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ORIGINAL PAPER

Association between exposure to heavy occupational lifting and cardiac structure and function: a cross-sectional analysis from the Copenhagen City Heart Study

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Abstract

To investigate cross-sectional associations between heavy occupational lifting and cardiac structure and function. Participants from the 5th round of the Copenhagen City Heart Study, aged < 65 years old, answering a questionnaire regarding occupational physical activity, heavy occupational lifting, use of anti-hypertensive and heart medication, and data on blood pressure, cardiac structure and function, from an echocardiographic examination, were included. Adjusted linear regressions and logistic regressions were applied to estimate the cross-sectional association between heavy occupational lifting and cardiac structure and function across all included participants and in groups stratified by hypertension status, and the risk for having abnormal values of cardiac structure and function. 2511 participants were included. The cross-sectional standardized associations between heavy occupational lifting and measures of cardiac structure and function showed a trends for raised left ventricular mass index (LVMi) (β 0.14, 99% CI − 0.03 to 0.31). The standardized associations stratified by hypertensive status showed significant associations between exposure to heavy occupational lifting and LVMi (β 0.20, 99% CI − 0.002 to 0.40) and a trend of a raised end-diastolic interventricular septal thickness (IVSd) (β 0.15, 99% CI − 0.03 to 0.33) among normotensives. Exposure to heavy occupational lifting increased the odds for an abnormal IVSd (OR 1.42, 99% CI 1.07–1.89). This cross-sectional study shows heavy occupational lifting to associate with indices of abnormal cardiac structure and function among normotensives, indicating an increased risk for cardiovascular disease.

Keywords Occupational physical activity · Cardiovascular disease · Manual handling · Blue-collar · Occupational epidemiology · Heavy lifting · Cohort study

Introduction

Studies have shown that workers exposed to high levels of occupational physical activity (OPA), often including occupational lifting (OL), have a higher risk of cardiovascular disease than sedentary workers [1, 2]. The background for this increased risk of cardiovascular disease is not fully understood due to a lack of knowledge of the physiologic mechanisms linking high levels of OPA to cardiovascular disease [3]. One of the proposed explanations is exposure to heavy OL [3]. From laboratory studies, it is known that a single lift acutely increases blood pressure (BP) [4]. Thus, heavy OL performed several times per workday, repeatedly over the workweek, as well as throughout a whole career, might explain the increased BP among workers reporting exposure to heavy OL [5, 6]. However, to understand the background for the higher risk of cardiovascular disease,

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investigations of mechanical changes in the cardiovascular system are needed.

Only one previous paper has investigated the associations between OL and cardiac structure and function assessed by echocardiography [7]. The paper by Nde and colleagues reported construction workers (n = 50), performing heavy load work including OL, to have a greater thickness of the interventricular septum and left ventricular (LV) posterior wall compared to office workers (n = 50) matched on weight, height, and age. An increased thickness of the septum and posterior wall is assumed to increase the risk of cardiovascular disease [8]. Previous studies investigating the effect of physical activity on echocardiographic measures did not separate activity into domains (work/leisure) or only included leisure-time physical activity (LTPA) [9, 10]. This study aims to investigate the association between heavy OL and cardiac structure and function in a very large cohort from the general population. The hypothesis of the study were that heavy OL would impair cardiac structure and function.

Methods

The Copenhagen City Heart Study (CCHS) has collected data on health by a large variety of biological, environmental, and lifestyle related factors across randomly selected Copenhagen citizens in five examination rounds. Data from the 5th (2011–2015) examination were included in this study. The collection of data was performed following the 2nd Declaration of Helsinki and was approved by the local ethical committee. All participants gave written informed consent.

Inclusion of participants

In total, 2511 participants were included. Inclusion criteria were; (1) answering the questionnaire item regarding OPA including exposure to heavy OL, (2) information on the use of anti-hypertensive or heart medication, (3) measurement of BP at the examination, and (4) aged 18 to < 65 years.

Assessment of exposure

The level of OPA was obtained by questionnaire: "Please describe your level of occupational physical activity within the past year" with the following response categories:" (1) predominantly sedentary; (2) sitting or standing, some walking; (3) walking, some handling of material; (4) heavy manual work" [11]. By answering 3 or 4, an additional question regarding heavy OL was applied: "Do you lift heavy burdens?" with the response categories: "(1) yes; (2) no". Classification of exposure to heavy OL was defined as answering "yes" to the question concerning heavy OL, and the reference group was defined as those participants answering 1, 2 and 3 or 4 to the level of OPA combined with answering "no" to the heavy OL question.

Assessment of cardiac structure and function

Cardiac structure and function were assessed by echocardiography, performed using Vivid 9 Ultrasound Systems from GE Healthcare (Horten, Norway) by experienced sonographers. Analyses of the echocardiography were performed using the commercially available software Echopac version 113.1.5 from GE Healthcare (Horten, Norway) by experienced blinded investigators.

LV interventricular septal thickness (IVSd), LV internal diameter, and LV posterior wall thickness (LVPWd) were measured in the parasternal long-axis view during end-diastole [12]. From LV chamber dimensions, LV mass (using the Devereux formula [13]) and LV mass index (LVMi) were estimated. LV ejection fraction (LVEF) was measured by a semi-automated function performed in the apical 4-chamber and 2-chamber views, tracing the endocardial border during the cardiac cycle. Also, in the apical 4-chamber and 2-chamber views, at end-systole, left atrial (LA) volumes were measured and LA volume index (LAVi) was calculated. Early (E) peak mitral inflow velocities was measured at the tips of the mitral leaflets by pulsed wave Doppler. Peak early (e') diastolic mitral annular velocity was measured by tissue Doppler imaging at the septal and lateral walls of the mitral annulus and averaged. The relative wall thickness (RWT) were calculated as (2 × posterior wall thickness)/(LV internal diameter at end of diastole) [12].

