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Moderate-and-vigorous physical activity from adolescence to adulthood and subclinical atherosclerosis in adulthood: prospective observations from the European Youth Heart Study

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ABSTRACT

Aim To investigate the independent associations between mean exposure to or the change in moderate-and-vigorous physical activity (PA) from adolescence to adulthood and subclinical atherosclerosis in adulthood.

Methods This was a prospective cohort study among Danish boys and girls (N=277) followed for up to 12 years (age 15.7 (0.4) at baseline) enrolled in the European Youth Heart Study. PA intensity was objectively measured at baseline and follow-up, and ultrasonography was performed on the Carotid arteries at follow-up. Data on carotid intima-media thickness (cIMT), Carotid Compliance and Young's Elastic Modules were used as outcome measures.

Results In the multivariable analyses (adjusted for personal—lifestyle and demographic factors) the mean exposure to moderate-and-vigorous PA from adolescence to adulthood was negatively associated with Young's Elastic Modules (β=-0.001×10^3 kPa (95% CI −0.0015 to −0.0002), p=0.02) and positively associated with Carotid Compliance (β=0.004 mm^2 kPa^{-1} (95% CI 0.002 to 0.008), p=0.003) and cIMT (β=0.0003 mm (95% CI 0.00001 to 0.0007), p=0.013). Increases in moderate-and-vigorous PA from adolescence to adulthood were negatively associated with Young's Elastic Modules in adulthood (β=-0.00007×10^3 kPa (95% CI −0.0012 to −0.0001), p=0.01). Furthermore, participants with the largest decline in moderate-and-vigorous PA from adolescence to adulthood displayed significantly less compliant arteries compared with the remaining sample (p<0.05).

Conclusions High mean exposure to moderate-and-vigorous PA levels and increases herein were independently associated with lower levels of carotid arterial stiffness in adulthood.

INTRODUCTION

Increased carotid intima-media thickness (cIMT) and arterial stiffness in adults are associated with future cardiovascular disease (CVD) events independent of conventional risk factors.[1 2] Observations in adults suggest that increased physical activity (PA) levels attenuate the age-related carotid arterial stiffening[3] the CVD risk decreases with increasing PA intensity.[4]

Large decreases in PA are observed across youth[5] but little is known about the effect of the decrease in PA on cardiovascular risk in healthy adults. Research indicates that change in PA levels across adolescence is related to adult cardiovascular risk markers rather than the childhood PA level.[6] Previous longitudinal studies, conducted on the association between PA and subclinical atherosclerosis later in life, have used subjective methods to assess PA.[7–10] Self-reported PA is particularly susceptible to recall bias and non-differential misclassification,[11] with the risk of over- and underestimating the potential association. Therefore longitudinal studies investigating the health impact of age-related decline using objectively measured PA are needed.

The primary aim of the study was twofold: (1) to examine the association between the mean exposure to moderate-to-vigorous intensity PA in adolescence and adulthood; or (2) the changes in moderate-and-vigorous PA intensity from adolescence to adulthood and adult levels of cIMT and arterial stiffness in a population-based sample of healthy individuals from the European Youth Heart Study. Secondarily, we examined the association between the change in and mean exposure to moderate-and-vigorous PA and traditional cardiovascular risk factors in adulthood.

METHODS

Participants and design

This is a prospective observational study using data from the European Youth Heart Study. The European Youth Heart Study is an international population-based mixed longitudinal study that addresses the biological, environmental demographic and lifestyle correlates and determinants of CVD risk factors in children and adolescents. Detailed description of the European Youth Heart Study and the sampling procedures has been provided in detail elsewhere.[12] In 1997–1998, 658 15-year-old adolescents were randomly sampled and invited to take part in the study. A total of 429 adolescents participated (65% of the sample). Another random sample of 771 15-year-old adolescents were invited to participate in 2003–2004, of whom 444 adolescents agreed to take part (58% of the sample). A 6 and 12-year follow-up wave was conducted in 2009–2010, where all invitees were reinvited to participate. At follow-up, ultrasonography was added to the protocol. A total of 650 subjects agreed to participate (44% of the first cohort and 48% of the second). The present study reports on 277 subjects with complete data on exposures and outcomes. Two hundred and seventy-one subjects had valid exposure and outcome variables for the secondary analysis. The study was approved by the Regional Scientific Ethical Committee for Southern

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Denmark and data were collected according to the Helsinki declaration. All participants gave a written informed consent.

