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ORIGINAL ARTICLE

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Longitudinal physical activity patterns and the development of cardiometabolic risk factors during adolescence

Tuula Aira[1] | **Sami Petteri Kokko**[1] | **Olli Juhani Heinonen**[2] | **Raija Korpelainen**[3,4,5] | **Jimi Kotkajuuri**[6] | **Jari Parkkari**[7,8] | **Kai Savonen**[9,10] | **Kerttu Toivo**[7] | **Arja Uusitalo**[11,12] | **Maarit Valtonen**[13] | **Jari Villberg**[1] | **Onni Niemelä**[14] | **Henri Vähä-Ypyä**[15] | **Tommi Vasankari**[15,16]

[1]Faculty of Sport and Health Sciences, Research Centre for Health Promotion, University of Jyväskylä, Jyväskylä, Finland

[2]Paavo Nurmi Centre & Unit for Health and Physical Activity, University of Turku, Turku, Finland

[3]Medical Research Center (MRC), University of Oulu and University Hospital of Oulu, Oulu, Finland

[4]Department of Sports and Exercise Medicine, Oulu Deaconess Institute Foundation sr., Oulu, Finland

[5]Center for Life Course Health Research, University of Oulu, Oulu, Finland

[6]Department of Mathematics and Statistics, University of Jyväskylä, Jyväskylä, Finland

[7]Tampere Research Center of Sports Medicine, Tampere, Finland

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

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

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

[11]Clinic for Sports and Exercise Medicine, Foundation for Sports and Exercise Medicine, Helsinki, Finland

[12]Department of Sports and Exercise Medicine, Clinicum, University of Helsinki, Helsinki, Finland

[13]Research Institute for Olympic Sports, Jyväskylä Finland, Jyväskylä, Finland

[14]Medical Research Unit and Department of Laboratory Medicine, Seinäjoki Central Hospital and University of Tampere, Tampere, Finland

[15]UKK Institute of Health Promotion Research, Tampere, Finland

[16]Faculty of Medicine and Health Technology, Tampere University, Tampere, Finland

Correspondence
Tuula Aira, Faculty of Sports and Health Sciences, University of Jyväskylä, PL 35, FI-40014 Jyväskylä, Finland.
Email: tuula.m.aira@jyu.fi

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Purpose: To examine the associations between longitudinal physical activity (PA) patterns and the development of cardiometabolic risk factors from adolescence to young adulthood.

Methods: This cohort study encompassed 250 participants recruited from sports clubs and schools, and examined at mean age 15 and 19. Device-measured moderate-to-vigorous PA was grouped into five patterns (via a data-driven method, using *inactivity maintainers* as a reference). The outcomes were: *glucose*, *insulin*, *homeostasis model assessment for insulin resistance* (*HOMA-IR*), *total cholesterol*, *HDL and LDL cholesterol*, *triglycerides*, *blood pressure*, and *body mass index* (*BMI*). Linear growth curve models were applied with adjustment for sex,

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age, fruit/vegetable consumption, cigarette/snuff use, and change in the device wear-time.

Results: Insulin and BMI increased among *decreasers from moderate to low PA* (β for insulin 0.23, 95% CI 0.03–0.46; β for BMI 0.90; CI 0.02–1.78). The concentration of HDL cholesterol decreased (β −0.18, CI −0.31 to −0.05) and that of glucose increased (β 0.18, CI 0.02–0.35) among *decreasers from high to moderate PA*. By contrast, among *increasers*, blood pressure declined (systolic β −6.43, CI −12.16 to −0.70; diastolic β −6.72, CI −11.03 to −2.41).

Conclusions: Already during the transition to young adulthood, changes in PA are associated with changes in cardiometabolic risk factors. Favorable blood pressure changes were found among PA *increasers*. Unfavorable changes in BMI, insulin, glucose, and HDL cholesterol were found in groups with decreasing PA. The changes were dependent on the baseline PA and the magnitude of the PA decline.

KEYWORDS

accelerometry, adolescent, blood pressure, body mass index, insulin resistance, young adults

1 | INTRODUCTION

Physical activity (PA) decreases the risk for chronic diseases such as cardiovascular diseases and type 2 diabetes,[1,2] which are among the leading causes of death worldwide.[3] Although the full cardiometabolic benefits of PA appear over the course of decades, some health outcomes have been observed at childhood and adolescence.[4–7] However, the latter observations are based on a limited number of studies using heterogeneous methods.[4,6]

The associations between PA and cardiometabolic outcomes in prospective studies are traditionally examined either by analyzing (i) *baseline PA* in relation to a follow-up cardiometabolic outcome or change in the outcome, or (ii) the association between a *change in PA* and a change in the cardiometabolic outcome.[4,5,8] However, neither of these approaches take account of the fact that distinct patterns of PA evolution might result in different cardiometabolic outcomes, depending on the *baseline PA level*, and also on the *magnitude of the change in PA*. Thus, there is a need to analyze differences in cardiometabolic risk between subgroups of adolescents with different PA changes over time (also, see Ref.[9]).

A growing number of studies have identified distinct longitudinal PA patterns (or trajectories) during adolescence, identifying, for example, groups maintaining high or low activity, and patterns of decreasing and increasing activity (as we did in our previous study when we created the PA groups used in the current study[10]).[11,12] Only a few have gone further and focused on possible differences in health outcomes between groups representing different PA patterns.[13–18] Some of these studies have used objective methods for PA assessment[15–17] or applied data-driven methods (algorithm- or model-based) to identify distinct PA patterns.[14,15,17] PA assessment via accelerometry is recommended over self-reported measurements, since it enables more reliable determination of e.g. the duration and intensity of the PA.[19] At the same time, data-driven methods are better able to identify genuinely heterogeneous PA patterns than are subjective methods (such as splitting into quartiles or using pre-determined levels of PA). To our knowledge there are no studies on simultaneous cardiometabolic risk factors between various PA patterns, with PA assessment conducted via accelerometry, and with patterns grouped via a data-driven method. Existing studies fulfilling the methodological requirements mentioned above have examined the association of PA patterns with the accumulation of body adiposity[17] and obesity development,[15] but blood biomarkers have not been studied in a similar manner. It should also be noted that the follow-up periods used in previous studies have varied: Oh et al.[17] followed adolescents between the ages 15 and 23, while the study of Kwon et al.[15] set the baseline at childhood (with follow-up from 5 to 19 years).