2D speckle tracking echocardiography (2DSTE) analysis was performed in the apical 4-chamber, 2-chamber, and 3-chamber views, by dividing the LV into 6 segments in each of the 3 views. The LV endocardial border was traced with a semi-automated function outlining a region of interest spanning from the epicardial to the endocardial border. In cases of inaccurate tracing, the region of interest was manually adjusted by the investigator. Global longitudinal strain (GLS) was calculated as the mean peak systolic value from the 18 segments of the LV. All strain parameters are reported as percentages.

To estimate the effect of OL on the risk of having abnormal cardiac structure and function, the following recommended cutoffs were used to dichotomize the echocardiographic measures into normal or abnormal. Normal values were classified as: LVEF for males at > 52% and > 54% for females; e' at ≥ 9 cm/s [12]; LAVi at 16–34 mL/m^2; IVSd and LVPWd for males at < 1.0 cm, and for females at < 0.9 cm; peak tricuspid regurgitation gradient at < 26 mmH [12], the deceleration time at 119–242 ms; GLS at > 20.0% [12]. LV hypertrophy was defined as LVMi > 115 g/m^2 for males and > 95 g/m^2 for females [12]. The LV geometry were

classified as normal (RWT < 0.42 cm and LMVi ≤ 95 g/m^2 for females and ≤ 115 g/m^2 for males), concentric remodeling (RWT > 0.42 cm and LMVi ≤ 95 g/m^2 for females and ≤ 115 g/m^2 for males), eccentric hypertrophy (< 0.42 cm and LMVi > 95 g/m^2 for females and > 115 g/m^2 for males) and concentric hypertrophy (RWT > 0.42 cm and > 95 g/m^2 for females and > 115 g/m^2 for males) [12].

Assessment of covariates

Previous literature has shown several factors to impact both exposures to OPA, such as OL, and cardiac structure and function. Therefore, these factors were included as covariates: sex (male/female) [14, 15]; age (categories of < 40; 50–59; 60–70 years) [16, 17]; body mass index (BMI) (categories of < 18.5; 18.5–24.9; 25.0–29.9; ≥ 30 kg/m^2) [14, 18] calculated from objectively measured body height and weight (kg/m^2); smoking (categories of nonsmoking and currently smoking) [19, 20]; length of education (no formal education; low educated up to 3 years; vocationally educated 1–3 years; higher educated; academically educated) [21]; the level of LTPA were collected by answering which of the following categories are the best fit (*mainly sedentary* "you spend most of your leisure time performing sedentary tasks"; *light physical active* "you go for a walk, use your bicycle a little or perform activity for at least 4 h per week"; *moderate physical active* " you are an active athlete, for at least 3 h/week; *strenuous physical active* "you take part in competitive sports, or perform moderate to vigorous activity more than 4 h/week") [11]; and level of cardiorespiratory fitness was self-rated in the following categories (lower; similar; higher cardiorespiratory fitness compared to peers of same sex and age) [22]. Hypertension was defined as self-reported use of any anti-hypertensive drugs or a measured systolic BP (SBP) ≥ 140 mmHg or DBP ≥ 90 mmHg [23]. BP was measured three times on the non-dominant arm after 5-min sitting by use of a London School of Hygiene sphygmomanometer [24]. The rate of the fall of the mercury column was set to 2 mm/s. The mean value of the three repeated measures was included in the classification of hypertension.

An additional factor for the description of the population were included in Table 1, being vital exhaustion. Vital exhaustion, a psychological measure characterized by fatigue and depressive symptoms were collected by 17 questionnaire items [25].

Statistical analysis

All statistical analyses were performed using statistical software SAS, version 9.4 (SAS Institute, Cary, NC, USA). The null hypothesis was that no differences in cardiac structure and function should be seen across groups stratified by hypertensive status and exposure to OL. As the presences of hypertension are known to affect cardiac structure and function by leading to increased thickness and mass of the LV, it was investigated whether hypertension modified the effect between exposures to heavy OL and cardiac structure and function, by the inclusion of an interaction term in the linear regression model. If the interaction turns out statistically significant the included population will be stratified by hypertensive status.

Differences between mean values of baseline characteristics and echocardiographic data in groups stratified by exposure to OL and hypertensive status were estimated by t-test for continuous outcomes and Chi-square test for categorical outcomes. Multivariable linear regression models were used to investigate the association between heavy OL and cardiac structure and function. The outcome variables were standardized (mean = 0 and standard deviation = 1), to enable interpretation of regression coefficients across outcomes. The cross-sectional analysis was adjusted for sex, age, BMI, smoking, education, level of LTPA and cardiorespiratory fitness. No exposure for OL was treated as a reference. The odds of having abnormal values of cardiac structure and function as a function of heavy OL were estimated by logistic regression performed in generalized estimating equation models. Statistical significance was defined as a two-sided p-value < 0.05, secondary a Bonferroni correction was applied, therefore the hypotheses were tested at a significance level of 0.01, with a corresponding confidence interval (CI) of 99%.

