Leisure time and occupational physical activity, resting heart rate, and mortality in the Arctic region of Norway. The Finnmark Study

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Abstract

Aims

This study examined the association of leisure time physical activity (LTPA), occupational physical activity (OPA), and resting heart rate (RHR) with all-cause and cardiovascular (CVD) mortality in Sami and non-Sami populations.

Study design

Longitudinal, observational population-based study.

Methods

The Finnmark 3 study cohort was examined in 1987-1988 and followed for all-cause and CVD mortality for 26 years. The cohort included 17697 men and women with a mean age of 47.2 years at baseline. LTPA and OPA were assessed with a validated questionnaire at baseline, whereas cause of death was obtained from the Norwegian Cause of Death Registry.

Results

A total of 1983 women and 3147 men died during follow-up. LTPA was linearly and inversely associated with all-cause mortality, but not CHD mortality. Compared to inactive subjects, all-cause mortality was significantly reduced by 16% in the active LTPA group (HR 0.84; 95% CI 0.76-0.92). Both for all-cause and CVD mortality, we observed a U-formed relationship with OPA, as participants in the walking and lifting group had significantly lower mortality than both the mostly sedentary and the heavy manual labour group ($p<0.05$). An increase in RHR by one beat per minute was associated with a 1.1 % increase in all-cause mortality (HR 1.011; 95% CI 1.009-1.013). The associations were similar in Sami and non-Sami subjects.

Conclusion

In this population-based study, LTPA was inversely associated with all-cause mortality, whereas RHR was positively associated with all-cause and CVD mortality. There was a U-shaped association between OPA and CVD and all-cause mortality.

Keywords

Physical activity; heart rate, ethnicity; indigenous; longitudinal; mortality; Sami

Introduction

The importance of regular physical activity to reduce the risk of cardiovascular disease (CVD) and death is well established.[1-3] Both physical activity[1, 4] and exercise capacity[5, 6] are inversely related to mortality. However, a majority of previous studies have included only leisure time physical activity (LTPA),[1, 4] and the association between occupational physical activity (OPA) and mortality is less studied. Some studies suggest that high levels of OPA are associated with increased mortality, particularly among men,[7-9] whereas other studies show benefits of high levels of OPA.[10, 11] Moreover, high levels of OPA in combination with low cardiorespiratory fitness seem to be associated with increased CVD mortality.[12]

When direct measures of exercise capacity are lacking, resting heart rate (RHR) which is an easily accessible clinical variable, may act as a proxy for physical fitness and physical activity in large cohorts.[13, 14] Findings indicate that high RHR is independently associated with increased all-cause and cardiovascular mortality,[15] and development of atherosclerosis.[16] However, the independent role of RHR for mortality is disputed.[15]

We address these issues in a large cohort of men and women in Finnmark County in northern Norway, which is characterized by a large minority of indigenous Sami people. Historically, Finnmark county has had the highest mortality rates and incidence of coronary heart disease in Norway. The life expectancy for the population is still somewhat lower than the average in Norway, but the gap is slowly diminishing.[17] We have previously shown that the Sami population had higher work physical activity levels and lower RHR than their Norwegian peers,[18] which could suggest possible ethnic interactions in the association between physical activity and mortality.

The main aim of this study was to examine the associations of LTPA, OPA and RHR with all-cause and CVD mortality. Additionally, we wanted to examine whether these associations differed between Sami and non-Sami populations.

Methods

The Finnmark Study

The Finnmark Study is a longitudinal, population-based study in northern Norway conducted in 1974-75 (Finnmark 1), 1977-78. (Finnmark 2), and 1987-88 (Finnmark 3).[19, 20] The present analyses are based on data from the third Finnmark Study in 1987-88, to which all residents in Finnmark County aged 40-62 years (born 1925-47) were invited, together with a subsample among inhabitants aged 20-39 (born 1948-67) (those invited to Finnmark 2 still living in Finnmark and 10 % of those who were not invited to Finnmark 2). In total, 22 941 inhabitants were invited, and 17 821 men and women (77.7%) attended Finnmark 3. Invitations were sent by letter and the participants were asked to answer three questionnaires, which were presented in Norwegian and Sami languages. Participation rate increased with age and women had higher participation rates than men. Moreover, participation varied by community.[20]

Selected sample for the present analyses

The present sample includes 17697 men and women, aged 20-62 at examination, with valid data on physical activity and covariates. Of these, 13590 participants reported ethnic affiliation, among which 2813 (20.7%) were categorized as Sami and 10777 (79.3%) as non-Sami, constituting a subsample.