Our aim was to examine how longitudinal PA patterns (based on moderate-to-vigorous PA (MVPA) and exhibited by *inactivity maintainers*, *activity maintainers*, *decreasers from moderate to low PA*, *decreasers from high to moderate PA*, and *increasers*)[10] were associated with the evolution of cardiometabolic risk factors (blood lipids, glucose metabolism, blood pressure, and body mass index (BMI)), from adolescence (mean age 15) to young adulthood (mean age

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19). The *inactivity maintainers* group was used as a reference. We hypothesized that decreasing PA is positively and increasing PA inversely associated with cardiovascular risk factor development, at least in terms of some risk factors.

2 | MATERIALS AND METHODS

2.1 | Study design and participants

This observational cohort study was part of the Health Promoting Sports Club study, conducted in 2013–2014 and 2017–2018.[20] At baseline, the participants (mean age 15 years) were recruited from sports clubs and schools in six large cities and surrounding communities, in different parts of Finland (Figure 1). The sports clubs represented the 10 most popular team and individual sports disciplines: basketball, floorball, ice hockey, soccer, cross-country skiing, gymnastics, orienteering, skating, swimming, and track and field (for more details of the study protocol see Ref.[20,21]).

The HPSC study was conducted in accordance with the Declaration of Helsinki, and received ethical approval from the Ethics Committee of the Healthcare District of Central Finland 20.12.2012 (23 U/2012), 15.12.2016. Written consent was required from the participants, and also from a guardian when the participant was aged under 18.

The participants first took part in electronic surveys on current health behaviors and health status. On the basis of power calculations, randomly selected participants were also invited to participate in pre-participation screening in one of the six national Centres of Excellence in Sports and Exercise Medicine. The pre-participation screening included screening by a physician and a fasting blood sample.[20] Instructions were also given on a hip-worn accelerometer, which the participants wore for 7 consecutive days during waking hours, except when bathing or during water activities. Surveys, clinical examinations and PA measurements were all conducted during the same season of the year.

Nearly two-thirds (65% of the eligible participants) of the baseline participants ($n = 583$) also took part in the study in 2017–2018 ($n = 371$) with similar measurements to those at baseline. The mean difference between the baseline and follow-up measurements was 3.8 years (SD 0.4, min 2.8, max. 5.1). In total, 254 participants provided valid accelerometer data and written consent for both measurement points (for more details, see Ref.[10]). Participants who were diagnosed as patients with type 1 diabetes were excluded ($n = 4$) as were also swimmers, who were not able to use the accelerometer during their swimming training (Figure 1). Out of those 250 participants, 239–250 at baseline and 242–250 at follow-up had valid data on cardiometabolic risk outcomes.

2.2 | Physical activity

Physical activity was measured via a Hookie accelerometer (AM20 Activity meter, Hookie Technologies Ltd.),

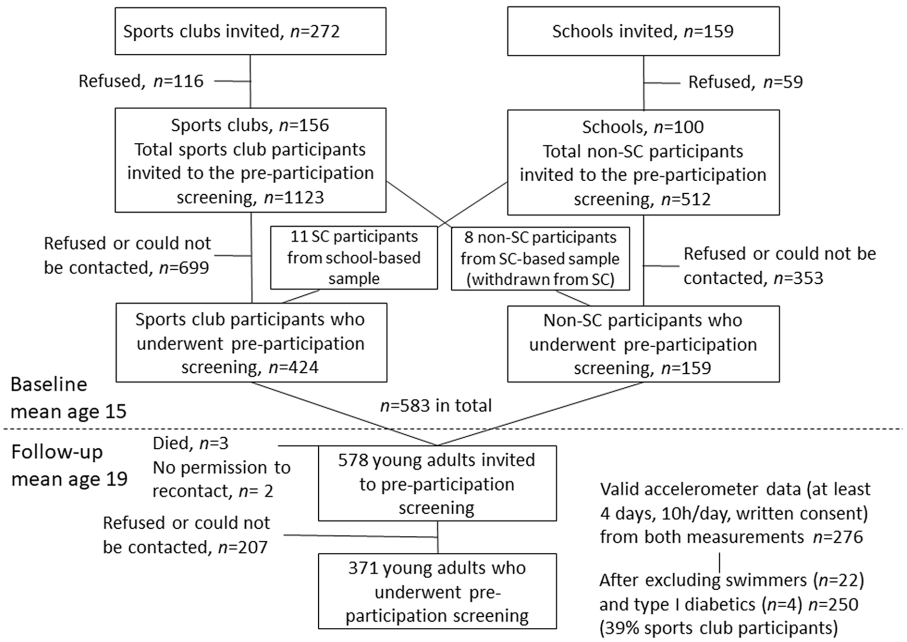

FIGURE 1 Flow chart of the data collection and study sample.

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which collected and stored tri-axial data as actual g-units with a 100 Hz sampling frequency. The Hookie accelerometer has been shown to be a valid measurement tool for both adolescents[22] and adults.[23] The data were analyzed in units of 6 s duration. Continuous quiescent time exceeding 30 min was taken to indicate nonwear of the device, as recommended also by others.[24] The PA analysis was based on mean amplitude deviation analyses (MAD), calculated from a resultant tri-axial raw acceleration signal, and converted to metabolic equivalents (METs).[23,25] The epoch-wise MET values were further smoothed by calculating the 1 min exponential moving average MET value for each epoch time point. Moderate-to-vigorous PA (MVPA) was defined as an MET value of ≥3.0.[22] For a valid measurement, at least 10 h per day and at least 4 days per week were required, as was also informed consent for the measurement.

