

Behav Genet (2011) 41:211–222
DOI 10.1007/s10519-010-9415-4

ORIGINAL RESEARCH

Exercise Participation in Adolescents and Their Parents: Evidence for Genetic and Generation Specific Environmental Effects

Marleen H. M. De Moor · Gonneke Willemsen ·
Irene Rebollo-Mesa · Janine H. Stubbe ·
Eco J. C. De Geus · Dorret I. Boomsma

Received: 5 November 2008 / Accepted: 1 November 2010 / Published online: 18 November 2010
© Springer Science+Business Media, LLC 2010

Abstract Individual differences in adolescent exercise behavior are to a large extent explained by shared environmental factors. The aim of this study was to explore to what extent this shared environment represents effects of cultural transmission of parents to their offspring, generation specific environmental effects or assortative mating. Survey data on leisure-time exercise behavior were available from 3,525 adolescent twins and their siblings (13–18 years) and 3,138 parents from 1,736 families registered at the Netherlands Twin Registry. Data were also available from 5,471 adult twins, their siblings and spouses similar in age to the parents. Exercise participation (No/Yes, using a cut-off criterion of 4 metabolic equivalents and 60 min weekly) was based on questions on type, frequency and duration of exercise. A model to analyze dichotomous data from twins, siblings and parents including differences in variance decomposition across sex and generation was developed. Data from adult twins and their spouses were used to investigate the causes of assortative mating (correlation between spouses = 0.41, due to phenotypic assortment). The heritability of exercise in the adult generation was estimated at 42%. The shared environment for exercise behavior in adolescents mainly

represents generation specific shared environmental influences that seem somewhat more important in explaining familial clustering in girls than in boys (52 versus 41%). A small effect of vertical cultural transmission was found for boys only (3%). The remaining familial clustering for exercise behavior was explained by additive genetic factors (42% in boys and 36% in girls). Future studies on adolescent exercise behavior should focus on identification of the generation specific environmental factors.

Keywords Exercise participation · Parents · Twins · Genetic transmission · Cultural transmission · Assortative mating · Adolescence · Adulthood

Introduction

A sedentary lifestyle increases the risk for cardiovascular morbidity and mortality (Berlin and Colditz 1990; Martinez-Gonzalez et al. 1999; Albright et al. 2000; Kaplan et al. 1996). In spite of this well-established fact, about half of the population in Western societies remains sedentary and does not exercise on a regular basis (Martinez-Gonzalez et al. 2001; Steptoe et al. 1997). Traditionally, studies on exercise behavior have focused on environmental determinants, such as health beliefs, social support, perceived lack of time and access to facilities (King et al. 1992). Twin studies have demonstrated that genetic factors also play a substantial role in adult exercise participation (Beunen and Thomis 1999; Lauderdale et al. 1997; Kujala et al. 2002; Stubbe et al. 2006; Eriksson et al. 2006). These studies show that individual differences in adult exercise behavior can be explained by a combination of additive genetic (A) and unique environmental (E) factors, with heritability estimates ranging between 35% and 80%.

Edited by Hermine Maes.

The Mx script developed for the analyses in this paper can be requested from the first author. The script is an extension of the Mx script for the factor model provided by Neale et al. (1994).

M. H. M. De Moor (✉) · G. Willemsen · I. Rebollo-Mesa ·
J. H. Stubbe · E. J. C. De Geus · D. I. Boomsma
Department of Biological Psychology, VU University
Amsterdam, van der Boechorststraat 1, 1081 BT Amsterdam,
The Netherlands
e-mail: mhm.de.moor@psy.vu.nl

In adolescents, shared environmental (C) factors are of major importance in explaining variation in exercise behavior. In a study of twins aged 13–20 years (Stubbe et al. 2005), exercise behavior in young adolescents (up to 16 years) was largely determined by shared environmental factors, accounting for 78–84% of the variance. The relatively small opposite-sex (DOS) twin correlation compared with the dizygotic (DZ) same-sex twin correlations suggested that the shared environmental factors influencing exercise in boys and girls are partly different. The influence of these factors rapidly wanes when adolescents become young adults and genetic factors start to become of importance. Other twin studies (Maia et al. 2002; Carlsson et al. 2006) also show that family resemblance in adolescent leisure-time exercise behavior is explained by a combination of additive genetic and shared environmental factors but that shared environmental factors cease to exert their influence in young adulthood.

The shared environmental factors in adolescence can represent several different types of environmental factors that are shared among offspring from the same family. For example, it could consist of the influence of parents on their children's behavior (through their own physical activity levels or social support), environmental factors that are shared within a family such as the neighborhood or socio-economic status or environmental factors that are specific to the adolescent generation, such as the influence of siblings, friends, peers or the school, or a combination of these factors. All these factors have appeared in the literature as correlates of adolescent exercise behavior (Sallis et al. 2000; Gustafson and Rhodes 2006). An alternative explanation for the shared environmental factors is non-random mating with respect to exercise behavior. If not explicitly modeled non-random mating will suggest the presence of shared environment in the classical twin design. In this design, it is assumed that the correlation between additive genetic factors in monozygotic (MZ) twin pairs equals unity and 0.5 in dizygotic (DZ) twin pairs (Neale and Cardon 1992). The value of 0.5 comes from the expected additive genetic correlation in first degree relatives under random mating (Falconer and Mackay 1996). Non-random mating refers to the phenomenon that the phenotypes of spouses are correlated. A number of studies have reported a significant spouse correlation for exercise behavior, ranging from 0.16 to 0.60 (Aarnio et al. 1997; Boomsma et al. 1989; Perusse et al. 1989; Perusse et al. 1988; Seabra et al. 2008).