Carotid arterial properties

The carotid arterial properties were assessed at follow-up using ultrasonography (GE Healthcare Logic C5 (GE Healthcare, Milwaukee, Wisconsin, USA), 7.5-MHz linear probe) according to guidelines for user procedures.[13] Before the measurements, participants rested for 10 min in a quiet temperature-controlled room. The arterial properties were assessed at the lateral and posterior position, 10 mm (cIMT) and 20 mm (for arterial stiffness) proximal to the beginning of the carotid bulb on both the right and the left common carotid arteries. cIMT was obtained at the far wall of the artery. Intrareader coefficients of variation were 5.7%, 4.5% and 4.5% for cIMT, systolic and diastolic diameter, respectively.

The analyses were performed by a blinded trained reader, using commercially available edge tracking software (Vascular Research Tools 5, Medical Imaging Applications, LLC). cIMT and artery diameters were obtained from the best quality image (out of seven). Peak-systolic (DS), end-diastolic (DD) arterial diameter and cIMT were obtained. The mean values of both positions and both sides were used for the subsequent analysis.

Brachial systolic (SBP) and diastolic (DBP) blood pressures were obtained from the right arm at the end of the examination in a supine position (Welch Allyn Vital Signs monitor 300 series, Kivex, Hoersholm, Denmark). Brachial pulse pressure (PP) was calculated as SBP−DBP. Carotid compliance coefficient (CC) and Young's elastic modules (YEM) were calculated as follows:[2 7 10]

1. $CC=\pi*(DS^2-DD^2)/(4*PP)$ in mm^2/kPa
2. $YEM=DD/(cIMT\times DC)$ in 10^3 kPa

The distensibility coefficient (DC) was calculated as $(2\times(DS-DD)\times DD+(DS-DD)^2)/(PP\times DD^2)$ (unit: 10^{-3}/kPa). YEM is a measure of intrinsic structure of the arterial wall. Carotid compliance and DC express the absolute and relative change in the vessel area per unit PP, respectively. Higher values of YEM indicate stiffer arteries whereas higher values of CC indicate more elastic arteries.[14]

Moderate-and-vigorous PA intensity

PA was assessed using a PA monitor. The Actigraph model AM7164 (Pensacola, Florida, USA) was used in 1997–1998 and 2003–2004, whereas the Actigraph model GT3X or model GT1M activity monitor (Pensacola) was used in 2009–2010. The participants were instructed to wear for at least five consecutive days and only remove it during showering, swimming or during night time sleep. Due to the limited storage capacity of the AM7164 monitor, 1 min epochs were employed.

Moderate-and-vigorous PA intensity was expressed as minutes per day spent at different intensity intervals. The minutes were adjusted proportionally to a full day of 14.5 h (the estimated awake time for this population), as described elsewhere.[15] The cut-points for time spent in moderate-and-vigorous PA intensity (4> Mets at age 15 years) were determined using published cut-points.[16] The cut-point for moderate-and-vigorous PA was thus >3000 counts/min. The mean PA intensity was calculated as the sum of the minutes spent at the adolescence and adulthood PA interval divided by two. The changes in PA intensity from adolescence to adulthood were calculated as the difference between the follow-up and the baseline PA level.

Cardiovascular risk factors

Anthropometric procedures and measurements of blood pressure have been described in detail elsewhere.[11 14] Mean arterial BP was calculated as $DBP\times((SBP-DBP)/3)$. Fasting blood samples (overnight) were taken in the morning from the antecubital vein. Prior to measurements individuals were rested for 5 min while seated. For the secondary analysis a continuous metabolic CVD risk z-score was calculated according to the definition by American Heart Association and the National Heart, Lung and Blood Institute definition of the metabolic syndrome.[13] The z-score included waist circumference, the mean of DBP and SBP, triglycerides, high-density lipoprotein (inverted) and fasting glucose were standardised and subsequently summed to create a continuous z-score. Standardisation in adulthood was done according to the baseline distribution (mean and SD) of each of the risk factors.