Exposure assessment

LTPA levels at baseline were assessed by the "Saltin-Grimby Physical Activity Level Scale"[21] with four mutually exclusive options. Due to a low number of participants

(n=224) answering the highest physical activity option, groups 3 and 4 were merged, leaving 3 groups for the analyses: *Inactive*, *Moderate* (walking, bicycling, etc. ≥4 hours a week), and *Active* (recreational sports etc. ≥4 hours a week or hard training or competitions several times a week).

OPA levels at baseline were also assessed by the "Saltin-Grimby Physical Activity Level Scale"[21] with four mutually exclusive options: *Mostly sedentary*, *Walking* (e.g. shop assistant, light industrial work, education), *Walking and lifting* (e.g. mailman, heavy industrial work, construction work), and *Heavy manual labour* (e.g. forestry work, heavy agriculture work, heavy construction work).

LTPA measured by the Saltin-Grimby Physical Activity Level Scale[21] shows satisfactory rank validity when validated against VO_{2max} and accelerometry.[13] In contrast to LTPA, no association between self-reported OPA and VO_{2max} was observed.[13]

RHR was measured during blood pressure monitoring (Dinamap, Criticon), sitting down after 4 minutes rest. Three measurements were taken with an interval of one minute, and the lowest of the three heart rate measurements was used in the analyses.

Covariates

Age was obtained from the National Population Registry. Data on daily smoking, previous cardiovascular diseases (myocardial infarction, angina pectoris, stroke), diabetes mellitus, and treatment for hypertension was self-reported (yes/no). Non-fasting blood samples were collected and analysed for serum total cholesterol and triglycerides using an enzymatic method (Hitachi auto analyser, Roche Diagnostic, Switzerland). Height and weight of all subjects were measured by the screening nurse and recorded. Systolic (SBP) and diastolic blood pressure (DBP) were measured

automatically by the Dinamap (Criticon) blood pressure monitor.[22] Three measurements were taken with an interval of one minute, and the mean value of the second and third measurements of blood pressure was used in the present analyses.

Ethnicity was dichotomized from four original categories into Sami (original categories Sami or Finnish/Sami) and non-Sami (original categories Norwegian or Finnish).[20] Classification was based on the two questions "Are two or more of your grandparents of Sami origin?" and "Are two or more of your grandparents of Finnish origin?" as shown in Supplemental table S1. Those who responded "unknown" were classified as missing.

Cause of death

Date and cause of death in the analytical cohort from date of attendance in 1987-1988 through 31.12.2013 were registered. CVD death was defined as ICD-9: 390-459 codes and ICD-10: I00-I99, using the underlying cause of death.

Statistical analysis

The associations of LTPA, OPA and RHR with CVD and all-cause mortality were estimated by Cox proportional hazard models with days-to-event as time axis, with hazard ratio (HR) and 95% confidence intervals (CI) as effect size. Proportional hazard assumptions were assessed by inspecting the log (-log) survival curves for the various physical activity categories. Model 1 tested the association between either LTPA, OPA, or RHR as exposure and all-cause or CVD mortality as outcome, adjusted for age, sex, smoking status, and BMI categories. Associations between LTPA and mortality were additionally adjusted for OPA, and vice versa. The model of RHR and mortality did not include OPA or LTPA. In model 2, we additionally adjusted for self-reported angina pectoris, myocardial infarction, cerebral insult, diabetes and anti-hypertensive medication, which represent possible mediators in the association between physical

activity and mortality. The analyses were repeated in a subsample (n=13590) with valid data on ethnicity, stratified by Sami or non-Sami.

By adding multiplicative interaction terms to the main multivariable models in Cox proportional hazard model, we assessed possible interactions between sex and LTPA, sex and OPA, ethnicity and LTPA, ethnicity and OPA, sex and RHR, ethnicity and RHR, and finally between LTPA and OPA, with OPA treated as a quadratic term. *P* values were two-sided with a significance level of 0.05. Data analyses were performed using IBM SPSS Statistics, version 24 (IBM Corporation, Armonk, NY, USA).

