Original research

Occupational physical activity and cardiovascular disease in the United States

Tyler D. Quinn ,[1,2] Patrick L. Yorio,[1] Peter M. Smith ,[3,4] Yongsuk Seo,[1] Geoffrey P. Whitfield,[5] Bethany Barone Gibbs[2]

► Additional material is published online only. To view please visit the journal online (http://dx.doi.org/10.1136/oemed-2020-106948).

[1]National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Pittsburgh, Pennsylvania, USA
[2]Health and Human Development, University of Pittsburgh School of Education, Pittsburgh, Pennsylvania, USA
[3]Institute for Work & Health, Toronto, Ontario, Canada
[4]Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada
[5]National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Correspondence to
Dr Tyler D. Quinn, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Pittsburgh, Pennsylvania, USA; yhh7@cdc.gov

Received 3 August 2020
Revised 26 January 2021
Accepted 30 January 2021
Published Online First 18 March 2021

To cite: Quinn TD, Yorio PL, Smith PM, *et al*. *Occup Environ Med* 2021;**78**:724–730.

ABSTRACT

Background Emerging evidence, predominately from European and Asian countries, describes opposing effects of occupational physical activity (OPA) and leisure-time physical activity (LTPA) on cardiovascular health. This analysis examined cardiovascular disease (CVD) prevalence associated with OPA and LTPA.

Methods This cross-sectional analysis of 2015 National Health Interview Survey data (n=16 974) employed logistic regression to estimate odds (OR) of self-reported CVD (coronary heart disease, heart attack, stroke or angina) with self-reported total occupational activity (TOA), occupational exertion (OE), occupational standing and walking (OSW) and LTPA. OPA was measured using two questions: 'How often does your job involve…' (1) 'repeated lifting, pushing, pulling or bending?' (OE) and (2) 'standing or walking around?' (OSW) with responses on a 5-item Likert scale (0=never, 4=always). TOA was categorised similarly after summing OE and OSW scores. LTPA was defined as 0, 1–149 or ≥150 min/week of moderate-to-vigorous activity. All models adjusted for common socioeconomic variables and additional analyses were stratified by sex, smoking status and LTPA.

Results Odds for CVD were higher when 'always' performing TOA (OR 1.99 95% CI 1.12 to 3.53), OE (OR 2.15, 95% CI 1.45 to 3.19) or OSW (OR 1.84, 95% CI 1.07 to 3.17) compared with 'never'. When restricting to never-smokers, odds for CVD were higher when 'always' performing TOA (OR 3.00, 95% CI 1.38 to 6.51) and OE (OR 3.00, 95% CI 1.80 to 5.02) versus 'never'.

Conclusion Associations of high OPA with CVD were equally apparent across sexes, stronger in lower LTPA levels and stronger in never-smokers. While uncontrolled confounding is still possible, even after extensive adjustment, the seemingly paradoxical adverse associations with OPA and CVD should be investigated further.

Key messages

What is already known about this subject?

► An occupational physical activity health paradox has been described in European and Asian populations. However, this literature remains limited by the lack of US data as well as potential residual confounding.

What are the new findings?

► This study adds an examination of a US sample to the occupational physical activity health paradox literature. Specifically, this study found that high amounts of occupational physical activity is associated with higher odds for cardiovascular disease. Interestingly, smoking does not appear to confound the relationship as the direct associations were more exaggerated among never-smokers. Lastly, associations were lessened in those reporting high levels of leisure-time physical activity.

How might this impact on policy or clinical practice in the foreseeable future?

► If proven true, the implications of an occupational physical activity health paradox on the modern workforce would be significant and would likely justify public health initiatives to limit or restructure occupational physical activity requirements to prioritise worker health.

INTRODUCTION

While leisure-time physical activity (LTPA) is known to have many health promoting effects,[1–6] occupational physical activity (OPA) has been associated with adverse health in some studies.[7–9] These contradictory observations have been termed the 'OPA health paradox.'[7] A recent meta-analysis found an 18% increased risk of all-cause mortality in men with high compared with low OPA.[7] Proposed mechanisms to explain the increased risk include increased 24-hour cardiovascular strain, insufficient recovery and psychological strain from OPA.[10]

To date, most previous work has been done in Europe and Asia[4 7] and confirming these results in other populations, such as the US, would add to the generalisability of the findings. Additionally, using all-cause mortality as an outcome may be inappropriate as many active occupations also have workplace hazards, increasing mortality. Using more specific outcomes such as current cardiovascular disease (CVD) would be advantageous to interpretation and potential intervention. Different types of OPA have not been explored which could have different effects on cardiovascular health.[11] Lastly, the current literature is limited by, and criticised for, the potential for uncontrolled confounding from concurrent health behaviours, most notably smoking status.[12]

To address these gaps, this analysis examined cross-sectional associations between self-reported OPA and LTPA with self-reported prevalence of CVD in a representative US population using the

2015 National Health Interview Survey (NHIS). Additional analyses were stratified/restricted by sex, smoking status and LTPA level.

