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Genetic and Environmental Influences on Individual Differences in Sedentary Behavior During Adolescence

A Twin-Family Study

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Objective: To investigate the degree to which genetic and environmental influences affect individual differences in sedentary behavior throughout adolescence.

Design: Cross-sectional twin-family design.

Setting: Data on self-reported sedentary behavior from Dutch twins and their nontwin siblings.

Participants: The total sample consisted of 5074 adolescent twins (aged 13-19 years) and 937 siblings (aged 12-20 years) from 2777 families.

Main Outcome Measures: Screen-viewing sedentary behavior was assessed with survey items about weekly frequency of television viewing, playing electronic games, and computer/Internet use. Based on these items, an overall score for screen-viewing sedentary behavior was computed.

Results: The genetic architecture of screen-viewing sedentary behavior differed by age. Variation in sedentary behavior among 12-year-olds was accounted for by genetic (boys: 35%; girls: 19%), shared environmental (boys: 29%; girls: 48%), and nonshared environmental (boys: 36%; girls: 34%) factors. Variation in sedentary behavior among 20-year-olds was accounted for by genetic (boys: 48%; girls: 34%) and nonshared environmental (boys: 52%; girls: 66%) factors.

Conclusion: The shift from shared environmental factors in the etiology of sedentary behavior among younger adolescents to genetic and nonshared environmental factors among older adolescents requires age-specific tailoring of intervention programs.

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ENGAGING IN SEDENTARY BEHAVIOR (SB), and screen-viewing behaviors in particular, has been identified as a risk factor for weight gain and metabolic disorders.[1-4] Studying SB during adolescence is of particular interest because overweight and obesity are likely to track into adulthood[5,6] and screen-viewing sedentary activities are dominant leisure time activities during adolescence.[7,8] To inform prevention and intervention strategies aiming to reduce time spent in sedentary activities, insight into the etiology of SB is important. The few available studies on the etiology of SB indicate that family environmental factors such as parental modeling (eg, rules and restrictions, parental SB) and availability of screen-viewing opportunities in the home are important correlates of engaging in SB in youth[9-11] but did not address possible genetic influences on the likelihood of engaging in SB.

With data from twins and their siblings, individual differences in SB can be decomposed as being due to genetic, shared environmental (environmental influences shared by members of the same family), and nonshared environmental (environmental influences unique to an individual) influences. In the only study, to our knowledge, examining the relative influence of genetic and environmental factors on individual differences in SB to date, Nelson et al[12] reported variation in adolescent SB to be accounted for by genetic (34%), shared environmental (10%), and nonshared environmental (56%) factors. When the same sample was reassessed during early adulthood, individual differences in SB were accounted for by genetic (32%) and nonshared environmental (68%) factors. These results indicate that shared environmental influences on individual differences in SB diminish during the transition from adolescence to early adulthood. Diminishing influence of shared

Table 1. Sample Constellation

	No. of Individuals	No. of Families	1 Twin	2 Twins	0 Twins + Sibling	1 Twin + Sibling	2 Twins + Siblings
MZM	1003	437	25	248	. . .	10	154
DZM	844	382	40	208	. . .	14	120
MZF	1424	627	44	357	. . .	12	214
DZF	1020	460	42	263	. . .	13	142
DOS	1737	820	125	439	. . .	34	222
Sibling only	42	42	. . .	. . .	42	. . .	. . .
Total	**6070**	**2768**	**276**	**1515**	**42**	**83**	**852**

Abbreviations: DOS, dizygotic opposite-sex twin pair; DZF, dizygotic female twin pairs; DZM, dizygotic male twin pair; ellipses, not applicable; MZM, monozygotic male twin pair; MZF, monozygotic female twin pair; Sibling only, families with data from nontwin siblings only; 1 Twin, families with data from 1 twin (incomplete twin pair); 2 Twins, families with data from complete twin pairs; 0 Twins + Sibling, families with data from 1 sibling; 1 Twin + Sibling, families with data from 1 twin (incomplete twin pair) and 1 sibling; 2 Twins + Siblings, families with data from a complete twin pair and 1 additional sibling.