Ethical permission

The Norwegian Data Inspectorate approved the Finnmark Study. The present study was approved by the Regional Committee for Medical Research Ethics in Northern Norway (REK nr. 2013/2249).

Results

In total, 17697 participants with a mean age of 47.2 years at baseline were included in the analyses. During a mean of 23.3 years of follow-up, 5130 participants (1983 women and 3147 men) died, of which 1764 were due to CVD. The crude mortality rate was 12.2 per 1000 person-years.

Table 1 shows the characteristics of the study population at baseline (Finnmark 3, 1987-1988) by LTPA and OPA. Compared with the non-Sami, a higher percentage of the Sami were categorized as inactive in leisure time (p=0.01), whereas the Sami were more active at work (p<0.001). Values of BMI, DBP, treatment for hypertension and RHR decreased significantly with increasing levels of LTPA.

LTPA and mortality

We found an inverse linear association between LTPA and all-cause mortality after adjustments ($p<0.001$) (Table 2, Model 1). Moderate LTPA was associated with a non-significant reduction in all-cause mortality, compared with the inactive group (HR 0.95; 95% CI 0.89-1.01; Model 1), and participants in the active LTPA group had 16% reduced risk (HR 0.84; 95% CI 0.76-0.92; Model 1). These inverse associations were slightly attenuated with further adjustment in model 2, and found both in Sami and non-Sami subjects (Table 2). A non-significant inverse, linear association between LTPA and CVD mortality was observed in both model 1 and 2 (Table 2). There were no interactions between sex or ethnicity and LTPA in any of the models. However, an interaction between LTPA and OPA was observed ($p<0.001$), as mortality decreased with increasing LTPA in the two lowest OPA categories; however, in the two highest levels of OPA, LTPA did not seem to influence mortality (Supplemental table S2).

OPA and mortality

The association of OPA with all-cause and CVD mortality was U-shaped (Table 3). Compared with the subjects with walking and lifting OPA, we found a 16% higher all-cause mortality in the mostly sedentary OPA group (HR 1.16; 95% CI 1.07-1.26; Model 1) and a 13% higher mortality in the heavy manual labour group (HR 1.13; 95% CI 1.01-1.26; Model 1) (Table 3). These associations were almost unchanged after further adjustments for CVD, diabetes and anti-hypertensive medication (Model 2, Table 3). The strength of the relationships was similar in Sami and non-Sami subjects.

There were no interactions with sex and ethnicity in any of the models. However, an interaction between OPA and LTPA was observed ($p<0.001$). When stratifying by LTPA levels, the overall U-shaped association between OPA and mortality was found in the

inactive and moderate LTPA groups. However, in the highest LTPA group (Active), we observed a different pattern, with a linearly increasing mortality with increasing OPA level (Supplemental table S2).

RHR and mortality

All-cause mortality increased by 1.1% for each beat per minute increase in RHR (HR 1.011; 95% CI 1.009-1.013; Model 1). Similar results were observed for CVD mortality (HR 1.007; 95% CI 1.004-1.011; Model 1), and after further adjustments for CVD and diabetes. These trends were seen in both ethnic groups. There were no interactions between sex or ethnicity and RHR in any of the models.

Discussion

In this prospective study over 26 years, LTPA reduced all-cause mortality in a linear dose-response relationship, whereas the association between LTPA and CVD mortality was not statistically significant. We found a U-shaped association between OPA and all-cause and CVD mortality, with moderate OPA levels associated with the lowest mortality. Furthermore, we found a linear relationship between RHR and all-cause and CVD mortality. The association of LTPA, OPA, and RHR with mortality did not differ between the Sami and non-Sami populations.

LTPA and mortality

Numerous studies have demonstrated an inverse association between LTPA level and all-cause mortality,[1, 4, 23] showing a 10-40% risk reduction with moderate physical activity. In the present study, participants with the highest LTPA level benefited most, as hard training at least 4 hours a week showed a 16% reduction in all-cause mortality compared to the non-significant 5% risk reduction in the moderate LTPA group. Our finding that LTPA is associated with reduced mortality in a dose-response manner is

also in accordance with results from pooled data from six studies in the NCI Cohort Consortium[1] and a meta-analyses of 22 cohort studies.[24] One possible explanation for this finding could be better general health and socio-economic status in the groups with highest LTPA levels. Unfortunately, data concerning socio-economic status were not available. On the other hand, numerous studies show strong evidence for an inverse and independent association between LTPA and mortality, supporting our findings.