METHODS

The NHIS is a cross-sectional face-to-face household survey conducted every year in the US. The sample adult survey, used for this analysis, is a subsection where one adult (aged ≥18 years) from each family is randomly selected to answer more extensive questions about their own health. The sample adult survey had a 2015 response rate of 55.2% with 33 672 individuals responding. Further details about the NHIS were published previously.[13]

Activity definitions

OPA was measured using two questions: (1) 'How often does your job involve repeated lifting, pushing, pulling or bending?' (occupational exertion (OE)) and (2) 'How often does your job involve standing or walking around?' (occupational standing and walking (OSW)). Participants responded to a 5-item Likert scale (0=never, 1=seldom, 2=sometimes, 3=often, 4=always). Total occupational activity (TOA) was calculated by summing these two scores and then categorising as: 0=never, 1 or 2=seldom, 3 or 4=sometimes, 5 or 6=often, 7 or 8=always.

Participants were asked to report moderate-intensity and vigorous-intensity activity as leisure-time bouts of ≥10 continuous minutes that 'cause only light sweating or a slight to moderate increase in breathing or heart rate' and 'cause heavy sweating or large increases in breathing or heart rate', respectively. Both moderate-intensity and vigorous-intensity aerobic activity were calculated as minutes/week. Moderate equivalent minutes/week were calculated as vigorous-intensity minutes/week multiplied by two and added to moderate-intensity minutes/week.[14 15] LTPA was defined in three categories as 0, 1–149 or ≥150 moderate-equivalent minutes/week.[5]

CVD definition

Each participant answered yes or no to the questions, 'Have you EVER been told by a doctor or other health professional that you had…': 'coronary heart disease,' 'a heart attack (also called myocardial infarction),' 'a stroke,' or 'angina, also called angina pectoris?' CVD was classified as presence or absence of any of the four diseases and used as the outcome variable.[16] The high validity of self-reported CVD in population-based studies has been reported previously.[17–19]

Covariate definitions

All models were adjusted for subject age, sex, working status, race/ethnicity, smoking status, alcohol consumption, family-income-to-poverty ratio, body mass index (BMI), education and US nativity. Age was defined continuously between 18 and 64 years. Sex was coded as male or female. Working status was defined as full time or part time (split at 35 hours/week). Race/ethnicity was categorised as Hispanic, non-Hispanic white, non-Hispanic black, non-Hispanic Asian and Non-Hispanic all other race groups. Smoking status was categorised as never, former, current or refused/not ascertained/don't know (a pack-years variable is not available in the NHIS dataset). Alcohol consumption was categorised as never, former, current and drinking status unknown. Family-income-to-poverty ratio was estimated using multiple imputations and defined as self-reported family income divided by the poverty threshold.[13] BMI was calculated as kg/m^2 using self-reported height and weight. Education was categorised as <high school diploma, high school diploma or equivalent, some college, college graduate, graduate degree or refused/not ascertained/don't know. US nativity was categorised as US born, not US born, or refused/not ascertained/don't know.

Sample

Those who did not answer the OPA questions due to not working (n=14 216) or refused to answer or didn't know the answer to the OPA questions (n=27) were excluded. Those aged ≥65 years (n=8378), with missing BMI (n=1145), with missing activity data (n=1259) or reporting not working (n=14 237) were also excluded. Sensitivity analyses were performed to confirm that the exclusion of individuals aged ≥65 years and with missing BMI data did not change the results in a meaningful way. Any missing CVD outcome data were also excluded (n=58 for coronary heart disease, n=43 for hypertension, n=25 for diabetes, n=39 stroke, n=102 for high cholesterol, n=61 for angina, n=34 for any other heart conditions and n=130 in total for composite CVD). After all exclusions from the total sample (n=33 672), the analytical sample size was n=16 974.

Analytical approach

Unadjusted prevalence of CVD was estimated and stratified across levels of the four physical activity variables. Logistic regression estimated the odds of having composite CVD across categorical levels of TOA, OE, OSW and LTPA, after adjustment for covariates. Analyses were repeated similarly after stratification by sex (female/male) and LTPA level. Stratification by education level was also completed and the results are provided in online supplemental table 1. Lastly, all associations were restricted to a sample of never-smokers. The linear trend of CVD prevalence was tested across increasing categories of the ordinal physical activity variables using linear regression and presented as p for trend.

TOA was the only OPA covariate used in the LTPA models due to collinearity between TOA and OE (r=0.89, p≤0.001) and OSW (r=0.88, p≤0.001). Similarly, when analysing the effect of OE and OSW on CVD, no other OPA variable was included due to high joint correlations between the OPA variables.

All estimates were made using the complex survey weighting and design variables described in the 2015 NHIS Survey Description Document.[13] STATA V.16.1 was used for all analyses and alpha level was set at 0.05.

RESULTS

The estimated weighted sample characteristics of the overall and sex-stratified samples are presented in table 1. The overall sample was 49.6% female, 40.6 years old and mostly (64.5%) non-Hispanic white who had never smoked (67.1%). CVD was reported by 2.5% of individuals. 33.7% of individuals reported 'always' performing TOA and 55% reported ≥150 of moderate-equivalent minutes/week.