environmental factors throughout adolescence has also been found for other behavioral traits, eg, exercise behavior[13,14] and psychopathology.[15,16]

In the present study, we assessed SB in a large sample of Dutch adolescent twins and their nontwin siblings in the age range of 12 to 20 years. Sedentary behavior was defined as the frequency of television viewing, playing electronic games, and engagement in personal computer/Internet activities. The main objective was to estimate the genetic and environmental contribution (ie, the genetic architecture) to individual differences in self-reported SB in adolescence as a function of age. In addition, we assessed whether there were sex differences in the genetic architecture of SB.

METHODS

PARTICIPANTS

The Netherlands Twin Registry (NTR) contacts Dutch families with young twins from all regions of the Netherlands within the first few months after birth of the twins with the request for registration in the NTR. In 2005, the NTR started to collect data on behavior, well-being, lifestyle, and health in adolescent twins and their nontwin siblings by self-report.[17,18] During adolescence, 14-, 16-, and 18-year-old twins and their nontwin siblings (aged 12-25 years) receive an online or a paper-and-pencil self-report survey, on written parental consent. A detailed description of the data collection procedures among adolescent twins registered with the NTR was reported elsewhere,[19] including nonresponse analyses showing that responding and nonresponding twins were comparable on several characteristics. This study was approved by an institutional review board on human research.

For the present study, data from twins born between 1986 and 1992 were included plus data from 1 additional nontwin sibling. From families with more than 1 additional sibling, we selected the sibling closest in age to the twins, which resulted in the exclusion of 150 siblings. Data on SB were available for a total sample consisting of 5090 twins (44% male) and 980 nontwin siblings (45% male) from 2768 families. In **Table 1**, the exact constellation of the participating families is presented. Age of the twins and siblings ranged between 12 and 20 years with a mean (SD) age of 15.93 (1.60) years. For 743 (39.0%) of the same-sex twin pairs, zygosity was determined based on blood group or DNA typing. Zygosity for the remaining same-sex twin pairs was determined by questionnaire items about physical similarities and confusion by family members and strangers, which were provided by parents at multiple times in previous questionnaires. These items allow for accurate determination of zygosity in 93% of same-sex twin pairs.[20]

MEASURES

Data on SB were primarily collected by one of us (N.V.), who was also instructed about the data collection procedures of the NTR. Participants were asked to report their weekly frequency of watching television, gaming, and engaging in personal computer/Internet activities during leisure time on 7-point scales (1: never; 2: once until now; 3: less than 1 time per week; 4: once a week; 5: a couple of days per week; 6: almost every day; 7: every day). Scores on these 3 items were summed to get an overall score for the weekly frequency of SB, ranging from 3 to 21. This variable was normally distributed (skewness: –0.03; kurtosis: 0.30). A factor analysis of the items indicated a single factor explaining 43.1% of the variance.

STATISTICAL ANALYSES

In the following section, the statistical methods are briefly described. A more elaborate description is presented in the eAppendix (http://www.archpediatrics.com). The amount of variation in SB due to additive genetic (A), shared environmental (C), and nonshared environmental (E) factors can be estimated by comparing the resemblance in SB between monozygotic (MZ) and dizygotic (DZ) twins and nontwin siblings. This is based on the fact that MZ twin pairs are genetically identical, whereas DZ twin pairs and twin-sibling pairs share on average 50% of their segregating genes. When the resemblance in SB in MZ twin pairs is higher than the resemblance in DZ twin or twin-sibling pairs, genetic variation is likely to influence individual differences in SB.[19,21] When MZ twin pairs resemble each other more than DZ twin and twin-sibling pairs, but not to the extent that would be expected based on their twice-larger genetic resemblance, this implies that shared environmental factors influence variation in SB.[19,21] Differences between MZ twins are attributed to nonshared environment.[19,21] This component also includes measurement error.

