MR JUUSO VÄISTÖ (Orcid ID : 0000-0001-7026-5934)

DR EERO A HAAPALA (Orcid ID : 0000-0001-5096-851X)

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Longitudinal associations of physical activity and sedentary time with cardiometabolic risk factors in children

Väistö J[1,2], Haapala EA[1,3], Viitasalo A[1], Schnurr TM[9], Kilpeläinen TO[9], Karjalainen P[1], Westgate K[7], Lakka H-M[1], Laaksonen DE[5], Ekelund U[7,8], Brage S[7], Lakka TA[1,4,6]

[1]Institute of Biomedicine, Physiology, University of Eastern Finland, Kuopio, Finland

[2] Institute of Dentistry, School of Medicine, University of Eastern Finland, Kuopio, Finland

[3]Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland

[4]Department of Clinical Physiology and Nuclear Medicine, Kuopio University Hospital and University of Eastern Finland, Kuopio, Finland

[5]Institute of Clinical Medicine, Internal Medicine, Kuopio University Hospital, Kuopio, Finland

[6] Kuopio Research Institute of Exercise Medicine, Kuopio, Finland

[7]MRC Epidemiology Unit, Institute of Metabolic Science, University of Cambridge School of Clinical Medicine, Cambridge, UK.

[8] Department of Sport Medicine, Norwegian School of Sport Sciences, Oslo, Norway.

[9] Novo Nordisk Foundation Center for Basic Metabolic Research, Section of Metabolic Genetics, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark

Correspondence author:
Juuso Väistö
e-mail: juuso.vaisto@uef.fi
telephone: +358 40 5109122

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ABSTRACT

Background

There are few prospective studies on the associations of changes in objectively measured vigorous physical activity (VPA_{Δ}), moderate-to-vigorous physical activity ($MVPA_{\Delta}$), light physical activity (LPA_{Δ}), and sedentary time (ST_{Δ}) with changes in cardiometabolic risk factors ($_{\Delta}$) in children. We therefore investigated these relationships among children.

Methods

The participants were a population sample of 258 children aged 6–8 years followed for 2 years. We assessed PA and ST by a combined heart rate and movement sensor; computed continuous age- and sex-adjusted z-scores for waist circumference, blood pressure, and fasting insulin, glucose, triglycerides, and high-density lipoprotein (HDL) cholesterol; and constructed a cardiometabolic risk score (CRS) of these risk factors. Data were analysed using linear regression models adjusted for age, sex, the explanatory and outcome variables at baseline, and puberty.

Results

VPA_{Δ} associated inversely with CRS_{Δ} (β=-0.209, p=0.001), body fat percentage $(BF\%)_{\Delta}$ (β=-0.244, p=0.001), $insulin_{\Delta}$ (β=-0.220, p=0.001), and $triglycerides_{\Delta}$ (β=-0.164, p=0.012) and directly with HDL $cholesterol_{\Delta}$ (β=0.159, p=0.023). $MVPA_{\Delta}$ associated inversely with CRS_{Δ} (β=-0.178, p=0.012), $BF\%_{\Delta}$ (β=-0.298, p=<0.001), and $insulin_{\Delta}$ (β=-0.213, p=0.006) and directly with HDL $cholesterol_{\Delta}$ (β=0.184, p=0.022). LPA_{Δ} only associated negatively with CRS_{Δ} (β=-0.163, p=0.032). ST_{Δ} associated directly with CRS_{Δ} (β=0.218, p=0.003), $BF\%_{\Delta}$ (β=0.212, p=0.016), and $insulin_{\Delta}$ (β=0.159, p=0.049).

Conclusions

Increased VPA and MVPA and decreased ST were associated with reduced overall cardiometabolic risk and major individual risk factors. Change in LPA had weaker associations with changes in these cardiometabolic risk factors. Our findings suggest that increasing at least moderate-intensity PA and decreasing ST decrease cardiometabolic risk in children.

1 Introduction

Insufficient levels of physical activity (PA) and excess sedentary time (ST) have been associated with overweight, insulin resistance, glucose intolerance, dyslipidaemia, elevated blood pressure, and the clustering of these cardiometabolic risk factors in children [1–3]. This clustering of risk factors among otherwise healthy children may be a biological marker of poor cardiometabolic health [4]. Moreover, an unfavorable cardiometabolic risk profile during childhood and adolescence has been linked to an increased risk of metabolic syndrome, type 2 diabetes, and cardiovascular disease in adulthood [5–7].

