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ORIGINAL ARTICLE

Regular exercise behaviour in youth is not related to current body mass index or body mass index at 7-year follow-up

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Summary

Objective

This population-based study aimed (1) to test the presence of an association between regular voluntary exercise behaviour (EB) that is performed in leisure time and body mass index (BMI) in youth and (2) to investigate the causal nature of this association using a longitudinal design in genetically informative subjects.

Design and methods

Both EB and BMI were assessed repeatedly over time in 21 458 twin individuals from the Netherlands Twin Register (47.5% male) – first by parental report (ages 7, 10 and 12) and subsequently through self-report surveys (ages 14, 16 and 18). EB was quantified as weekly metabolic equivalent of task hours.

Results

Correlations over time were higher for BMI than for EB ($r \approx 0.70$ vs. $r \approx 0.35$) across 12 different follow-up periods. Cross-sectionally, regular involvement in EB was not associated with lower BMI in childhood and in genetically identical twin pairs discordant for EB; the exercising twin did not have a lower BMI than the non-exercising twin. Longitudinally, linear and quadratic relationships between EB and BMI were non-significant. Changes in EB over time did not induce opposite changes in BMI.

Conclusions

No consistent association between regular EB and BMI was observed from ages 7 to 18 years.

Keywords: Adolescence, childhood, energy balance, physical activity.

Introduction

Childhood obesity is a major health concern of our time. Olds *et al*. estimated the average prevalence of overweight in almost 500 000 children and adolescents of nine countries to range between 13.5% and 37.4% (1). Being overweight as a child not only impairs health in the short run (2) but also increases morbidity and premature mortality in adulthood (3). Attempts to intervene and curb the obesity epidemic have aimed at increasing energy expenditure and/or decreasing energy intake, particularly the consumption of high-caloric food. Interventions that target youth tend to favour an increase in energy expenditure as opposed to a decrease in energy intake, owing to its positive effects on general health and possible risks associated with energy intake modification (e.g. compromise of growth and facilitation of eating disorders) (4,5).

A wealth of studies on the relationship between energy expenditure and body composition in youth has focused on daily physical activity (PA). These observational studies have produced rather mixed outcomes, and studies with null findings might be underrepresented as a result of a publication bias towards significant results (6–9). Admittedly, the majority of studies have methodological flaws such as a cross-sectional design that prohibits conclusions on cause–effect relationships and small sample sizes

(8,9). However, a systematic review on the effect of PA interventions in over 36 000 children did not support an obesity-reducing effect (10). The use of suboptimal measurement instruments is another potential flaw. Survey-based assessments of PA have shown poor agreement with actual measures of energy expenditure, which may reduce power to detect a relation between PA and body composition (11). However, even studies using objective PA measurements including doubly labelled water, step counts or accelerometers have not produced systematic evidence for this link (12), although sample sizes in these studies have by necessity been far more modest than those in survey studies.

Measuring PA by survey in youth is particularly challenging because of children's complex activity patterns that include spontaneous PA (e.g. fidgeting); PA related to transportation (bicycling), school or work; PA related to indoor and outdoor play; and all structured and unstructured exercise activities (11). These are even difficult to capture by surveys that rely on subjective recall of a complex set of activities. The present study, therefore, uses a different approach by focusing only on regular voluntary exercise behaviour (EB) that is performed in leisure time and in structured settings, like health clubs, recreational outdoor activities and team sports. Because the exercise activities are both voluntary and often scheduled at regular times, recall is easier than less salient activities like the amount of walking or moderately intensive household activities during the day. Self-reported EB measured with surveys has indeed been shown to have high test–retest reliability (13).

Obesity can be defined in a myriad of ways, but body mass index (BMI) has become the standard for defining and assessing overweight in both adults and children, and it is directly associated with negative long-term health consequences (14,15). Although not a perfect indicator of body fatness in thin children (16), it has been shown to strongly correlate with skin-fold thickness, body fat percentage and total fat mass in children and adolescents (17). BMI is therefore the most feasible approximation of body composition that can be assessed in large population-based samples.

Cross-sectional and longitudinal relationships were assessed between regular EB and BMI in a very large genetically informative longitudinal data set, with data of monozygotic (MZ) and dizygotic (DZ) twins aged 7, 10, 12, 14, 16 and 18 years. The expectation of this study is that higher levels of EB in childhood and adolescence will lead to lower levels of BMI. This would be reflected in significant cross-sectional and longitudinal associations between the (changes in the) two traits, even when accounting for genetic pleiotropy or confounding by latent environmental factors.