Resemblance in SB is expressed in twin and twin-sibling correlations and these were estimated for each of the 5 sex × zygosity groups as well as for twin-sibling pairs. Because twin and twin-sibling correlations are corrected for age, they represent twin and twin-sibling correlations at the mean age in the sample. To assess age and sex differences in mean levels and variation in SB, these were estimated conditional on sex and age.

Genetic structural equation modeling in the software package Mx[22] was used to estimate the contribution of A, C, and E to variation in SB (**Figure 1**). A moderator model as de-

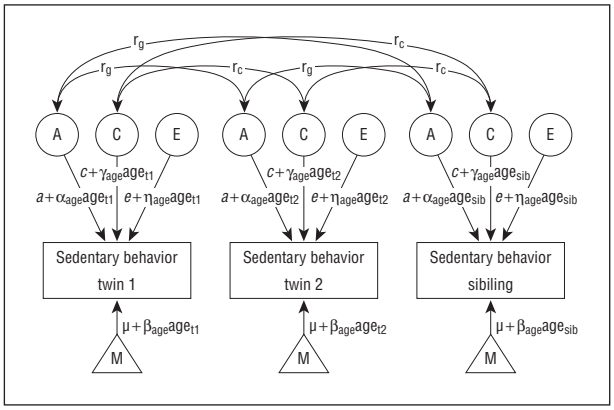

Figure 1. Genetic model for sedentary behavior with moderating effects of age on genetic and environmental path coefficients. Squares represent measured sedentary behavior. Triangles represent mean level of sedentary behavior (M). The total variance in sedentary behavior is modeled as caused by additive genetic influences (A), common or shared environment (C), and nonshared environment (E). Under this model, *a*, *c*, and *e* represent the unmoderated genetic, shared environmental, and nonshared environmental path coefficients, respectively, and the α, γ, and η coefficients represent the moderating effects of age. If, for example, α is significantly different from zero, the magnitude of A changes as a linear function of age. Path coefficients *a*, *c*, and *e*, as well as the α, γ, and η coefficients, were allowed to differ for boys and girls. Genetic correlation (r_g): monozygotic twin pairs = 1; dizygotic twin pairs and twin-sibling pairs = 0.5; shared environmental correlation (r_c) = 1. t1 Indicates the first twin of a twin pair; t2, the second twin of a twin pair; and sib, sibling.

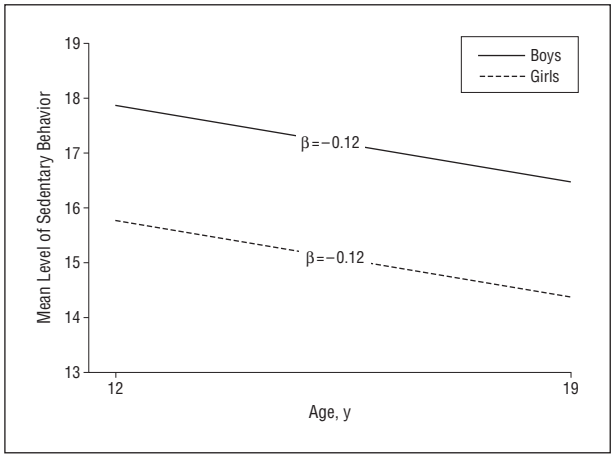

Figure 2. Changes in mean levels of sedentary behavior as a function of sex and age. Standardized regression coefficient β = −0.12 r^2 = 0.014.

Table 2. Twin and Twin-Sibling Correlations Corrected for Age and Their 95% CIs for Sedentary Behavior

	r	95% CI
MZ male	0.47	0.40-0.53
DZ male	0.25	0.14-0.34
MZ female	0.58	0.52-0.63
DZ female	0.44	0.34-0.51
DZ opposite sex	0.25	0.18-0.31
Brother-brother	0.25	0.16-0.33
Sister-sister	0.28	0.22-0.34
Brother-sister	0.29	0.19-0.38

Abbreviations: CI, confidence interval; DZ, dizygotic; MZ, monozygotic.