The consequences of assortative mating depend on the mechanisms that lead to the spouse correlation in exercise behavior. These include social interaction, social homogamy and phenotypic assortment (Heath and Eaves 1985; Reynolds et al. 2006; van Leeuwen et al. 2008). Social interaction refers to the phenomenon that spouses mutually

influence each other because they spend time together. Their phenotypes are not necessarily correlated at the time they first meet, but they become more similar during the course of their relationship. Therefore, if social interaction explains the spouse correlation, it is expected that the spouse correlation increases when partners are together for a longer time. Social homogamy is the tendency that individuals coming from similar social backgrounds are more likely to meet and marry each other. This process would also induce a positive spouse correlation. A third type of explanation for a positive spouse correlation is phenotypic assortment, which occurs when individuals select each other based on the phenotype under study (or a correlated phenotype). If phenotypic assortment is present, this increases the additive genetic variance in the offspring generation and the covariance between additive genetic factors in first degree relatives. If this increased genetic resemblance is not accounted for in the twin model, and an additive genetic correlation of 0.5 is assumed, this leads to overestimation of shared environmental effects.

Extending the classical twin design with additional family members such as non-twin siblings and parents (Fulker 1989; Eaves et al. 1978; Heath et al. 1985; Boomsma and Molenaar 1987; Maes et al. 2009; Keller et al. 2009) makes it possible to partition the shared environmental effects found in adolescent exercise behavior into the effects of the parental phenotype on offspring behavior (vertical cultural transmission), environmental effects that are shared among offspring but non-shared with the parents (horizontal cultural transmission) and the effects of assortative mating. These effects can be separated by modeling the correlations between parents, between parents and their offspring and between twins and siblings. A significant spouse correlation suggests that some of the shared environmental effects may be explained by assortative mating. If the parent-offspring correlations are larger than what would be expected under genetic transmission alone this could imply that the shared environmental effects found in adolescents are the result of vertical cultural transmission. A lower parent-offspring correlation compared with DZ twin and sibling correlations can indicate that part of the environmental effects is shared between twins and siblings only, that part of the genetic factors acts in a dominant manner, or that different genetic factors affect exercise in the two generations.

There are a few studies that have examined the familial resemblance for exercise behavior in twins and their parents (Aarnio et al. 1997; Boomsma et al. 1989; Perusse et al. 1989) but all of these studies made the assumption that the etiology of exercise in parents and offspring is the same. However, as outlined above, we now know that there is a large shift from shared environmental to genetic factors in the transition from adolescence to adulthood. In this

study, we readdressed parent-offspring resemblance, taking into account possible differences in variance components between the generations. We used structural equation modeling techniques developed for extended kinships (Neale et al. 1994; Fulker 1989; Eaves et al. 1978; Phillips and Fulker 1989; Boomsma and Molenaar 1987; Maes et al. 2009) to test whether the shared environmental factors in adolescents are best explained by the effects of vertical cultural transmission, horizontal cultural transmission, assortative mating or a combination of these mechanisms. To this end, we made use of data on exercise participation collected from families registered at the Netherlands Twin Registry at different time points. Between 1991 and 1995 parent-offspring exercise data were collected from 3,525 adolescent twins and siblings and 3,138 parents. Between 1997 and 2004 exercise data were collected in 4,364 adult twins and siblings and 1,107 spouses that were in a similar age range as the parents in the parent-offspring sample (30–65 years).

Data from twin-spouse pairs were analyzed to test for different explanations of the spouse correlation. Data on duration of relationship in twin-spouse pairs were available to test whether the spouse correlation could be explained by social interaction. To test whether social homogamy or phenotypic assortment explained the spouse correlation, the twin-cotwin's spouse correlations were computed as a function of zygosity. Next, a parent-offspring model was fitted to the parent-offspring data including differences in variance decomposition across generations and sex. Data from adult twins and siblings were added to simultaneously estimate the heritability of exercise in the parental generation. Exercise participation was a dichotomous phenotype and therefore the data were analyzed with a threshold model.

Methods

Participants

This study was part of an on-going study on health, lifestyle and personality in twins and their family members (siblings, parents and spouses of twins) who are voluntarily registered with the Netherlands Twin Register (NTR) (Boomsma et al. 2002, 2006). Adolescent and young adult twins were recruited through city councils during 1990 and 1992. Since 1991, every 2–3 years the participants receive a mailed questionnaire, including questions about exercise participation. The exercise data from the first three surveys (1991, 1993 and 1995) were used to obtain a dataset in adolescent twins. In 1991 and 1993, surveys were sent out to twins and their parents; in 1995 non-twin siblings also completed the questionnaire. A cross-sectional dataset with