The formulation of the longitudinal PA patterns has previously been described in detail.[10] Based on two valid MVPA measurement periods, a k-means algorithm for longitudinal data (KmL)[26] was applied to identify homogenous subgroups, that is, clusters that were as heterogeneous as possible in comparison with each other. The PA patterns were: *inactivity maintainers*, *activity maintainers*, *decreasers from moderate (to low) PA*, *decreasers from high (to moderate) PA*, *increasers* (Figure 2).

2.3 | Clinical examination

In the pre-participation screening, trained personnel (with a specialization in healthcare) measured the height and weight (wearing light clothes without shoes) of the participants to the nearest 0.5 cm and 0.1 kg. BMI was calculated as weight (kg)/height $(m)^2$. The participant was seated, and after at least 5 min of rest, blood pressure was measured via a validated, cuff-style oscillometric (automated) device.[27] A second measurement was taken at an interval of 1 min. A third measurement was obtained if there was more than 10 mm Hg difference in systolic or diastolic pressure between the first and second measurement. The average of two (or three) measurements was used in the analyses.

2.4 | Laboratory analyses

Venous blood samples were taken after participants had fasted for at least 10 h. Serum and plasma were separated by centrifugation (2000 *g* for 10 min) and stored at −75°C prior to analysis. Concentrations of fasting plasma glucose, serum total cholesterol, high- and low-density cholesterol (HDL, LDL), and triglyceride were analyzed using standard enzymatic methods on a Cobas c702 clinical chemistry analyzer (Roche Diagnostics). Serum insulin concentrations were determined using electrochemiluminescence technology on Cobas e801, according to the instructions of the manufacturer (Roche Diagnostics). All measurements were carried out in an SFS-EN ISO 15189:2013 accredited laboratory.

The homeostasis model assessment for insulin resistance (HOMA-IR) was assessed via the formula: (fasting serum insulin (mU/L)×fasting plasma glucose (mmol/L))/22.5.[28]

2.5 | Other variables

Participants reported their smoking, alcohol consumption, snuff use, and eating behavior in the electronic survey. Combined information covering at least weekly cigarette and/or snuff use was applied in the analysis (yes/no). Because fruit and vegetable intake is associated with a reduced risk for cardiovascular disease[29] and reflects healthy eating overall, a fruit and vegetable index[30] was applied in the analyses. The index ranged from 0 to 14, where value 0 represented no fruit and vegetable consumption and value 14 consumption of both fruit and vegetables at least once a day (for more details, see Ref.[30,31]). The assessment of family affluence was based on a previously validated Family Affluence Scale score, which was calculated from participants' answers to questions on four common consumption indicators of material deprivation (cars, bedrooms, computers, vacations).[32]

2.6 | Statistical analyses

To characterize the PA patterns cross-sectionally,[33] the Kruskal–Wallis test (with post hoc Dunn's test, adjusted by the Bonferroni correction for multiple tests) and the Chi-square test were used to reveal the differences in mean values and in frequencies. The significance of the change over time was calculated by the Wilcoxon signed rank test. The independent samples *t*-test and Mann Whitney U test were performed in the loss-to-follow up analysis. All the descriptive data analyses were conducted using SPSS version 26.

To further study the differences between PA patterns with regard to cardiometabolic risk factors, linear growth curve analyses (nlme package in R Studio; version 4.0.3)[34] were conducted. The baseline level and the slope (rate of change over time) of each cardiometabolic risk factor were explained by (in addition to the PA pattern) sex, age, fruit and vegetable consumption, snuff and/or cigarette use, plus the change in the device wear-time between the

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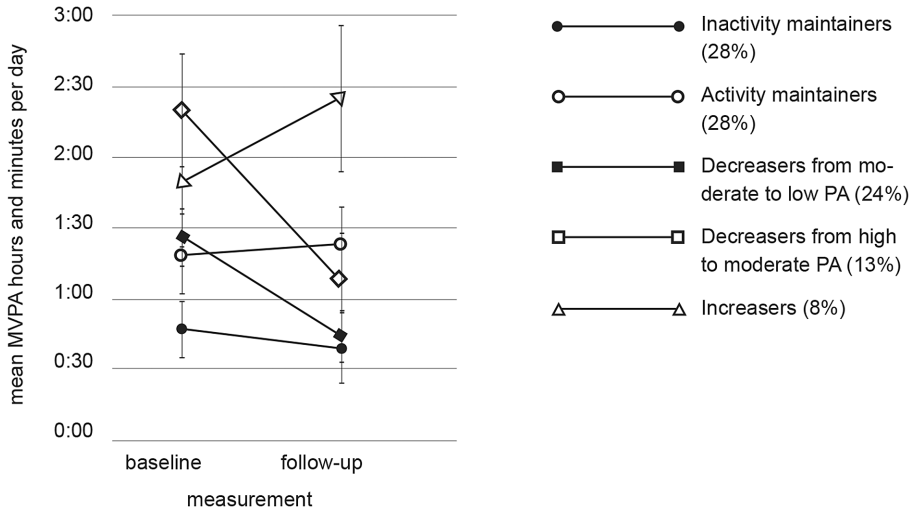

FIGURE 2 Longitudinal physical activity patterns (the data are mean and standard deviation).[10]

baseline and follow-up measurements (to eliminate the effect on the results of differences in device usage time). The change in cardiometabolic risk factors over time by PA pattern was also examined (i.e., with time nested within participant as a random effect). The growth curve analysis accommodates missing data by creating estimates based on all available data for each participant. Moreover, the baseline values of the outcome are reflected in the intercept of the model, ensuring correction of any possible regression to the mean.