Results

The response rate in the 5th round of the CCHS was 49.3%, and from the 4543 participants attending the examination 2511 participants were included in this study (Fig. 1). The 2511 participants included in the analysis differed from the 2032 participants excluded from the analysis (see supplementary table 1).

The baseline characteristics of the participants included in the analysis had a mean age of 45 years, 56% were women and 9% were exposed to OL (Table 1). All baseline characteristics except for exposure to heavy OL and smoking differed between normotensives and hypertensives (Table 1).

The presence of hypertension modified the associations between exposure to heavy OL and cardiac structure and function (p < 0.05 for all), thus all analyses was stratified by hypertension status. Among the normotensive participants the mean values of measures of cardiac structure and function in groups stratified according to exposure to OL showed significantly lower levels of e' (p = 0.001), and increased IVSd (p = 0.002) among participants exposed to OL compared to those not. Also, a higher proportion (58.0% vs. 45.3%, p = 0.002) of

Table 1 Baseline characteristics of the included participants for the cross-sectional analysis

	All, N = 2511				Normotensives, n = 1532				Hypertensives, n = 979				*p*
	Mean	SD	n (%)	Range	Mean	SD	n (%)	Range	Mean	SD	n (%)	Range	
Age (years)	**45.4**	**12.9**		**20.0–65.0**	**41.7**	**12.4**		**20.0–65.0**	**51.1**	**11.6**		**21.0–65.0**	**0.001**
Sex (%female)			**1408 (56.1)**				**970 (63.3)**				**438 (44.7)**		**0.001**
Body mass index (kg/m^2)	**25.0**	**4.2**		**16.3–56.3**	**24.1**	**3.8**		**16.3–56.3**	**26.5**	**4.3**		**17.2–51.7**	**0.001**
Smoking (%current smokers)			469 (18.7)				274 (17.9)				195 (19.9)		0.20
Systolic blood pressure (mmHg)	**133.4**	**18.5**		**88.0 – 215.0**	**122.4**	**10.2**		**88.0–139.0**	**150.5**	**15.2**		**100.0–215.0**	**0.001**
Diastolic blood pressure (mmHg)	**79.2**	**11.0**		**42.0–131.0**	**74.1**	**7.7**		**42.0–89.0**	**87.3**	**10.4**		**47.0–131.0**	**0.001**
Blood pressure ≥ 90/ ≥ 140 mmHg			**899 (35.8)**				**–**				**899 (91.8)**		
Using anti-hypertensive medication			**258 (10.3)**				**–**				**258 (26.4)**		
Hypertensive (% ≥ 90/ ≥ 140 mmHg or using anti-hypertensive medication)			**979 (39.0)**				**–**				**–**		
Left ventricular hypertrophy[1] (%)			**229 (9.1)**				**94 (6.1)**				**135 (13.8)**		**0.001**
Education													
No formal education			**231 (9.2)**				**141 (9.2)**				**90 (9.2)**		**0.001**
Low educated, < 3 years			**207 (8.2)**				**122 (8.0)**				**85 (8.7)**		
Vocational education, 1–3 years			**396 (15.8)**				**202 (13.2)**				**194 (19.8)**		
Higher education, > 3 years			**633 (25.2)**				**368 (24.0)**				**265 (27.1)**		
Academic education			**1039 (41.4)**				**695 (45.4)**				**344 (35.1)**		
Occupational physical activity													**0.001**
Predominantlysedentary			**1175 (46.8)**				**752 (49.1)**				**423 (43.2)**		
Sitting or standing, some walking			**815 (32.5)**				**485 (31.7)**				**330 (33.7)**		
Walking, some handling of material			**467 (18.6)**				**273 (17.8)**				**194 (19.8)**		
Heavy manual work			**54 (2.2)**				**22 (1.4)**				**32 (3.3)**		
Occupational heavy lifting (%yes)			228 (9.1)				131 (8.6)				97 (9.9)		0.25
Leisure-time physical activity													**0.001**
Inactive/light physical active < 2 h/week			**157 (6.3)**				**83 (5.4)**				**74 (7.6)**		
Light physical active 2–4 h/week			**857 (34.1)**				**488 (31.9)**				**369 (37.7)**		
Light physical active > 4 h/week OR Moderate to vigorous physical activity 2–4 h/week			**1217 (48.5)**				**789 (51.5)**				**428 (43.7)**		
Moderate to vigorous physical activity > 4 h/week			**275 (11.0)**				**170 (11.1)**				**105 (10.7)**		
Vital exhaustion (sum, 0–17)	**3.2**	**3.5**		**0.0–17.0**	**3.4**	**3.6**		**0.0–17.0**	**2.8**	**3.3**		**0.0–17.0**	**0.003**
Cardiorespiratory fitness (% similar level as peers)			**1188 (47.3)**				**723 (47.2)**				**465 (47.5)**		**0.04**

Significant ($p < 0.05$) different mean values in the stratified groups of normotensives and hypertensive participants are marked by bold