adolescent (13–18 years old) twins and siblings and their parents was created by selecting from each twin family the data of the most recent survey. If the most recent survey of a twin family contained missing data on exercise for one of the twins (incomplete twin pair) and an earlier survey contained complete information of a twin pair, data from this earlier survey were selected instead. This ensured a maximum of complete twin pairs in the dataset. Half-siblings, non-biological siblings and parents, twins with missing zygosity and all subjects with missing data on exercise, sex or age were excluded (less than 2%). This resulted in a dataset with 3,360 twins, 165 siblings and 3,138 parents from 1,736 families. Of the 3,360 twins, there were 1,667 twin pairs of which both twins had valid exercise data, 292 were monozygotic male (MZM), 239 dizygotic male (DZM), 393 monozygotic female (MZF), 266 dizygotic female (DZF) and 477 opposite-sex (DOS) pairs. Mean age of the twins was 16.4 (SD = 1.1), mean age of the siblings was 16.0 (SD = 1.5) and of the parents 45.5 (SD = 4.6). Of all family members, 53.1% were women. To compute the heritability of exercise in the parents, additional data from 3,225 twins and 1,139 siblings between 30 and 65 years (mean age 39.9, SD = 9.4) collected in 1997, 2000, 2002 and 2004 were analyzed. Of the 3,225 twins, there were 1,187 twin pairs of which both twins had exercise data, 153 were MZM, 81 DZM, 503 MZF, 244 DZF and 526 DOS pairs. Data on exercise participation and duration of relationship from 1,107 twin-spouse pairs collected in 2002 and 2004 were available to test for different explanations of the spouse correlation for exercise participation.

Zygosity determination

Zygosity was determined by DNA typing for 29.1% of the adolescent same-sex twin pairs and for 36.6% of the adult same-sex pairs. For the other same-sex twin pairs, zygosity was based on eight items on physical similarity and the frequency of confusion of the twins by parents, other family members and strangers. Agreement between zygosity based on these items and zygosity based on DNA was 97% (Willemsen et al. 2005).

Measurements

Leisure-time exercise participation was measured with a number of questions. The first question “Do you participate in exercise regularly?” could be answered with ‘Yes’ or ‘No’. If the participants responded affirmative, further information on type, frequency and duration of exercise was gathered. Reported non-leisure time activities, such as walking or biking to work, were not counted as exercise. All remaining exercise activities were assigned a metabolic

equivalent (MET) value, using Ainsworth's Compendium of physical activity (Ainsworth et al. 2000). A MET score of 1 corresponds to the rate of energy expenditure when at rest (1 kcal/kg/h). Subjects were classified as regular exercisers if they participated in exercise with at least 4 MET for 60 min weekly for at least 10 months during the past year. Subjects were classified as non-exercisers otherwise. This dichotomous variable was used in the analyses.

Statistical analyses

Structural equation modeling in Mx (Neale et al. 2006) was employed for all analyses. Threshold models were fitted to the raw ordinal data using maximum likelihood. In threshold models it is assumed that the ordinal variable has an underlying liability with a continuous and standard normal distribution. For dichotomous traits, there is one threshold that divides the liability distribution into two discrete categories (i.e., 'regular exerciser' or 'non-exerciser'). This threshold is based on the prevalence of the different categories in the population. Sex and generation differences in the threshold were allowed. We tested whether the prevalence for exercise participation differs in fathers, mothers, sons and daughters. Within each sex by generation group, age was modeled as a covariate on the threshold to account for any remaining variability in prevalence of exercise as a function of age. The variance of the liability distribution of exercise participation was constrained to one in all types of relatives.

In a first set of analyses, the spouse correlations for exercise participation were estimated in the parents of adolescent offspring and in the adult spouse-twin pairs. We evaluated whether these spouse correlations could be constrained to be equal. Next, three different explanations for the spouse correlation were tested: social interaction, social homogamy and phenotypic assortment (Heath and Eaves 1985; Reynolds et al. 2006). If social interaction explains the spouse correlation, it is expected that the spouse correlation increases as a function of the duration of the relationship. If social homogamy processes drive the spouse correlation, it is expected that the correlation between a twin with the cotwin's spouse is the same for MZ and DZ twin pairs, since twins within a pair come from the same social background. It is further expected that these twin-cotwin's spouse correlations equal the twin-spouse correlations. If assortment is mainly phenotypic, the twin-cotwin's spouse correlations are expected to be the product of the twin correlation and the twin-spouse correlation (Heath and Eaves 1985). If the phenotype is heritable, the MZ twin correlation is smaller than one but larger than the DZ twin correlation. Therefore, it is expected that

the twin-spouse correlation is larger than the MZ twin-cotwin's spouse correlation, which in turn is larger than the DZ twin-cotwin's spouse correlation.

Secondly, in a saturated model we estimated the tetrachoric correlations for exercise participation in adolescent twins, siblings and their parents, and in the adult twins and siblings. In total, 15 correlations were estimated: 1 correlation between the parents, 4 parent-offspring correlations (father-son, father-daughter, mother-son and mother-daughter), 5 adolescent twin and sibling correlations (MZM, DZM/male sibling, MZF, DZF/female sibling and DOS/opposite sex sibling) and 5 adult twin and sibling correlations. In both adolescent and adult twins, the correlations between DZ twins, between twins and their non-twin siblings and between non-twin siblings were constrained to be equal.