The outcome variables triglyceride, HOMA-IR, and insulin were log-transformed using $\log(x+1)$ transformation to improve the normal distribution of the residuals. A multiple of the identity positive-definite matrix (PdIdent) was set as a correlation structure in all the models for the same reason. Age was centred at 15, with age 15 corresponding to time = 0. Additional analyses were performed by adjusting the cardiometabolic risk factors also for BMI (centred) and for the season of the year when measured, in order to assess whether the associations were independent of BMI and seasonal variation. The significance level was set at $p < 0.05$ in all the statistical tests.

3 | RESULTS

The study sample was 60% female and at baseline two thirds (68%) of the participants lived in families with high affluence (Table 1). At follow-up, 64% of the young adults were studying (39% in a general upper secondary school, 9% in a vocational school, and 16% in a university or university of applied sciences), while 20% had entered working life. Table 1 illustrates more characteristics of the participants by PA patterns, including health behaviors, and the amount of MVPA.

The study participants and those lost to follow-up at the second measurement did not differ in any of the studied baseline cardiometabolic risk factors, or in terms of MVPA or family affluence (see Table S1). However, males, and those who reported lower school achievement, were more likely not to participate in the post-measurement (see Table S1). Table 2 presents the differences in cardiometabolic risk factors between PA patterns at baseline, at follow-up, and in development over time.

3.1 | Growth curve analyses

To further study the differences in cardiometabolic risk factors between PA patterns, growth curve analyses were conducted with adjustment for sex, age, fruit and vegetable consumption, snuff and/or cigarette use, plus the change in the device wear-time (see Table 3, Table S2 with confidence intervals).

3.2 | Baseline

The *decreasers from moderate to low PA* had lower baseline values in insulin, HOMA-IR and BMI as compared to the *inactivity maintainers* (the reference group) (Table 3, Figure 3). The *decreasers from high to moderate PA* had lower glucose, insulin and HOMA-IR levels, and higher HDL cholesterol at baseline as compared to the corresponding levels among the *inactivity maintainers*. There was no significant difference in the baseline levels of any of the studied cardiometabolic risk parameters between

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TABLE 1 Characteristics of the study participants by physical activity patterns.

	All	Inactivity maintainers (A)	Activity maintainers (B)	Decreasers from moderate to low PA (C)	Decreasers from high to moderate PA (D)	Increasers (E)	*p*
***n* (%)**	250 (100)	69 (28)	69 (28)	60 (24)	32 (13)	20 (8)	
Age, mean years (SD)							
Baseline	15.5 (0.6)	15.6 (0.5)	15.5 (0.6)	15.3 (0.5)	15.5 (0.5)	15.4 (0.5)	0.138
Follow-up	19.4 (0.6)	19.4 (0.7)	19.4 (0.7)	19.2 (0.7)	19.5 (0.6)	19.5 (0.5)	0.130
Males, *n* (%)	100 (40)	19 (28)	24 (35)	22 (37)	26 (81)	9 (45)	<0.001
MVPA mean/day: hours and minutes (SD)							
Baseline	1:22 (0:33)	0:47 (0:12)	1:19 (0:17)	1:26 (0:12)	2:20 (0:24)	1:50 (0:28)	A < B–E <0.001, B < D <0.001, B < E 0.004, C < D <0.001
Follow-up	1:05 (0:34)	0:39 (0:15)	1:24 (0:15)	0:44 (0:11)	1:07 (0:21)	2:25 (0:31)	A < B,D,E <0.001, B > D 0.041, B < E 0.047, C < B,D,E <0.001, D < E <0.001
MVPA change, mean minutes (SD)	−16.5 (37.7)	−7.9 (19.4)	5.2 (22.7)	−40.9 (17.3)	−67.8 (29.7)	34.7 (44.3)	
Education & employment status, *n* (%)							
Follow-up							
Studying	159 (64)	40 (59)	46 (67)	44 (73)	18 (56)	11 (58)	0.768[a]
Working	50 (20)	16 (24)	13 (19)	9 (15)	8 (25)	4 (21)	
Other[b]	39 (16)	12 (17)	10 (15)	7 (12)	6 (19)	4 (21)	
Living with parents, *n* (%)							
Follow-up	170 (68)	48 (70)	47 (68)	38 (63)	23 (72)	14 (74)	0.885
Living in urban area, *n* (%)							
Baseline	161 (66)	35 (53)	49 (71)	42 (70)	21 (70)	14 (70)	0.169
Follow-up	197 (79)	51 (74)	57 (83)	49 (82)	27 (84)	13 (68)	0.455
Self-reported school grade average: good to excellent (grades 8–10 in grading 4–10), *n* (%)							
Baseline	203 (83)	51 (77)	57 (83)	51 (85)	27 (90)	17 (85)	0.643
High family affluence, *n* (%)							
Baseline	152 (62)	36 (55)	44 (64)	38 (63)	22 (73)	12 (60)	0.505
Reached menarche or Tanner stage P4, *n* (%)							
Baseline	238 (95)	67 (97)	66 (96)	56 (93)	29 (91)	20 (100)	0.529

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TABLE 1 (Continued)

	All	Inactivity maintainers (A)	Activity maintainers (B)	Decreasers from moderate to low PA (C)	Decreasers from high to moderate PA (D)	Increasers (E)	*p*
Snuff and/or cigarette use at least weekly, *n* (%)							
Baseline	7 (3)	4 (6)	0	3 (5)	0	0	0.154
Follow-up	24 (10)	8 (12)	4 (6)	8 (13)	3 (9)	1 (5)	0.626
Alcohol consumption at least weekly, *n* (%)							
Baseline	1 (0.4)	0	0	1 (2)	0	0	
Follow-up	20 (8)	5 (7)	6 (9)	5 (8)	3 (9)	1 (5)	0.995
Fruit & vegetable consumption index, mean (SD)							
Baseline	9.1 (4.3)	7.9 (4.6)	9.9 (3.9)	8.6 (4.4)	10.6 (3.7)	10.0 (4.2)	ns
Follow-up	9.2 (4.0)	7.5 (4.2)	10.6 (3.6)	8.6 (4.0)	10.8 (3.0)	10.0 (3.6)	A < B <0.001, A < D 0.003, C < D 0.039

Note: *p*-values have been assessed using Chi-square test or Fisher's exact test (in cases of sparse data) for categorical variables. The Kruskal-Wallis test was used in analyzing differences in mean values between PA patterns cross-sectionally (with post hoc Dunn's test adjusted by the Bonferroni correction for multiple tests).