Cross-sectional studies have linked higher levels of objectively measured PA [1,8,9], especially more vigorous PA [10–12], with lower cardiometabolic risk in children and adolescents. To the best of our knowledge, there are few prospective studies on the associations of objectively measured PA [13–15], particularly PA at different intensity levels [13–15], with cardiometabolic risk factors in children. The results of these studies suggest that at least moderate-intensity PA is required to reduce cardiometabolic risk among children [16].

Screen time assessed by questionnaires, a widely used measure of ST, has been directly associated with cardiometabolic risk factors in cross-sectional studies among children and adolescents [3,17,18]. However, cross-sectional studies have found weak or no associations of objectively measured total ST with cardiometabolic risk factors in children [9,19,20]. Moreover, on the basis of recent reviews there is limited evidence from longitudinal studies on the associations of objectively measured total ST with clustered cardiometabolic risk factors in children [21,22].

We investigated the cross-sectional and longitudinal associations of objectively measured total PA energy expenditure (PAEE), PA at different intensity levels, and total ST with overall cardiometabolic risk and individual cardiometabolic risk factors in a 2-year follow-up study in a general population of children.

2 Methods

2.1 Study design and study population

The present analyses are based on the baseline and 2-year follow-up data from the Physical Activity and Nutrition in Children (PANIC) Study that is an ongoing controlled PA and dietary intervention study (ClinicalTrials.gov NCT01803776) in a representative population sample of primary school children from the city of Kuopio, Finland. Altogether 736 children aged 6–8 years who started the first grade in primary schools in 2007–2009 were invited in the baseline examinations, and 512 (70%) of them participated. Complete data on variables used in the statistical analyses were available for 399 children at baseline (198 girls, 201 boys) and for 258 children at 2-year follow-up (140 girls, 118 boys). The Research Ethics Committee of the Hospital District of Northern Savo approved the study protocol. All participating children gave their assent, and their parents or caregivers gave a written informed consent.

2.2 Assessment of PA and ST

PA and ST were assessed using a combined heart rate and movement sensor (Actiheart, CamNtech Ltd, Papworth, UK), a light and waterproof device [23] which is attached to the chest with standard electrocardiogram (ECG) electrodes (Bio Protech Inc, Wonju, South Korea). The monitor was set to record heart rate and body movement in 60-second epochs.

The participants were instructed to carry on with their usual behaviour and to wear the monitor during all daily activities, including sleep, shower, sauna, and swimming. The activity patterns of school children are known to vary markedly between weekdays and weekend days [24]. The participants were therefore requested to wear the monitor continuously for a minimum of four consecutive days, including two weekdays and two weekend days, to obtain more representative information on PA and ST. Altogether 73% of the participants at baseline and 63% of the participants at 2-year follow-up wore the Actiheart monitor for at least four days the average wear time being 4.7 days at baseline and 4.1 days at 2-year follow-up. We accepted PA and ST data for the statistical analyses if there was a minimum of 48 hours of activity recording in weekday and weekend day hours that included at least 12 hours from morning (3 am - 9 am), noon (9 am - 3 pm), afternoon (3 pm - 9 pm), and night (9 pm - 3 am) to avoid potential bias from over-representing specific times and activities of the days.

Heart rate data were cleaned [25] and individually calibrated with sleeping heart rate and parameters obtained from maximal exercise tests [26,27] performed by the Ergoselect 200K® electromagnetic bicycle ergometer (Ergoline, Bitz, Germany) and the Cardiosoft® V6.5 Diagnostic System ECG device (GE Healthcare Medical Systems, Freiburg, Germany). The heart rate data were finally combined with trunk acceleration data in a branched equation model to estimate activity intensity time-series [28], as described previously [29]. PAEE was calculated by integrating the intensity time-series, where time distribution of activity intensity was generated by using standard metabolic equivalents (METs) in 0.5 increments. Sleep duration was analysed from the Actiheart recordings by a trained exercise specialist and confirmed by a physician, if necessary. The time of falling asleep was defined as accelerometer counts decreasing to zero and heart rate to a plateau level. The time of waking

up was defined as accelerometer counts increasing and remaining above zero and heart rate increasing and remaining above the plateau level. We defined total ST as time spent in activity ≤1.5 METs excluding sleep and light PA (LPA), moderate PA (MPA), and vigorous PA (VPA) as time spent in activity >1.5 and ≤4.0 METs, > 4.0 and ≤7.0 METs, and >7.0 METs, respectively, by defining 1 MET as 71.2 J/min/kg. Moderate-to-vigorous PA (MVPA) included MPA and VPA.