Thirdly, the liability variance of exercise participation of individuals in the parental and offspring generation was decomposed into genetic and environmental variances, while modeling the effects of assortative mating between parents. We used the factor model described by Neale and colleagues (Neale et al. 1994; Maes et al. 2009), which builds on the work from Fulker, Heath, Eaves and others (Heath et al. 1985; Fulker 1989; Phillips and Fulker 1989; Eaves et al. 1978). We extended this model to account for generation differences in the variance decomposition of exercise. The variance components in the parents were estimated by simultaneously modeling the data from adult twins and siblings of comparable age with the parents of adolescent twins. This was done by constraining the parameters of the parents to the parameters of the adult twins/siblings, assuming that there were no birth cohort differences in heritability. Standard twin models (Neale and Cardon 1992) with quantitative and qualitative sex differences in parameters were fitted to the data of the adult twins/siblings. The liability variances in the adult twins, siblings and parents were constrained to 1 and were modeled as a function of additive genetic variance (A), non-additive genetic variance (D) and unique environmental variance (E). The additive genetic, non-additive genetic and unique environmental factors in the parental generation were standardized and had a variance of 1. The estimates of the path loadings in the fathers (a_{FA}^* , d_{FA}^* , e_{FA}^*) and mothers (a_{MO}^* , d_{MO}^* , e_{MO}^*) were constrained to the path loadings in the adult twins/siblings of comparable age. In the adult twin/sibling data, we additionally estimated the additive genetic correlation in opposite sex pairs ($r_{A,FA,MO}$, bound between 0 and 0.5).

To test whether C in the adolescents was due to vertical cultural transmission, horizontal cultural transmission or assortative mating, a set of models with assortment and cultural transmission effects were fitted to the data. The path diagram of the full model is shown in Fig. 1a for opposite-sex twins and their parents. The adult twins and

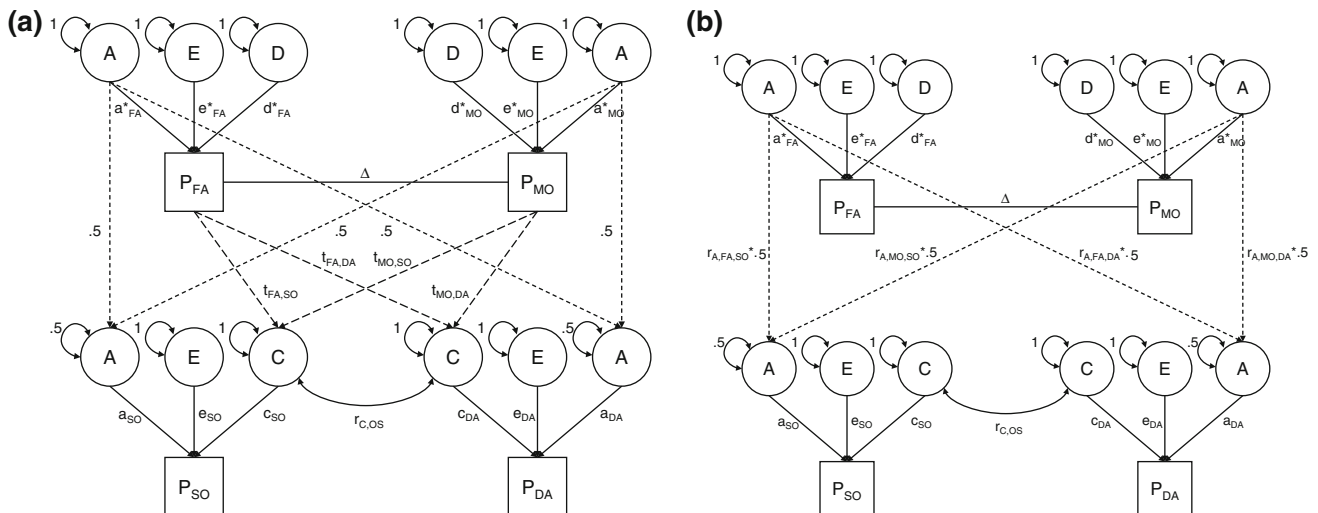


Fig. 1 Two types of path models used for exercise participation in adolescent twins, siblings and their parents. **a** PaFth model with estimated cultural transmission effects and genetic transmission effects constrained to 0.5. **b** Path model with estimated genetic correlations across generations and cultural transmission effects constrained to zero. P = Phenotype, FA = Father, MO = Mother, SO = Son, DA = Daughter, A = Additive genetic factors, E = Non-shared environmental factors, D = Non-additive genetic factors, C = Total of environmental factors shared among offspring, Δ = co-path used to represent phenotypic assortment, a = additive genetic path loading, e = non-shared environmental path loading, c = shared environmental path loading, d = non-additive genetic path loading, t = vertical

cultural transmission path, r_A = genetic correlation across generations, $r_{C,OS}$ = correlation between residual shared environmental factors in opposite-sex pairs. *The parameters in the parents are constrained to the parameters in the adult twins and siblings. *Note:* The total variances of A and C in the sons and daughters were a function of the fixed residual variances (0.5 and 1, respectively) plus the variance due to genetic or cultural transmission. The variances of A differ in boys and girls when genetic correlations across generations are estimated and sex differences in these correlations are allowed; the variances of C differ in boys and girls when cultural transmission with sex differences is estimated

siblings and any additional adolescent siblings are omitted from the diagram for clarity of presentation. The spouse correlation was modeled as phenotypic assortment based on the first set of analyses in the adult twin-spouse pairs and is represented as a co-path in the model (Δ) (Cloninger 1980). This co-path implicitly represents the many correlations induced by assortment between genetic and environmental deviations of spouses (Eaves et al. 2005). This induces an additive genetic correlation between first degree relatives that is larger than 0.5.