Abbreviation: PA, physical activity.

[a]The *p*-value represents the difference between the groups in bold font (studying, working, other) and PA patterns.

[b]For example for those who are unemployed, temporarily laid off, or doing military service.

the *activity maintainers* and the reference group, or between the *increasers* and the reference group (Table 3).

3.3 | Change over time

The development over time in HOMA-IR and in insulin among the *decreasers from moderate to low PA* differed from the corresponding development among the *inactivity maintainers*, hence showing unchanged HOMA-IR (vs. a decrease among the inactivity maintainers), and increased insulin (vs. a decrease among the inactivity maintainers; see Table 3, Figure 3). BMI increased among the *decreasers from moderate to low PA*, whereas BMI was essentially unchanged (stable) among the *inactivity maintainers*.

The decrease over time in systolic and diastolic blood pressure among the *increasers* differed from the fairly stable trend among the *inactivity maintainers* (Table 3, Figure 3). There was no difference in the development of the cardiometabolic risk factors over time between the *activity maintainers* and the *inactivity maintainers*.

3.4 | Adjustment for BMI and the measurement season

After adjusting the analyses for BMI, the results mainly remained unchanged. However, some associations no longer reached statistical significance, namely HOMA-IR and insulin at baseline among the *decreasers from high to moderate PA*, and the rate of change in insulin among the *decreasers from moderate to low PA* (see Table S3). Adjustment for the measurement season did not alter the main results (Table S4).

4 | DISCUSSION

This longitudinal cohort study indicated changes in cardiometabolic risk from adolescence to young adulthood (from age 15 to 19) that were associated with changes in PA. The results also highlighted the importance of the baseline level and of the magnitude of the PA change. Unfavorable changes in insulin and BMI occurred among *decreasers from moderate to low PA*, as compared to those who maintained a relatively low level of PA over time. There were also unfavorable changes in glucose and HDL cholesterol among *decreasers from high to moderate PA*. By contrast, among *increasers*, there were favorable changes in systolic and diastolic blood pressure, both of which decreased over time.

The changes in cardiometabolic risk factors were relatively small, suggesting no immediate cause for

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TABLE 2 Cardiometabolic risk factors at baseline and follow-up across PA patterns (mean (SD)).

	All (women $n = 144–150$, men $n = 95–100$)		Inactivity maintainers (A)		Activity maintainers (B)		Decreasers from moderate to low PA (C)		Decreasers from high to moderate PA (D)		Increasers (E)		p (adj.) for differences between PA patterns	
	Baseline	**Follow-up**	**Baseline**	**Follow-up**	**Baseline**	**Follow-up**	**Baseline**	**Follow-up**	**Baseline**	**Follow-up**	**Baseline**	**Follow-up**	**Baseline**	**Follow-up**
Glucose (mmol/L)	**4.94** (0.33)	**4.77*** ** (0.38)	**4.95** (0.28)	**4.68*** ** (0.33)	**4.93** (0.40)	**4.78*** (0.40)	**4.93** (0.32)	**4.79*** (0.38)	4.95 (0.29)	4.87 (0.40)	4.97 (0.25)	4.78 (0.36)	ns	ns
Insulin (mU/L)	**11.42** (5.31)	**9.72*** ** (4.48)	**13.33** (6.55)	**10.56*** ** (5.23)	**11.42** (5.37)	**9.57*** (5.23)	10.73 (4.00)	10.50 (4.98)	**9.63** (3.66)	**7.82**** (2.38)	9.70 (4.01)	8.21 (4.40)	**D < A***	ns
HOMA-IR	**2.53** (1.25)	**2.08*** ** (1.02)	**2.95** (1.53)	**2.22*** ** (1.15)	**2.53** (1.28)	**2.05**** (0.84)	2.37 (0.95)	2.27 (1.23)	**2.12** (0.82)	**1.69**** (0.52)	2.17 (0.98)	1.75 (0.93)	**D < A***	ns
Cholesterol (mmol/L)	3.83 (0.67)	3.87 (0.79)	3.86 (0.70)	3.88 (0.76)	3.95 (0.76)	3.97 (0.83)	3.82 (0.58)	3.98 (0.82)	3.57 (0.59)	3.49 (0.73)	3.72 (0.53)	3.77 (0.58)	ns	**D < B,C***
LDL cholesterol (mmol/L)	1.98 (0.58)	1.95 (0.64)	1.99 (0.62)	1.91 (0.61)	2.09 (0.64)	2.03 (0.64)	1.94 (0.54)	2.03 (0.71)	1.84 (0.51)	1.81 (0.65)	1.86 (0.41)	1.84 (0.50)	ns	ns
HDL cholesterol (mmol/L)	1.46 (0.30)	1.50 (0.35)	1.46 (0.38)	1.53 (0.38)	1.46 (0.32)	1.54 (0.36)	1.49 (0.28)	1.50 (0.33)	**1.41** (0.26)	**1.31**** (0.29)	1.47 (0.33)	1.59 (0.32)	ns	**D < A,B,E***
Triglycerides (mmol/L)	**0.85** (0.40)	**0.92*** (0.45)	0.92 (0.44)	0.98 (0.45)	0.87 (0.40)	0.89 (0.51)	**0.79** (0.31)	**0.99**** (0.44)	0.72 (0.26)	0.82 (0.82)	0.87 (0.87)	0.77 (0.31)	ns	ns
Systolic blood pressure (mm Hg)	**117.1** (10.89)	**121.2*** ** (11.89)	**116.4** (11.50)	**121.3*** ** (12.48)	116.6 (10.19)	118.5 (11.31)	**116.0** (10.69)	**121.9*** ** (11.78)	**120.3** (11.31)	**127.4**** (10.65)	119.4 (10.80)	118.4 (11.01)	ns	**A,E < D*** **B < D****
Diastolic blood pressure (mm Hg)	**67.4** (7.12)	**72.6*** ** (7.94)	**68.8** (6.17)	**74.4*** ** (8.60)	**67.5** (7.12)	**71.9*** ** (7.09)	**66.3** (6.77)	**73.7*** ** (7.45)	**66.2** (9.09)	**72.9*** ** (8.34)	66.7 (7.54)	65.7 (5.43)	ns	**E < A,C*** ** **E < B**** **E < D***
Body mass index (kg/m^2)	**21.1** (2.82)	**23.2*** ** (3.32)	**21.8** (3.05)	**23.6*** ** (3.91)	**21.5** (2.90)	**23.4*** ** (3.41)	**20.4** (2.83)	**23.1*** ** (3.32)	**20.8** (2.08)	**23.0*** ** (1.86)	**20.5** (2.06)	**22.1*** ** (2.37)	**C < A***	ns