[1]Hypertrophy defined as LVMi > 115 g/m^2 for males and > 95 g/m^2 for females

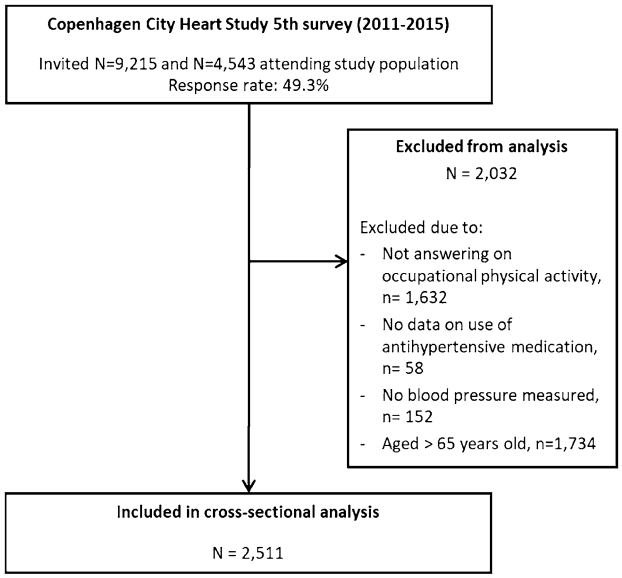

Fig. 1 Flow of the inclusion of participants for the cross-sectional analysis in the fifth examination of the Copenhagen City Heart Study

those exposed to OL had an abnormal IVSd compared to those not. Furthermore, deceleration time showed a trend towards being increased among those exposed to OL compared to the non-exposed ($p = 0.04$). Among the hypertensive participants, we found a trend towards a lower mean level of GLS ($p = 0.08$) and e´ ($p = 0.09$) seen among those exposed to OL in comparison to the non-exposed (Table 2).

The linear regression models showed trends of OL to raise LVMi ($p = 0.03$), and LAVi ($p = 0.06$). The models stratified by hypertensive status showed significant positive associations between exposure to OL and LVMi ($p = 0.001$) and a trend of raised IVSd ($p = 0.02$). Among the hypertensive participants, no associations between exposure to OL and measures of cardiac structure and function were seen (Table 3).

Among all participants and the normotensives, the odds of having an abnormal IVSd (all OR 1.23, 99% CI 0.98–1.53; normotensives OR 1.42, 99% CI 1.07–1.89) and deceleration time (all OR 1.30, 99% CI 0.98–1.74; normotensive OR 1.41, 99% CI 0.95–2.08) were higher when exposed to OL. The risk of an abnormal e' was 45% higher among normotensives exposed to heavy OL and on the contrary, 25% lower for hypertensives exposed to heavy OL. And among the hypertensive participants only, did the risk of having LV hypertrophy increase by exposure to heavy OL (OR 1.41, 99% CI 1.05–1.90) (Table 4).

Discussion

We investigated the cross-sectional associations between heavy OL and cardiac structure and function and found that OL was associated with higher LV mass and LV wall thickness. Furthermore, we found a 42% higher risk of having an abnormal thickness of the IVSd, 41% higher risk of abnormally prolonged deceleration time, and 45% higher risk of abnormal e' among normotensives exposed to heavy OL compared to normotensives not exposed to heavy OL (Table 4). These findings suggest that OL is associated with morphological changes leading to a higher risk of LV physiological re-modeling, related to the volume of the physical activity included in the OL, and thus cardiovascular disease and mortality [8, 26]. On the contrary, OL was associated with a 25% lower risk of having an abnormal e' among hypertensives. Hence, these findings indicate OL to hazardously affect LV re-modeling towards concentric hypertrophy, among normotensives, similar to the re-modeling seen among hypertensives and Olympic weight lifters [27]. Previous studies show hypertensive patients to tend to develop septal thickening of the basal part of the LV [28], where IVSd are measured. Increased wall stress may induce LV remodeling, the variance of regional wall stress variance can be explained by the law of Laplace, defining wall stress to be dependent on LV pressure, and modified by local LV geometry such as LV wall thickness and myocardial curvature, being maximal at the basal septum [29]. Thus, normotensives exposed to heavy OL may experience increased wall stress as previously reported [30], due to the intensity, frequency and duration of the OPA including lifting. On the contrary, among the hypertensives, no visible effects associated with exposure to OL can be seen separate from those expected to originate from the presence of hypertension in itself (Fig. 2).

LVMi is a strong independent predictor of incident cardiovascular disease [8, 12, 26]. Thus a higher risk for cardiovascular disease would be expected among normotensives exposed to OL due to the association with increases in LVMi.

In the field of exercise physiology, harmful changes in heart morphology are observed among athletes performing long term high-intensity endurance training, but especially among athletes performing static exercises [31] as well as among Olympic weight lifters, but in most cases, not among those performing resistance training [27]. These associations reported here could reflect the harmful changes in heart morphology seem among the Olympic weight lifters as the mass and thickness of the LV, as well as the risk of LV hypertrophy seems to be increased among normotensives exposed to OL combined with moderate

Table 2 Measures of cardiac structure and function by self-rated exposure to heavy occupational lifting