Methods and Procedures

Participants

The study is based on longitudinal research of the Netherlands Twin Register (NTR) (18). Registered twins and family members are primarily Caucasian and live in all regions of The Netherlands (rural and urban areas). The large majority of twins are registered with the NTR as newborns. Both mothers and fathers (the latter after age 2) are invited to complete surveys about their twins' health, lifestyle and behaviour at birth and when the children are approximately 2 ('survey 2'), 3, 5, 7, 10 and 12 years old. The twins are subsequently approached to complete self-report surveys when they are 14 ('survey 14'), 16 and 18 years old. Individuals with diseases or disabilities that may prevent them from being physically active (e.g. hemiplegia or heart disease) were excluded from the analyses ($N = 346$). Subsequently, an injury at the time of assessment led to an exclusion of the exercise data for that specific survey ($N = 419$ for survey 14, $N = 371$ for survey 16 and $N = 72$ for survey 18). The final dataset comprised 21 458 individuals born between 1984 and 2001 (47.5% male). Table 1 presents the number of individuals with within-trait and cross-trait data on EB and BMI for the entire study. Data on both EB and BMI were available for 3089 individuals on survey 7 (522 complete MZ and 1005 complete DZ twin pairs), 4444 on survey 10 (759 complete MZ and 1425 complete DZ twin pairs), 10 261 on survey 12 (1855 complete MZ and 3153 complete DZ twin pairs), 7171 on survey 14 (1120 complete MZ and 1759 complete DZ twin pairs), 4256 on survey 16 (669 complete MZ and 875 complete DZ twin pairs) and 2949 on survey 18 (464 complete MZ and 606 complete DZ twin pairs).

The longitudinal structure included 2-year follow-ups (surveys 10 and 12, 12 and 14, 14 and 16, and 16 and 18), a 3-year follow-up (7 and 10), 4-year follow-ups (10 and 14, 12 and 16, and 14 and 18), a 5-year follow-up (7 and 12), 6-year follow-ups (10 and 16, and 12 and 18) and a 7-year follow-up (7 and 14). Twin individuals and/or their parents provided informed consent to take part in the research. If individuals decide not to participate in a specific survey, they can always re-enter on a subsequent survey. The main reason given for non-participation is 'time constraints'. The data collection protocol was approved by the Medical Research Ethics Committee of the VU University Medical Centre (no. 2010/284).

Measures

Exercise behaviour was consistently assessed by parental report for surveys 7, 10 and 12 and by self-report for

Table 1 The number of individuals with within-trait/cross-trait data on weekly MET hours and sdsBMI, both cross-sectionally and across different follow-up periods, for male (M) and female (F) individuals separately

	Surveys	Within-trait				Cross-trait			
		MET hours		sdsBMI		MET hours and sdsBMI		ΔMET hours and ΔsdsBMI*	
		M	F	M	F	M	F	M	F
Cross-sectionally	7	1947	1971	1504	1593	1500	1589		
	10	2941	2970	2221	2229	2216	2228		
	12	6033	6313	5063	5247	5031	5230		
	14	3480	4611	3234	4339	3071	4100		
	16	1966	2796	1854	2757	1742	2514		
	18	1106	1988	1085	1930	1057	1892		
2-year follow-up	10 and 12	1205	1231	827	854	951	970	824	853
	12 and 14	2597	3238	2168	2653	2464	3101	2054	2525
	14 and 16	1027	1584	955	1547	969	1570	867	1334
	16 and 18	495	904	491	891	483	871	453	813
3-year follow-up	7 and 10	497	489	325	326	375	365	323	324
4-year follow-up	10 and 14	750	980	580	779	672	932	543	724
	12 and 16	1523	2105	1331	1867	1459	2084	1250	1704
	14 and 18	294	614	278	552	280	584	268	535
5-year follow-up	7 and 12	525	514	351	349	408	387	351	349
6-year follow-up	10 and 16	198	300	156	246	174	292	143	217
	12 and 18	681	1252	614	1085	669	1202	602	1067
7-year follow-up	7 and 14	413	505	309	412	369	477	288	382

*For example, for the 2-year follow-up '10 and 12', the following variables were available: MET hours for survey 10, MET hours for survey 12, sdsBMI for survey 10 and sdsBMI for survey 12.
MET, metabolic equivalent of task; BMI, body mass index.

surveys 14, 16 and 18. A list of 17 common exercise activities was provided, plus the option to add activities. Individuals were asked to indicate for each activity (a) whether or not their child/they participated in the activity and, if so, (b) for how many years, (c) for how many months a year, (d) how many times a week and (e) how many minutes each time. Participants had to have been active in the activity during the past half year, and only activities that were conducted for a minimum of 3 months a year were included (thereby excluding ski holidays, sailing camps and similar). In addition, activities related to transportation (walking and biking) were excluded. Activities during compulsory physical education classes were also excluded.

