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Longitudinal associations between parental and offspring's leisure-time physical activity: The Young Finns Study

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ABSTRACT

Purpose: The longitudinal influence of parental leisure-time physical activity (LTPA) on their offspring's LTPA is poorly understood. This study examined the longitudinal associations between parental LTPA and offspring's LTPA at two-time intervals.

Method: Child (offspring) participants (N=3596) were enrolled from the Cardiovascular Risk in Young Finns Study in 1980. Their LTPA was self-rated through nine phases from baseline to 2018 and categorized by year into youth (1980–1986) and adult (1992–2018) LTPA. Parental LTPA was assessed with a single self-reported question at three phases from 1980 to 1986. Latent growth curve modeling stratified by gender was fitted to estimate the potential pathways between parental LTPA and offspring's youth and adult LTPA.

Results: Higher initial levels of paternal and maternal LTPA were independently associated with greater initial levels of youth and adult LTPA of offspring in both genders respectively, except

maternal LTPA that did not associate with male offspring's adult LTPA. The initial levels of paternal LTPA were directly related to changes in male offspring's youth LTPA after adjusting for age, residential place, paternal education and occupation, having siblings, and offspring's body mass index.

Conclusion: Our study demonstrates that the initial levels of parental LTPA are directly linked to the initial levels of offspring's LTPA during youth and adulthood, while changes in parental LTPA are unrelated to changes in offspring's youth and adult LTPA for either gender over time. These results imply that higher initial levels of LTPA in parents may serve as a predictor of offspring's LTPA across life stages.

Key words: leisure-time physical activity, parents, offspring, latent growth curve modeling, follow-up

1 BACKGROUND

Lack of leisure-time physical activity (LTPA) may contribute to development of obesity and other cardiovascular risk factors in young people[1], which lead to increased risk of all-cause mortality in adults.[2] A recent longitudinal study applying trajectories of LTPA has found that the prevalence of Finnish children and adolescents who maintain their high-level LTPA to adulthood is only 6.6%.[3] Additionally, the high amounts and intensities of LTPA decline dramatically with age around the world.[4] It is important, therefore, to establish why such declines may occur and what key influences are operating on LTPA during childhood.

Systematic reviews have demonstrated inconclusive evidence on the influence of parental modeling on offspring's LTPA in cross-sectional studies.[5,6] Some studies have reported associations between parent and child LTPA,[7,8] while other studies have found no such associations.[9–11] There is also an interaction with mixed gender pairs in parent and child LTPA;

paternal LTPA has a modest direct effect on child's LTPA in both genders,[12] and maternal LTPA has a similar effect related to child's LTPA.[13,14] In our longitudinal studies, we have found that compared with maternal LTPA, paternal LTPA is a better predictor of child's LTPA later in life.[15,16] Other research has suggested that high parental LTPA predicts a high level of LTPA from childhood to adulthood.[17] Furthermore, parental role modeling has a positive impact on children's LTPA in same gender pairs (i.e., fathers with sons, mothers with daughters),[18,19] but the associations are not significant in opposite gender pairs.[18] However, there are important factors concerning the influence of parental modelling on children's LTPA that remain insufficiently explored, and this may explain the inconsistencies in the findings. A plausible explanation for these inconsistencies may include research design, study methodology, self-report methods of behaviors, sample sizes, children of different ages, and variability in LTPA patterns.[20,21] For instance, previous findings of longitudinal studies have revealed that parental self-reported physical activity (PA) predict children's organized PA over time and paternal PA predict adolescents' overall PA.[21] In contrast, a recent study has indicated that paternal and maternal objectively-assessed moderate-to-vigorous-intensity PA (MVPA) at baseline are not associated with children's MVPA over 4 years and change in parent MVPA does not predict change in child MVPA.[20] The evidence on the mechanisms for the longitudinal associations of parental LTPA on offspring's LTPA has also not been thoroughly investigated.