Online supplemental table 2 presents the unadjusted prevalence of CVD across categories of OPA and LTPA. Prevalence of CVD was highest in the 'always' categories of TOA, OE and OSW (3.2%, 3.6% and 3.0%, respectively) compared with the other levels. Significant linear trends were detected in TOA, OE and OSW (all p<0.05). CVD prevalence was lowest among the highest LTPA group (2.0%) with a significant p for trend (p=0.002) (online supplemental table 2).

Table 2 presents the adjusted odds for CVD by OPA and LTPA levels. The odds for composite CVD were significantly higher in those reporting 'always' performing TOA, OE and

Table 1 Sample characteristics

	Overall	Female	Male
Sample size	16 974	8412	8562
Age (years)	40.6	40.6	40.5
Cardiovascular disease	2.5%	2.1%	2.9%
Occupational status			
Full time	79.3%	72.4%	85.1%
Part time	20.7%	27.6%	14.9%
Total occupational activity			
Never	6.0%	6.7%	5.3%
Seldom	15.6%	17.5%	13.9%
Sometimes	22.0%	22.7%	21.3%
Often	22.8%	23.3%	22.3%
Always	33.7%	29.7%	37.2%
Leisure-time physical activity			
0 min	27.0%	27.0%	27.0%
1–149 min	18.1%	20.5%	16.0%
≥150 min	55.0%	52.5%	57.0%
Ratio of family income to poverty threshold	4.5*	4.4*	4.6*
Body mass index	27.9	27.5	28.3
Alcohol drinking status			
Never	15.8%	18.7%	13.3%
Former	9.0%	8.8%	9.2%
Current	74.6%	72.0%	76.9%
Drinking status unknown	0.5%	0.5%	0.5%
Smoking status			
Never	67.1%	70.4%	64.3%
Former	17.7%	15.4%	19.7%
Current	15.1%	14.1%	15.9%
Refused/not ascertained/don't know	0.1%	0.1%	0.1%
Education			
<High school diploma	8.3%	6.5%	9.9%
High school diploma	21.7%	19.0%	24.0%
Some college	19.1%	19.3%	18.9%
College graduate	36.9%	39.2%	34.8%
Graduate degree	13.7%	15.8%	12.0%
Refused/not ascertained/don't know	0.2%	0.2%	0.3%
Race			
Hispanic	16.7%	14.8%	18.3%
Non-Hispanic white	64.5%	64.7%	64.3%
Non-Hispanic black	11.7%	13.2%	10.4%
Non-Hispanic Asian	6.2%	6.3%	6.2%
Non-Hispanic all other	0.9%	0.9%	0.9%
Nativity			
US born	81.5%	83.1%	80.1%
Not US born	18.4%	16.8%	19.8%
Refused/not ascertained/don't know	0.01%	0.01%	0.01%

Data presented as either estimated frequency or mean.
*Estimated using multiple imputations.

OSW compared with 'never' (OR=1.99, p=0.019, OR=2.15, p=0.001 and OR 1.84, p=0.027, respectively). Odds for CVD were significantly higher in those reporting 'seldom' and 'often' performing OE compared with 'never' (OR=1.75, p=0.005 and OR=1.60, p=0.042, respectively). Significant positive trends were found between CVD and TOA, OE and OSW (all p<0.01). LTPA level was not associated with CVD (p>0.05).

Sex stratification

Table 3 presents the odds for CVD associated with TOA, OE, OSW and LTPA by sex. Odds for CVD were higher in men reporting 'always' performing TOA compared with 'never' (OR=2.47, p=0.041) but not for any other TOA categories in either sex. Odds for CVD were higher in those reporting 'always' performing OE compared with 'never' in both females and males (OR=2.32, p=0.002 and OR=1.97, p=0.017, respectively). Females reporting 'seldom' performing OE had higher odds for CVD (OR=2.06, p=0.014) compared with 'never'. Significant positive trends were observed between CVD prevalence and TOA, OE and OSW for both sexes (all p<0.05). LTPA level was not associated with CVD in either sex.

Restriction to never-smokers

Table 4 presents the adjusted odds for CVD associated with OPA and LTPA in never-smokers. The unadjusted prevalence of CVD in never-smokers was 1.78% (not shown). In never-smokers, the odds for CVD were significantly higher in the those 'always' performing TOA and OE compared with 'never' (OR=3.00, p=0.006 and OR=3.00, p=0.001, respectively). Odds of CVD were higher among those reporting 'often' and 'seldom' performing OE compared with 'never' (OR=2.50, p=0.002 and OR=2.24, p=0.002, respectively). Significant positive trends were observed between CVD prevalence and TOA, OE and OSW (all p<0.01). Odds for CVD remained non-significant across LTPA levels in never-smokers (all p>0.05).