Each activity was subsequently recoded into its metabolic equivalent of task (MET), reflecting the energy expended during a specific activity as a multiple of energy expenditure at rest (approximately 1 kcal kg^{-1} h^{-1}). For individuals younger than 18 years, METs were taken from the Ridley *et al*. compendium of energy expenditures for youth, whereas for individuals of 18 years or older, they were taken from Ainsworth et al.'s compendium compendium of physical activities (19,20). The product of the MET score, weekly frequency and duration was summed over all exercise activities that an individual engaged in. If participants indicated an unrealistically large number of MET hours a week (>250), these were truncated at 250 MET hours ($N = 16$). For all surveys, if either exercise frequency or duration was missing while the other was provided, it was replaced with the median of that activity within the respective age group.

BMI (weight in kg/ (height in m)^2) was calculated based on reported height and weight. BMI was standardized with the software package Growth Analyser RCT (2011, version 4.0.28., Growth Analyser B.V., Rotterdam, the Netherlands), based on sex-specific and age-specific BMI scores of the Dutch population (sdsBMI). As the correlations between mothers' and fathers' ratings were high at all ages (ranging from 0.82 to 0.88 for EB and from 0.98 to 1 for BMI), their average rating was used. If mean sdsBMI values were outside the range of ±5, height, weight, BMI and sdsBMI were excluded for that person at the respective survey ($N = 16$).

Statistical analyses

In a first set of analyses, tracking over time of both EB and sdsBMI was examined by test–retest correlation across the 2-, 3-, 4-, 5-, 6- and 7-year follow-up periods. So-called saturated models were fitted in the structural equation

software OpenMx (21). Obviously, twin pairs are more similar to each other than strangers. This affects not only the correlations between twins but also the variances. Saturated models make it possible to calculate correlations while taking into account familial relatedness and even differences in genetic relatedness between MZ and DZ twins.

In a second set of analyses, the cross-sectional association between EB and sdsBMI was computed in each of the six age groups. Sex was entered to the model as a first predictor. To allow for a possible threshold effect, where EB is effective only above a certain exercise intensity, both linear and quadratic relationships with BMI were tested. Weekly MET hours were *z*-transformed (second predictor), then squared (third predictor) and added to the model as additional predictors. Generalized estimating equations were used to correct for familial relatedness.

Under the causal hypothesis, MZ twins discordant for EB should also be discordant for BMI. In these pairs, there is complete matching of age, sex and full genetic background, and even the part of the environment that is shared by siblings (family, neighbourhood and parental socioeconomic status) is better matched than in any other possible design. Therefore, in a third set of analyses, it was tested whether the MZ twins that were discordant for EB (one twin exercised much more than the other) also showed intrapair differences in BMI. To optimize statistical power either by increasing sample size or by increasing the expected effect size, discordant twin pairs were selected in each of the six age groups by two different methods. For method 1, EB-discordant MZ pairs were selected, for which the amount of weekly MET hours of one individual was equal to or greater than the median within survey and sex, whereas the other individual scored lower than this median. For method 2, we divided all individuals into five categories based on the amount of EB they engaged in. The first category comprised non-exercisers (zero MET hours), and the remaining individuals were divided into quartiles. Next, EB-discordant MZ pairs with a minimum difference of two categories were selected (e.g. one member in category 2 and the co-twin in category 4 or 5). Paired *T*-tests were used to test the hypothesis that the twin with the lower EB would have a higher sdsBMI compared with his or her co-twin.

The fourth set of analyses tested the longitudinal prediction of sdsBMI by EB across the 2-, 3-, 4-, 5-, 6- and 7-year follow-up periods. A final set of analyses focused on the correlations between *changes* in EB (weekly MET hours at time point 2 − weekly MET hours at time point 1) and *changes* in sdsBMI (sdsBMI at time point 2 − sdsBMI at time point 1). This was performed cross-sectionally at all six time points and longitudinally across all available time lags. Significance was tested with a liberal alpha level of 0.001 throughout.

The use of longitudinal twin data allows a more robust test of causal hypotheses about the nature of an association between two traits than the standard longitudinal study (13,22,23). While it is usually assumed that EB causally influences BMI, causality could also run the other way around (24), e.g. because overweight individuals might not enjoy exercising. More importantly, there may be underlying (genetic and/or environmental) factors influencing EB at baseline but also BMI at follow-up, which could create the illusion of causality in a standard longitudinal approach. In a genetically informative study, the true causal nature of these associations can be additionally tested by significance of both the genetic and environmental correlations (13,22,23). Therefore, in case systematic regression effects were found, causality would be tested by confirming that effects of all the latent genetic and environmental factors on baseline EB were transmitted to follow-up sdsBMI.