Covariates

Family history of CVD events (paternal or maternal, yes/no) and parental educational levels were obtained by a parental self-report using the International Standard Classification of Education (ISCED) (UNESCO 1997). As the details obtained from the description of education were insufficient, the ISCED scale was combined into three new groups (1=level 1–2; 2=level 3–4 and 3=level 5–7). The highest parental educational level of the mother or father was used in the analysis. Current smoking status (yes/no) was obtained in adulthood. Weekly frequency of soft drinks, fruit and vegetable intake was obtained by a self-report in adolescence using a computer-based questionnaire.[12] At baseline and follow-up, TV time during leisure was obtained by a self-report.[17 18]

Statistics

Baseline descriptive statistics were calculated for participants with valid data on exposures and outcomes and for excluded participants. Exclusion criteria were (1) drop-out, (2) drop-in and (3) incomplete or invalid data on relevant exposures or outcomes.

Associations were analysed using multiple-linear regression analyses. The analysis of the mean exposure across adolescence was adjusted for gender, height and follow-up time (cohort) (model 1). The analysis of the change in moderate-and-vigorous PA was further adjusted for the baseline moderate-and-vigorous PA level. As we did not observe any gender and/or cohort interaction ($p>0.1$), the associations are presented for both gender and cohorts combined. Additionally, we adjusted the models for parental educational level, family history of CVD, adult smoking, baseline fruit, vegetable and soft drink consumption and TV-viewing time (model 2). Standard linear regression diagnostics were performed. As studies have shown that adiposity and mean arterial pressure may confound or mediate the association between PA and arterial stiffness, we additionally adjusted for the change in WC (ΔWC) and mean arterial BP across youth and their respective baseline levels.[7 19]

All statistical analyses were performed in STATA 11.2 (STATA Corp. Fort Valton Texas, USA) with $\alpha=0.05$ (two-sided).

RESULTS

The baseline characteristics of the participants and the excluded subjects are described in table 1. None of the participants reported any CVD. Moderate-and-vigorous PA decreased by 6.1 (22.0) min/day from adolescence to adulthood and mean moderate-and-vigorous PA was 20.6 (11.7) min/day. No

Table 1 Baseline characteristics of participants and excluded subjects

	Male				Female			
	N	Participants	N	Excluded	N	Participants	N	Excluded
Age	116	15.7 (0.4)	283	15.7 (0.4)	161	15.6 (0.4)	313	15.6 (0.4)
Parental educational level (%low/middle/high)	114	7/75/18	283	12/59/29	158	9/68/23	313	16/59/26
Familial CVD prevalence (%)	116	13	283	5	161	33	313	8*
Sugar sweetened drinks (servings/week)**	116	2.8 (1.6)	283	3.2 (2.1)	159	1.8 (1.6)	308	2.1 (1.7)
Fruit and vegetables (servings/week)**	114	7.7 (3.8)	280	8.0 (3.7)	159	9.8 (3.5)	308	8.7 (3.8)*
TV time**	114	2.7 (1.3)	280	2.5 (1.4)	159	1.7 (1.3)	313	2.2 (1.3)*
Biological maturity (%1/2/3/4/5)	114	0/2/1/63/34	280	0/1/1/46/52*	159	0/0/5/71/24	313	0/0/4/65/31
Physical activity								
MVPA (min/day)**	116	22.3 (13.1 to 36.6)	172	22.1 (8.7 to 35.7)	161	16.8 (9.2 to 28.4)	209	14.8 (8.0 to 26.7)
HPA (counts/min)**	116	409 (163)	172	394 (211)	161	524.9 (85.2)	209	580.4 (102.7)*
Daily wear time (h)	116	14.3 (1.0)	172	13.8 (1.3)*	161	14.2 (1.0)	209	13.8 (1.4)*
Accepted days	116	4.6 (0.9)	172	4.3 (1.2)	161	4.6 (1.0)	209	4.4 (1.5)
Metabolic CVD risk factors								
BMI	116	20.8 (2.3)	283	21.0 (2.8)	161	20.8 (2.7)	313	21.3 (3.0)
Waist circumference**	116	74.1 (6.1)	283	73.4 (7.9)	161	70.0 (6.1)	313	71.8 (7.4)
Systolic BP (mm Hg)**	116	113.4 (10.3)	283	114.8 (10.8)	161	106.7 (8.3)	107	106.9 (7.9)
Diastolic BP (mm Hg)**	116	61.1 (7.2)	283	61.6 (6.2)	161	62.6 (5.6)	107	63.3 (6.0)
Glucose (mmol/l)**	113	5.2 (0.4)	103	5.3 (0.5)	158	5.0 (0.4)	102	5.0 (0.4)
HDL (mmol/l)**	113	1.3 (0.3)	103	1.3 (0.3)	158	1.5 (0.3)	102	1.4 (0.4)
Triglyceride (mmol/l)	113	0.87 (0.54)	103	0.95 (0.49)	158	0.91 (0.40)	102	0.96 (0.41)
Mean arterial pressure	113	78.4 (7.5)			158	77.2 (5.8)		