Participation in LTPA at different phases of life can be viewed through a life-course approach that examines an individual's life history and observes how early events influence future decisions and events. In a life-course perspective on physical activity,[22,23] physical activity is viewed as having dynamic and interactive relationships that involve a series of interlocking social, emotional and developmental trajectories. There is inconclusive evidence that LTPA in childhood or adolescence seems to carry over into adulthood.[24–27] Tracking of LTPA during life-course tends to have low to moderate inter-age correlations and the tracking correlations diminish with increasing interval between the measurement points.[28] The low correlations may reflect a considerable within-individual variability in LTPA participation over longer periods of time, along with physical and social environmental changes.[29,30] So far, however, there are large gaps in our understanding of cause or consequence between parent and child LTPA over time.[9,13]

The focus of cross-sectional studies has been to evaluate the association of parental LTPA with offspring's LTPA, and few studies have examined the association of parental LTPA on maintaining offspring's LTPA using longitudinal data. Latent growth curve modeling (LGCM) is a method that allows for the prediction of interindividual (between-person) variability based on intraindividual (within-person) change over time.[31] However, there are no longitudinal studies that have analyzed the relationship of temporal changes in parental LTPA with changes in offspring's LTPA over time. The purpose of the present study, therefore, was to determine whether initial levels and changes of parental LTPA link to initial levels and changes of youth (offspring aged 9–18 years) LTPA from 1980 to 1986 and adult (aged 21–56 years) LTPA from 1992 to 2018 in the context of mixed-gender interactions.

2 METHODS

2.1 Participants

We used data from the ongoing longitudinal prospective Cardiovascular Risk in Young Finns Study (YFS)[32] which consisted of six cohorts born in 1962, 1965, 1968, 1971, 1974, and 1977 when participants were 3, 6, 9, 12, 15, and 18 years of age respectively. All participants were randomly selected (N = 3596, 83% of those invited) from the five Finnish university cities with medical schools (Helsinki, Kuopio, Oulu, Tampere and Turku) and their surrounding communities at study baseline in 1980. They were followed up in 1983, 1986, 1989, 1992, 2001, 2007, and 2011. A detailed description of the YFS has been reported elsewhere.[26,32] The 2018 physical activity questionnaire was recently completed. The study design and number of offspring participants and their parents in this study are presented in Table 1. Self-report questionnaires were available in 1980, 1983 and 1986 to assess LTPA in parents and their children aged 9–18 years, 9–18 years and 9–15 years, respectively. Data for 1989 were excluded from the analysis because parental LTPA was only available for the two youngest cohorts between 12 and 15 years and assessment components of offspring's LTPA in adulthood underwent some changes thereafter. LTPA data from 1992 to 2018 were used for the offspring from the age of 21 years. Ethics approval was obtained from the ethics committees of each of the five participating universities and written informed consent was obtained from all participants in accordance with the Helsinki Declaration.

2.2 Leisure-time physical activity

The offspring self-reported LTPA was grouped into two categories based on measurement years. Youth LTPA (1980–1986) for children and adolescents (aged 9–18 years) consisted of the frequency and intensity of LTPA, participation in sports club training, participation in sport competitions, and habitual ways of spending leisure time. Each item was coded from 1 to 3 (1 = inactivity or very low activity, 2 = moderately intensive or frequent activity, and 3 = frequent or vigorous activity), except for participation in sport competitions (1 = no and 2 = yes) and then the five variables were combined into an index (5–14). The LTPA of the children when adults (1992–2018, aged 21–56 years) was assessed through questions concerning the intensity of LTPA, frequency of moderate-to-vigorous LTPA, hours spent on moderate-to-vigorous LTPA, average duration of a LTPA session, and participation in organized LTPA. The items were coded from 1 (low) to 3 (high) and then summed to the index (5–15). Both the indices indicated that higher scores represented higher levels of LTPA. The detailed description of the questionnaire and the scoring of PA indices has been published elsewhere.[3,25,26]

Parental LTPA was assessed with a single question: "How much do you engage in leisure-time physical activity?" The response alternatives were "a little bit or none", "somewhat" and "regular" which were coded as 1, 2, and 3, respectively, 1 representing mostly reading, watching TV, listening to radio, going to cinema or restaurant, and spending time socializing with other people, 2 hunting, fishing, gardening, and spending time outdoors with family or occasionally conducting PA, and 3 regular sports such as running, skiing, cycling, ball games, swimming, gymnastics, and weight lifting in either recreational or competitive activities. The same question was completed separately for fathers and mothers from 1980 to 1986.