Joint associations between OPA and LTPA

Table 5 presents the adjusted odds for CVD by level of TOA, OE and OSW stratified by LTPA level. Results stratified by LTPA were of similar magnitude to the overall results. Associations between high CVD with TOA and OE were more apparent in those reporting 1–149 min/week of LTPA. Significant positive trends between CVD prevalence and TOA, OE and OSW were found in those reporting 1–149 min/week of LTPA; however, similar trends were not found among those meeting LTPA guidelines. The odds for CVD were higher in those 'always' performing TOA, OE and OSW compared with 'never' in those with 1–149 min/week of LTPA (OR=10.87, p=0.001; OR=4.78, p=0.001; OR=5.21, p=0.009, respectively). Similarly, odds for CVD were higher in those 'often' performing TOA, OE and OSW compared with 'never' in those with 1–149 min/week of LTPA (OR=6.47, p=0.006; OR=4.60, p=0.002; OR=4.60, p=0.017; respectively). With 0 min/week of LTPA, odds for CVD were higher in those 'always' performing OE compared with 'never' (OR=2.08, p=0.046) and significant positive trends were found between CVD and TOA as well as OSW (p<0.05). No significant associations were observed between OPA and CVD in those meeting LTPA guidelines (all p>0.05).

DISCUSSION

This analysis examined cross-sectional associations between prevalent CVD associated with TOA, OE, OSW and LTPA in a nationally representative sample of US working adults. Those reporting 'always' performing TOA, OE and OSW had higher odds of prevalent CVD than those reporting 'never'. Both sexes had similar, direct associations between high levels of OE and CVD. Direct associations between CVD and high TOA, OE and OSW seem to be of similar magnitude in never-smokers. Lastly, higher odds for CVD with high OE were more distinct in those reporting 0 and especially 1–149 min/week of LTPA as compared with ≥150 min/week.

Table 2 Adjusted odds of cardiovascular disease by frequency of leisure-time and occupational physical activity

	Total occupational activity			Occupational exertion			Occupational standing			Leisure-time physical activity			
	OR	P value	95% CI	OR	P value	95% CI	OR	P value	95% CI		OR	P value	95% CI
Never	1.00	--	--	1.00	--	--	1.00	--	--	0 min	1.00	--	--
Seldom	1.10	0.785	(0.56 to 2.13)	**1.75**	**0.005**	**(1.18 to 2.60)**	1.14	0.694	(0.59 to 2.19)	1–149 min	1.16	0.469	(0.77 to 1.74)
Sometimes	1.35	0.343	(0.72 to 2.52)	0.96	0.833	(0.63 to 1.44)	1.03	0.929	(0.52 to 2.04)	≥150 min	0.95	0.737	(0.69 to 1.29)
Often	1.59	0.146	(0.85 to 2.98)	**1.60**	**0.042**	**(1.02 to 2.53)**	1.25	0.484	(0.66 to 2.37)	--	--	--	--
Always	**1.99**	**0.019**	**(1.12 to 3.53)**	**2.15**	**0.001**	**(1.45 to 3.19)**	**1.84**	**0.027**	**(1.07 to 3.17)**	--	--	--	--
P for trend	0.001			0.002			0.001				0.479		

All models adjusted for age, sex, working status, race/ethnicity, smoking status, alcohol consumption, family income-to-poverty ratio, BMI, education, US nativity, leisure-time activity category and total occupational activity; all strata had at least 80 cases of reported CVD; boldface text indicates significance (p<0.05).
BMI, body mass index; CVD, cardiovascular disease.

High amounts of OPA have repeatedly been associated with detrimental cardiovascular and overall health as compared with little or no OPA among European and Asian populations.[7–9] This is in stark contrast to the well-established, health-enhancing effects of LTPA.[2] These findings, although cross-sectional, provide some initial evidence that a similar paradoxical effect may exist in the US. While further investigation is necessary, these findings align with a previous study that demonstrated a non-significant 25% increased HR for all-cause mortality in those reporting high OPA compared with low OPA in National Health and Nutrition Examination Survey III.[4] However, as mentioned before, all-cause mortality may be confounded by other hazards in high OPA occupations. Another previous study found that OPA had no effect on risk for coronary heart disease.[6] However, the different findings could reflect reliance on reported occupation to estimate OPA level rather than the NHIS's method of self-reported OPA. Another longitudinal study of Canadian workers found a 232% increased risk of heart disease in those with predominately standing occupations compared with sitting.[20] The current overall NHIS sample results align with the Canadian findings in the direction and magnitude of effect of OSW. Effects of OPA on cardiovascular health could be similar between the two countries, strengthening international generalisability despite potential differences in OPA and culture across countries.

One possible explanation for the OPA health paradox is insufficient recovery time during and after OPA performed, therefore, increasing the 24-hour cardiovascular load.[10] Consistent with that theory, those reporting 'always' and 'often' performing TOA, OE and OSW generally had higher odds for CVD compared to those with low OPA. It is reasonable to infer that those who 'always' perform OPA are most susceptible to inadequate recovery.

LTPA was not found to be significantly associated with prevalent CVD in this study. However, non-significant associations were largely in the expected direction and in agreement with previous literature suggesting a negative relationship.[2 21 22] The non-significant results in this study may be due to the known measurement error of the self-reported LTPA data in the NHIS.[23] Additionally, given that associations between OPA and CVD were different across LTPA levels, further consideration should be given to potential interactions between the two domains of activity in the same model in future examinations.