Resemblance between parents and offspring was modeled by genetic and vertical cultural transmission. Parents transmit half of their additive genetic effects to their offspring (represented by the path fixed to 0.5 going from A in the parents to A in the children) and none of their non-additive genetic effects. If there is no assortative mating, 50% of the additive genetic variance in the offspring generation is due to the parents and the remaining 50% is due to recombination, as represented by the 0.5 residual variance of A in the offspring. If there is assortative mating, this total variance of A is increased. More formally, the total variance of A was constrained to $0.5 + 0.5 \cdot a_{FA}^* \Delta^* a_{MO} + 0.5$ in boys and girls. Vertical cultural transmission was modeled as the effect of the parental phenotype on the environment that is shared among

offspring. The effect of vertical cultural transmission was allowed to be different depending on the sex of both the parent and the child. The total variance of C in the offspring was computed as the variance induced by the vertical cultural transmission effects plus the residual variance. The residual variance represents horizontal cultural transmission and was constrained to 1. Thus, the total variance of C in boys was constrained to $t_{FA,SO}^2 + t_{MO,SO}^2 + 2 \cdot t_{FA,SO}^* \Delta^* t_{MO,SO} + 1$. A similar constraint was employed for the total variance of C in girls. A consequence of simultaneous genetic and vertical cultural transmission is that A and C become correlated. The covariance between A and C in boys was constrained to $0.5 \cdot a_{FA}^* t_{FA,SO} + 0.5 \cdot a_{FA}^* \Delta^* t_{MO,SO} + 0.5 \cdot a_{MO}^* t_{MO,SO} + 0.5 \cdot a_{MO}^* \Delta^* t_{FA,SO}$. Similarly, this covariance was given by $0.5 \cdot a_{FA}^* t_{FA,DA} + 0.5 \cdot a_{FA}^* \Delta^* t_{MO,DA} + 0.5 \cdot a_{MO}^* t_{MO,DA} + 0.5 \cdot a_{MO}^* \Delta^* t_{FA,DA}$ in girls.

The liability variances in the offspring were modeled as a function of A, C and E. Different path loadings were allowed in boys (a_{SO}, c_{SO}, e_{SO}) and girls (a_{DA}, c_{DA} and e_{DA}) to model quantitative sex differences. The phenotypic variance in boys was given by $var(P_{SO}) = 1 = a_{SO} var(A_{SO}) + c_{SO} var(C_{SO}) + 2 \cdot a_{SO} cov(A_{SO}, C_{SO}) + e_{SO}^2$. Similarly, the phenotypic variance in girls

was given by $\text{var}(P_{DA}) = 1 = a_{DA} \text{var}(A_{DA})^* a_{DA} + c_{DA} \text{var}(C_{DA})^* c_{DA} + 2^* a_{DA} \text{cov}(A_{DA}, C_{DA})^* c_{DA} + e_{DA}^2$. Qualitative sex limitation was modeled in C, in accordance with the previous finding that the lower OS correlation in adolescents can be explained by qualitative sex differences in C rather than A (Stubbe et al. 2005). This was done by fixing the correlation between residual variances of C in same-sex pairs to 1, but estimating this correlation in opposite-sex pairs ($r_{C,OS}$, bound between 0 and 1).

In this first model (Fig. 1a, which is further referred to as model 1), we tested whether dominance in the parental generation was significant (Fig. 1a with: $d_{FA}^* = 0, d_{MO}^* = 0$, model 2), whether there were quantitative and qualitative sex differences in the variance decomposition in the parental generation (Fig. 1a with: $a_{FA}^* = a_{MO}^*, c_{FA}^* = e_{MO}^*, d_{FA}^* = d_{MO}^*, r_{A,FA,MO} = 0.5$, model 3), and whether the constraints of models 2 and 3 were allowed in combination (Fig. 1a with: $a_{FA}^* = a_{MO}^*, e_{FA}^* = e_{MO}^*, d_{FA}^* = d_{MO}^* = 0, r_{A,FA,MO} = 0.5$, model 4).

In addition to the models described above, we explored an alternative set of models in which we allowed for the possibility that different genes are expressed in both generations. This was modeled by estimating the genetic correlations between parents and offspring, allowing for sex differences in these correlations ($r_{A,FA,SO}, r_{A,MO,SO}, r_{A,FA,DA}, r_{A,MO,DA}$, bound between 0 and 1, see Fig. 1b). This model is further referred to as model 5. A model that estimates the genetic correlations simultaneously with the cultural transmission effects is not identified, since for both types of estimates the information comes from the parent-offspring correlations. Therefore, the cultural transmission effects were constrained to zero. Estimation of the genetic correlations has consequences for the constraints employed for the variances of A in the offspring. The total variance of A in boys was constrained to $0.25^* r_{FA,SO}^2 + 0.25^* r_{MO,SO}^2 + 0.5^* r_{FA,SO}^* r_{MO,SO}^* a_{FA}^* \Delta^* a_{MO} + 0.5$. In a similar way, we constrained the total variance of A in girls.