Data are means (SD) or median (IQR).
*p<0.05 for difference between participants and excludes subjects (including drop-outs and subjects with missing data at baseline or follow-up).
**p<0.05 for gender differences in participants (with complete data).
BMI, body mass index; BP, blood pressure; CVD, cardiovascular disease; HDL, high-density lipoprotein; HPA, mean habitual physical activity; MVPA, moderate-and-vigorous physical activity; VPA, vigorous physical activity.

differences in change were observed between men and women (p>0.05); however, men spent more time in moderate-and-vigorous intensity compared with women (5.4 (1.4) min/day, p=0.0002). Table 2 describes the follow-up values of subclinical atherosclerosis.

Baseline moderate-and-vigorous PA was positively associated with adult IMT (std. β=0.12, p=0.02) and CC (std. β=0.11, p=0.04), but not YEM (p>0.1) adjusted for gender, body height and cohort. At follow-up (cross-sectional analysis) moderate-and-vigorous PA was significantly associated with YEM (std. β=−0.16, p=0.009) and marginally associated with cIMT (std. β=0.10, p=0.07) and CC (std. β=0.11, p=0.05) adjusted for gender, body height and cohort.

The mean exposure to moderate-and-vigorous PA was significantly associated with all outcomes (p<0.05) adjusted for gender and cohort (table 3). For every 1-SD increment (11 min/day) of the mean exposure to moderate-and-vigorous PA, cIMT and CC were increased by 0.17 SD in adulthood (p<0.05), whereas YEM was decreased by –0.16 SD in adulthood (p<0.05).

Table 2 Arterial properties at follow-up

	Men	Women
N	116	161
Intima media thickness (mm)	0.57 (0.04)*	0.56 (0.03)
Carotid compliance coefficient (mm^2/kPa)	1.5 (0.3)*	1.3 (0.3)
Young's elastic modules (10^3×kPa)	0.28 (0.07)*	0.26 (0.06)

*Significant gender differences (p<0.05).

For a 1-SD (22 min/day) increase in moderate-and-vigorous PA from adolescence to adulthood, YEM was reduced by –0.23 SD (p<0.05) in adulthood, adjusted for gender, cohort and baseline moderate-and-vigorous PA. The change in moderate-and-vigorous PA was marginally positively associated with CC (std. β=0.17, p=0.06; model 1). Adjustment for the remaining covariates (model 2) did not alter the associations substantially. Additional adjustment for the change in waist circumference or mean arterial pressure and the respective baseline values did not alter the associations materially (data not shown). Replacing waist circumference with body mass index did not change this.

Figure 1 shows the association between the gender- and cohort-adjusted standardised values for CC, YEM and cIMT across quintiles of mean exposure to and changes in moderate-and-vigorous PA. The upper three quintiles of PA change (with little or an increase) displayed less stiff and more compliant carotid arteries compared with the group with the largest decline (p<0.05). Furthermore, only the participants with the highest mean exposure to moderate-and-vigorous PA had significantly higher cIMT compared with the least active group (p<0.05). Table 4 shows the mean minutes of and change in moderate-and-vigorous PA across quintiles of the respective exposure.

In the secondary analysis a 1-min/day decline in moderate-and-vigorous PA was associated with a 0.04-unit (95% CI −0.08 to −0.01, p=0.01, std. β=0.24) increased metabolic syndrome z-score in adulthood adjusted for gender, cohort and baseline moderate-and-vigorous PA. Further adjustment of the remaining covariates did not change the association. We did not observe a significant association between the mean exposure to

Table 3 Associations between mean physical activity intensity across youth and adult measures of subclinical atherosclerosis