scribed by Purcell[23] was fitted to the data in which age (*z* score) was regressed on the genetic (*a*), shared environmental (*c*), and nonshared environmental (*e*) path coefficient. In Figure 1, regression weights of age are presented by the α, γ, and η coefficients. The unmoderated estimates of *a*, *c*, and *e* represent the parameter estimates used to calculate the variance components A, C, and E at the mean age in the sample. To assess sex differences in the genetic architecture of SB, regression coefficients of age as well as path coefficients *a*, *c*, and *e* were allowed to differ for boys and girls, and it was tested whether constraining these parameter estimates to be equal for boys and girls reduced model fit significantly. The significance of the age effects was assessed by testing whether constraining the regression weights of age at zero resulted in a significant deterioration of model fit. To assess whether genetic and shared environmental factors contributed significantly to variation in SB, we tested whether constraining the unmoderated parameter estimates *a* and *c* at zero resulted in a significant deterioration of model fit. The fit of submodels was compared by means of the log-likelihood ratio test and the Akaike Information Criterion, keeping the model with the lowest Akaike Information Criterion as the best-fitting model.[22]

RESULTS

Figure 2 presents the mean levels of SB as a function of sex and age. The means (SD) of SB were 17.1 (2.9) and 15.0 (2.4) for boys and girls, respectively. Sedentary behavior was significantly higher for boys than girls (χ^2_1=755.56; $P<.05$). A significant effect of age was found on SB (χ^2_1=81.54; $P<.05$), indicating higher levels of SB in younger participants. A significant negative effect of age was found on the variance (χ^2_1=19.66; $P<.05$), indicating that variance in SB declines with increasing age. Variation in SB was also larger for boys than girls (χ^2_1=104.58; $P<.05$).

Twin and twin-sibling correlations for SB are presented in **Table 2**. Dizygotic twin correlations were not significantly different from twin-sibling correlations (χ^2_3=5.41; $P=.14$). Monozygotic twin correlations were significantly higher than the DZ twin/twin-sibling correlations for boys (χ^2_1=21.22; $P<.05$) and girls (χ^2_1=25.45; $P<.05$). This suggests that individual differences in SB are influenced by genetic factors. For boys, DZ twin/twin-sibling correlations were about half the MZ twin correlation, suggesting that shared environmental factors play no role in explaining variation in SB. For girls, DZ twin/twin-sibling correlation was higher than half the MZ twin correlation, suggesting shared environmental influence. Because the correlation structure suggested sex differences in the genetic architecture of SB, genetic modeling was started with an ACE model with different parameter estimates for boys and girls.

Table 3 presents the model fitting results of the genetic models. In model 2, the moderation effects of age on the path coefficients of SB were constrained to be equal between boys and girls, which did not reduce model fit significantly. This suggests that there are no sex differences in the magnitude of age effects on the genetic architecture of SB. Model 3 tested the statistical significance of the moderation effects of age on the path coefficients of SB, which resulted in a significant deterioration of model fit, indicating that the magnitude of genetic, shared environmental, and nonshared environmental effects on variation in SB changes as a function of age.

Table 3. Model Fitting Results for Sedentary Behavior

Model	vs Model	–2LL	*df*	χ^2	Δdf	*P* Value	AIC
1 ACE model	. . .	27977.981	6008	. . .	. . .	. . .	. . .
2. $\alpha_{boys}=\alpha_{girls}$, $\gamma_{boys}=\gamma_{girls}$, $\eta_{boys}=\eta_{girls}$[a]	1	27982.151	6011	4.17	3	.24	–3.19
3. $\alpha_{boys}=\alpha_{girls}=0$, $\gamma_{boys}=\gamma_{girls}=0$, $\eta_{boys}=\eta_{girls}=0$	2	28019.761	6014	37.61	3	<.001	28.42
4. $a_{boys}=a_{girls}$, $c_{boys}=c_{girls}$, $e_{boys}=e_{girls}$	2	28091.621	6014	109.47	3	<.001	100.28
5. $a_{boys}=0$	2	28006.402	6012	24.25	1	<.001	19.06
6. $a_{girls}=0$	2	28004.646	6012	22.50	1	<.001	17.31
7. $c_{boys}=0$	2	27987.018	6012	4.87	1	.03	–0.32
8. $c_{girls}=0$	2	27990.879	6012	8.73	1	<.001	3.54