Sex stratification

Though a recent meta-analysis found OPA to be associated with higher mortality among men (HR 1.18, 95% CI 1.05 to 1.34), no such association was observed in women (HR 0.90, 95% CI 0.80 to 1.01).[7] This inconsistency, despite proposed

Table 3 Sex-stratified odds of cardiovascular disease by occupational physical activity and leisure-time physical activity

	Total occupational activity			Occupational exertion			Occupational standing			Leisure-time physical activity			
	OR	P value	95% CI	OR	P value	95% CI	OR	P value	95% CI		OR	P value	95% CI
Female													
Never	1.00	--	--	1.00	--	--	1.00	--	--	0 min	1.00	--	--
Seldom	0.61	0.293	(0.25 to 1.53)	**2.06**	**0.014**	**(1.16 to 3.67)**	0.73	0.533	(0.28 to 1.95)	1–149 min	0.99	0.984	(0.56 to 1.78)
Sometimes	0.99	0.973	(0.42 to 2.31)	0.95	0.871	(0.48 to 1.85)	1.11	0.824	(0.43 to 2.90)	≥150 min	0.78	0.365	(0.45 to 1.35)
Often	1.24	0.599	(0.55 to 2.78)	1.89	0.068	(0.95 to 3.73)	1.14	0.763	(0.49 to 2.64)	--	--	--	--
Always	1.46	0.320	(0.69 to 3.07)	**2.32**	**0.002**	**(1.37 to 3.93)**	1.62	0.200	(0.77 to 3.37)	--	--	--	--
P for trend	p=0.008			p=0.012			p=0.009				p=0.312		
Male													
Never	1.00	--	--	1.00	--	--	1.00	--	--	0 min	1.00	--	--
Seldom	1.67	0.290	(0.65 to 4.31)	1.54	0.123	(0.89 to 2.68)	1.47	0.399	(0.60 to 3.64)	1–149 min	1.33	0.291	(0.78 to 2.25)
Sometimes	1.71	0.244	(0.69 to 4.19)	0.93	0.806	(0.52 to 1.68)	0.91	0.836	(0.37 to 2.22)	≥150 min	1.08	0.705	(0.73 to 1.58)
Often	1.90	0.183	(0.74 to 4.88)	1.41	0.285	(0.75 to 2.65)	1.26	0.611	(0.51 to 3.11)	--	--	--	--
Always	**2.47**	**0.041**	**(1.04 to 5.91)**	**1.97**	**0.017**	**(1.13 to 3.44)**	1.89	0.105	(0.87 to 4.08)	--	--	--	--
P for trend	0.090			0.035			0.030				0.936		

All models adjusted for age, working status, race/ethnicity, smoking status, alcohol consumption, family income-to-poverty ratio, BMI, education, US nativity, leisure-time activity category and total occupational activity; all strata had at least 80 cases of reported CVD; boldface text indicates significance (p<0.05).
BMI, body mass index; CVD, cardiovascular disease.

Table 4 Adjusted odds of cardiovascular disease by frequency of leisure-time and occupational physical activity in never-smokers

	Total occupational activity			Occupational exertion			Occupational standing			Leisure-time physical activity			
	OR	P value	95% CI	OR	P value	95% CI	OR	P value	95% CI		OR	P value	95% CI
Never	1.00	--	--	1.00	--	--	1.00	--	--	0 min	1.00	--	--
Seldom	1.47	0.378	(0.62 to 3.44)	**2.24**	**0.002**	**(1.35 to 3.72)**	1.10	0.830	(0.45 to 2.69)	1–149 min	1.19	0.531	(0.69 to 2.08)
Sometimes	1.74	0.179	(0.77 to 3.91)	1.07	0.829	(0.57 to 2.01)	1.25	0.609	(0.53 to 2.98)	≥150 min	0.91	0.675	(0.59 to 1.42)
Often	2.29	0.056	(0.98 to 5.37)	**2.50**	**0.002**	**(1.41 to 4.44)**	1.49	0.324	(0.67 to 3.32)	--	--	--	--
Always	**3.00**	**0.006**	**(1.38 to 6.51)**	**3.00**	**0.001**	**(1.80 to 5.02)**	2.06	0.061	(0.97 to 4.40)	--	--	--	--
P for trend	0.001			0.001			0.006				0.454		

All models adjusted for age, sex, working status, race/ethnicity, alcohol consumption, family income-to-poverty ratio, BMI, education, US nativity, leisure-time activity category and total occupational activity; all strata had at least 80 cases of reported CVD; boldface text indicates significance (p<0.05).
BMI, body mass index; CVD, cardiovascular disease.

mechanisms that should affect both sexes, limits confidence in the OPA health paradox. Contrasting with previous literature, the current sex-stratified results demonstrate associations that are largely similar across sexes.[7 24–26] The differing results could be attributed to differences in US workplace job/task roles across sexes compared with other countries. Potentially, women who perform high levels of OPA in the US have similar cardiovascular responses as men due to more equalised work/rest ratios or job task training compared with women in other countries. Further investigation is certainly warranted, though our consistent results across sexes are supportive of proposed mechanisms for the OPA health paradox.