	Model 1			Model 2		
Variable	**β (95% CI)**	**Std. β**	**p Value**	**β (95% CI)**	**Std. β**	**p Value**
Mean moderate-and-vigorous physical activity						
Intima media thickness (mm)	0.0004 (0.0001 to 0.001)	0.17	0.004	0.0003 (0.0001 to 0.0007)	0.15	0.013
Carotid compliance (mm^2/kPa)	0.004 (0.001 to 0.007)	0.17	0.004	0.004 (0.002 to 0.008)	0.17	0.003
Young's elastic modules (10^3 kPa)	−0.001 (−0.0016 to −0.0002)	−0.16	0.007	−0.001 (−0.0015 to −0.0002)	−0.16	0.01
Change in moderate-and-vigorous physical activity						
Intima media thickness (mm)	0.0002 (−0.0002 to 0.0005)	0.16	0.08	0.00024 (−0.00002 to 0.00050)	0.17	0.07
Carotid compliance (mm^2/kPa)	0.002 (−0.0001 to 0.0046)	0.17	0.06	0.002 (−0.0004 to 0.0050)	0.16	0.11
Young's elastic modules (10^3 kPa)	−0.0007 (−0.0012 to −0.0002)	−0.25	0.01	−0.0007 (−0.0012 to −0.0001)	−0.25	0.01

Model 1 is adjusted for gender, body height and cohort. Change if further adjusted for baseline levels of exposure.
Model 2 is equal to Model 1 but additionally adjusted for parental educational status, weekly servings of soft drinks, vegetables and fruits, family history of cardiovascular disease, adult smoking status and baseline TV-viewing time.

moderate-and-vigorous PA and the metabolic syndrome z-score (std. β=−0.08, p=0.19). Associations between change in and mean exposure to moderate-and-vigorous PA and the individual CVD risk factors in adulthood are described in an online supplementary table.

DISCUSSION

In this prospective study we observed that higher mean exposure to moderate-and-vigorous PA and an increase in moderate-and-vigorous PA from adolescence to adulthood were consistently associated with lower levels of carotid stiffness in adulthood independent of adiposity, mean arterial BP, demographic-, personal- and lifestyle factors. Furthermore, an increase in moderate-and-vigorous PA was associated with lower levels of metabolic CVD risk in adulthood. Therefore our observations suggest that prevention of the age-associated decline in PA from adolescence to adulthood could slow down the atherosclerotic process at an early age in healthy people.

Our observations that the mean exposure to moderate-and-vigorous PA was associated with lower levels of carotid stiffness are supported by a study from the Amsterdam Growth and Health Longitudinal Study (AGHLS).[7] They observed that participants with stiffest carotid arteries reported less time on vigorous and more light-to-moderate self-reported PA, compared with the participants with less stiff carotid arteries. Our findings are in contrast to a smaller study based on AGHLS data by Ferreira *et al*,[10] who did not observe an association between changes in the total PA level from adolescence to adulthood and arterial stiffness in adulthood. However, the latter observations were based on a small sample and used subjectively assessed PA. Experimental studies in young healthy people[20 21] and elderly[22] indicate that moderate-and-vigorous exercise can decrease both peripheral and central arterial stiffness. Therefore it is possible the high intensity PA is required to prevent arterial stiffness. Thus, the discrepancy to the study by Ferreira *et al* could be ascribed to the use of mean PA levels instead of PA intensity. The effect sizes observed across measures of arterial stiffness, although statistically significant, were small and thus clinical significance is uncertain. In a recent study, Yang *et al*[2] reported that a 1-SD increase in arterial stiffness was associated with a 15–50% increased risk of stroke. Thus, even small reductions in carotid arterial stiffness may correspond to important health gains later in life.

As cIMT is a consistent independent predictor of future CVD events in adults, it was surprising to observe that the participants accumulating the most moderate-and-vigorous PA across youth had the largest cIMT. Our observations are supported by earlier cross-sectional associations where cIMT was increased in normotensive exercise trained subjects compared with sedentary subjects.[23 24] However, our observations are in contrast to previous longitudinal observations from two Finnish cohorts. Juonala *et al*[8] observed an inverse association between self-reported childhood PA and adult cIMT change over a 6-year period among adults Finns. Pahkala *et al*[9] observed an inverse association between increases in and maintenance of a high leisure time PA and slow aortic IMT progression from age 13 to 17. cIMT not only reflects early atherosclerosis but also non-atherosclerotic compensatory enlargement to other exposures.[25] Therefore, it is possible that other exposures, such as increased exercise, are likely to explain our observation. Exercise training increases shear stress and mechanical strain acutely.[26 27] The increased mechanical loading during long-term exposures to high-intense exercise induces an increase in the smooth muscle cell cross-sectional area, thus cIMT, in order for the artery to maintain 'tensional homeostasis'.[28] This is consistent with our findings as only the participants with the highest mean exposure to moderate-and-vigorous PA, but not participants with the highest increase, displayed a higher cIMT. Further studies are needed to investigate the effect of PA on cIMT in young individuals.