Abbreviations: *a*, genetic path coefficient; ACE model, ACE model with moderating effects of age and different parameter estimates for boys and girls; AIC, Akaike Information Criterion; *c*, shared environmental path coefficient; *e*, nonshared environmental path coefficient; ellipses, not applicable; –2LL, –2 log likelihood; Δdf, *df* of χ^2 test.

[a] Most parsimonious model.

Model 4 tested whether constraining the genetic, shared environmental, and nonshared environmental parameter estimate to be equal for boys and girls led to a significant deterioration of model fit. There appeared to be significant sex differences in the magnitude of the genetic, shared environmental, and nonshared environmental path coefficients, suggesting that the contribution of genetic, shared environmental, and nonshared environmental factors to individual differences in SB differs for boys and girls. Therefore, parameter estimates *a*, *c*, and *e* were allowed to differ between boys and girls.

Models 5 through 8 tested whether constraining the genetic or shared environmental parameter estimate to zero would reduce model fit significantly. The contribution of additive genetic and shared environmental factors to individual differences in SB was statistically significant for boys and girls. The log-likelihood ratio tests and the Akaike Information Criterion pointed to the ACE model with significant age effects on the path coefficients as the most parsimonious models for boys and girls. This indicates that individual differences in adolescent SB are accounted for by additive genetic, shared environmental, and nonshared environmental factors and that the genetic architecture of adolescent SB differs as a function of age.

Figure 3 presents the unstandardized (parts A and C) and standardized (parts B and D) contributions of genetic, shared environmental, and nonshared environmental effects to variation in SB as a function of age and sex, derived from the most parsimonious model (model 2). The contribution of shared environmental effects to variation in SB was larger among younger compared with older adolescents. The absolute contribution of genetic and nonshared environmental effects to variation in SB (Figure 3A and C) was similar at all ages, whereas the relative contribution of genetic and nonshared environmental effects showed substantial increase among older adolescents (Figure 3B and D). This is due to the diminishing part of variation accounted for by shared environmental factors among younger adolescents, leading to an overall decreased variation in SB throughout adolescence.

COMMENT

In a large sample of Dutch adolescent twins and their nontwin siblings, we found that older adolescents are less frequently engaged in SB (television viewing, gaming, and Internet activities) than younger adolescents and that boys were more often sedentary than girls. Variation in SB was accounted for by genetic, shared environmental, and nonshared environmental factors. Heritability of SB was larger in boys than in girls. In addition, we found that the genetic architecture changed throughout adolescence. Shared environmental effects on SB were larger among younger adolescents, whereas genetic and nonshared environmental effects were larger among older adolescents.

Our finding that the frequency of sedentary activities was lower in older compared with younger adolescents corresponds with the results of other studies.[24,25] Additional analyses showed that the lower overall SB score observed among older adolescents was due to a lower frequency of television viewing and gaming among older participants. An explanation for the overall lower levels of screen-viewing activities among older adolescents may be that other activities increasingly compete with these sedentary activities. Sex differences in the overall SB score were mainly due to a higher frequency of playing electronic games for boys. Even though boys were more frequently engaged in sedentary activities, this does not mean that boys are physically less active than girls, since boys are also more engaged in exercise than girls.[13,14]

The main aim of the present study was to assess to what extent genetic and environmental factors affect SB during adolescence. Age significantly modified the genetic architecture of SB. As adolescents grow older and become more independent, individual differences in their SB are increasingly determined by their genetic makeup and factors from their personal environment. In other words, changes in the social and economical environments of adolescents may cause some adolescents to spend less time on sedentary activities, whereas for others, sedentary activities remain dominant leisure time activities. Additional analyses in our data indicated that adolescents enrolled in lower secondary education reported higher levels of SB than those enrolled in moderate and high secondary education. Lower levels of SB were also associated with engagement in after-school employment. Higher levels of SB were reported by participants who frequently spend time in social interaction with peers and going out during the week vs those reporting to be less frequently engaged in these activities.