Restriction to never-smokers

Another major criticism of the OPA health paradox is that uncontrolled confounding due to related health behaviours could remain.[26] A recent criticism proposed discrete smoking categorisation as contributing to the relationships seen.[12] The current analysis attempted to address this by presenting the relationships in a restricted sample of never-smokers. Among never-smokers, we found that the direct associations between TOA and OE with CVD were of similar magnitude and direction compared with the overall sample. Additionally, a potential dose–response relationship was observed between OPA and CVD in never-smokers (all p<0.01). This suggest that uncontrolled confounding by smoking status is likely not driving the relationships seen, however, future investigation is warranted to fully understand the robustness of the associations against confounding by smoking status.

While covariates were chosen based on previous studies, the influence of socioeconomic status is difficult to completely control.[7 27–30] Even with multifactorial classification of socioeconomic status (race/ethnicity, education, income, nativity, etc), representation of multidimensional and institutionalised phenomena such as race and socioeconomic status using discrete self-reported categorisations may fall short.[31] While analyses included adjustment and stratification, uncontrolled confounding may still exist and it must be acknowledged as an

Table 5 Adjusted odds of cardiovascular disease by occupational physical activity stratified by level of leisure-time physical activity

	Total occupational activity			Occupational exertion			Occupational standing		
	OR	P value	95% CI	OR	P value	95% CI	OR	P value	95% CI
0 min of leisure-time physical activity									
Never	1.00	--	--	1.00	--	--	1.00	--	--
Seldom	2.46	0.227	(0.57 to 10.67)	2.08	0.061	(0.97 to 4.46)	1.25	0.738	(0.34 to 4.61)
Sometimes	1.86	0.385	(0.46 to 7.55)	0.86	0.729	(0.38 to 1.98)	1.41	0.602	(0.39 to 5.11)
Often	3.29	0.094	(0.81 to 13.34)	1.27	0.584	(0.54 to 3.02)	1.00	0.999	(0.32 to 3.14)
Always	3.37	0.072	(0.90 to 12.64)	**2.08**	**0.046**	**(1.01 to 4.25)**	2.18	0.148	(0.76 to 6.27)
P for trend	0.031			0.147			0.038		
1–149 min of leisure-time physical activity									
Never	1.00	--	--	1.00	--	--	1.00	--	--
Seldom	3.73	0.079	(0.86 to 16.21)	1.93	0.218	(0.68 to 5.52)	3.48	0.088	(0.83 to 14.56)
Sometimes	**4.61**	**0.029**	**(1.17 to 18.16)**	1.11	0.861	(0.36 to 3.39)	2.21	0.295	(0.50 to 9.73)
Often	**6.47**	**0.006**	**(1.70 to 24.63)**	**4.60**	**0.002**	**(1.79 to 11.80)**	**4.60**	**0.017**	**(1.31 to 16.08)**
Always	**10.87**	**0.001**	**(2.87 to 41.15)**	**4.78**	**0.001**	**(1.86 to 12.27)**	**5.21**	**0.009**	**(1.52 to 17.79)**
p for trend	0.003			0.001			0.027		
≥150 min of leisure-time physical activity									
Never	1.00	--	--	1.00	--	--	1.00	--	--
Seldom	0.68	0.333	(0.31 to 1.49)	1.55	0.086	(0.94 to 2.54)	0.89	0.784	(0.38 to 2.07)
Sometimes	1.05	0.898	(0.51 to 2.16)	0.97	0.907	(0.53 to 1.75)	0.77	0.517	(0.34 to 1.72)
Often	0.83	0.625	(0.39 to 1.77)	1.08	0.821	(0.55 to 2.14)	1.01	0.988	(0.47 to 2.17)
Always	1.07	0.848	(0.54 to 2.12)	1.52	0.128	(0.89 to 2.59)	1.31	0.427	(0.67 to 2.57)
p for trend	0.393			0.331			0.104		

All models adjusted for age, sex, working status, race/ethnicity, smoking status, alcohol consumption, family income-to-poverty ratio, BMI, education and US nativity; all strata had at least 80 cases of reported CVD; boldface text indicates significance (p<0.05).
BMI, body mass index; CVD, cardiovascular disease.

ongoing limitation to this study and investigations of the OPA health paradox in general.

Joint associations between OPA and LTPA

The joint associations explored provided insight on the potential interaction between OPA and LTPA and their associations with CVD. At higher levels of LTPA the association between OPA and CVD was lessened. This aligns somewhat with proposed mechanisms for the OPA health paradox where OPA does not improve fitness, increasing 24-hour cardiovascular strain on days with OPA.[10 32] Under this hypothesised mechanism, those with high LTPA would be associated with high fitness and therefore would be less susceptible to cardiovascular strain during OPA. Two previous studies demonstrated a significantly increased resting and 24-hour ambulatory systolic blood pressure after a 16-week moderate-intensity aerobic exercise programme of 60 min/week in hospital cleaners with high OPA.[33 34] The current results agree with these studies, concluding that added LTPA of less than 150 min/week to already high levels of OPA is potentially detrimental to cardiovascular health. This suggests that the amount of LTPA performed in conjunction with OPA is critical. It may be that LTPA <150 min/week is detrimental to those with high OPA due to increased cardiovascular load while LTPA ≥150 min/week is beneficial due to improved fitness. Furthermore, temporality of the LTPA and OPA exposures may be important for cardiovascular risk because fitness changes would directly affect the interaction of OPA and cardiovascular strain.