An important strength of the current study was that we were able to control for important confounding factors, and we used objectively measured PA at baseline and follow-up. There are also some limitations to the study. First, the drop-out and participants with incomplete data could have introduced a selection bias. Selection bias only occurs if associations of PA intensity and the levels of subclinical atherosclerosis are different between participants, and individuals who have missing data or are lost to follow-up. Excluded subjects did not differ from the participants in their baseline levels of exposure variables. We further performed a post hoc sensitivity analysis, where the estimates were weighed according to the probability of participating at follow-up given they participated at baseline (using the *pweight* option in Stata). The estimates were weighed according to overweight, parental educational status and smoking status at baseline. This did not change the effect sizes materially. Therefore, we do not suspect that selection bias explains our observations. Second, the activity monitor does not capture activities such as bicycling, weight bearing activities and swimming very well. Third, a measurement period of ~4 days might not represent the participant's true PA activity level. This would introduce a random error thus underestimate the association

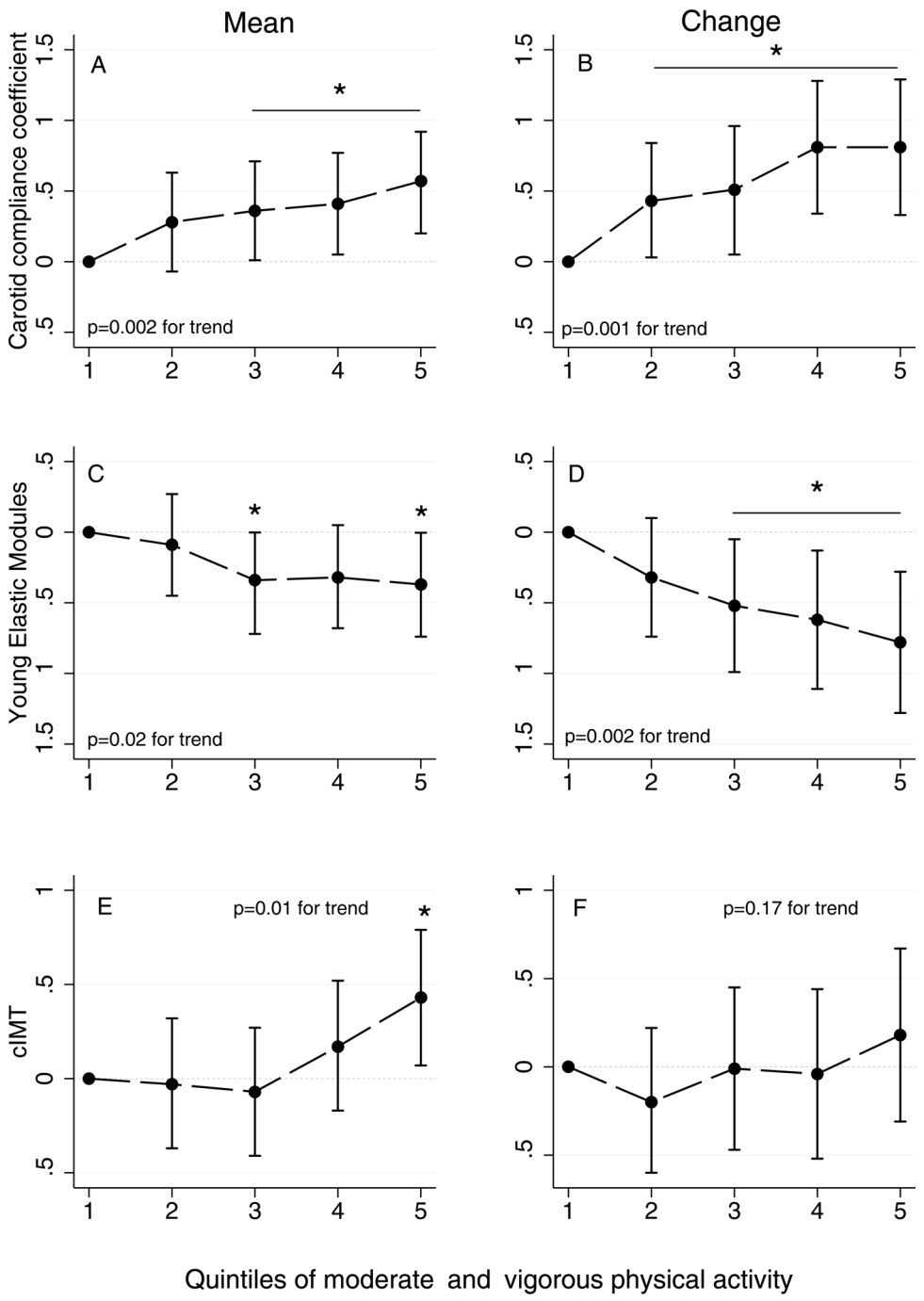

Figure 1 Standardised difference between the lowest (1) and the higher quartiles of mean (left colon) and change (right colon) in moderate-and-vigorous physical activity (95% CI) of carotid compliance (A and B), Young's elastic modules (C and D) and carotid intima-media thickness (cIMT) (E and F).

Table 4 Mean exposure to or change in moderate-and-vigorous physical activity minutes across quintiles of exposure

Quintile	Mean	Change
1	6.9 (2.3)	−36.6 (17.9)
2	13.5 (1.8)	−14.6 (3.8)
3	19.1 (1.4)	−4.7 (1.9)
4	25.5 (2.4)	3.2 (2.9)
5	38.5 (9.1)	20.5 (14.0)

Data are means (SD).

between intensity and outcome, but not introduce a bias. Furthermore, we have shown that the Actigraph models AM7164 and the GT models applied in this study are not fully compatible (45). However, this problem is primarily evident at lower intensities. As we focused on higher intensities and the different models were not mixed within waves of follow-up, we do not suspect this to affect the associations. However, it might have underestimated the absolute change of PA. Third, as we used brachial blood pressure for calculation of arterial stiffness the pulse-pressure might be overestimated and thus overestimate our measure of arterial stiffness (42). This bias would be

random as the cohort was homogeneous according to age. Furthermore, pulse-pressure amplifications may differ across gender and CVD risk (43, 44) this would potentially introduce a differential bias if the size of the amplification is related to PA level and confounding factors in this population of healthy young adults. Finally, the observational design of our study precludes us from excluding the possibility of unknown or residual confounding, such as crude measures of diet.