2.3 Assessment of cardiometabolic risk factors and calculation of cardiometabolic risk score

The children attended the examinations in the research facility between 7 am and 10 am having fasted for 12 hours. A research nurse measured blood pressure, assessed body size and composition, and took blood samples. Body height was measured three times using a calibrated wall-mounted stadiometer to accuracy of 0.1 cm the children standing in the Frankfurt plane without shoes. We used the mean of the nearest two values in the analyses. Body weight was measured twice using the Inbody 720® bioimpedance device (Biospace, Seoul, Korea) to accuracy of 0.1 kg the children having emptied the bladder and wearing light underwear [30]. We used the mean of the two values in the analyses. Body mass index (BMI) was calculated by dividing body weight (kg) with body height (m) squared. BMI - standard deviation score (BMI-SDS) was calculated using Finnish reference data [31]. Body fat percentage was measured using the Lunar® dual-energy x-ray absorptiometry (DXA) device (Lunar Prodigy Advance; GE Medical Systems, Madison, WI, USA) the children being in the non-fasting state, having emptied the bladder, and being in light clothing with all metal objects removed [32]. The assessment of fasting serum insulin and fasting plasma glucose, triglycerides, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL)

cholesterol has been explained previously [33]. Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated using the formula fasting serum insulin (mU/L) x fasting plasma glucose (mmol/L) / 22.5 [34]. Blood pressure was measured manually from the right arm using a calibrated Heine Gamma® G7 aneroid sphygmomanometer (Heine, Optotechnic GmbH, Herrsching, Germany) to accuracy of two mmHg. The measurement protocol included a rest of five minutes and thereafter three measurements in the sitting position at 2-minute intervals. The mean of all three measurements was used as the systolic and diastolic blood pressure. We calculated the cardiometabolic risk score using continuous z-score variables adjusted for age and sex using the formula $Z_{\text{waist circumference}} + Z_{\text{insulin}} + Z_{\text{glucose}} + Z_{\text{triglycerides}} - Z_{\text{HDL cholesterol}} + Z_{\text{the average of systolic and diastolic blood pressure}}$, a larger score indicating a higher cardiometabolic risk [35].

2.4 Assessment of pubertal status

A research physician assessed pubertal status using a 5-stage scale described by Tanner [36]. The boys were defined having entered clinical puberty if their testicular volume assessed by an orchidometer was ≥4 (Stage ≥2). The girls were defined having entered clinical puberty if their breast development had started (Stage ≥2).

2.5 Statistical analysis

The statistical analyses were performed using the IBM SPSS Statistics software, Version 25.0 (IBM Corp. Armonk, NY, USA). To normalise skewed distributions, a natural logarithmic transformation was performed for waist circumference, body fat percentage, triglycerides,

and HDL cholesterol, and a square root transformation was performed for insulin. Differences in basic characteristics between sexes were tested using the independent samples T-test for continuous variables with normal distributions, the Mann–Whitney U-test for continuous variables with skewed distributions, and the Chi-square test for categorical variables. The cross-sectional associations of total ST, LPA, MVPA, VPA, and PAEE with the cardiometabolic risk score and independent cardiometabolic risk factors at baseline were analysed using linear regression models adjusted for age, sex, and puberty. The longitudinal associations of changes in total ST, LPA, MVPA, VPA, and PAEE with changes in the cardiometabolic risk score and independent cardiometabolic risk factors during 2-year follow-up were analysed using linear regression models adjusted for age, sex, and the explanatory and outcome variables at baseline as well as the change in pubertal status during 2-year follow-up. We adjusted the longitudinal data for the explanatory and outcome variables at baseline to control for variation in their initial levels that could affect the changes in these variables and thereby partly explain the associations observed. We additionally adjusted the cross-sectional data for baseline body fat percentage and the longitudinal data for baseline body fat percentage and the change in body fat percentage during 2-year follow-up to study whether body fat content partly explained the associations observed. Adjustment for baseline lean body mass or the change in lean body mass had no effect on the cross-sectional or longitudinal associations, so these data are not presented in the results. Associations with P-values <0.05 were considered statistically significant.