Results

Figure 1 depicts the means and standard deviations (SD) of weekly MET hours, height, weight and BMI, split by survey and sex (the exact numbers can be found in the Supporting Information, including the means and SDs of age and sdsBMI). Both the means and the SDs of EB increased from childhood up to survey 16 for boys and survey 14 for girls, but the means were lower at survey 18. Boys spent more MET hours a week on EB than girls, and overall they were taller and heavier than girls. The sdsBMI was close to the expected mean of 0 and standard deviation of 1 across all surveys. Tracking over time tended to decrease with increasing time intervals for both traits but was consistently higher for sdsBMI compared with EB (Table 2). For EB, the median cross-time correlation was 0.43 for the 2-year interval, 0.42 for the 3-year interval, 0.34 for the 4-year interval, 0.26 for the 5-year interval, 0.32 for the 6-year interval and 0.16 for the 7-year interval. For sdsBMI, this was 0.78, 0.67, 0.72, 0.69, 0.62 and 0.60, respectively. Table 3 depicts the cross-sectional association of sdsBMI by the linear ($-0.07 < B < 0.10$) and quadratic ($-0.02 < B < 0.01$) effects of weekly MET hours. The linear effect of EB on sdsBMI was significant for survey 12 with a negative sign ($B = -0.07$). Thus, more EB was associated with lower BMI. At survey 18, however, a significant positive (linear) effect was found ($B = 0.10$), signalling higher BMI in adolescents with more EB. Effect sizes were very small. All other linear and quadratic relationships across the surveys were non-significant. The overall absence of a

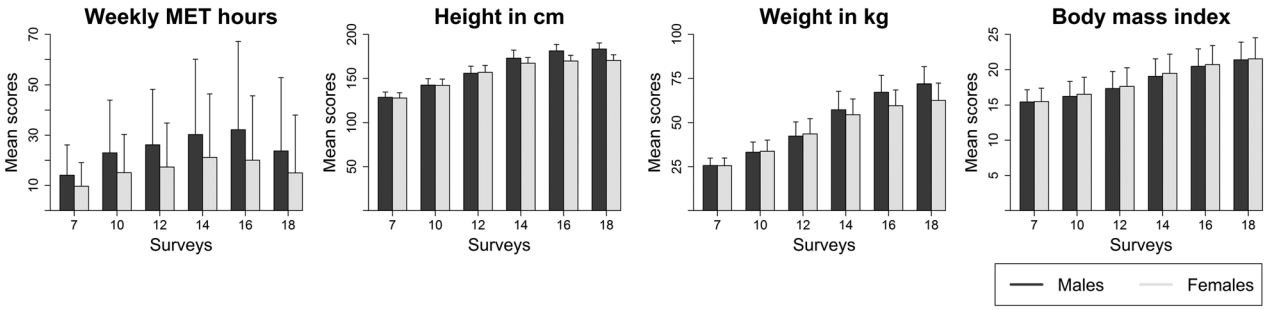

Figure 1 Means and standard deviations of weekly MET hours, height, weight and body mass index, split by survey and sex.

Table 2 Cross-time correlations (99% CI) for weekly MET hours and sdsBMI, for male and female individuals separately

Longitudinal follow-up time	Surveys	MET hours		sdsBMI	
		Male	Female	Male	Female
2-year interval	10 and 12	0.34 (0.26, 0.40)	0.38 (0.31, 0.44)	0.79 (0.76, 0.82)	0.79 (0.76, 0.82)
	12 and 14	0.39 (0.34, 0.44)	0.43 (0.39, 0.47)	0.74 (0.72, 0.77)	0.77 (0.74, 0.78)
	14 and 16	0.43 (0.35, 0.49)	0.58 (0.53, 0.62)	0.74 (0.70, 0.77)	0.81 (0.79, 0.83)
	16 and 18	0.53 (0.44, 0.61)	0.45 (0.39, 0.52)	0.77 (0.72, 0.81)	0.85 (0.83, 0.87)
3-year interval	7 and 10	0.41 (0.28, 0.51)	0.42 (0.31, 0.51)	0.66 (0.58, 0.72)	0.67 (0.59, 0.74)
4-year interval	10 and 14	0.27 (0.17, 0.37)	0.30 (0.21, 0.38)	0.72 (0.67, 0.76)	0.73 (0.68, 0.76)
	12 and 16	0.38 (0.31, 0.44)	0.40 (0.34, 0.46)	0.64 (0.60, 0.68)	0.71 (0.68, 0.74)
	14 and 18	0.23 (0.09, 0.36)	0.42 (0.32, 0.50)	0.72 (0.64, 0.78)	0.75 (0.70, 0.79)
5-year interval	7 and 12	0.29 (0.16, 0.41)	0.23 (0.11, 0.34)	0.67 (0.59, 0.73)	0.71 (0.64, 0.76)
6-year interval	10 and 16	0.18 (-0.02, 0.35)	0.27 (0.08, 0.43)	0.54 (0.35, 0.67)	0.68 (0.58, 0.75)
	12 and 18	0.36 (0.25, 0.45)	0.40 (0.32, 0.47)	0.57 (0.49, 0.63)	0.67 (0.63, 0.71)
7-year interval	7 and 14	0.18 (0.02, 0.33)	0.14 (0.00, 0.26)	0.59 (0.48, 0.67)	0.60 (0.52, 0.67)