CONCLUSION

In conclusion, high mean exposure to and increases in moderate-and-vigorous PA from adolescence to adulthood were independently associated with lower levels of carotid arterial stiffness and metabolic CVD risk in adulthood. The mean exposure to moderate-and-vigorous PA was positively associated with cIMT in adulthood. Further studies are needed to fully understand the effects of high intense PA on carotid arterial remodelling in young healthy individuals. Our observations suggest that emphasis should be put on reducing the age-related decline and increasing the mean exposure to moderate-and-vigorous PA from adolescence to adulthood.

What are the new findings?

- Slowing the rate of the age-related decline in moderate-and-vigorous physical activity (PA) could slow down the development of atherosclerosis in healthy young individuals.
- Maintaining a high level of moderate-and-vigorous PA could slow down the development of atherosclerosis in healthy young individuals.
- Our observations suggest that slowing the rate of decline or increasing the accumulated PA level could prevent atherosclerosis independent of adiposity, systolic blood pressure, demographic-, personal- and lifestyle factors including TV viewing.

How might it impact clinical practice in the near future?

- Policies should focus on slowing the rate of decline of PA from adolescence to adulthood in the young healthy population.
- Clinicians should continuously throughout youth promote increasing a high PA level alongside promoting a reduction of TV viewing and unhealthy foods in the young healthy population.
- Clinicians could use the moderate-and-vigorous PA level as a screening tool for high cardiovascular disease risk in healthy young people.

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Contributors MR-L conceptualised the study, collected data, carried out the initial analyses, drafted the manuscript and approved the final manuscript as submitted. AG conceptualised the study, collected data, reviewed and revised the manuscript and approved the final manuscript as submitted. PLK coordinated, collected data and supervised data collection at baseline (2003), interpreted observations, critically reviewed the manuscript for important intellectual content, and approved the final manuscript as submitted. KF designed the study, coordinated and supervised data collection at baseline (1997), interpreted observations, critically reviewed the manuscript for important intellectual content and approved the final manuscript as submitted. LBA designed the study, interpreted observations, supervised data collection at follow-up (2009), critically reviewed the manuscript for important intellectual content and approved the final manuscript as submitted.

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