In this second set of models, we performed three tests. First, we tested whether dominance in the parental generation was significant (Fig. 1b with: $d_{FA}^* = 0, d_{MO}^* = 0$, model 6). Second, we tested whether there were significant

quantitative and qualitative sex differences in variance decomposition in the parental generation (Fig. 1b with: $a_{FA}^* = a_{MO}^*, e_{FA}^* = e_{MO}^*, d_{FA}^* = d_{MO}^*, r_{A,FA,MO} = 0.5, r_{A,FA,SO} = r_{A,MO,SO} = r_{A,FA,DA} = r_{A,MO,DA}$, model 7). Third, we tested whether the combination of constraints with regard to dominance and sex differences in the parental generation was allowed (Fig. 1b with: $a_{FA}^* = a_{MO}^*, e_{FA}^* = e_{MO}^*, d_{FA}^* = d_{MO}^* = 0, r_{A,FA,MO} = 0.5, r_{A,FA,SO} = r_{A,MO,SO} = r_{A,FA,DA} = r_{A,MO,DA}$, model 8).

These parent-offspring models were fitted to the data of adolescent twins and siblings and their parents simultaneously with the data of adult twins and siblings. For the model with the best fit (i.e., the lowest AIC), further constraints were imposed to arrive at a most parsimonious model (model 9). The fit of the constrained models was evaluated by means of the log-likelihood ratio test (LRT). The difference in minus two times the log-likelihood (-2LL) between two nested models has a χ^2 distribution and the degrees of freedom (df) equals the difference in df between the two models. If the χ^2 -test yielded a *p*-value larger than 0.05 the fit of the constrained model was not significantly worse than the fit of the more complex model and the constrained model was kept as the most parsimonious and best fitting model.

Results

Prevalence of exercise participation in adolescents and adults

The prevalence of exercise participation in different age groups for men and women is given in Table 1. The percentage of regular exercisers in adolescents is larger than in adults ($\chi^2 = 13.16, df = 2, p < 0.01$). Further, the percentage of regular exercisers in adolescent boys is larger than in girls ($\chi^2 = 9.45, df = 1, p < 0.01$). The percentage of regular exercisers in adult men is not different from the percentage of exercisers in women ($\chi^2 = 0.89, df = 1, p = 0.35$). Within each group of sons, daughters, fathers and mothers, there is a significant decrease in prevalence of exercise participation with age ($\chi^2 = 47.73, df = 1, p < 0.001$).

Table 1 Prevalence of exercise participation (%) in adolescent twins and siblings and their parents (data from 1991, 1993 and 1995) and in adult twins, siblings and spouses (data from 1997, 2000, 2002, 2004)

Exercise participation	Adolescent twins and siblings Mean age 16.4 (SD = 1.2)		Parents of adolescent twins Mean age 45.5 (SD = 4.6)		Adult twins, siblings and spouses Mean age 43.4 (SD = 6.2)	
	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%
Men	1,636	68.3	1,488	39.2	2,114	49.1
Women	1,889	61.6	1,650	40.8	3,357	49.2
Total	3,525	64.7	3,138	40.1	5,471	49.2

N total number of individuals, % percentage of regular exercisers

Table 2 Tetrachoric twin-spouse and twin-cotwin's spouse correlations for exercise participation as a function of duration of relationship and zygosity

	<5 years	5–10 years	10–15 years	≥15 years	
Twin-spouse correlations as a function of duration of relationship					
Number of pairs	245	360	198	255	
Tetrachoric correlation	0.61	0.34	0.42	0.47	
95% Confidence interval	0.45; 0.74	0.18; 0.48	0.22; 0.59	0.30; 0.62	
	MZM	DZM	MZF	DZF	
Twin-cotwin's spouse correlations as a function of zygosity of the twin pair					
Number of pairs	144	63	352	155	174
Tetrachoric correlation	0.20	−0.21	0.16	−0.04	0.04
95% Confidence interval	−0.03; 0.42	−0.57; 0.23	0.02; 0.31	−0.29; 0.20	−0.18; 0.26

Twin-cotwin's spouse correlations in MZM = monozygotic male twin pairs, DZM = dizygotic male twin pairs, MZF = monozygotic female twin pairs, DZF = dizygotic female twin pairs and DOS = dizygotic opposite-sex twin pairs

Spouse correlations for exercise participation in adults

The spouse correlation in parents of adolescent twins is estimated at 0.41 (95% confidence interval (CI) 0.34; 0.48). The spouse correlation in the adult twin cohort is 0.44 (95% CI 0.37; 0.53). These correlations were not significantly different ($\chi^2 = 0.69$, $df = 1$, $p = 0.41$).

To investigate whether the spouse correlations result from social interaction, the correlations were computed as a function of duration of the relationship. The correlations are given in Table 2. There is no clear increase in spouse correlations as a function of length of the relationship. Constraining the twin-spouse correlations to be equal across different groups of duration of relationship was permitted ($\chi^2 = 6.59$, $df = 3$, $p = 0.09$). Thus, social interaction does not seem to provide a good explanation for the spouse correlations.

To further investigate the causes of assortment, the correlations of twins with the cotwin's spouses as a

function of zygosity were estimated (see Table 2). The estimates of the MZ twin-cotwin's spouse correlations are larger than of the DZ twin-cotwin's spouse correlations, although these differences were not statistically significant ($\chi^2 = 3.80$, $df = 1$, $p = 0.05$). Further, the twin-cotwin's spouse correlations in MZ pairs were significantly lower than the twin-spouse correlation ($\chi^2 = 18.01$, $df = 2$, $p < 0.001$). Given this pattern of correlations, the correlation in spouses for exercise participation seems best explained by phenotypic assortment.