Note: Correlations are corrected for family relatedness.
CI, confidence interval; MET, metabolic equivalent of task; BMI, body mass index.

Table 3 Cross-sectional association of sdsBMI by the linear and quadratic effects of weekly MET hours

Survey	Predictors	Unstandardized beta (99.9% CI)	*p*-value
7	Sex*	0.04 (−0.08, 0.17)	0.28
	MET hours	−0.02 (−0.11, 0.07)	0.37
	MET hours squared	0.00 (−0.03, 0.03)	1.00
10	Sex	0.03 (−0.07, 0.14)	0.29
	MET hours	−0.01 (−0.08, 0.07)	0.79
	MET hours squared	0.00 (−0.02, 0.02)	0.73
12	Sex	−0.06 (−0.13, 0.01)	0.01
	MET hours	−0.07 (−0.12, −0.03)	1.34E−7
	MET hours squared	0.01 (−0.01, 0.02)	0.04
14	Sex	0.01 (−0.07, 0.10)	0.61
	MET hours	−0.01 (−0.06, 0.04)	0.55
	MET hours squared	0.00 (−0.01, 0.01)	0.97
16	Sex	−0.03 (−0.15, 0.08)	0.34
	MET hours	0.07 (−0.01, 0.15)	4.28E−3
	MET hours squared	0.00 (−0.02, 0.02)	0.80
18	Sex	0.01 (−0.13, 0.16)	0.74
	MET hours	0.10 (0.01, 0.19)	3.51E−4
	MET hours squared	−0.02 (−0.04, 0.00)	1.63E−3

*0 = male, 1 = female; dependent variable: sdsBMI.
BMI, body mass index; MET, metabolic equivalent of task; CI, confidence interval.

Table 4 Comparison of sdsBMI in monozygotic twin pairs discordant for EB

Survey	Status of twin	Discordance based on median split				Discordance based on ≥2 quintiles			
		MET hours (SD)	sdsBMI (SD)	*N* pairs	*p*-value*	MET hours (SD)	sdsBMI (SD)	*N* pairs	*p*-value*
7	Higher EB	14.34 (6.08)	−0.05 (1.24)	34	0.22	11.93 (4.44)	0.00 (1.08)	13	1.0
	Lower EB	4.52 (3.16)	−0.21 (1.41)			2.63 (2.64)	0.01 (1.04)		
10	Higher EB	26.07 (11.27)	0.00 (1.06)	65	0.16	29.10 (9.51)	0.21 (1.04)	25	0.50
	Lower EB	8.43 (5.01)	−0.08 (1.10)			6.59 (4.72)	0.15 (1.17)		
12	Higher EB	28.62 (13.78)	−0.26 (1.10)	166	0.17	32.09 (15.51)	−0.16 (1.17)	68	0.92
	Lower EB	10.03 (6.93)	−0.21 (1.13)			6.31 (6.83)	−0.15 (1.29)		
14	Higher EB	38.71 (25.82)	−0.04 (0.98)	184	0.30	44.54 (28.76)	0.09 (1.07)	69	0.61
	Lower EB	9.77 (7.85)	0.02 (1.04)			7.19 (8.28)	0.04 (0.95)		
16	Higher EB	40.04 (29.36)	0.01 (0.96)	120	0.19	46.89 (25.58)	0.01 (0.88)	60	0.81
	Lower EB	7.51 (7.73)	−0.06 (0.92)			9.45 (11.07)	0.03 (0.89)		
18	Higher EB	29.13 (26.33)	−0.02 (1.06)	99	0.21	39.89 (27.04)	0.05 (1.13)	37	0.49
	Lower EB	2.33 (4.06)	−0.12 (1.03)			5.54 (9.66)	−0.04 (1.05)		

*p-value of the comparison of sdsBMI between the higher-EB and lower-EB twins. The p-value for EB was consistently <0.001.
BMI, body mass index; EB, exercise behaviour; MET, metabolic equivalent of task; SD, standard deviation.

relationship between EB and sdsBMI was reconfirmed in the MZ twin pairs discordant for EB as shown in Table 4. . Whether discordance was defined as EB at/above or below the sex-specific median of each survey or as a difference of at least two exercise categories, the sdsBMI of the twin who exercised more was not significantly different from that of the twin who exercised less at any age.