Note: The Kruskal–Wallis test was used in analyzing the differences in mean values between physical activity patterns cross-sectionally (with post hoc Dunn's test adjusted by the Bonferroni correction for multiple tests), and the Wilcoxon Signed Rank test was used to analyze differences over time. Numbers in bold indicate significant differences over time, and letters in bold (in the last two columns) indicate significant difference between physical activity patterns. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Abbreviations: HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment for insulin resistance; LDL, low-density lipoprotein; PA, physical activity.

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TABLE 3 Growth curve models for cardiometabolic risk factors (assessing baseline level and change over time).

	Glucose (mmol/L)	Insulin (mU/L)[a]	HOMA-IR[a]	Cholesterol (mmol/L)	LDL cholesterol (mmol/L)	HDL cholesterol (mmol/L)	Triglycerides (mmol/L)[a]	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Body mass index
	β	*β*	*β*	*β*	*β*	*β*	*β*	*β*	*β*	*β*
Baseline level										
Intercept	**5.22*** **	**16.07*** **	**1.48*** **	**3.77*** **	**2.10*** **	**1.23*** **	**0.89*** **	**124.13*** **	**71.28*** **	**21.39*** **
PA pattern B	−0.12	−0.21	−0.19	0.10	0.09	0.01	0.005	1.82	0.06	−0.36
PA pattern C	−0.15	**−0.33** **	**−0.25** **	−0.14	−0.12	0.10	−0.12	−2.38	−3.71	**−2.13** **
PA pattern D	**−0.31***	**−0.29***	**−0.24***	0.05	−0.08	**0.24***	−0.10	−4.20	−3.00	−1.39
PA pattern E	−0.14	−0.29	−0.18	−0.19	−0.22	−0.02	0.04	6.71	4.55	−0.76
Sex (female)	**−0.23*** **	0.04	−0.01	**0.33*** **	0.13	**0.22*** **	0.01	**−12.02*** **	−1.21	−0.23
Age (15 years)	−0.04	−0.003	−0.002	0.06	0.03	0.01	0.02	1.55	**2.03** **	**0.54***
Rate of change										
Time	−0.09	−0.20	−0.18	−0.19	−0.17	0.04	−0.05	−0.85	−2.14	−0.22
Time by PA pattern B	0.10	0.11	0.11	−0.001	0.02	0.002	−0.03	−2.91	−1.05	0.14
Time by PA pattern C	0.11	**0.23***	**0.18***	0.13	0.13	−0.06	0.07	0.81	1.77	**0.90***
Time by PA pattern D	**0.18***	0.09	0.10	−0.15	0.03	**−0.18** **	0.01	1.59	0.60	0.31
Time by PA pattern E	0.13	0.08	0.03	0.08	0.09	0.06	−0.07	**−6.43***	**−6.72** **	−0.38

Note: Linear growth curve models, adjusted for change in the device wear-time, fruit and vegetable consumption, and weekly cigarette and/or snuff use. The intercept in the models represents the baseline level (mean) of the risk factor in the reference group (inactivity maintainers), when all the exposure variables are 0—in other words, among men, at age 15 (since age was centred), when fruit and vegetable consumption and weekly cigarette/snuff use were 0. *Time* represents the impact of time (mean slope) on the risk factors between baseline and follow-up in the reference group (inactivity maintainers), and *time by PA pattern* (B,C,D,E) the corresponding rate of change in the other PA patterns as compared to the rate of change in the reference group.

Abbreviations: B, activity maintainers; BP, blood pressure; C, decreasers from moderate to low PA; D, decreasers from high to moderate PA; E, increasers; HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment-insulin resistance; LDL, low-density lipoprotein; PA, physical activity; *β*, unstandardized regression coefficients.

[a]Back-transformation from the natural logarithm(log($x+1$)); numbers in bold indicate significant differences, *$p<0.05$; **$p<0.01$; ***$p<0.001$.