Parent-offspring correlations for exercise participation

The correlations among adult twins and siblings, parents and adolescent twins and siblings are given in Table 3. In the adult twins and siblings, there were no significant sex differences in the correlations ($\chi^2 = 0.75$, $df = 3$, $p = 0.86$). The resemblance between parents and their

Table 3 Tetrachoric correlations for exercise participation between adult twins and siblings and between adolescent twins, siblings and their parents

	Correlations in parental generation					Parent-offspring correlations					Correlations in offspring generation				
	MZM	DZM/ sib	MZF	DZF/ sib	DOS/ sib	Fa– Mo	Fa– So	Mo– So	Fa– Da	Mo– Da	MZM	DZM/ sib	MZF	DZF/ sib	DOS/ sib
Number of complete pairs	153	226	503	711	691	1,444	1,438	1,566	1,599	1,792	292	305	393	338	580
Tetrachoric correlation	0.47	0.23	0.46	0.17	0.13	0.41	0.36	0.18	0.21	0.29	0.85	0.66	0.88	0.69	0.45
95% CI, lower bound	0.24	0.03	0.33	0.05	0.01	0.34	0.27	0.09	0.12	0.20	0.76	0.52	0.81	0.56	0.34
95% CI, upper bound	0.66	0.42	0.57	0.28	0.25	0.48	0.44	0.27	0.30	0.36	0.91	0.78	0.92	0.79	0.56

Fa = father, Mo = mother, So = son, Da = daughter, MZM = monozygotic male twin pairs, DZM/sib = dizygotic male twin and non-twin sibling pairs, MZF = monozygotic female twin pairs, DZF/sib = dizygotic female twin and non-twin sibling pairs, DOS/sib = dizygotic opposite-sex twin and non-twin sibling pairs, 95% CI = 95% confidence interval

offspring is largest in father-son pairs and lowest in mother-son pairs. Correlations between fathers and sons, and between mothers and daughters and also between fathers and daughters, and between mothers and sons could be constrained to be equal ($\chi^2 = 2.40$, $df = 2$, $p = 0.30$), but the father-son and mother-daughter correlations are significantly larger than the father-daughter and mother-son correlations ($\chi^2 = 10.58$, $df = 1$, $p < 0.01$).

In the adolescent offspring, there were no sex differences in the MZ correlations ($\chi^2 = 0.77$, $df = 1$, $p = 0.38$) and DZ/sibling correlations ($\chi^2 = 1.47$, $df = 1$, $p = 0.23$), but the DOS/sibling correlation was significantly smaller than the same-sex DZ/sibling correlations ($\chi^2 = 14.69$, $df = 2$, $p < 0.001$), indicating that there are no quantitative sex differences, but there are qualitative sex differences for exercise participation in adolescents. This seems consistent with the sex differences found in the parent-offspring correlations. The MZ and DZ/sib correlations in boys and girls are high, confirming that shared environmental factors play an important role in exercise participation in adolescents. However, the MZ correlations are significantly larger than the DZ/sib correlations in boys ($\chi^2 = 6.37$, $df = 1$, $p < 0.05$) and in girls ($\chi^2 = 5.29$, $df = 1$, $p < 0.05$), indicating that genetic factors are also of importance.

Parent-offspring modeling of exercise participation

Model fitting results for the different parent-offspring models are given in Tables 4, 5 and 6. The models with cultural transmission effects show lower AIC's than the

models with genetic correlations across generations, and therefore provide a better fit to the data. Model 4, in which the same AE model is assumed in fathers and mothers, is the most parsimonious model with the lowest AIC. In this model, 42% (95% CI: 33%; 50%) of the variance in adult exercise was explained by additive genetic factors and 58% (95% CI: 50.0%; 68%) by non-shared environmental factors. The vertical cultural transmission path from father to son was positive and significant ($\chi^2 = 5.62$, $df = 1$, $p < 0.05$). The path from mother to son was also significant but negative ($\chi^2 = 4.18$, $df = 1$, $p < 0.05$). The vertical cultural transmission paths from father and mother to daughter were not significant ($\chi^2 = 1.10$, $df = 1$, $p = 0.29$ and $\chi^2 = 0.72$, $df = 1$, $p = 0.40$, respectively). The correlation between residual variances of the shared environmental latent factor in opposite-sex pairs was estimated at 0.61 and significantly lower than 1 ($\chi^2 = 6.03$, $df = 1$, $p = 0.01$), suggesting that besides sex specific effects of the parental phenotype on boys and girls, there are also qualitative differences between boys and girls in the generation specific shared environmental factors. The additive genetic effects in boys and girls could not be omitted from the model ($\chi^2 = 17.36$, $df = 2$, $p < 0.001$).

The model fit and parameter estimates of the best fitting constrained model are provided in the last row of Tables 4, 5 and 6. Under this model, 42% of the variance of exercise participation in adolescent boys was explained by additive genetic factors, 41% by generation specific shared environmental factors, 3% by vertical cultural transmission and 14% by unique environmental factors. In girls, 36% of the variance was explained by additive genetic factors, 52% by

Table 4 Model fitting results for exercise participation in adolescent twins and siblings and their parents, and adult twins and siblings