				Normotensives							
	All			Without occupational lifting			With occupational lifting				
	n	Mean (SD)	Abnormal n (%)	n	Mean (SD)	Abnormal n (%)	n	Mean (SD)	Abnormal n (%)	Δ	*p*
LVEF (%)	2314	57.12 (5.48)	487 (19.4)	1298	57.53 (5.16)	241 (17.2)	122	56.80 (5.85)	28 (21.4)	0.73	0.24
LVMi(g/m^2)	2453	82.54 (18.44)	361 (14.4)	1371	78.74 (17.68)	153 (10.9)	128	81.70 (14.85)	20 (15.3)	− 2.96	0.13
GLS (%)[¤]	2392	− 20.15 (2.07)	1266 (50.4)	1349	− 20.38 (2.02)	767 (54.7)	123	− 20.35 (2.05)	69 (52.7)	0.03	0.87
e (cm/s)[#]	2450	12.51 (3.55)	434 (17.3)	**1367**	**13.60 (3.31)**	**118 (8.4)**	**128**	**12.92 (3.62)**	**23 (17.6)**	**0.68**	**0.001**
LAVi (mL/m^2)	2471	23.35 (7.05)	418 (16.6)	1377	22.87 (6.84)	233 (16.6)	130	23.18 (7.73)	24 (18.3)	− 0.31	0.66
IVSd (cm)[*]	2457	1.00 (0.21)	1413 (56.3)	**1375**	**0.95 (0.18)**	**634 (45.3)**	**128**	**0.98 (0.17)**	**76 (58.0)**	**− 0.03**	**0.002**
LVPWd (cm)	2457	0.90 (0.17)	756 (30.1)	1375	0.86 (0.14)	312 (22.3)	128	0.87 (0.14)	34 (26.0)	− 0.01	0.31
RWT (cm)	2456	0.38 (0.07)	630 (25.1)	1375	0.37 (0.06)	255 (18.2)	128	0.37 (0.07)	29 (22.1)	0	0.47
Tr_maxpg (mmHg)	1210	19.72 (4.37)		714	18.92 (3.97)	–	55	18.55 (3.53)	–	0.37	–
Deceleration time (ms)[#]	2417	187.79 (40.80)	247 (9.8)	*1353*	*184.37 (38.07)*	*111 (7.9)*	*126*	*191.31 (39.93)*	*17 (13.0)*	*−6.94*	*0.04*
Left ventricular hypertrophy[1]			143 (5.7)	–	–	82 (5.9)	–	–	12 (9.2)	−3.30	0.13
Normal geometry[2]			1690 (67.3)			*1054 (75.2)*			*89 (67.9)*	*7.30*	*0.06*
Concentric remodelling[3]			518 (20.6)			*224 (16.0)*			*27 (20.6)*	*− 4.60*	
Eccentric hypertrophy[4]			116 (4.6)			*51 (3.6)*			*10 (7.6)*	*− 4.00*	
Concentric hypertrophy[5]			111 (4.4)			*30 (2.1)*			*2 (1.5)*	0.60	

	Hypertensives							
	Without occupational lifting			With occupational lifting				
	n	Mean (SD)	Abnormal n (%)	n	Mean (SD)	Abnormal n (%)	Δ	*p*
LVEF (%)	803	56.64 (5.81)	194 (22.0)	91	55.89 (5.97)	24 (24.7)	0.75	0.64
LVMi(g/m^2)	861	87.80 (18.41)	167 (18.9)	93	90.92 (19.73)	21 (21.6)	− 3.12	0.46
GLS (%)[¤]	*828*	*− 19.83 (2.09)*	*395 (44.8)*	*92*	*− 19.46 (2.00)*	*35 (36.1)*	*0.37*	*0.08*
e (cm/s)[#]	*860*	*10.89 (3.28)*	*271 (30.7)*	*95*	*11.09 (3.08)*	*22 (22.7)*	*− 0.80*	*0.09*
LAVi (mL/m^2)	868	23.95 (7.21)	148 (16.8)	96	24.87 (7.23)	13 (13.4)	− 0.92	0.38

Table 2 (continued)

	Hypertensives							
	Without occupational lifting			With occupational lifting				
	n	Mean (SD)	Abnormal n (%)	n	Mean (SD)	Abnormal n (%)	Δ	*p*
IVSd (cm)*	861	1.08 (0.20)	633 (71.8)	93	1.10 (0.19)	70 (72.2)	− 0.02	0.86
LVPWd (cm)	861	0.96 (0.21)	376 (42.6)	93	0.96 (0.16)	34 (35.1)	0	0.19
RWT (cm)	860	0.41 (0.08)	311 (35.3)	93	0.40 (0.07)	35 (36.1)	0.01	0.87
Tr_maxpg (mmHg)	390	21.08 (4.64)		51	21.65 (5.18)		− 0.54	
Deceleration time (ms)#	845	192.67 (42.49)	105 (11.9)	93	188.49 (57.27)	14 (14.4)	− 14.78	0.47
Left ventricular hypertrophy[1]			*116 (13.2)*			*19 (19.6)*	*− 6.40*	*0.07*
Normal geometry[2]			497 (56.3)			50 (51.5)	4.80	0.34
Concentric remodelling[3]			243 (27.6)			24 (24.7)	2.90	
Eccentric hypertrophy[4]			47 (5.3)			8 (8.2)	− 2.90	
Concentric hypertrophy[5]			68 (7.7)			11 (11.3)	− 3.60	

The models stratified on hypertensive status are adjusted for sex, age, BMI, smoking, education, level of cardiorespiratory fitness and leisure time physical activity. The model among all included participants is additionally adjusted for hypertensive status. Significantly different ($p < 0.01$) mean levels in groups exposed and non-exposed to heavy occupational lifting are marked by bold; and italics indicate a trend to difference ($p < 0.10$) in mean levels in groups exposed and non-exposed to heavy occupational lifting

LVEF Left ventricular ejection fraction, *LVMi* Left ventricular mass index, *GLS* Global longitudinal strain, *e* peak early diastolic mitral annular velocity, *LAVi* Left atrial volume index, *IVSd* end-diastolic interventricular septal thickness, *LVPWd* end-diastolic left ventricular posterior wall thickness, *RWT* Relative wall thickness, *Tr_maxpg* Peak Tricuspid regurgitation gradient