3 Results

3.1 Basic characteristics

Girls were shorter and lighter and had higher body fat percentage, smaller waist circumference, higher fasting serum insulin, and lower fasting plasma glucose compared to boys (Table 1). Furthermore, girls had longer total ST, higher LPA, and lower MVPA, VPA, and PAEE than boys.

3.2 Cross-sectional associations at baseline

Higher total ST was associated with a higher cardiometabolic risk score, higher body fat percentage, waist circumference, insulin, HOMA-IR, glucose, systolic blood pressure, and diastolic blood pressure, and lower HDL cholesterol adjusted for age, sex, and puberty (Table 2). The associations of total ST with the cardiometabolic risk score (β=0.116, P=0.013), insulin (β=0.138, P=0.006), and HOMA-IR (β=0.141, P=0.005) attenuated but remained statistically significant after further adjustment for body fat percentage. Total ST was not related to other cardiometabolic risk factors adjusted for body fat percentage.

LPA was inversely associated with the cardiometabolic risk score, body fat percentage, waist circumference, insulin, HOMA-IR, glucose, and diastolic blood pressure and was directly related to HDL cholesterol adjusted for age, sex, and puberty (Table 2). The associations of LPA with the cardiometabolic risk score (β=-0.101, P=0.022), insulin (β=-0.101, P=0.034), and HOMA-IR (β=-0.110, P=0.022) weakened but remained statistically significant after additional adjustment for body fat percentage. LPA was not associated with other cardiometabolic risk factors adjusted for body fat percentage.

Lower MVPA was associated with a higher cardiometabolic risk score and higher body fat percentage, waist circumference, insulin, HOMA-IR, triglycerides, LDL cholesterol, systolic blood pressure, and diastolic blood pressure adjusted for age, sex, and puberty (Table 2).The associations of MVPA with insulin (β=-0.125, P=0.018), HOMA-IR (β=-0.120, P=0.025), and systolic blood pressure (β=-0.114, P=0.043) weakened but remained statistically significant after further adjustment for body fat percentage. MVPA was not related to other cardiometabolic risk factors adjusted for body fat percentage.

The inverse associations of VPA with the cardiometabolic risk score, body fat percentage, waist circumference, insulin, HOMA-IR, and LDL cholesterol were slightly weaker and the direct association between VPA and HDL cholesterol was slightly stronger than the relationships of MVPA to these cardiometabolic risk factors after adjustment for age, sex, and puberty (Table 2). The associations of VPA with cardiometabolic risk factors were no longer statistically significant after further adjustment for body fat percentage.

Lower PAEE was associated with a higher cardiometabolic risk score and higher body fat percentage, waist circumference, insulin, HOMA-IR, triglycerides, LDL cholesterol, systolic blood pressure, and diastolic blood pressure adjusted for age, sex, and puberty (Table 2). The relationships of PAEE to cardiometabolic risk score (β=-0.103, P=0.038), insulin (β=-0.146, P=0.006), HOMA-IR (β=-0.148, P=0.006), and systolic blood pressure (β=-0.130, P=0.022) weakened but remained statistically significant after further adjustment for body fat percentage. PAEE was not associated with other cardiometabolic risk factors adjusted for body fat percentage.

3.3 Longitudinal associations during 2-year follow-up

Increased total ST was associated with an increased cardiometabolic risk score and increased body fat percentage, waist circumference, and insulin adjusted for age, sex, total ST, and respective cardiometabolic risk factors at baseline and incident puberty (Table 3). The association of increased total ST with an increased cardiometabolic risk score weakened but remained statistically significant after further adjustment for baseline body fat percentage and the change in body fat percentage (β=0.155, P=0.029). The change in total ST was not related to the change in insulin after adjustment for baseline body fat percentage and the change in body fat percentage.