The longitudinal linear and quadratic relationships between EB and sdsBMI can be found in Table 5. Linear effects ranged from $B = -0.03$ to 0.14, and quadratic effects from -0.03 to 0.01. All but one of the relationships were non-significant. Counter to our expectation, the one significant relationship ($B = 0.14$) suggested that the high EB at survey 16 predicted *higher* BMI at survey 18. Finally, Table 6 depicts the correlations between the change in EB and change in sdsBMI for all the possible longitudinal combinations. Based on 99% confidence intervals, all but one of the correlations were not significantly different from zero ($-0.14 < B < 0.10$). An increase in EB in girls aged 12 to 16 led to an unexpected increase in sdsBMI ($B = 0.07$). As there was no consistent relationship between EB and sdsBMI, investigating the possibility of a causal relationship with longitudinal multivariate genetic modelling was deemed to be redundant.

Discussion

This study examined the relationship between regular EB and BMI in youth, using repeated surveys in a population-based sample of 7-, 10-, 12-, 14-, 16- and 18-year-old twin individuals. Based on the hypothesis that regular exercise is a causal determinant of obesity, higher levels of EB in childhood and adolescence were expected to be associated with lower levels of BMI at all ages, and changes in EB across time were expected to predict opposite changes in BMI. The availability of twin data would have allowed an explicit test of the causal nature of these associations (13,22,23). Under the causal hypothesis, EB–BMI associations should derive from significant cross-trait correlations between the genetic and environmental factors influencing either trait. Moreover, genetically identical twins discordant for EB should have also been discordant for BMI, such that the twin with the highest level of exercise would have been leaner than the co-twin, in spite of an identical genome and a shared family environment.

None of our expectations were borne out by the data. Cross-sectionally, the linear and quadratic effects of EB on sdsBMI were mostly non-significant. In addition, there was no compelling evidence for a longitudinal association between EB and BMI. Increases in EB across time were not paralleled by decreases in sdsBMI, nor were decreases in EB paralleled by increases in sdsBMI. Further twin modelling of the causal nature of the association was considered moot, as no association was present.

Notwithstanding their counterintuitive nature, the results are rather well aligned with previous work. The few longitudinal studies focusing specifically on EB (as opposed to general PA) in large population-based samples found no robust association between EB and BMI (25–27), with one exception (28). Taken together, the current evidence does not point towards a lack of regular leisure time exercise as a major source of obesity in youth.

This does not, of course, preclude that other forms of PA have an effect on BMI. We deliberately choose to focus on the narrow trait of voluntary EB in leisure time. This salient voluntary behaviour can be reliably assessed by self-report through surveys on a scale of tens of

Table 5 Longitudinal prediction of sdsBMI by the linear and quadratic effects of weekly MET hours

Longitudinal follow-up time	Surveys	Predictors	Unstandardized beta (99.9% CI)	p-value
2-year interval	10 and 12	Sex*	0.00 (−0.16, 0.17)	0.95
		MET hours	−0.01 (−0.14, 0.11)	0.71
		MET hours squared	0.01 (−0.03, 0.05)	0.60
	12 and 14	Sex	0.03 (−0.07, 0.13)	0.26
		MET hours	−0.03 (−0.09, 0.04)	0.20
		MET hours squared	0.01 (−0.01, 0.04)	0.07
	14 and 16	Sex	−0.06 (−0.21, 0.08)	0.17
		MET hours	0.08 (−0.02, 0.17)	0.01
		MET hours squared	−0.01 (−0.04, 0.02)	0.18
	16 and 18	Sex	0.14 (−0.07, 0.35)	0.02
		MET hours	0.14 (0.01, 0.27)	5.92E−4
		MET hours squared	−0.01 (−0.04, 0.01)	0.09
3-year interval	7 and 10	Sex	0.02 (−0.22, 0.26)	0.80
		MET hours	−0.02 (−0.18, 0.13)	0.64
		MET hours squared	−0.03 (−0.09, 0.02)	0.05
4-year interval	10 and 14	Sex	0.02 (−0.17, 0.20)	0.78
		MET hours	0.03 (−0.10, 0.16)	0.51
		MET hours squared	0.00 (−0.06, 0.05)	0.81
	12 and 16	Sex	−0.03 (−0.15, 0.10)	0.48
		MET hours	0.03 (−0.05, 0.11)	0.24
		MET hours squared	0.01 (−0.03, 0.05)	0.54
	14 and 18	Sex	0.04 (−0.23, 0.30)	0.66
		MET hours	0.03 (−0.14, 0.20)	0.55
		MET hours squared	0.01 (−0.03, 0.04)	0.64
5-year interval	7 and 12	Sex	−0.06 (−0.31, 0.19)	0.45
		MET hours	0.01 (−0.17, 0.19)	0.81
		MET hours squared	−0.02 (−0.08, 0.05)	0.38
6-year interval	10 and 16	Sex	0.07 (−0.24, 0.39)	0.45
		MET hours	0.08 (−0.15, 0.32)	0.24
		MET hours squared	−0.03 (−0.14, 0.08)	0.35
	12 and 18	Sex	0.06 (−0.12, 0.25)	0.25
		MET hours	0.05 (−0.06, 0.15)	0.17
		MET hours squared	−0.01 (−0.05, 0.04)	0.51
7-year interval	7 and 14	Sex	0.08 (−0.15, 0.32)	0.25
		MET hours	0.05 (−0.11, 0.20)	0.32
		MET hours squared	−0.02 (−0.07, 0.04)	0.34