The categories of walking, some handling of material AND heavy manual work, but no heavy lifting are collapsed. The models stratified on hypertensive status are adjusted for sex, age, BMI, smoking and education

[2]Normal geometry defined as LVMi < 115 g/m^2 for males and < 95 g/m^2 for females and RWT ≤ 0.42 cm

[3]Concentric remodeling defined as LVMi < 115 g/m^2 for males and < 95 g/m^2 for females and RWT > 0.42 cm

[4]Eccentric hypertrophy defined as LVMi > 115 g/m^2 for males and > 95 g/m^2 for females and RWT ≤ 0.42 cm

[5]Concentric hypertrophy defined as LVMi > 115 g/m^2 for males and > 95 g/m^2 for females and RWT > 0.42 cm

Table 3 Associations between self-reported heavy occupational lifting and standardized values of indicators of heart morphology, adjusted for sex, age, BMI, smoking, education, level of cardiorespiratory fitness and leisure time physical activity

	All				Normotensives				Hypertensives			
	n	β	99% CI	*p*	n	β	99% CI	*p*	n	β	99% CI	*p*
LVEF (%)	2292	− 0.03	− 0.22 to 0.15	0.66	1405	− 0.05	− 0.29 to 0.18	0.57	887	0.12	− 0.30 to 0.31	0.98
LVMi(g/m^2)	*2434*	*0.14*	*− 0.02 to 0.30*	*0.03*	**1487**	**0.20**	**0.002–0.40**	**0.001**	947	0.05	− 0.22 to 0.32	0.61
GLS (%)[¤]	2369	− 0.01	− 0.18 to 0.17	0.90	1456	− 0.02	− 0.25 to 0.21	0.80	913	0.11	− 0.26 to 0.30	0.84
e (cm/s)[#]	2426	− 0.01	− 0.13 to 0.12	0.89	1478	0.06	− 0.22 to 0.09	0.29	948	0.06	− 0.13 to 0.26	0.40
LAVi (mL/m^2)	*2451*	*0.07*	*− 0.05 to 0.32*	*0.06*	1494	0.09	− 0.12 to 0.35	0.20	957	0.17	− 0.13 to 0.46	0.14
IVSd (cm)[*]	2434	0.07	− 0.08 to 0.22	0.23	*1487*	*0.07*	*− 0.03 to 0.34*	*0.02*	947	− 0.05	− 0.31 to 0.21	0.63
LVPWd (cm)	2434	0.04	− 0.13 to 0.20	0.59	1487	0.07	− 0.10 to 0.24	0.29	947	− 0.004	− 0.34 to 0.33	0.98
RWT (cm)	2442	− 0.02	− 0.15 to 0.11	0.78	1493	0.01	− 0.14 to 0.17	0.87	949	− 0.06	− 0.30 to 0.17	0.59
Tr_maxpg (mmHg)	1197	0.01	− 0.25 to 0.27	0.92	760	− 0.13	− 0.46 to 0.19	0.29	437	0.20	− 0.23 to 0.64	0.23
Deceleration time (ms)[#]	2393	0.07	− 0.14 to 0.23	0.50	1462	0.14	− 0.09 to 0.37	0.13	931	− 0.09	− 0.41 to 0.23	0.46

No exposure to heavy occupational lifting was reference. Significant associations ($p < 0.01$) are marked by bold, and italics indicate a trend to difference ($p < 0.10$)

The categories of walking, some handling of material AND heavy manual work, but no heavy lifting are collapsed. The models stratified on hypertensive status are adjusted for sex, age, BMI, smoking and education. The model among all included participants is additionally adjusted for hypertensive status

LVEF Left ventricular ejection fraction, *LVMi* Left ventricular mass index, *GLS* Global longitudinal strain, *e* peak early diastolic mitral annular velocity, *LAVi* Left atrial volume index, *IVSd* end-diastolic interventricular septal thickness, *LVPWd* end-diastolic left ventricular posterior wall thickness, *RWT* Relative wall thickness, *Tr_maxpg* Peak Tricuspid regurgitation gradient

Table 4 Odd ratios of prevalence of abnormal levels of indicators of heart morphology by self-rated exposure to heavy occupational lifting, adjusted for sex, age, BMI, smoking, and education. No exposure to heavy occupational lifting was reference

	All				Normotensives				Hypertensives			
	n	OR	99% CI	*P*	n	OR	99% CI	*p*	n	OR	99% CI	*p*
LVEF (%)	2300	0.99	0.78–1.24	0.88	1410	0.98	0.71–1.35	0.89	890	0.98	0.69–1.39	0.90
LVMi (g/m^2)	2443	1.17	0.91–1.51	0.10	1493	1.15	0.81–1.65	0.31	950	1.17	0.81–1.68	0.27
GLS (%)[¤]	2378	0.98	0.80–1.21	0.84	1462	1.03	0.78–1.35	0.79	916	0.92	0.67–1.26	0.50
e (cm/s)[#]	2436	0.98	0.74–1.29	0.85	*1485*	*1.45*	*0.95–2.21*	*0.02*	*951*	*0.75*	*0.51–1.08*	*0.04*
LAVi (mL/m^2)	2461	0.95	0.74–1.23	0.61	1501	1.03	0.75–1.43	0.79	960	0.81	0.53–1.24	0.21
IVSd (cm)[*]	*2361*	*1.23*	*0.98–1.53*	*0.02*	**1432**	**1.42**	**1.07–1.89**	**0.002**	929	0.96	0.67–1.38	0.78
LVPWd (cm)	2369	0.99	0.80–1.23	0.90	1449	1.10	0.82–1.48	0.40	920	0.89	0.65–1.23	0.36
RWT (cm)	2425	1.04	0.88–1.23	0.67	1481	1.05	0.83–1.34	0.67	944	1.02	0.80–1.30	0.88
Deceleration time (ms)*	*2303*	*1.30*	*0.98–1.74*	*0.02*	*1469*	*1.41*	*0.95–2.08*	*0.02*	815	1.18	0.77–1.80	0.33
Left ventricular hypertrophy[1]	*2443*	*1.30*	*1.04–1.61*	*0.02*	1493	1.15	0.82–1.61	0.43	*950*	*1.41*	*1.05–1.90*	*0.02*