Interestingly, the decreasing mortality with increasing LTPA was only found in the two lowest OPA categories; in higher levels of OPA, LTPA did not seem to influence mortality (Supplemental table S2).

The weaker association between LTPA and CVD mortality compared to all-cause mortality is in contrast to other studies.[1, 3] One possible explanation could be low statistical power. Moreover, high prevalences of CVD in the Arctic region[25] may have influenced the effect of physical activity on CVD mortality in this population.[26]

OPA and mortality

We found a 13-16% lower all-cause mortality in the walking and lifting group compared with the sedentary OPA group and the heavy manual labour group, suggesting a U-shaped association between OPA and mortality. However, this U-shape was found only in the inactive and moderate LTPA groups, whereas participants in the active LTPA groups showed a linearly increasing mortality with increasing OPA level.

The Danish National Work Environment Cohort study[7] observed an increasing all-cause mortality with increasing OPA in men, whereas female workers showed a U-shaped association. The association between OPA and mortality is inconsistent in cohort studies, although many indicate an increased mortality among those with high OPA. This

has led researchers to propose a "physical activity paradox",[27] suggesting that OPA and LTPA have opposite effects on CVD health and mortality. Possible reasons for this include the characteristics of OPA, such as low intensity, long duration, static postures, and heavy lifting. Furthermore, OPA may elevate 24-hour heart rate and blood pressure.[27]

RHR and mortality

Our finding that high RHR is related to a significant increase in all-cause and CVD mortality is in accordance with findings from several meta-analyses and large cohort studies showing that elevated RHR is independently associated with increased of all-cause and cardiovascular mortality,[15, 28, 29] even when controlling for familial factors.[30] A study showing that high RHR trajectories were associated with the highest risk of death, although only in men, further highlight these findings.[31] A plausible explanation for the increased mortality risk with higher RHR is dysfunctional autonomic nervous activity[15] and detrimental effects on progression of atherosclerosis caused by higher RHR.[16]

Physical activity and mortality in Sami and non-Sami populations

The associations between physical activity and mortality did not differ with ethnicity in our study. In the 1980's, the percentage of employees in primary industries (reindeer industry, agriculture and fishing) was approximately 20 % among the Samis compared to about 8% amongst the Arctic population at large.[32] However, the numbers of employees in the reindeer industry decreased with 16% from 1990 to 2008,[33] indicating that the industries of the Sami and non-Sami populations became more similar during follow-up. This may have levelled out the differences in physical activity and RHR found between the Sami and non-Sami in 1986-87.[18] In addition, the Sami and non-Sami populations seem to be rather homogeneous regarding lifestyle and CVD risk factors.[34,

[35] To our knowledge, no previous studies have examined the association between physical activity and mortality among other indigenous people.

Limitations and strengths

Self-reported physical activity may be subject to misclassification errors. We did not have updated information on the exposure variables, which could lead to misclassification due to changes in exposure during follow-up. Moreover, we do not have information about socio-economic status (SES). Physical activity levels increase with education level,[36] and higher social class is often associated with longevity,[37] thus introducing SES as potential confounder that could explain some of the association between physical activity and mortality. Moreover, the observed associations could be influenced by unmeasured confounders such as diet and genotype.

Strengths of this cohort study are the prospective design with 26 years follow-up time in a population with a large Sami minority, a large number of mortality cases, and minimal loss to follow-up. The study involves rigorous outcome ascertainment, with a high degree of completeness of the Norwegian Cause of Death Registry, covering about 98% of all deaths in Norway.[38] The validity of CVD mortality in Norway shows substantial agreement with autopsy findings.[39]

Conclusion

In this population-based study, LTPA was inversely associated with all-cause mortality, whereas a U-shaped association was observed between OPA and CV and all-cause mortality. Elevated RHR was associated with higher all-cause and CVD mortality. The findings applied to both the Sami and non-Sami populations.

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Conflict of interest

None.

Authors' contributions

RH, BM and BKJ contributed to the design, statistical analysis and drafting of the manuscript. RH, BM, BKJ and MLL revised the manuscript critically. All the authors approved the manuscript for publication and are accountable for all aspects of the work.