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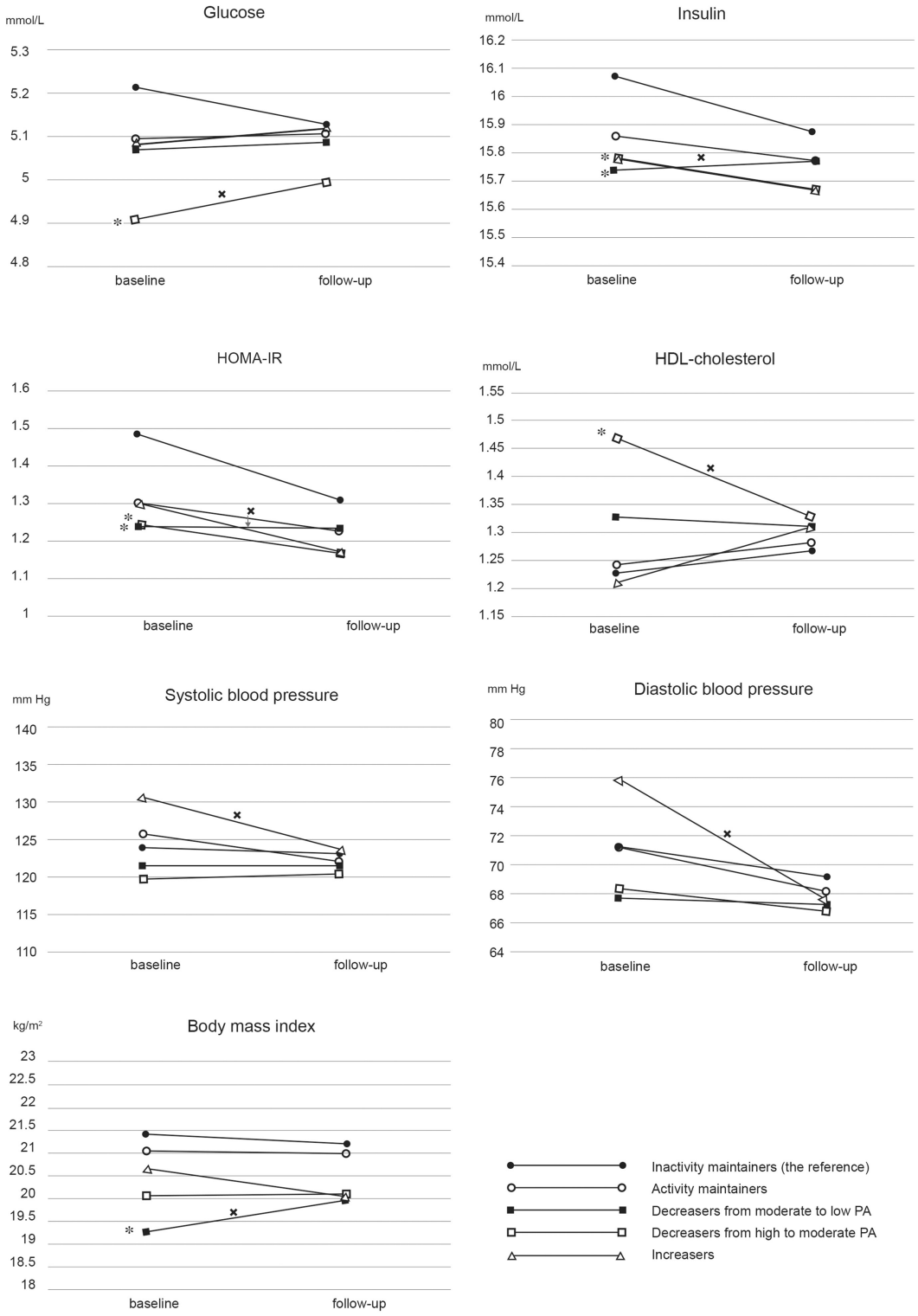

FIGURE 3 Development over time of cardiometabolic risk factors across longitudinal physical activity patterns. Development over time of cardiometabolic risk factors across longitudinal physical activity patterns among non-smoking/non-snuff-using men with no fruit/vegetable consumption. Estimated using the parameters of the linear growth curve models after adjusting for the change in the device wear-time. The symbol * represents a statistically significant difference in the baseline level (intercept). The symbol × represents a statistically significant change over time (slope) as compared to the reference (inactivity maintainers). HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment for insulin resistance; PA, physical activity.

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cardiometabolic health concerns in the population exhibiting a decreasing PA. This was expected given that the study sample consisted for the most part of relatively active young people. The results do, however, demonstrate PA effects on cardiometabolic health from adolescence to early adulthood (findings that are independent of sex, fruit and vegetable consumption, or cigarette/snuff use). One should also take into account the baseline level of the outcome. For example, among *decreasers from high to moderate PA* the levels of HDL cholesterol, and glucose were more favorable at baseline than those of the reference group. Hence, the unfavorable changes in these risk factors did not result in particularly poor follow-up results.

The transition to young adulthood is one of the most fundamental periods of life in terms of PA-related behavior, given that PA may be influenced by e.g. moving out of the childhood family, cohabiting, starting further studies or entering working life.[35,36] During a 30-year follow-up[37] it was found that a low level of MVPA among young adults was associated with increased odds of diabetes, a higher level of triglycerides, and a lower level of HDL cholesterol in later life. The present study adds to this, indicating that the *risk* for cardiometabolic diseases is not just something that will occur far in the future, insofar as changes in PA were associated with changes in cardiometabolic risk already at adolescence.

An increase in BMI is generally a sign of increased cardiometabolic risk; however, during adolescence the increase is not necessarily an unfavorable result, since it may arise from an increase in lean body mass. Moreover, during adolescence, the increase in BMI might also be a result of normal body development and maturation. This might be the case among *decreasers from moderate to low PA*, bearing in mind that the BMI was lower at baseline as compared to the *inactivity maintainers*. On the other hand, no statistical difference was found between the participants according to the stage of puberty they had reached (Table 1).

The decrease in blood pressure among *increasers*—who were physically active already at baseline—is logical. However, due to the small number of persons exhibiting this PA pattern, the finding needs confirmation from further studies with a larger study sample. The small number of *increasers* may also be one explanation for the lack of other significant differences in cardiometabolic risk factors at baseline or over time in this PA pattern.