Strengths

The current analysis had several strengths. First, the multidimensional and stepped categorisation of TOA, OE and OSW allowed for more specific analysis of the association with CVD. This provided information about the modality and pattern of activity that could be compared with proposed mechanisms.[10] Also, this study used a population-based and nationally representative survey design to allow for generalisation of results to the civilian non-institutionalised US population. Lastly, this study provided novel stratified or restricted analyses by sex, smoking and LTPA level. These subanalyses provide valuable steps forward in addressing the concerns with uncontrolled confounding and disparate findings regarding the OPA health paradox. However, future research should confirm these findings with improved methods including longitudinal designs and improved measurement of OPA, LTPA and CVD.

Limitations

This cross-sectional analysis is limited by the inability to establish temporality, making causal inference difficult. However, OPA exposure is likely stable over time making the cross-sectional analysis stronger (ie, reflecting historical longitudinal exposure to OPA). Second, selection bias is likely present where individuals with CVD may self-select out of high activity occupations. However, this bias would work towards the null making the presented estimates conservative. CVD prevalence was assessed using self-report at one time using a yes/no question without providing information about the duration, severity or cause of the disease. However, high validity of self-reported CVD in population-based studies has been reported previously.[17–19] TOA, OE, OSW and LTPA were self-reported and subject to recall and social desirability biases. Though, over-reporting is more likely in activity recall which would bias the current results towards the null, thus resulting in more conservative estimates.[35] TOA, OE and OSW were assessed using only two questions referring to the amount and certain type of activity done at work and the LTPA questionnaire restricts to activities of ≥10 min and may miss sporadic activity.[2 5] Objective measurement of OPA and LTPA could address these limitations in the future. Lastly, additional covariates outside of what is available in the NHIS may be helpful in future analyses to address remaining concerns around uncontrolled confounding. Specific covariates of interest for future analyses include occupational tenure, other occupational exposures such as particulate exposure and family history of CVD.

CONCLUSIONS

The current study examined the associations between TOA, OE, OSW and LTPA with CVD. Increased odds for CVD were observed in those reporting high amounts of TOA, OE and OSW. The direct association between CVD and OE was apparent in both sexes. Interestingly, smoking does not appear to confound the relationship between OPA and CVD as the direct associations were more exaggerated among never-smokers. Lastly, associations between CVD and OE were lessened in those reporting high levels of LTPA.

These data support the potential existence of an OPA health paradox in the US. Limitations to the research investigating the paradox remain and future studies should explore this topic using epidemiological and experimental methods to develop the understanding of a potential causal framework and mechanistic pathways. If proven true, however, the implications of such a paradox on the modern workforce would be significant and would likely justify public health initiatives to limit or restructure OPA requirements to prioritise worker health.

Acknowledgements Data Source: NCHS, National Health Interview Survey, 2015. The results of this study are presented clearly, honestly and without fabrication, falsification or inappropriate data manipulation.

Contributors TDQ, PLY, YS and BBG contributed to analytical design development, analysis and manuscript authorship. PMS and GPW contributed to critical manuscript review and authorship.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Disclaimer The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention. The authors' analyses, interpretations, and conclusions do not necessarily represent the views of the National Center for Health Statistics (NCHS), which is responsible only for the initial data.

Competing interests BBG discloses grant funding from the Agency for Healthcare Quality Research, the American Heart Association, the National Institutes of Health, and the Tomayko Fund.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Public data source: NCHS, National Health Interview Survey, 2015.

ORCID iDs
Tyler D. Quinn http://orcid.org/0000-0002-0172-062X
Peter M. Smith http://orcid.org/0000-0001-8286-4563