The change in LPA was inversely associated with the change in the cardiometabolic risk score adjusted for age, sex, LPA, and the cardiometabolic risk score at baseline and incident puberty (Table 3). This relationship was no longer statistically significant after additional adjustment for baseline body fat percentage and the change in body fat percentage. The change in LPA was not associated with the changes in individual cardiometabolic risk factors adjusted for age, sex, LPA, respective cardiometabolic risk factors at baseline, and incident puberty (Table 3).

Increased MVPA was associated with a decreased cardiometabolic risk score, reduced body fat percentage, waist circumference, insulin, and HOMA-IR, and increased HDL cholesterol adjusted for age, sex, MVPA, and respective cardiometabolic risk factors at baseline and incident puberty (Table 3). These relationships were no longer statistically significant after further adjustment for baseline body fat percentage and the change in body fat percentage.

Increased VPA was associated with a decreased cardiometabolic risk score, reduced body fat percentage, waist circumference, insulin, HOMA-IR, and triglycerides, and increased HDL cholesterol adjusted for age, sex, VPA, and respective cardiometabolic risk factors at baseline and incident puberty (Table 3). The inverse associations of the change in VPA with the changes in the cardiometabolic risk score (β=-0.143, P=0.017), insulin (β=-0.161, P=0.016), HOMA-IR (β=-0.157, P=0.020), and triglycerides (β=-0.135, P=0.042) and the direct relationship between the change in VPA and the change in HDL cholesterol (β=0.146, P=0.043) weakened but remained statistically significant after additional adjustment for baseline body fat percentage and the change in body fat percentage.

The change in PAEE was inversely associated with the changes in the cardiometabolic risk score, body fat percentage, waist circumference, insulin, HOMA-IR, and triglycerides and was directly related to the change in HDL cholesterol adjusted for age, sex, PAEE, and respective cardiometabolic risk factors at baseline and incident puberty (Table 3). The inverse associations of the change in PAEE with the changes in the cardiometabolic risk score (β= -0.156, P=0.019) and insulin (β= -0.153, P=0.038) and the direct relationship between the change in PAEE and the change in HDL cholesterol (β= 0.180, P=0.022) remained similar after additional adjustment for baseline body fat percentage and the change in body fat percentage. The inverse associations of the change in PAEE with the changes in HOMA-IR and triglycerides were no longer statistically significant after further adjustment for these measures of body fat content.

4 Discussion

The main finding of this longitudinal study is that increased VPA, MVPA, and PAEE as well as decreased ST were associated with a reduced cardiometabolic risk score and decreased body fat percentage, waist circumference, fasting serum insulin, and HOMA-IR during 2-year follow-up in a general population of children. Moreover, increased VPA, MVPA, and PAEE were associated with elevated plasma HDL cholesterol, and increased VPA was related to decreased plasma triglycerides. However, increased LPA was associated only with a decreased cardiometabolic risk score. We also found cross-sectional associations of lower VPA, MVPA, LPA, and PAEE as well as longer total ST with a higher cardiometabolic risk score and most of the individual cardiometabolic risk factors in children.

Our cross-sectional findings on the inverse associations of MVPA and VPA with overall cardiometabolic risk, body fat content, and insulin resistance in children are in accordance with the results of some earlier cross-sectional studies [1,20,37]. The results of the few previous prospective studies on the associations of objectively measured PA with cardiometabolic risk factors in paediatric populations suggest that at least moderate-intensity PA is required to reduce cardiometabolic risk among children[13,15]. Consistent with these prospective findings, we observed that increased VPA and MVPA were associated with decreased overall cardiometabolic risk, body fat content, and insulin resistance but also with reduced dyslipidaemia during 2-year follow-up in a general population of children.

Previous studies have mainly focused on the associations of MVPA, VPA, and total PA with cardiometabolic risk factors, and therefore little is known about the relationships of LPA to cardiometabolic risk factors [38]. However, there are some earlier cross-sectional and prospective studies on the associations of LPA with cardiometabolic risk factors in children, but the results of these studies have been inconsistent [16,39]. In our cross-sectional analyses, lower LPA was associated with higher overall cardiometabolic risk, body fat content, insulin resistance, and fasting plasma glucose as well as lower plasma HDL cholesterol. In the longitudinal analyses, however, increased LPA was associated only with decreased overall cardiometabolic risk.