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Table 1 Participant characteristics at baseline by leisure time and occupational physical activity. The Finnmark Study

	Overall	**Leisure time physical activity (n=17697)**				**Occupational physical activity (n=17697)**				
		Inactive	**Moderate**	**Active**	**P for linear trend****	**Mostly sedentary**	**Walking**	**Walking and lifting**	**Heavy manual labour**	**P for linear trend****
Overall (%)	17697 (100)	4684 (26.5)	10431 (58.9)	2582 (14.6)		5767 (32.6)	6729 (38.0)	3679 (20.8)	1522 (8.6)	
Sex					<0.001					<0.001
Men (%)	8951 (50.6)	2316 (25.9)	4764 (53.2)	1871 (20.9)		3221 (36.0)	2387 (26.7)	2021 (22.6)	1322 (14.8)	
Women (%)	8746 (49.4)	2368 (27.1)	5667 (64.8)	711 (8.1)		2546 (29.1)	4342 (49.6)	1658 (19.0)	200 (2.3)	
Age (years)	47.2 (9,2)	46.5 (9.2)	47.9 (9.2)	45.3 (9.4)	0.014	47.2 (9.1)	47.9 (9.2)	45.8 (9.4)	47.2 (8.9)	<0.001
Ethnicity*					0.012					<0.001
Non-Sami (%)	10777 (79.3)	2682 (24.9)	6523 (60.5)	1572 (14.6)		3778 (35.1)	4111 (38.1)	2138 (19.8)	750 (7.0)	
Sami	2813 (20.7)	832 (29.6)	1533 (54.5)	448 (15.9)		790 (28.1)	1033 (36.7)	638 (22.7)	352 (12.5)	
Body height (cm)	167.3 (9.3)	166.8 (9.4)	166.8 (9.2)	170.6 (9.1)	<0.001	168.8 (9.4)	165.3 (9.2)	167.5 (9.1)	170.5 (8.3)	0.287
Body weight (kg)	72.5 (13.2)	73.2 (14.3)	71.8 (13.0)	73.8 (11.5)	0.700	73.7 (13.6)	70.6 (13.0)	72.6 (12.7)	75.9 (12.0)	0.010
BMI (kg/m^2)	25.8 (4.0)	26.3 (4.5)	25.8 (3.9)	25.3 (3.1)	<0.001	25.8 (4.0)	25.8 (4.2)	25.8 (3.9)	26.1 (3.4)	0.015
Systolic blood pressure (mmHg)	135.4 (18.5)	135.2 (18.5)	135.7 (18.8)	134.5 (17.0)	0.979	134.7 (18.4)	135.5 (19.1)	135.0 (17.7)	138.4 (18.1)	<0.001
Diastolic blood pressure (mmHg)	81.4 (11.3)	81.9 (11.3)	81.6 (11.3)	79.9 (10.8)	<0.001	81.8 (11.2)	81.1 (11.3)	80.9 (11.0)	82.3 (11.5)	0.587
RHR (beats/min)	72.7 (13.1)	74.7 (13.0)	73.0 (12.9)	67.7 (12.5)	<0.001	72.5 (13.3)	73.2 (13.1)	72.6 (12.7)	71.2 (12.5)	0.040
Cholesterol (mmol/l)	6.59 (1.36)	6.59 (1.37)	6.63 (1.36)	6.42 (1.36)	0.002	6.53 (1.35)	6.61 (1.37)	6.55 (1.38)	6.79 (1.34)	<0.001

Triglycerides (mmol/l) ***	1.86 (1.30)	1.97 (1.47)	1.83 (1.24)	1.80 (1.21)	<0.001	1.89 (1.20)	1.79 (1.33)	1.88 (1.37)	2.00 (1.36)	0.054
Smokers (%)	8721 (49.3)	2698 (57.6)	4963 (47.6)	1060 (41.1)	<0.001	2818 (48.9)	3071 (45.6)	1968 (53.5)	864 (56.8)	<0.001
Hypertension treatment (%)	1393 (7.9)	422 (9.0)	852 (8.2)	119 (4.6)	<0.001	493 (8.5)	570 (8.5)	233 (6.3)	97 (6.4)	0.001
Myocardial infarction (%)	327 (1.8)	90 (1.9)	210 (2.0)	27 (1.0)	0.042	145 (2.5)	118 (1.8)	42 (1.1)	22 (1.4)	<0.001
Angina pectoris (%)	710 (4.0)	192 (4.1)	466 (4.5)	52 (2.0)	0.001	271 (4.7)	296 (4.4)	96 (2.6)	47 (3.1)	<0.001
Cerebral insult (%)	125 (0.7)	37 (0.8)	74 (0.7)	14 (0.5)	0.277	57 (1.0)	53 (0.8)	9 (0.2)	6 (0.4)	<0.001
Diabetes mellitus (%)	234 (1.3)	78 (1.7)	136 (1.3)	20 (0.8)	0.002	91 (1.6)	96 (1.4)	33 (0.9)	14 (0.9)	0.008