REFERENCES

1 Leitzmann MF, Park Y, Blair A, *et al*. Physical activity recommendations and decreased risk of mortality. *Arch Intern Med* 2007;167:2453–60.
2 Physical Activity Guidelines Advisory Committee. *Physical activity guidelines Advisory Committee scientific report*. Washington, DC: US Department of Health and Human Services, 2018.
3 Durstine JL, Gordon B, Wang Z, *et al*. Chronic disease and the link to physical activity. *J Sport Health Sci* 2013;2:3–11.
4 Richard A, Martin B, Wanner M, *et al*. 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;12:184–92.
5 Physical Activity Guidelines Advisory Committee. *2018 physical activity guidelines for Americans*. Washington D.C: U.S. Department of Health and Human Services, 2018.
6 Folsom AR, Arnett DK, Hutchinson RG, *et al*. Physical activity and incidence of coronary heart disease in middle-aged women and men. *Med Sci Sports Exerc* 1997;29:901–9.
7 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;52:1320–6.
8 Holtermann A, Mortensen OS, Burr H, *et al*. The interplay between physical activity at work and during leisure time--risk of ischemic heart disease and all-cause mortality in middle-aged Caucasian men. *Scand J Work Environ Health* 2009;35:466–74.
9 Holtermann A, Hansen JV, Burr H, *et al*. The health paradox of occupational and leisure-time physical activity. *Br J Sports Med* 2012;46:291–5.
10 Holtermann A, Krause N, van der Beek AJ, *et al*. 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;52:149–50.
11 Gupta N, Hallman DM, Dumuid D, *et al*. Movement behavior profiles and obesity: a latent profile analysis of 24-h time-use composition among Danish workers. *Int J Obes* 2020;44:409–17.
12 Shephard RJ. Is there a 'recent occupational paradox' where highly active physically active workers die early? Or are there failures in some study methods? *Br J Sports Med* 2019;53:1557–9.
13 National Center for Health Statistics. *Survey Desciption*. Hyattsville, Maryland: National Health Interview Survey, 2015.
14 Centers for Disease Control and Prevention. *2008 physical activity guidelines for Americans: centers for disease control and prevention*, 2008.
15 Description S. *National health interview survey, 2017*. Hyattsville, Maryland: National Center for Health Statistics, 2018.
16 Sabanayagam C, Shankar A. Sleep duration and cardiovascular disease: results from the National health interview survey. *Sleep* 2010;33:1037–42.
17 Lampe FC, Walker M, Lennon LT, *et al*. Validity of a self-reported history of doctor-diagnosed angina. *J Clin Epidemiol* 1999;52:73–81.
18 Walker MK, Whincup PH, Shaper AG, *et al*. Validation of patient recall of doctor-diagnosed heart attack and stroke: a postal questionnaire and record review comparison. *Am J Epidemiol* 1998;148:355–61.
19 Okura Y, Urban LH, Mahoney DW, *et al*. Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure. *J Clin Epidemiol* 2004;57:1096–103.
20 Smith P, Ma H, Glazier RH, *et al*. The relationship between occupational standing and sitting and incident heart disease over a 12-year period in Ontario, Canada. *Am J Epidemiol* 2018;187:27–33.
21 Wahid A, Manek N, Nichols M, *et al*. Quantifying the association between physical activity and cardiovascular disease and diabetes: a systematic review and meta-analysis. *J Am Heart Assoc* 2016;5:e002495.
22 Sattelmair J, Pertman J, Ding EL, *et al*. Dose response between physical activity and risk of coronary heart disease. *Circulation* 2011;124:789–95.
23 Control CfD,. *Prevention. health behaviors of adults: United States, 2011–2014*, 2014.
24 Etemadi A, Abnet CC, Kamangar F, *et al*. Impact of body size and physical activity during adolescence and adult life on overall and cause-specific mortality in a large cohort study from Iran. *Eur J Epidemiol* 2014;29:95–109.
25 Wanner M, Tarnutzer S, Martin BW, *et al*. Impact of different domains of physical activity on cause-specific mortality: a longitudinal study. *Prev Med* 2014;62:89–95.
26 Shaikh RA, Sikora A, Siahpush M, *et al*. Occupational variations in obesity, smoking, heavy drinking, and non-adherence to physical activity recommendations: findings from the 2010 National health interview survey. *Am J Ind Med* 2015;58:77–87.
27 Petersen CB, Eriksen L, Tolstrup JS, *et al*. Occupational heavy lifting and risk of ischemic heart disease and all-cause mortality. *BMC Public Health* 2012;12:1070.
28 Holtermann A, Mortensen OS, Burr H, *et al*. Physical demands at work, physical fitness, and 30-year ischaemic heart disease and all-cause mortality in the Copenhagen male study. *Scand J Work Environ Health* 2010;36:357–65.
29 Hu G-C, Chien K-L, Hsieh S-F, *et al*. Occupational versus leisure-time physical activity in reducing cardiovascular risks and mortality among ethnic Chinese adults in Taiwan. *Asia Pac J Public Health* 2014;26:604–13.
30 Krause N, Arah OA, Kauhanen J. Physical activity and 22-year all-cause and coronary heart disease mortality. *Am J Ind Med* 2017;60:976–90.
31 Williams DR, Mohammed SA, Leavell J, *et al*. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci* 2010;1186:69–101.
32 Hallman DM, Birk Jørgensen M, Holtermann A. On the health paradox of occupational and leisure-time physical activity using objective measurements: effects on autonomic imbalance. *PLoS One* 2017;12:e0177042.
33 Korshøj M, Lidegaard M, Skotte JH, *et al*. Does aerobic exercise improve or impair cardiorespiratory fitness and health among cleaners? a cluster randomized controlled trial. *Scand J Work Environ Health* 2015;41:140–52.
34 Korshøj M, Krause N, Clays E, *et al*. Does Aerobic Exercise Increase 24-Hour Ambulatory Blood Pressure Among Workers With High Occupational Physical Activity?-A RCT. *Am J Hypertens* 2017;30:444–50.
35 Sallis JF, Saelens BE. Assessment of physical activity by self-report: status, limitations, and future directions. *Res Q Exerc Sport* 2000;71:1–14.