Earlier cross-sectional studies have shown direct associations of screen time assessed by questionnaires with cardiometabolic risk factors among children and adolescents [3,10]. However, previous cross-sectional studies have found weak or no associations of objectively measured total ST with overall cardiometabolic risk, body fat content, plasma HDL cholesterol, and blood pressure among children [21,40,41]. We observed direct cross-sectional associations of total ST with the cardiometabolic risk score, body fat content, and insulin resistance in children. Importantly, there are few earlier longitudinal studies on the associations of objectively measured total ST with cardiometabolic risk factors in children [21,41]. In our longitudinal analyses, ST was directly associated only with overall cardiometabolic risk among children.

The beneficial associations of PA and the harmful associations of ST with some cardiometabolic risk factors in children have been found to be partly explained by body fat content [13,42]. In line with these results, we observed that body fat content partly explained the

cross-sectional and longitudinal associations of PA at different intensity levels and ST with cardiometabolic risk factors. Importantly, the longitudinal associations of VPA and PAEE with overall cardiometabolic risk, insulin resistance, and dyslipidaemia and the longitudinal association between ST and overall cardiometabolic risk remained even after controlling for body fat content. However, the longitudinal associations of LPA and MVPA with cardiometabolic risk factors did not persist after taking body fat content into account. These findings suggest that the associations of increased vigorous PA with decreased overall cardiometabolic risk, insulin resistance, and dyslipidaemia and the association between decreased ST and reduced overall cardiometabolic risk are explained also by other physiological mechanisms than change in body fat content. One of these mechanisms could be enhanced insulin sensitivity in skeletal muscle in response to exercise training [43,44]. The results of some studies suggest that increasing at least moderate-intensity PA improves insulin sensitivity independent of adiposity not only in adults but also in children and adolescents [2,45]. Moreover, there is some evidence that sedentary behaviour impairs endothelial function, increases oxidative stress, and elevates blood pressure independent of adiposity among children and youth [9,19,20,40,46].

The strengths of the present study include the relatively large population sample of children, the longitudinal study design with 2-year follow-up, the objective and valid assessment of free-living movement behaviour, as well as the comprehensive and detailed assessment of cardiometabolic risk factors and possible confounding factors. These characteristics of the study enabled us to investigate and compare the magnitude of the associations of the changes in PA at different intensity levels and the change in ST with the changes in a number of cardiometabolic risk factors in a general population of children. Even though we used a

longitudinal approach, however, we cannot draw conclusions about the causality of the relationships of PA and ST with cardiometabolic risk factors among children.

A limitation of our study is that the assessment of movement behaviour by the Actiheart® monitor, which combines information of heart rate and body movements, may overestimate LPA and underestimate ST because not only PA but also sympathetic activation at rest increases heart rate [47–49]. In case of some children it may also have been difficult to determine the time of falling asleep or waking up which may entail some error into ST estimation, as well. Moreover, Actiheart® is a method for measuring the intensity and energy expenditure of PA but not different types of PA that reflect PA as a behaviour. Another limitation of the study is the use of relatively long epochs during the Actiheart® recording, because 60-second epochs may have limited our ability to detect the relatively common intermittent bouts of PA among children [50].

5 Conclusion

Our longitudinal study showed that increased VPA, MVPA, and PAEE and decreased total ST were associated with reduced overall cardiometabolic risk and major individual cardiometabolic risk factors, including adiposity, insulin resistance, and dyslipidaemia, among school-aged children. Increased LPA had weaker associations with changes in cardiometabolic risk factors. These findings suggest that increasing at least moderate-intensity PA provides additional cardiometabolic benefits, including decreased overall cardiometabolic risk, reduced adiposity, improved insulin sensitivity, decreased plasma triglycerides, and increased plasma HDL cholesterol, beyond less intensive PA among

children. Our observations also suggest that not only increasing at least moderate-intensity PA but also decreasing total ST improves cardiometabolic health in children.

6 Perspectives

The findings of our longitudinal study emphasize increasing at least moderate-intensity PA and decreasing total ST to improve cardiometabolic health in general populations of school-aged children. Long-term follow-up and intervention studies starting in childhood and having objective measures of PA and ST and comprehensive assessments of cardiometabolic risk factors are needed to provide further evidence for the beneficial effects of increasing PA and decreasing sedentary ST in reducing cardiometabolic risk later in life.