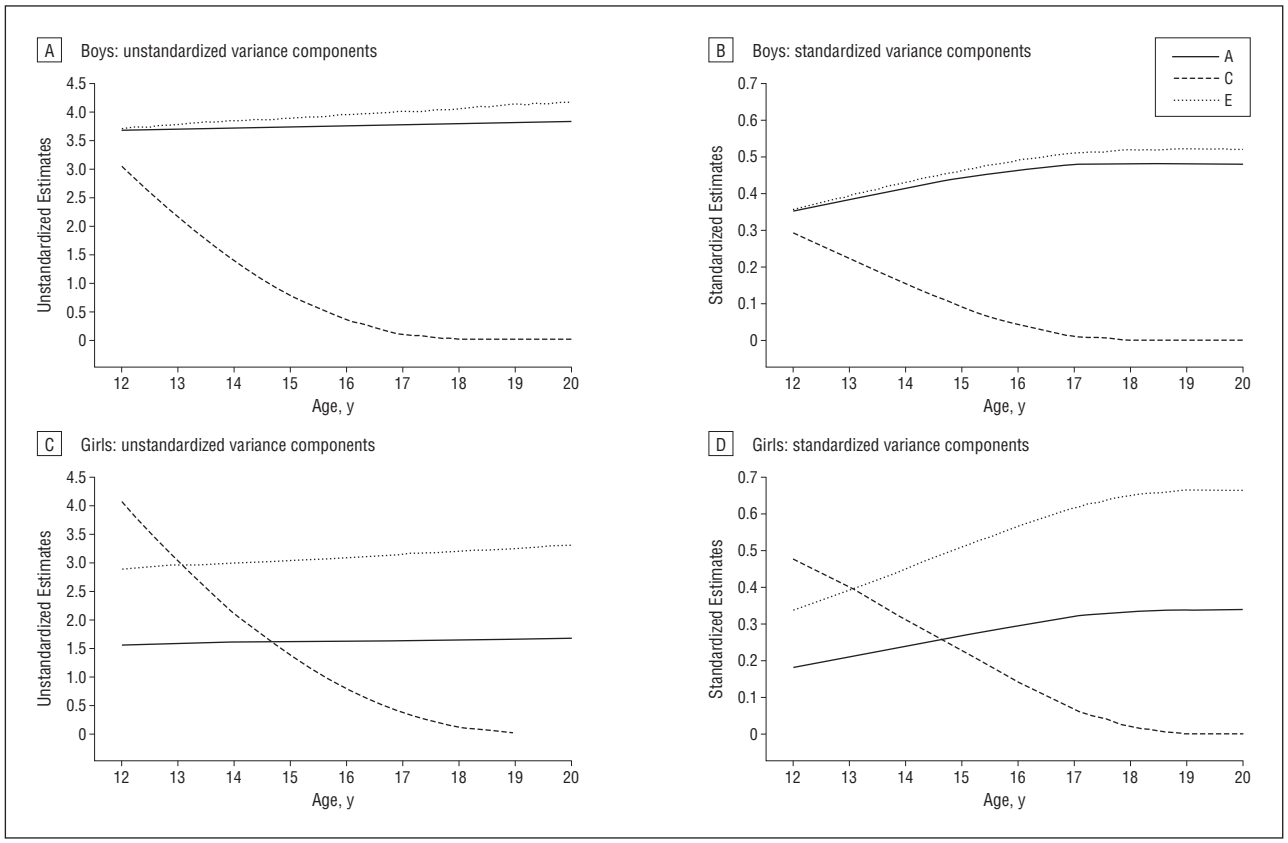

Figure 3. Changes in the absolute and relative contribution of genetic, shared environmental, and nonshared environmental effects to variation in sedentary behavior as a function of age for boys and girls.