Significant associations ($p < 0.01$) are marked by bold, and italics indicate a trend to difference ($p < 0.10$)

The categories of walking, some handling of material AND heavy manual work, but no heavy lifting are collapsed. The models stratified on hypertensive status are adjusted for sex, age, BMI, smoking and education. The model among all included participants is additionally adjusted for hypertensive status

LVEF Left ventricular ejection fraction, *LVMi* Left ventricular mass index, *GLS* Global longitudinal strain, *e* peak early diastolic mitral annular velocity, *LAVi* Left atrial volume index, *IVSd* end-diastolic interventricular septal thickness, *LVPWd* end-diastolic left ventricular posterior wall thickness, *RWT* Relative wall thickness

[1]Hypertrophy defined for males as LVMi > 115 g/m^2 and > 95 g/m^2 for females

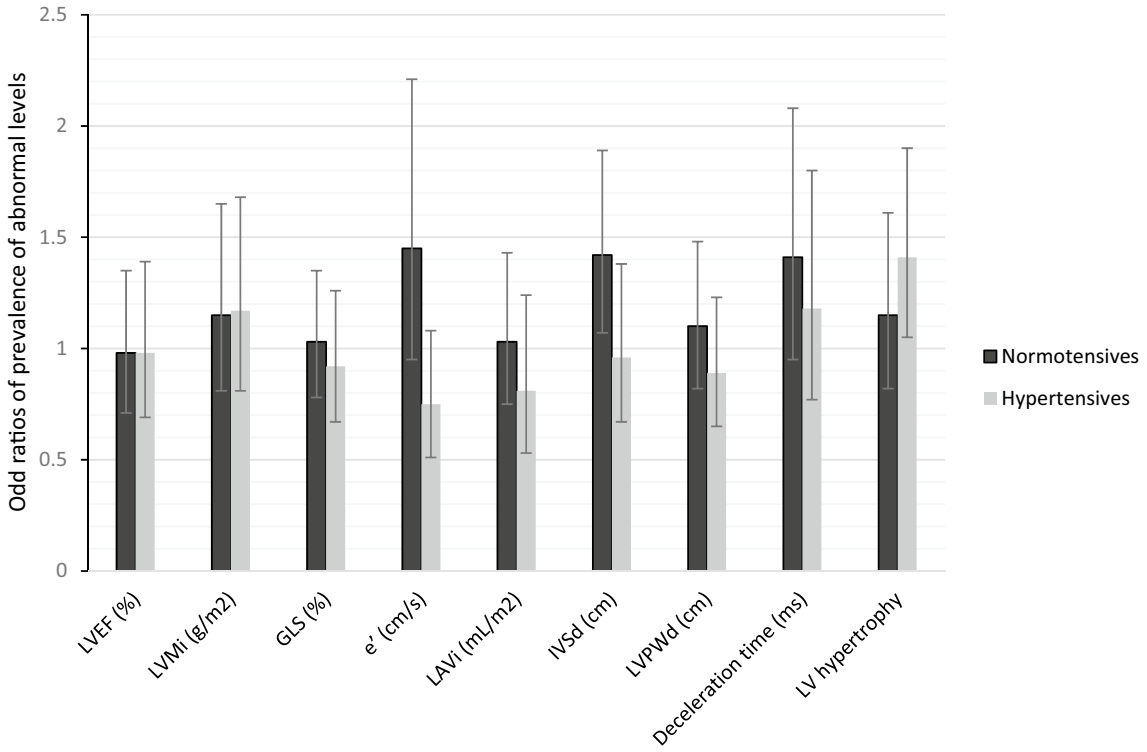

Fig. 2 Odd ratios of prevalence of abnormal levels of indicators of heart morphology by self-rated exposure to heavy occupational lifting, adjusted for sex, age, BMI, smoking, and education. Error bars indicate the 99% confidence intervals of the presented odd ratios

to high OPA. Among hypertensives are the risk of LV hypertrophy increased by exposure to heavy OL (OR 1.41, 99% CI 1.05–1.90) (Table 4). Also, are the prevalalence of normal LV geometry lower among those exposed to heavy OL, irrespective of them being normotensive or hypertensive (Table 2). A combination of high OPA and OL could indicate insufficient recovery, as the worker performing high volumes of OPA (5 days/week, 7–8 h/day) might not recover properly between bouts of OPA, which could also be expected among athletes performing long term high-intensity endurance training. Also the exposure to high volumes of OPA, including heavy OL, associates to increased arterial stiffness [32], 24 h blood pressure [5, 33] and progression of artherosclerosis [30]. All of these factors, jointly with the harmful changes in heart morphology, leading to increases in the risk for cardiovascular disease [8, 34].