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Compliance with Ethical Standards

Juuso Väistö, Eero Haapala, Anna Viitasalo, Theresia Maria Schnurr, Tuomas Kilpeläinen, Panu Karjalainen, Kate Westgate, Hanna-Maaria Lakka, David Laaksonen, Ulf Ekelund, Søren Brage, Timo Lakka declare that they have no conflicts of interest. All aspects of the PANIC study were approved by the Research Ethics Committee of the Hospital District of Northern Savo. Written informed consent was acquired from the parent/caregiver of each child and every child provided assent to participation.

Ethical approval

All procedures were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual parents or guardians while children gave their assent to the study.

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Table 1 Baseline descriptive characteristics of the study population

Variable	Girls (n=198)	Boys (n=201)	All (n=399)	p-value for difference
Demographics				
Age (years)	7.6 ± 0.4	7.7 ± 0.4	7.6 ± 0.4	0.082
Anthropometrics				
Weight (kg)	26 ± 4.6	27.3 ± 4.7	26.7 ± 4.7	**0.003**
Height (cm)	127.5 ± 5.5	129.9 ± 5.5	128.7 ± 5.7	**<0.001**
Body mass index standard deviation score[1]	-0.109 ± 0.99	0.06 ± 1.11	-0.02 ± 1.05	0.104
Waist circumference (cm)	55.4 ± 5.4	57.2 ± 5.2	55.3 ± 5.4	**<0.001**
Body fat percentage (%)	21.7 ± 7.2	17.2 ± 7.8	19.4 ± 7.8	**<0.001**
Pubertal status (%)*				
Prepubertal	97,5	98,5	98,0	0.462
Pubertal	2,5	1,5	2,0	
Biochemical markers				
HOMA Insulin Resistance	1.00 ± 0.51	0.94 ± 0.60	0.97 ± 0.56	0.056
Fasting serum insulin (mU/L)	4.64 ± 2.16	4.24 ± 2.5	4.44 ± 2.34	**0.013**
Fasting plasma glucose (mmol/L)	4.75 ± 0.39	4.88 ± 0.36	4.81 ± 0.38	**<0.001**
Fasting plasma triglycerides (mmol/L)	0.61 ± 0.24	0.59 ± 0.24	0.60 ± 0.24	0.156
Fasting plasma HDL cholesterol (mmol/L)	1.57 ± 0.29	1.62 ± 0.31	1.59 ± 0.30	0.106
Fasting plasma LDL cholesterol (mmol/L)	2.38 ± 0.51	2.30 ± 0.5	2.34 ± 0.51	0.115
Fasting plasma total cholesterol (mmol/L)	4.28 ± 0.6	4.21 ± 0.62	4.25 ± 0.61	0.134
Systolic blood pressure (mmHg)	99.8 ± 7.3	100.4 ± 7.2	100 ± 7.3	0.413
Diastolic blood pressure (mmHg)	60.9 ± 7.6	61.6 ± 7.0	61.2 ± 7.3	0.380
Cardiometabolic risk score	-0.22 ± 3.33	0.09 ± 3.64	-0.07 ± 3.48	0.361
Objectively measured movement behaviors				
Total sedentary time (min/d)	241 ± 128	225 ± 125	233 ± 127	**<0.001**
Light physical activity (min/d)	522 ± 108	499 ± 101	510 ± 105	**0.010**
Moderate to vigorous physical activity (min/d)	97 ± 53	135 ± 67	116 ± 63	**<0.001**
Moderate physical activity (min/d)	81 ± 45	105 ± 57	93 ± 53	**<0.001**
Vigorous physical activity (min/d)	16 ± 16	31 ± 26	23 ± 23	**<0.001**
PAEE (kJ/kg/day)	89.7 ± 27.4	105.3 ± 33.3	97.6 ± 31.5	**<0.001**

Values are means and standard deviations for all variables. P-values are from independent samples t-test for variables with normal distributions or Mann–Whitney U-test for variables with skewed distribution and chi-square test for categorical variables. [1]Based on Finnish reference values. *Pubertal status using Tanner 5-stage criteria

Table 2 Cross-sectional associations of baseline PA with baseline cardiometabolic factors adjusted for age, sex and puberty in 398 children