2.3 Covariates

Age, residential place (urban vs. rural) and having siblings (no vs. yes) were queried at the baseline questionnaire. Offspring's height and weight were measured at the baseline study visit and body mass index (BMI) was calculated as weight (kg)/height (m^2). Parental educational level was self-reported and applied as completed school years. Self-reported information on parental occupation was classified into three categories based on the criteria of the Central Statistical Office of Finland: manual (builders, metal workers, nannies, etc.), lower non-manual (civil servants, specialized and skilled workers, etc.), and upper non-manual (administrators, managers, academics, etc.).

2.4 Statistical analysis

Data analyses were performed using R 4.0.2,[33] Mplus 7 statistical package[34] and an external R package MplusAutomation version 0.7-3.[35] Gender comparisons for study variables were calculated using Student's t-test and Chi-squared test. A joint model of three LGCMs was fitted to examine whether longitudinal changes in parental LTPA between time intervals 1980–1986 affected changes in offspring's youth LTPA (1980–1986) and adult LTPA (1992–2018). This approach estimated initial levels (intercept) and slopes (growth parameters) of parental LTPA and offspring's youth and adult LTPA as node-specific latent variables (Figure 1). Parental LGCM was conducted on the categorical response using a probit link function.[36] Offspring's LTPA in youth and adulthood were modelled by standard LGCMs. Changes in the response variables were assumed to be linear when the other variables were held constant, and time scores for each observed their variable were chosen to correspond to measurement intervals from baseline to each follow-up measurement year (0, 3 and 6 for parental LTPA; 0, 3 and 6 for offspring's youth LTPA; and 0, 9, 15, 19, and 26 for offspring's adult LTPA; respectively). All models were adjusted for baseline age, residential place, parental education and occupation, having siblings, and offspring's baseline BMI. The possible effect modification of the initial levels and changes of parental LTPA on the initial levels and changes of offspring's youth and adult LTPA was tested using gender-stratified analyses with model-estimated beta coefficients. Level of significant was $P < 0.05$.

Missing data were assumed to be missing at random (MAR) and were considered missing as a function of observed covariates and observed outcomes,[34] because exclusion of the missing data from the final analysis might significantly reduce statistical power and lead to biased estimation results.[37] Maximum likelihood with robust standard errors was used to estimate the sample correlations and the parameters of the models. The method produced unbiased parameter estimates under MAR. The comparative fit index (CFI), the Tucker-Lewis Index (TLI), the root-mean-square error of approximation (RMSEA), and the standardized root-mean-square residual (SRMR) were used to assess goodness of fit of the models. To interpret these indices, the CFI and TLI values were close to 0.90, and the RMSEA and SRMR values were below 0.08.[38]

3 RESULTS

3.1 Descriptive characteristics of study variables

Of all 3596 offspring participants, 2939 (53.5% females) had information on LTPA in at least one follow-up study (Table 1). Over 85% of fathers and mothers completed a LTPA questionnaire at least once between study years 1980 and 1986. To gain valid results from the sampling, at least one observation of a parent's LTPA was separately required for at least one observation of offspring's LTPA in youth and in adulthood. On average, male offspring had higher levels of youth LTPA than female offspring (Table 2). Compared to female offspring, male offspring were more likely to have fathers in manual work. No other gender differences were observed.