Overall, the presented results show that cardiac structure and function among normotensives exposed to OL mimics the indicies of cardiac structure and function seen among hypertensives. Because lifting tasks require static muscle contraction combined with external loads, it may be assumed that the acute occlusion of the arteries, during static muscle contractions, and related effects on the total peripheral resistancet, gives rise to morphological changes of the heart, like those seen among hypertensives, where the heart also pumps against a raised total peripheral resistance due to lack of elasticity of the arteries [35].

No associations were observed among hypertensives from exposure to OL, except from an increased risk for LV hypertrophy (Table 4). These results might be explained by the healthy worker selection bias [36]. The healthy worker selection bias describes the migration of workers not being sufficiently capable or fit to perform the work tasks, out of occupational groups with high OPA into more sedentary occupational groups [36]. Thus, as OL mostly are performed in occupational groups also exposed to high levels of OPA, and both OL and high levels of OPA, give rise to increased SBP, which gives rise to angina [37], it may be assumed that workers suffering from angina or such would migrate to less strenuous occupational groups. Thus, this migration might also explain the low prevalence of LV hypertrophy seen among the hypertensives (Table 1) [38]. Also, it might be assumed that the impact from exposure to heavy OL on the cardiovascular system only impact the heart morphology a little among this group already having an increased afterload and presence of hypertrophy.

Perspectives/practical/clinical implications

The impact of OL on cardiovascular health is rarely investigated and thus, understanding the physiological mechanisms leading to higher cardiovascular mortality among blue-collar workers is lacking. Surveys report 33% of the European workforce [39] and 31% of Danish workers [40] to be exposed to OL, thus knowledge of the impact on cardiovascular health from OL is essential for developing preventive initiatives. In spite of the statistiscal significant differences among normotensives with and without exposure to heavy OL (Table 2) this may not reflect relevant clinical differences. Additionally, could these statistical differences reflect subtle myocardial changes in remodeling which in combination with other risk factors may lead to clinically important risk on the long term. Thus, these statistical significant differences may be clinically relevant as an early marker of myocardial remodeling, helping to guide clinicians to intervene before the participant develops a clinical cardiovascular disease.

Thus, in the cases where it is possible to reduce or even remove OL from the occupational exposures by the use of technical solutions or altered work tasks, the potentially harmful effects from OL on heart morphology could be preventable. Therefore, the reduction of OL should be incorporated in the primary and secondary prevention of cardiovascular disease among blue-collar workers.

Strenghts and lilmitations

The randomly selected study population is a strength. Because the laws and regulations regarding OL include the entire Danish nation, these results may be generalized, to some extent, to the entire Danish workforce. Yet, only 9.1% of the participants stated to be exposed to heavy OL, which is low compared to the Danish Work and Health Survey, where 31% of the respondents state to be exposed to OL for $\geq$ 25% of work time [40]. Future investigations should consider a study design independent from participation, e.g. a job-exposure matrix (JEM), being a tool for assessing level of occupational exposures linked to specific job titles. The use of a JEM for determining the exposure could also hold the possibility of investigating the effect of cumulative exposure, as a JEM may hold information on both exposure profile according to job title, and the duration of exposure [41]. This cross-sectional association cannot draw any causal conclusions. Also, the following limitations should be taken into account when interpreting the results; i) the self-reported exposure to heavy OL might be affected by recall bias [42], also the self-reported OL lacks to quantify the lifted burden; thus, future studies should consider to use technical monitors could diminish the bias from lack of recall; ii) the study sample is primarily Caucasian which limits the generalizability of the results; iii) the BP measurement was performed during the one-day data collection at the clinic and therefore could masked hypertension and white-coat hypertension bias the measured BP. Conclusively, these findings need to be verified by future studies investigating relations between the accumulation of exposure from OL and indicators of heart morphology.

Conclusion

This cross-sectional study found that OL was associated with indices of abnormal cardiac structure and function among normotensives. Moreover, exposure to OL among normotensives was associated with changes in cardiac structure and function indicating an increased risk for cardiovascular disease. Thus, a reduction of OL would be assumed to reduce risk for cardiovascular disease and mortality among normotensive workers. Future studies should consider to investigate exposure to occupational lifting as a physiologic explanation for the physical activity paradox in health.

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Authors contributions MK designed the study, applied for and recieved funding. MK performed the statistical analyses and drafted the manuscript. KGS, MCHL and NDJ performed the data processing from raw material to data for statistical analyses. JLM merged the data and contributed to the statistical analyses. PS commented on the design for the study and the manuscript, as well as lead the initial data collection. TBS contributed to the drafting of the manuscript and supervised the interpretation of the statistical analyses.

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Availability of data and material Requests for data should be applied to the steering group for the Copenhagen City Heart Study, which can be reached by contact to either Peter Schnohr or Jacob Louis Marott.

Code availability Availability of coding for the statistical analysis can be delivered by request to Mette Korshøj.

Declarations

Conflict of interest Nothing to disclose.

Ethical approval The Danish Data Protection Agency approved the Copenhagen City Heart Study (Approval No.: 2001-54-0280; 2007-

58-0015, 2012-58-0004, HEH-2015-045, Isuite 03741), as well as the National Committee on Health Research Ethics approved the data collection (Approval No.: VEK: H-KF 01–144/01 31104).

Consent to participate All participation was voluntary and conducted in agreement with the Declaration of Helsinki, and written consent from all participants were colledted prior to data collection.

Consent for publication COI for all authors are attached.

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