	Cardio Metabolic Risk Score	***Body fat percentage (%)***	***Waist Circumference (cm)***	***Fasting Serum Insulin (mU/l)***	***HOMA-IR***	***Fasting plasma glucose (mmol/L)***	***Fasting plasma triglycerides (mmol/L)***	***Fasting plasma HDL cholesterol (mmol/L)***	***Fasting plasma LDL cholesterol (mmol/L)***	***Systolic blood pressure (mmHg)***	***Diastolic blood pressure (mmHg)***
	Standardized Regression Coefficient (p-value)										
Sedentary Time (min/day)	0.298	0.359	0.295	0.262	0.262	0.103	0.096	-0.104	0.098	0.118	0.172
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**0.039**	0.058	**0.040**	0.051	**0.019**	**0.001**
Light Physical Activity (min/day)	-0.203	-0.191	-0.171	-0.173	-0.180	-0.109	-0.060	0.100	-0.029	-0.030	-0.114
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**0.029**	0.238	**0.047**	0.569	0.556	**0.024**
Moderate to Vigorous Physical Activity (min/day)	-0.285	-0.391	-0.319	-0.263	-0.255	-0.035	-0.113	0.076	-0.159	-0.182	-0.145
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	0.500	**0.032**	0.152	**0.002**	**0.001**	**0.006**
Vigorous Physical Activity (min/day)	-0.221	-0.324	-0.271	-0.186	-0.196	-0.056	-0.045	0.113	-0.108	-0.093	-0.087
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	0.287	0.394	**0.032**	**0.041**	0.078	0.102
PAEE (kJ/kg/day)	-0.322	-0.435	-0.352	-0.289	-0.287	-0.068	-0.107	0.088	-0.155	-0.200	-0.174
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	0.192	**0.041**	0.092	**0.003**	**<0.001**	**0.001**

Table 3 Longitudinal associations of changes in total sedentary time, light, moderate-to-vigorous, and vigorous physical activity, and physical activity energy expenditure with changes in cardiometabolic risk factors adjusted for adjusted for age, sex, and the explanatory and outcome variables at baseline as well as the change in pubertal status during 2-year follow-up in 258 children

	Δ*Cardio Metabolic Risk Score*	Δ*Body fat percentage (%)*	Δ*Waist Circumference (cm)*	Δ*Fasting Serum Insulin (mU/l)*	Δ*HOMA-IR**	Δ*Fasting plasma glucose (mmol/L)*	Δ*Fasting plasma triglycerides (mmol/L)*	Δ*Fasting plasma HDL cholesterol (mmol/L)*	Δ*Fasting plasma LDL cholesterol (mmol/L)*	Δ*Systolic blood pressure (mmHg)*	Δ*Diastolic blood pressure (mmHg)*
	Standardized Beta Coefficient (p-value)										
Δ**Total *Sedentary Time (min/day)***	0.218	0.212	0.250	0.159	0.136	0.101	0.113	-0.146	0.019	0.080	0.008
	0.003	**0.016**	**0.004**	**0.049**	0.093	0.137	0.144	0.080	0.219	0.304	0.919
Δ***Light Physical Activity (min/day)***	-0.163	-0.132	-0.169	-0.125	-0.114	-0.103	-0.074	0.083	0.088	-0.072	-0.038
	0.032	0.142	0.056	0.134	0.170	0.137	0.355	0.333	0.991	0.367	0.623
Δ***Moderate to Vigorous Physical Activity (min/day)***	-0.178	-0.298	-0.225	-0.213	-0.194	-0.054	-0.122	0.184	-0.128	0.053	0.089
	0.012	**<0.001**	**0.007**	**0.006**	**0.013**	0.415	0.102	**0.022**	0.128	0.475	0.216
Δ*Vigorous Physical Activity (min/day)*	-0.209	-0.244	-0.173	-0.220	-0.213	-0.101	-0.164	0.159	-0.021	0.030	0.069
	0.001	**0.001**	**0.016**	**0.001**	**0.002**	0.076	**0.012**	**0.023**	0.770	0.647	0.271
Δ***PAEE (kJ/kg/day)***	-0.244	-0.371	-0.294	-0.237	-0.218	-0.092	-0.168	0.190	-0.078	0.008	0.064
	<0.001	**<0.001**	**<0.001**	**0.001**	**0.003**	0.130	**0.016**	**0.011**	0.321	0.908	0.345