3.2 Longitudinal relations between parent and offspring LTPA

In the father-offspring LTPA models (Table 3), the fully adjusted models fitted the data well for father-son LTPA (CFI = 0.977; TLI = 0.955; SRMR = 0.036; RMSEA = 0.027) and for father-daughter LTPA (CFI = 0.977; TLI = 0.955; SRMR = 0.031; RMSEA = 0.023). Higher initial levels of paternal LTPA were associated with a greater initial level of offspring's youth LTPA of boys ($\beta = 0.21$, $p < 0.001$) and girls ($\beta = 0.16$, $p < 0.001$) and offspring's adult LTPA of men ($\beta = 0.22$, $p < 0.001$) and women ($\beta = 0.19$, $p = 0.003$), respectively, independent of baseline age, residential place, paternal education and occupation, having siblings, and offspring's baseline BMI. The initial levels of paternal LTPA were directly associated with changes in offspring's youth LTPA of boys ($\beta = 0.13$, $p = 0.045$) after adjusting for potential covariates. There were no other significant pathways of initial levels and changes of LTPA for both parents and offspring at either time interval.

In the mother-offspring LTPA models (Table 4), the fully adjusted models fitted the data well for mother-son LTPA (CFI = 0.98; TLI = 0.96; SRMR = 0.037; RMSEA = 0.026) and for mother-daughter LTPA (CFI = 0.975; TLI = 0.951; SRMR = 0.031; RMSEA = 0.024). Higher initial levels of maternal LTPA were independently associated with a higher initial level of offspring's youth LTPA of boys ($\beta = 0.14$, $p = 0.001$) and girls ($\beta = 0.23$, $p < 0.001$) and offspring's adult LTPA of women ($\beta = 0.18$, $p = 0.003$), respectively. No other significant associations were found in the remaining paths.

4 DISCUSSION

To our knowledge, this is the first prospective study to examine longitudinal associations between parental LTPA and offspring's youth and adult LTPA across time. We found that higher initial levels of paternal and maternal LTPA were associated with greater initial levels of offspring's youth and adult LTPA for both genders, except maternal LTPA that did not associate with offspring's adult LTPA in men. These associations remained significant after adjusting for important covariates. The initial levels of paternal LTPA were also significantly related to changes in male offspring's youth LTPA when controlling for the covariates. This suggests that there is evidence of a direct relationship between parental LTPA and offspring's LTPA in youth and adulthood, although changes in parental LTPA do not alter the change of offspring's LTPA over time.

The results supported the hypothesis that higher initial levels of paternal and maternal LTPA were independently associated with more favorable initial levels of offspring's youth LTPA in both genders. Our results are in line with previous studies[13,18,19] that have indicated gender-specific associations between the parental and offspring LTPA (father-son, mother-daughter). Our results also extend the previous findings by showing associations for opposite gender pairs (father-daughter, mother-son). However, these results are not in line with some studies which have not shown significant relationships between parental and children's or adolescents' LTPA.[9–11] In systematic reviews and meta-analyses of large-scale studies, parental physical activity has been found to be a determinant of activity levels in childhood but not in adolescence.[6] Paternal physical activity was more strongly associated with sons' physical activity than maternal physical activity, but parental gender does not moderate the association between parental and daughters' physical activity.[5] We found that paternal LTPA was more strongly related to LTPA in sons than in daughters, whereas maternal LTPA was more strongly linked to LTPA in daughters than in sons. These differences may be partially explained by a need to comply with gender stereotypes: in Finland, boys are expected to engage in soccer, ice-hockey and floorball, and girls are expected to involve in dancing, gymnastics and horse riding[39]. Our findings highlight the need for more research that addresses the roles of parent gender and child gender in the process of sports socialization.

The present study provides new insight, suggesting that the LTPA levels of parents and offspring with the same-gender are significantly higher than that of those with the opposite-gender. Additionally, children are influenced by their parental support toward LTPA and appear to be responsive to parental role modeling.[5,6] The proposed mechanism for initial levels of parental LTPA with initial levels of offspring's youth LTPA has been suggested to explain how parental involvement contributes to offspring's participation in LTPA during the early years of life. However, revealing the precise pathways underlying the association between parental and offspring's LTPA from childhood to adulthood (such as genetic and environmental factors) still need further studies. For example, the role of various types of social support requires further study to see how parent support of physical activity for their children may differ from actual parental physical activity participation. It seems logical that some parents may be highly supportive yet rather inactive.