It is somewhat surprising that there was no statistically significant difference in any of the studied cardiometabolic risk factors between the *activity maintainers* and the *inactivity maintainers*, although the baseline values of insulin and HOMA-IR were very close to significance (Table S2). One could have expected that the baseline or follow-up levels of the risk factors would show a benefit from the maintenance of relatively high PA, but this was not indicated in the study. One explanation might be that the reference group was not extremely inactive (with mean MVPA 47 min/day at age 15), even if it was below the PA recommendation for children and adolescents, that is, at least 60 min/day of MVPA.[38] The population in the present study represented young people who were more active than the average in Finland, with the mean daily MVPA being 8 min more than that found in a previous population-based study.[10,39] Furthermore, 39% of the participants in the present study were still participating in sports club as young adults. The result demonstrates that the consequences of both decreased and increased PA can be seen even when the reference group is that of young people who maintain a relatively low—but not extremely low—level of activity from adolescence to young adulthood. This can be considered an important and new finding.

4.1 | Comparisons with other studies and perspectives for future research

A few previous studies on objectively measured PA during youth have examined cardiometabolic health outcomes between distinct data-based PA patterns[15,17]; however, no studies have so far examined the associations of PA patterns with blood biomarkers. Moreover, the self-reporting of PA,[14] or the lack of a data-driven method for grouping PA patterns[16]—or both[13,18]—have lessened comparability to the present study. Nevertheless, our finding indicating an increase in BMI among *decreasers from moderate to low PA* is consistent with previous studies showing increased accumulation of body adiposity[17] and obesity development[15] among groups displaying decreasing activity.

It should be noted that little attention has so far been paid to *changes* in health indicators. Overall, it has been more common to examine the level of the health outcome at follow-up, for example in young adults.[13,14,17] Undoubtedly, comparisons at follow-up are useful; however, going beyond these, examination of the change in health indicators over time—via a valid methodology for prospective research—can broaden knowledge of the phenomena in question. Growth curve modeling (growth mixture modeling), latent profile analysis, and related approaches (as used in this article) have been recommended also by other researchers in efforts to gain a better understanding of the nuances in young people's health.[40] Mention has also been made of the need for studies on health outcomes beyond adiposity (e.g. using blood biomarkers[5]).

An interesting perspective for future research would be analysis of the healthy thresholds of PA, that is, how much PA is needed to maintain cardiometabolic health benefits

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during the transition to young adulthood. In one study, 88 min of daily MVPA was concluded to be necessary to prevent clustering of risk factors in 15-year-old adolescents.[41] A further research gap has been mentioned regarding PA guidelines, which have been criticized changing too rapidly as adolescents turn into 18-year-olds.[6] In fact, the same PA guidelines are still issued for young adults and for older people approaching retirement age, in the absence of any proper research evidence from young adults. Another unsolved question is how much of a *change in PA* is needed to modify the cardiometabolic status of growing adolescents.

4.2 | Strengths and limitations

The main strengths of this study are the use of prospective and diverse data on cardiometabolic risk factors from a less-studied period of life (see also Ref.[5,6,11]), data-driven determination of longitudinal PA patterns measured via accelerometry, and the research methodology that enabled analysis of changes in cardiometabolic outcomes over time. Furthermore, adjustment by other health behaviors increased the quality of this study.

However, the results of this study should be interpreted with caution for several reasons. Females and adolescents with higher school achievement were over-represented in the study sample, and it encompassed only two measurement points. The number of persons in some of the PA patterns was small, which decreased the statistical power. On the other hand, the recruitment of participants from six different regions of Finland (from 100 schools and 156 sports clubs) increased the quality of this study, even if the results are not directly generalizable to the entire Finnish age cohort in question. The study sample was also somewhat more active than would be found among Finnish young people on average – although this was also a strength, insofar as the data also allowed analysis of PA changes from high baseline levels. While accelerometry is the state-of-the-art method to assess PA via a device,[42] one should bear in mind the related challenges; for example, the 7-day measurement period might not be representative of the habitual behavior of every participant. Moreover, use of the device tends to underestimate the vigorous PA occurring in certain types of PA, such as skating and weight training (see also Ref.[21])—even though moderate and vigorous PA were combined in our analyses.

5 | PERSPECTIVES

The study provided new evidence on how different longitudinal PA patterns are associated with changes in cardiometabolic risk factors already during adolescence and young adulthood (between ages 15 and 19). A favorable (decreasing) trend emerged in the systolic and diastolic blood pressure of adolescents whose PA increased over time, as compared to those who maintained a relatively low level of PA. Correspondingly, unfavorable changes in insulin and BMI occurred among *decreasers from moderate to low PA*, and in glucose and HDL cholesterol among *decreasers from high to moderate PA*. Hence, it seems that different changes in cardiometabolic risk factors may be expected in relation to differing baseline PA levels and the magnitude of the change in PA. Cardiometabolic risk factors are known to change during adolescence and sufficiency of MVPA may influence the changes in these risk factors. One can therefore suggest that the promotion of PA may be highly beneficial to individuals in this age-bracket, especially for those with greater cardiometabolic risk.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT

The datasets generated and/or analyzed during the current study are not publicly available, since they contain confidential personal details and health information; however, they are available from the corresponding author on reasonable request.

ORCID

Tuula Aira https://orcid.org/0000-0001-9770-1732
Sami Petteri Kokko https://orcid.org/0000-0001-9436-5681
Olli Juhani Heinonen https://orcid.org/0000-0002-1722-1729
Raija Korpelainen https://orcid.org/0000-0002-3627-0542

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Jari Parkkari https://orcid.org/0000-0001-5211-9845
Kai Savonen https://orcid.org/0000-0002-6871-8153
Maarit Valtonen https://orcid.org/0000-0001-8883-2255
Henri Vähä-Ypyä https://orcid.org/0000-0001-7537-2501
Tommi Vasankari https://orcid.org/0000-0001-7209-9351

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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