Data are presented as mean (SD) or number (%).

BMI: body mass index; RHR: resting heart rate

*n=13590

** Age-adjusted

*** Triglycerides were log transformed when computing the p-value

Table 2 All-cause and cardiovascular mortality by self-reported leisure time physical activity. The Finnmark Study

			All-cause mortality			
			Model 1		**Model 2**	
	n	**N deaths**	**HR (95% CI)**	**P for linear trend**	**HR (95% CI)**	**P for linear trend**
Overall	17697	5130		< 0.001		0.009
Inactive	4684	1392	1.0		1.0	
Moderate	10431	3126	0.95 (0.89, 1.01)		0.96 (0.90, 1.02)	
Active	2582	612	0.84 (0.76, 0.92)		0.87 (0.79, 0.96)	
Non-Sami*	10777	3035		0.001		0.010
Inactive	2682	789	1.0		1.0	
Moderate	6523	1907	0.94 (0.86, 1.02)		0.94 (0.87, 1.03)	
Active	1572	339	0.80 (0.71, 0.91)		0.84 (0.74, 0.96)	
Sami*	2813	815		0.188		0.252
Inactive	832	244	1.0		1.0	
Moderate	1533	449	0.92 (0.79, 1.08)		0.94 (0.80, 1.10)	
Active	448	122	0.87 (0.69, 1.09)		0.88 (0.70, 1.10)	

			Cardiovascular mortality			
			Model 1		Model 2	
	n	N deaths	HR (95% CI)	P for linear trend	HR (95% CI)	P for linear trend
Overall	17697	1764		0.240		0.816
Inactive	4684	471	1.0		1.0	
Moderate	10431	1070	0.96 (0.86, 1.08)		0.99 (0.88, 1.10)	
Active	2582	223	0.91 (0.77, 1.07)		0.98 (0.84, 1.16)	
Non-Sami*	10777	999		0.141		0.381
Inactive	2682	260	1.0		1.0	
Moderate	6523	623	0.95 (0.82, 1.10)		0.95 (0.82, 1.11)	
Active	1572	116	0.84 (0.67, 1.05)		0.91 (0.73, 1.14)	
Sami*	2813	311		0.759		0.640
Inactive	832	88	1.0		1.0	
Moderate	1533	168	0.96 (0.74, 1.26)		0.98 (0.75, 1.28)	
Active	448	55	1.08 (0.76, 1.53)		1.11 (0.78, 1.58)	

Model 1: Adjusted for age, sex, smoking status, BMI and occupational physical activity.

Model 2: Adjusted for age, sex, smoking status, BMI, self-reported angina pectoris, myocardial infarction, cerebral insult, diabetes, anti-hypertensive medication and occupational physical activity.

* n=13590

Table 3 All-cause and cardiovascular mortality by self-reported occupational physical activity. The Finnmark Study

			All-cause mortality			
			Model 1		**Model 2**	
	n	**N deaths**	**HR (95% CI)**	**P for non-linear trend**	**HR (95% CI)**	**P for non-linear trend**
Overall	17697	5130		0.003		0.020
Mostly sedentary	5767	1758	1.16 (1.07, 1.26)		1.13 (1.04, 1.22)	
Walking	6729	1915	1.11 (1.03, 1.20)		1.08 (1.00, 1.17)	
Walking and lifting	3679	924	1.0		1.0	
Heavy manual labour	1522	533	1.13 (1.01, 1.26)		1.14 (1.02, 1.27)	
Non-Sami	10777	3035		0.052		0.073
Mostly sedentary	3778	1125	1.15 (1.04, 1.28)		1.14 (1.03, 1.26)	
Walking	4111	1130	1.09 (0.98, 1.21)		1.08 (0.97, 1.20)	
Walking and lifting	2138	530	1.0		1.0	
Heavy manual labour	750	250	1.14 (0.98, 1.32)		1.16 (1.00, 1.35)	
Sami	2813	815		0.167		0.371
Mostly sedentary	790	242	1.19 (0.97, 1.45)		1.14 (0.93, 1.39)	
Walking	1033	282	1.23 (1.01, 1.50)		1.16 (0.95, 1.42)	
Walking and lifting	638	158	1.0		1.0	
Heavy manual labour	352	133	1.25 (0.99, 1.58)		1.21 (0.96, 1.53)	