Importantly, in this study, the initial levels of paternal LTPA directly contributed to changes in male offspring's LTPA during youth, the association remained significant after the covariates were taken into account. The results partially support our hypothesis that the initial paternal LTPA levels are associated with more favorable increased youth LTPA in male offspring over 6 years, compared to the initial maternal LTPA levels. The findings suggest that the initial levels of paternal LTPA may have modest predictive power in the process of increased LTPA of male offspring in youth, which then prove sufficient to sustain male offspring's youth LTPA as they grow older.

We also tested whether initial levels of parental LTPA predict initial levels of offspring's adult LTPA. We found that higher initial levels of paternal LTPA predicted greater initial levels of offspring's adult LTPA of both genders, while higher initial levels of maternal LTPA predicted higher initial levels of offspring's adult LTPA of women only. These findings are in line with our previous research demonstrating that parental LTPA predicts their children's LTPA later in life.[15–17] This suggests that the initial parental LTPA levels may have the potential to enhance the initiation of offspring's adult LTPA, which provide a unique insight into the underlying behavioral mechanisms that influence adoption and maintenance of LTPA among adult offspring. These longitudinal path models may prove to be useful for identifying parental behaviors as potential targets of intervention to sustain and improve adherence to their offspring's LTPA for the long-

term. These results further suggest that the relationship between parental LTPA and offspring's adult LTPA is not driven solely by their actual LTPA levels. The parental LTPA accounted for only little of the variance in offspring's adult LTPA, indicating that life circumstances and other personal (e.g., values and beliefs) and environmental (e.g., neighborhood and community) factors might explain additional variance in offspring's adult LTPA levels.

We did not observe direct associations of changes in parental LTPA with changes in offspring's LTPA either in youth or adulthood. This may imply that changes in parental LTPA do not fully represent a direct association with changes in offspring's LTPA and, therefore, should not be the only way to achieve the offspring's LTPA over time. On the other hand, all three phases of parental LTPA were coded according to activity level as an ordered categorical variable with a probit link function. Changes in parental LTPA from baseline to follow-up was very small, as was changes in offspring's LTPA. This may cause an absence of significant changes in both LTPAs over time. It is also possible that the change of parental LTPA is not linearly associated with the change of offspring's LTPA, although at this point this is only speculation. Thus, increases in parental LTPA over time may not necessarily relate to increases in offspring's LTPA at two-time intervals.

Taken together, these findings suggest that the initial levels of parental LTPA have stronger links with the initiation of offspring's youth and adult LTPA than with the maintenance of their LTPA. Thus, changes in parental LTPA over time may be indicative of, but do not necessarily associate with, changes in offspring's youth and adult LTPA. In general, the initial parental LTPA has been identified as an important contributor to children's LTPA levels[5] and related to the development of LTPA during childhood or adolescence[20,40] and throughout adulthood.[15–17] Alternatively, initial parental LTPA levels are more likely to exert a direct relationship with initial levels of offspring's youth and adult LTPA than to accumulate over two-time intervals. There is also empirical evidence that gender likely acts as a moderator in the associations for LTPA levels between father-sons and mother-daughters,[18,19] but this evidence on parental LTPA change interventions to improve offspring's youth and adult LTPA changes is scarce and inconclusive. Therefore, it is not enough to rely only on a single measure of parental LTPA as an indication of changes in offspring's LTPA over time.

The present study has several strengths, including its prospective cohort design over a 31-year follow-up period, a representative population-based sample of Finnish children and adolescents and their parents, repeated measures of LTPA, and major potential covariates. The LGCM approach used in this study also allowed for simultaneous analysis of the intercepts and slopes of parental LTPA, offspring's youth and adult LTPA, and the degree of correspondence between growth parameters across time, resulting in substantial reductions in misclassification bias. Several potential covariates that could affect the association between parental and offspring's LTPA were controlled for in the analysis.

