chinese context: occupational physical activity and mortality in 142,302 urban working adults from the China Kadoorie Biobank study. *Lancet Reg Heal - West Pac*. 2022;23:100457. doi:10.1016/j.lanwpc.2022.100457
20. Holtermann A, Krause N, van der Beek AJ, Straker L. The physical activity paradox: six reasons why Occupational Physical Activity (OPA) does not confer the cardiovascular health benefits that leisure time physical activity does. *Br J Sports Med*. 2018;52(3):149–150. doi:10.1136/bjsports-2017-097965
21. Cillekens B, Lang M, van Mechelen W, et al. How does occupational physical activity influence health? An umbrella review of 23 health outcomes across 158 observational studies. *Br J Sports Med*. 2020;54(24):1474–1481. doi:10.1136/bjsports-2020-102587
22. Franklin BA, Eijsvogels TMH, Pandey A, Quindry J, Toth PP. Physical activity, cardiorespiratory fitness, and cardiovascular health: a clinical practice statement of the ASPC Part I: bioenergetics, contemporary physical activity recommendations, benefits, risks, extreme exercise regimens, potential maladaptations. *Am J Prev Cardiol*. 2022;12:100424. doi:10.1016/j.ajpc.2022.100424
23. Bahls M, Ittermann T, Ewert R, et al. Physical activity and cardiorespiratory fitness-a ten-year follow-up. *Scand J Med Sci Sports*. 2021;31(3):742–751. doi:10.1111/sms.13882
24. Weston KS, Wisløff U, Coombes JS. High-intensity interval training in patients with lifestyle-induced cardiometabolic disease: a systematic review and meta-analysis. *Br J Sports Med*. 2014;48(16):1227–1234. doi:10.1136/bjsports-2013-092576
25. Crochemore-Silva I, Knuth AG, Mielke GI, Wendt A de FJJ. *Physical Activity in Low- and Middle Income Countries*. Routledge; 2021. https://www.routledge.com/Physical-Activity-in-Low--and-Middle-Income-Countries/Siefken-Varela-Waqanivalu-Schulenkorf/p/book/9780367362232
26. Horta BL, Gigante DP, Goncalves H, et al. Cohort profile update: The 1982 Pelotas (Brazil) Birth Cohort Study. *Int J Epidemiol*. 2015;44(2):441a–441e. doi:10.1093/ije/dyv017
27. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *J Clin Epidemiol*. 2008;61(4):344–349. doi:10.1016/j.jclinepi.2007.11.008
28. Cora L, Marshall AL, Sjöström M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sport Exerc*. 2003;35(8):1381–1395. doi:10.1249/01.MSS.0000078924.61453.FB

29. Lima NP, Bassani DG, Magalhães EIS, Barros FC, Horta BL. Breastfeeding and maternal cardiovascular risk factors: 1982 Pelotas Birth Cohort. *Sci Rep*. 2019;9(1):13092. doi:10.1038/s41598-019-49576-1
30. Monteiro CA, Cannon G, Moubarac JC, Levy RB, Louzada MLC, Jaime PC. The UN decade of nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutr*. 2018;21(1):5–17. doi:10.1017/S1368980017000234
31. Holtermann A, Marott JL, Gyntelberg F, et al. Occupational and leisure time physical activity: risk of all-cause mortality and myocardial infarction in the Copenhagen City Heart Study. a prospective cohort study. *BMJ Open*. 2012;2(1):556. doi:10.1136/bmjopen-2011-000556
32. Holtermann A, Schnohr P, Nordestgaard BG, Marott JL. The physical activity paradox in cardiovascular disease and all-cause mortality: the contemporary copenhagen general population study with 104 046 adults. *Eur Heart J*. 2021;42(15):1499–1511. doi:10.1093/eurheartj/ehab087
33. Jørgensen K. Permissible loads based on energy expenditure measurements. *Ergonomics*. 1985;28(1):365–369. doi:10.1080/00140138508963145
34. Korshøj M, Lidegaard M, Kittel F, et al. The relation of ambulatory heart rate with all-cause mortality among middle-aged men: a prospective cohort study. *PLoS One*. 2015;10(3):729. doi:10.1371/journal.pone.0121729
35. Stergiou D, Duncan E. Atrial Fibrillation (AF) in endurance athletes: a complicated affair. *Curr Treat Options Cardiovasc Med*. 2018;20(12):98. doi:10.1007/s11936-018-0697-9
36. Kasapis C, Thompson PD. The effects of physical activity on serum C-reactive protein and inflammatory markers: a systematic review. *J Am Coll Cardiol*. 2005;45(10):1563–1569. doi:10.1016/j.jacc.2004.12.077
37. Samitz G, Egger M, Zwahlen M. Domains of physical activity and all-cause mortality: systematic review and dose-response meta-analysis of cohort studies. *Int J Epidemiol*. 2011;40(5):1382–1400. doi:10.1093/ije/dyr112
38. Coenen P, Huysmans MA, Holtermann A, et al. Do highly physically active workers die early? A systematic review with meta-analysis of data from 193 696 participants. *Br J Sports Med*. 2018;52(20):1320–1326. doi:10.1136/bjsports-2017-098540
39. Whelton PK, He J, Appel LJ, et al. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. *JAMA*. 2002;288(15):1882–1888. doi:10.1001/jama.288.15.1882
40. Ettehad D, Emdin CA, Kiran A, et al. Blood pressure lowering for prevention of cardiovascular disease and death: a systematic review and meta-analysis. *Lancet*. 2016;387(10022):957–967. doi:10.1016/S0140-6736(15)01225-8
41. Bundy JD, Li C, Stuchlik P, et al. Systolic blood pressure reduction and risk of cardiovascular disease and mortality: a systematic review and network meta-analysis. *JAMA Cardiol*. 2017;2(7):775–781. doi:10.1001/jamacardio.2017.1421
42. Virani SS, Alonso A, Aparicio HJ, et al. Heart disease and stroke statistics-2021 update: a report from the American Heart Association. *Circulation*. 2021;143(8):e254–e743. doi:10.1161/CIR.0000000000000950
43. Lavie CJ, Ozemek C, Carbone S, Katzmarzyk PT, Blair SN. Sedentary behavior, exercise, and cardiovascular health. *Circ Res*. 2019;124(5):799–815. doi:10.1161/CIRCRESAHA.118.312669
44. Hallal PC, Gomez LF, Parra DC, et al. Lessons learned after 10 years of IPAQ use in Brazil and Colombia. *J Phys Act Heal*. 2010;7(suppl 2):259. doi:10.1123/jpah.7.s2.s259
45. Strain T, Wijndaele K, Sharp SJ, Dempsey PC, Wareham N, Brage S. Impact of follow-up time and analytical approaches to account for reverse causality on the association between physical activity and health outcomes in UK Biobank. *Int J Epidemiol*. 2020;49(1):162–172. doi:10.1093/ije/dyz212
46. Darras P, Mattman A, Francis GA. Nonfasting lipid testing: the new standard for cardiovascular risk assessment. *CMAJ*. 2018;190(45):E1317–E1318. doi:10.1503/cmaj.180804
47. Preto R, Luís S, Cardoso VC, et al. Profile of three Brazilian birth cohort studies in. *Brazilian J Med Biol Res*. 2007;40(9):1165–1176. www.bjournal.com.br
48. Schmidt MI, Duncan BB, Mill JG, et al. Cohort profile: longitudinal study of adult health (ELSA-Brasil). *Int J Epidemiol*. 2015;44(1):68–75. doi:10.1093/ije/dyu027