The relative larger contribution of genetic and nonshared environmental factors to variation in SB among older adolescents is not due to an increase in genetic and nonshared environmental variation, since the absolute contribution of genetic and nonshared environmental effects was similar at all ages. The relative increased importance of genetic and nonshared environmental effects on SB was due to the diminishing contribution of shared environmental effects to variation in SB, leading to an overall decrease in the variation in SB with increasing age.

The substantial influence of genetic factors on adolescent SB might have important implications for intervention strategies aiming at reduction of screen-time activities. For adolescents, sedentary activities such as television viewing and engagement in personal computer and Internet activities are frequent leisure time activities.[7,8] The substantial genetic influence on SB suggests that there is a genetic liability toward such sedentary activities, which might complicate prevention and intervention strategies. It may be that interventions aiming to reduce the availability and accessibility of screen-time opportunities, ie, interventions that restrict opportunities to act according to a genetically defined "preference," have better perspectives than health education–like interventions trying to educate and convince youngsters to change their behavior. Earlier studies indicated that adolescents with easier access to screen-time opportunities, eg, having a television in the bedroom, are more likely to engage in screen-time activities.[26,27] However, restricting certain sedentary activities, particularly in late adolescence, is not feasible and it may not make adolescents give up sedentary activities but rather result in compensatory forms of sedentary activities.[25] Interventions focusing on offering alternative activities and promoting regular interruptions of SB may have more potential. Recently, evidence was found that SB of prolonged duration, but not interrupted SB, is unfavorable for metabolic disorders.[28]

Shared environmental factors contributed substantially to individual differences in SB among younger adolescents and largely explain the difference in total variance in SB between younger and older adolescents. Shared environmental factors may include the influence of parents' SB and parental monitoring of their children's SB. Such factors have already been shown to be associated with SB in youth.[9-11] The importance of shared environmental effects on SB among younger adolescents supports prevention and intervention strategies to target families rather than the individual adolescent.

The genetic architecture of adolescent SB has been addressed in a previous study.[12] We expanded on that study by explicitly modeling age moderation and sex differences but our findings are generally consistent with those reported by Nelson et al.[12] They also found a contribution of shared environmental factors that disappeared with increasing age and additionally assessed changes in the genetic architecture of SB after adolescence during the transition to young adulthood. Nonshared environmental factors were found to become increasingly important at the expense of genetic factors during this transition.

While interpreting the results, the following limitation should be kept in mind. The composite score that was used as a measure of SB has not previously been validated. It was based on self-reported weekly frequency of 3 sedentary activities while duration of these activities was not taken into account. An objective measure of SB (eg, accelerometry) would have been more reliable. However, to enable genetic analyses, large sample sizes are needed, and therefore, self-report survey research is the most feasible way of data collection.

Our data showed that variation in adolescent SB was largely accounted for by genetic and nonshared environmental factors, whereas shared environmental factors account for a substantial part of the variation among younger adolescents. The shift from shared environmental factors in the etiology of SB among younger adolescents to genetic and nonshared environmental factors among older adolescents has consequences for intervention programs that aim to reduce SB. These require specific tailoring to age groups and need to focus on peers and parents in early adolescence but on the youngsters themselves at later ages.

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Author Contributions: Dr van der Aa (the principal investigator) had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* van der Aa, Bartels, te Velde, de Geus, and Brug. *Acquisition of data:* van der Aa, Bartels, and Boomsma. *Analysis and interpretation of data:* van der Aa, Bartels, te Velde, Boomsma, and de Geus. *Drafting of the manuscript:* van der Aa, Bartels, and Brug. *Critical revision of the manuscript for important intellectual content:* van der Aa, Bartels, te Velde, Boomsma, de Geus, and Brug. *Statistical analysis:* van der Aa, Bartels, and Boomsma. *Obtained funding:* Bartels, te Velde, and Brug. *Administrative, technical, and material support:* Bartels and te Velde. *Study supervision:* Bartels, te Velde, and Brug.
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Online-Only Material: The eAppendix is available at http://www.archpediatrics.com.

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