We also recognize that the study has some limitations. First, because data on parental LTPA levels were self-reported with a single question over time, we were not able to estimate a random measurement error. Second, offspring's self-reported LTPA did not capture certain domains of physical activity (e.g., school physical education, commuting and work-related physical activity), leading to an underestimation of the overall physical activity levels. Further research using multiple questions to assess LTPA for parents or monitoring using wearable devices for both generations is needed. There is also a need for more research on the influence of parental role models for children's other activities such as active commuting. Third, the study focused on the residential environment as a covariate, so other potential covariates such as home (e.g., screen devices), neighborhood (e.g., parks and playgrounds), school (e.g., policies), and community (e.g., sports facilities) environments were not examined. To optimize explanation of parental and their children's LTPA, these factors also warrant future research. Finally, all participants are white European with a relatively high family income, thus the results may not be generalizable to other populations with diverse ethnicities and socioeconomic status.

5 PERSPECTIVE

This study highlights a positive association between parental LTPA and offspring's LTPA during different life phases. The findings of this study are partially consistent with the hypothesized model that higher initial levels of parental LTPA are independently associated with greater initial levels of offspring's youth and adult LTPA. Changes in parental LTPA are unrelated to changes in offspring's youth and adult LTPA for either gender over time. These results imply that higher initial levels of LTPA in parents may serve as a predictor of offspring's LTPA across life stages.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available online at http://youngfinnsstudy.utu.fi/.

Figure legend:

Figure 1. A latent growth curve model for intercepts and slopes of parental LTPA, offspring youth LTPA and offspring's adult LTPA.

Table 1. Study design and number of parents and offspring

Year	N_{Father}	N_{Mother}	$N_{Male\ offspring}$	$N_{Female\ offspring}$	Age of offspring participants															
1980	2,580	2,864	949	1,084	3	6	**9**	**12**	**15**	**18**										
1983	2,203	2,475	825	977		6	**9**	**12**	**15**	**18**	21									
1986	1,123	1,279	758	883			**9**	**12**	**15**	18	21	24								
1992			597	779					15	18	**21**	**24**	**27**	**30**						
2001			1,053	1,293								**24**	**27**	**30**	**33**	**36**	**39**			
2007			932	1,158										**30**	**33**	**36**	**39**	**42**	**45**	
2011			818	1,037											**34**	**37**	**40**	**43**	**46**	**49**
2018			833	1044													**41**	**44**	**47**	**50**
N‡	2,635	2,906	1,367	1,572																

N‡ is the total sample size used in models. Age in bold font represents offspring linked with their parental leisure-time physical activity in the early years and with own leisure-time physical activity in the late years.

Table 2. Characteristics of study variables by gender			
Variable	Male offspring	Female offspring	$p^{\ddagger}$
Mean and standard deviation	(n = 1367)	(n = 1572)	
Youth age (years)	10.6 (4.9)	10.8 (4.9)	0.443
Mean youth's LTPA	9.5 (1.8)	8.6 (1.4)	<0.001
Mean adult's LTPA	8.9 (1.7)	8.9 (1.4)	0.565
Youth BMI (kg/m^2)	18.0 (3.2)	17.9 (3.0)	0.495
Paternal education (years)	10.1 (3.8)	9.9 (3.8)	0.385
Maternal education (years)	10.4 (3.4)	10.2 (3.3)	0.090
Percentages			
Residence			
Urban	47.0	46.1	0.662
Rural	53.0	53.9	
Paternal LTPA			

Low	22.4	22.7	0.589
Moderate	57.1	55.4	
High	20.5	22.0	
Maternal LTPA			
Low	29.1	31.3	0.431
Moderate	54.3	52.4	
High	16.6	16.3	
Paternal occupation			
Manual	41.0	38.3	0.030
Lower non-manual	17.1	21.2	
Upper non-manual	41.8	40.5	
Maternal occupation			
Manual	33.2	30.9	0.177
Lower non-manual	36.9	40.2	
Upper non-manual	29.9	28.8	
Having siblings	84.5	85.8	0.366

[‡]The *p*-values for gender difference, Student's t-test or Chi-squared test. Youth LTPA (1980–1986) and adult LTPA (1992–2018) were summed to derive average LTPA. BMI, body mass index; LTPA, leisure-time physical activity.