*0 = male, 1 = female; dependent variable: sdsBMI.
BMI, body mass index; MET, metabolic equivalent of task; CI, confidence interval.

thousands of participants, which is a major asset for causal modelling in a longitudinal twin design. Furthermore, EB presents a well-defined and feasible target for intervention. From adolescence onwards, exercise activities in leisure time are the major source of exercise bouts for the majority of people with sufficient intensity and duration to increase or maintain cardiorespiratory fitness and to induce positive health outcomes. Habit formation in this domain can be maintained across the life course (in contrast to school-based physical education or free child play), and a large meta-analysis of 80 prospective studies in adults testing the effects of exercise on mortality in 1 338 143 participants (118 121 deaths) showed that the risk reduction per unit of time increase was largest for (moderate-to-)vigorous exercise (29).

Nonetheless, (high-intensity) leisure time exercise activities may only account for up to 25% of the total daily activity-induced energy expenditure (30). Other aspects of PA could still prove possible determinants of BMI in childhood and adolescence. Obvious aspects are the parts of daily PA in children and adolescents that were excluded here, including standing time, light activities and moderate-to-vigorous activities like cycling or walking to school, physical education classes (between 1 and 3 h a week in the Netherlands), free play, dance and household or job-related PA (Dutch children can work up to 4 h a week on non-schooldays from age 13 onwards). There might be no difference in calories burned between a child that participates in scheduled exercise activities but is largely sedentary the remaining time and

Table 6 Correlations (99% CI) between *change* in weekly MET hours (MET hours at time point 2 − MET hours at time point 1) and *change* in sdsBMI (sdsBMI at time point 2 − sdsBMI at time point 1), for male and female individuals separately

Longitudinal follow-up time	Surveys	Correlation between ΔMET hours and ΔsdsBMI*	
		Male	Female
2-year interval	10 and 12	−0.01 (−0.11, 0.09)	0.02 (−0.08, 0.12)
	12 and 14	−0.01 (−0.07, 0.06)	−0.03 (−0.09, 0.03)
	14 and 16	−0.02 (−0.11, 0.07)	0.01 (−0.06, 0.08)
	16 and 18	0.01 (−0.11, 0.14)	0.05 (−0.04, 0.14)
3-year interval	7 and 10	−0.03 (−0.19, 0.12)	0.01 (−0.14, 0.15)
4-year interval	10 and 14	0.10 (−0.02, 0.21)	0.03 (−0.09, 0.14)
	12 and 16	0.05 (−0.03, 0.13)	0.07 (0.01, 0.14)
	14 and 18	0.06 (−0.10, 0.21)	−0.01 (−0.12, 0.11)
5-year interval	7 and 12	−0.06 (−0.24, 0.12)	−0.14 (−0.30, 0.02)
6-year interval	10 and 16	0.09 (−0.14, 0.31)	0.10 (−0.09, 0.27)
	12 and 18	0.05 (−0.06, 0.16)	−0.01 (−0.10, 0.07)
7-year interval	7 and 14	0.05 (−0.13, 0.23)	0.01 (−0.13, 0.14)

*For example, for '10 and 12', this would be (MET hours at age 12 − MET hours at age 10) × (sdsBMI at age 12 − sdsBMI at age 10).
CI, confidence interval; MET, metabolic equivalent of task; BMI, body mass index.

a child that does not participate in scheduled exercise but is actively playing and commuting to school. The effect of each aspect of PA on BMI, their change over time (e.g. free play might be more important in younger children) and their relationships with each other should be investigated more closely (31).