			Cardiovascular mortality			
			Model 1		Model 2	
	n	N deaths	HR (95% CI)	P for non-linear trend	HR (95% CI)	P for non-linear trend
Overall	17697	1764		0.013		0.160
Mostly sedentary	5767	634	1.26 (1.10, 1.45)		1.17 (1.02, 1.34)	
Walking	6729	640	1.18 (1.02, 1.35)		1.09 (0.95, 1.25)	
Walking and lifting	3679	298	1.0		1.0	
Heavy manual labour	1522	192	1.14 (0.95, 1.36)		1.15 (0.96, 1.38)	
Non-Sami*	10777	999		0.064		0.216
Mostly sedentary	3778	395	1.27 (1.06, 1.52)		1.20 (1.00, 1.44)	
Walking	4111	356	1.13 (0.94, 1.36)		1.08 (0.90, 1.30)	
Walking and lifting	2138	166	1.0		1.0	
Heavy manual labour	750	82	1.10 (0.85, 1.44)		1.14 (0.87, 1.49)	
Sami*	2813	311		0.612		0.856
Mostly sedentary	790	88	1.06 (0.76, 1.46)		0.95 (0.68, 1.32)	
Walking	1033	106	1.22 (0.89, 1.69)		1.07 (0.78, 1.48)	
Walking and lifting	638	63	1.0		1.0	
Heavy manual labour	352	54	1.12 (0.78, 1.62)		1.06 (0.73, 1.54)	

Model 1: Adjusted for age, sex, smoking status, BMI and leisure time physical activity.

Model 2: Adjusted for age, sex, smoking status, BMI, self-reported angina pectoris, myocardial infarction, cerebral insult, diabetes, anti-hypertensive medication and leisure time physical activity.

* n=13590

Supplemental table S1: Classification of ethnicity. The Finnmark Study 1987-88

(n=13590)

	Are two or more of your grandparents of Finnish origin? Yes	Are two or more of your grandparents of Finnish origin? No
Are two or more of your grandparents of Sami origin? Yes	Sami (original category Finnish/Sami)	Sami (original category Sami)
Are two or more of your grandparents of Sami origin? No	Non-Sami (original category Finnish)	Non-Sami (original category Norwegian)

Sami: n=2813 (20.7%)
Non-Sami (Norwegian, Finnish): n=10777 (79.3%)

Supplemental table S2: *HR for mortality for combined LTPA and OPA categories, adjusted for age, sex, smoking, and BMI*

	OPA 1 (Sedentary)	**OPA 2 (Walking)**	**OPA 3 (Walking and lifting)**	**OPA 4 (Heavy manual labour)**
LTPA 1 (Inactive) HR (95.0% CI)	1.29 (1.08, 1.55)	1.15 (0.95, 1.40)	0.89 (0.72, 1.11)	1.12 (0.88, 1.43)
N (n deaths)	1897 (632)	1584 (441)	847 (194)	356 (125)
LTPA 2 (Moderate) HR (95.0% CI)	1.14 (0.95, 1.36)	1.10 (0.92, 1.31)	1.01 (0.84, 1.21)	1.06 (0.87, 1.30)
N (n deaths)	3114 (971)	4330 (1275)	2210 (589)	777 (291)
LTPA 3+4 (Active) HR (95.0% CI)	0.82 (0.66, 1.04)	0.95 (0.77, 1.18)	1.0 (Ref)	1.17 (0.91, 1.49)
N (n deaths)	756 (155)	815 (199)	622 (141)	389 (117)

*Reference category is Active LTPA (cat. 3+4) and Walking and lifting OPA (cat 3) which showed the lowest HR when testing main effects.