Table 3. Associations of initial levels and slopes of paternal leisure-time physical activity with offspring's youth and adult leisure-time physical activity

	Paternal LTPA								
	Model I†					Model II‡			
	Level		Slope			Level		Slope	
Offspring's LTPA	*β* (*SE*)	*p*	*β* (*SE*)	*p*		*β* (*SE*)	*p*	*β* (*SE*)	*p*
Youth LTPA									
Boys									
Level	0.21 (0.05)	<0.001				0.21 (0.05)	<0.001		
Slope	0.16 (0.06)	0.010	0.07 (0.11)	0.529		0.13 (0.06)	0.045	0.04 (0.12)	0.720
Girls									
Level	0.16 (0.04)	<0.001				0.16 (0.04)	<0.001		
Slope	0.07 (0.05)	0.168	0.05 (0.07)	0.516		0.08 (0.06)	0.149	0.04 (0.08)	0.571
Adult LTPA									
Men									
Level	0.27 (0.05)	<0.001	-0.11 (0.14)	0.413		0.22 (0.06)	<0.001	-0.14 (0.14)	0.345
Slope	-0.02 (0.09)	0.832	0.32 (0.21)	0.124		0.04 (0.09)	0.680	0.31 (0.21)	0.141
Women									

Level	0.19 (0.06)	0.002	0.18 (0.12)	0.123		0.19 (0.06)	0.002	0.17 (0.11)	0.125
Slope	-0.03 (0.08)	0.682	-0.24 (0.16)	0.138		-0.07 (0.08)	0.383	-0.23 (0.16)	0.144

†Model I was adjusted for baseline age.

‡Model II was additionally adjusted for residential place, paternal education and occupation, having siblings, and offspring's baseline body mass index.

β, standardized regression coefficient; SE, standard error; LTPA, leisure-time physical activity.

Table 4. Associations of initial levels and slopes of maternal leisure-time physical activity with offspring's youth and adult leisure-time physical activity

	Maternal LTPA								
	Model I†					Model II‡			
	Level		Slope			Level		Slope	
Offspring's LTPA	*β* (*SE*)	*p*	*β* (*SE*)	*p*		*β* (*SE*)	*p*	*β* (*SE*)	*p*
Youth LTPA									
Boys									

Level	0.14 (0.04)	0.001				0.14 (0.04)	0.002		
Slope	0.02 (0.06)	0.741	-0.04 (0.16)	0.786		0.00 (0.06)	0.946	-0.04 (0.14)	0.772
Girls									
Level	0.23 (0.04)	<0.001				0.23 (0.04)	<0.001		
Slope	0.03 (0.06)	0.612	0.05 (0.09)	0.566		0.02 (0.06)	0.738	0.05 (0.09)	0.557
Adult LTPA									
Men									
Level	0.06 (0.06)	0.306	-0.18 (0.20)	0.358		0.04 (0.06)	0.481	-0.14 (0.17)	0.407
Slope	0.04 (0.08)	0.665	-0.14 (0.29)	0.634		0.05 (0.08)	0.552	-0.16 (0.26)	0.522
Women									
Level	0.18 (0.06)	0.002	-0.01 (0.13)	0.910		0.18 (0.06)	0.003	-0.05 (0.13)	0.707
Slope	-0.11 (0.08)	0.172	0.13 (0.17)	0.432		-0.14 (0.08)	0.077	0.15 (0.17)	0.373

†Model I was adjusted for baseline age.

‡Model II was additionally adjusted for residential place, maternal education and occupation, having siblings, and offspring's baseline body mass index.

β, standardized regression coefficient; SE, standard error; LTPA, leisure-time physical activity.