A second obvious aspect of PA that could influence BMI in childhood and adolescence is the amount of physical *in*activity, or sedentary behaviours. Sedentary behaviours are defined as activities that are performed sitting or reclining and cost ≤1.5 times the basal metabolic rate. In adults, many negative health outcomes, including high BMI, have been reported to follow from sedentary behaviours, independent of PA levels (32–34). Similar detrimental effects appear to occur already in children and adolescents, although the evidence is still incomplete (35). Importantly, the association between sedentary behaviours and EB itself is weak at best, at least in adults (36). Hence, high EB can co-occur with low levels of sedentary behaviours and vice versa, distorting potential causal effects of EB on BMI.

Although future investigation may reveal an effect on BMI of these aspects of daily physical (in)activity other than EB, the results of the present study are also rather well aligned with a major role of the alternative determinant of BMI: eating behaviour patterns. Bleich, Ku and Wang reviewed the literature on the relative contribution of energy intake and energy expenditure to obesity in childhood and adolescence, with mixed results (6). A main reason for the blurry picture were the large differences between studies in terms of methods used and populations studied. Furthermore, the assessment of food intake is prone to reporting errors. To overcome this problem, Waxman and Stunkard observed four obese boys, their non-obese brother and a peer for 4–5 months in their natural environments and monitored their eating behaviour and PA (37). They concluded that – compared with their controls – energy intake was higher, but energy expenditure was not lower in the obese boys. Model-based equations on the association between energy intake, energy expenditure and energy balance, both at the population level and at the individual level, confirm energy intake as the factor driving the obesity epidemic (38). For instance, Westerterp *et al*. reviewed studies that were based on the doubly labelled water method from the early 1980s onwards and concluded that energy expenditure has not decreased since then in spite of the substantial increase in the prevalence of obesity (39). Moreover, activity-related energy expenditure in modern day humans does not deviate from that of other terrestrial mammals, after taking differences in body size into account (40).

In adults, there is now good evidence that increases in energy intake can come about as a compensatory reaction to EB itself (41). Exercise may increase the amount of food that an individual eats, and it may amplify the preference for high-fat, energy-dense foods. Moreover, starting an exercise programme might lead to less non-exercise activity – either owing to physiologically caused fatigue or because of a feeling that one can afford to rest more because of the activity. Westerterp *et al*. highlight that humans are better at compensating for a negative energy imbalance compared with a positive energy imbalance (39). It is not known whether compensatory eating occurs in children or adolescents, but if the twins participating in this study indeed show increased eating with

higher levels of EB, this could have caused the absence of an EB–BMI relationship. Unfortunately, no food intake was assessed in any of the survey waves.

Apart from the absence of food intake data, this study had further limitations that should be noted. First of all, individual differences in basal metabolic rate (BMR) were not assessed, although in sedentary subjects, they can account for around 60% of the total energy expenditure (30). BMR is not currently a feasible target for intervention, but it may be a determinant of individual differences in BMI (42). Secondly, we did not correct for growth and maturation. Body height and body weight change dramatically as children develop from age 7 into adulthood. Growth and maturation may affect both BMI and PA (and vice versa), thereby undermining the detection of a relationship between the two. These limitations were somewhat attenuated by using sdsBMI, which provides a standardized ranking of the participants using sex-specific and age-specific BMI scores of the Dutch population, but such standardization does not remove the effects of variance in growth and maturation that can exist within each sex/age stratum. In addition, BMI contains both fat mass and fat-free mass, and we could not separate the effects of exercise on these compartments here. Although large effects of EB on fat-free mass are not anticipated in children and adolescents, they could potentially have masked parallel reductions in fat mass. Finally, we assessed height and weight by parental report and self-report, which can induce a reporting bias. However, self-reported body height and weight have been shown to be strongly correlated to actual body height and weight (43). Also, the use of self-report allowed us to create the largest genetically informative longitudinal data set in the world uniformly assessing EB and BMI across the entire age range from childhood to young adulthood, which is a major strength of the study.

In conclusion, we found no evidence for a cross-sectional or longitudinal association between EB and BMI across childhood and adolescence. Alternative determinants of BMI such as BMR, other aspects of daily PA and sedentary behaviour, but prominently also energy intake, are likely to be more important. It should be explicitly mentioned here that this does not detract from the value of encouraging regular EB in youth, as it has been shown to have many other favourable effects on health, even in the absence of an effect on body weight, and should thus still be promoted (41). Claiming a primary role for EB in the variation of childhood and adolescent BMI, however, may foster false expectations.

Conflict of Interest Statement

No conflict of interest statement.

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