Model	-2LL	df	#par	AIC
Models with cultural transmission and fixed genetic transmission (see Fig. 1a):				
1. Figure 1a	13959.45	11005	28	-8050.50
2. Figure 1a with: $d_{FA}^* = 0$, $d_{MO}^* = 0$	13960.34	11007	26	-8053.66
3. Figure 1a with: $a_{FA}^* = a_{MO}^*$, $e_{FA}^* = e_{MO}^*$, $d_{FA}^* = d_{MO}^*$, $r_{A,FA,MO} = 0.5$	13960.39	11009	24	-8057.61
4. Figure 1a with: $a_{FA}^* = a_{MO}^*$, $e_{FA}^* = e_{MO}^*$, $d_{FA}^* = d_{MO}^* = 0$, $r_{A,FA,MO} = 0.5$,	13962.35	11010	23	-8057.66
Models with estimated genetic transmission and no cultural transmission (see Fig. 1b):				
5. Figure 1b	13963.94	11001	28	-8038.06
6. Figure 1b with: $d_{FA}^* = 0$, $d_{MO}^* = 0$	13963.98	11003	26	-8042.02
7. Figure 1b with: $a_{FA}^* = a_{MO}^*$, $e_{FA}^* = e_{MO}^*$, $d_{FA}^* = d_{MO}^*$, $r_{A,FA,MO} = 0.5$ $r_{A,FA,SO} = r_{A,MO,SO} = r_{A,FA,DA} = r_{A,MO,DA}$	13973.16	11008	21	-8042.85
8. Figure 1b with: $a_{FA}^* = a_{MO}^*$, $e_{FA}^* = e_{MO}^*$, $d_{FA}^* = d_{MO}^* = 0$, $r_{A,FA,MO} = 0.5$, $r_{A,FA,SO} = r_{A,MO,SO} = r_{A,FA,DA} = r_{A,MO,DA}$	13974.48	11009	20	-8043.52
Best fitting constrained model:				
9. Figure 1a with: $a_{FA}^* = a_{MO}^*$, $e_{FA}^* = e_{MO}^*$, $d_{FA}^* = d_{MO}^* = 0$, $r_{A,FA,MO} = 0.5$, $t_{FA,DA} = 0$, $t_{MO,DA} = 0$	13964.77	11012	21	-8059.23

-2LL = -2 log-likelihood, df = degrees of freedom, #par = number of free parameters, AIC = akaike information criterion, a = additive genetic path, e = non-shared environmental path, d = non-additive genetic path, r_A = genetic correlation, FA = father, MO = mother, SO = son, DA = daughter

Table 5 Parameter estimates from parent-offspring models for exercise participation between parents and offspring and in the offspring generation

Model	$t_{FA,SO}$	$t_{MO,SO}$	$t_{FA,DA}$	$t_{MO,DA}$	var (AS)	var (CT _{SO})	cov (A _{SO} ,C _{SO})	var (CT _{DA})	cov (A _{DA} ,C _{DA})	a _{SO}	c _{SO}	e _{SO}	a _{DA}	c _{DA}	e _{DA}	r _{C,OS}
Models with cultural transmission and fixed genetic transmission (see Fig. 1a):																
1.	0.22	-0.16	-0.13	0.13	0.06	0.05	0.03	0.02	-0.01	0.61	0.64	0.39	0.62	0.69	0.35	0.58
2.	0.21	-0.25	-0.13	0.05	0.09	0.06	-0.02	0.01	-0.04	0.61	0.66	0.39	0.61	0.70	0.35	0.61
3.	0.35	-0.14	-0.01	0.16	0.03	0.10	0.06	0.03	0.04	0.62	0.61	0.38	0.62	0.66	0.35	0.54
4.	0.22	-0.25	-0.11	0.05	0.08	0.07	-0.01	0.01	-0.03	0.61	0.65	0.39	0.61	0.70	0.35	0.61
Models with estimated genetic transmission and no cultural transmission (see Fig. 1b):																
5.	1	0.33	0.55	1	0.03/0.05	0 ^a	0 ^a	0 ^a	0 ^a	0.74	0.65	0.37	0.66	0.71	0.34	0.72
6.	1	0.32	0.53	1	0.03/0.05	0 ^a	0 ^a	0 ^a	0 ^a	0.74	0.65	0.37	0.65	0.72	0.34	0.72
7.	1	1	1	1	0.06	0 ^a	0 ^a	0 ^a	0 ^a	0.66	0.63	0.38	0.64	0.67	0.35	0.54
8.	0.92	0.92	0.92	0.92	0.08	0 ^a	0 ^a	0 ^a	0 ^a	0.63	0.68	0.38	0.61	0.71	0.35	0.57
Best fitting constrained model:																
9.	0.24	-0.26	-	-	0.08	0.08	-0.01	0 ^a	0 ^a	0.62	0.64	0.38	0.58	0.72	0.35	0.57

Δ = Assortative mating path, t = cultural transmission path, r_A = genetic correlation across generations, var(AS) = part of the variance of the additive genetic factor that is explained by assortment, var(CT) = part of the variance of the shared environmental factor that is explained by vertical cultural transmission, cov(A,C) = covariance between the total variances of A and C, a = additive genetic path, c = shared environmental path, e = non-shared environmental path, $r_{C,OS}$ = correlation between residual variances of C in opposite-sex pairs, FA = father, MO = mother, SO = son, DA = daughter

^a In the absence of cultural transmission, the values of var(CT) and cov(A,C) are given

Note: The variances of AS differ in boys and girls when genetic correlations across generations are estimated and sex differences in these correlations are allowed

Table 6 Parameter estimates from parent-offspring models for exercise participation in the parental generation

Model	Δ	a _{FA}	d _{FA}	e _{FA}	a _{MO}	d _{MO}	e _{MO}	r _{A,FA,MO}
Models with cultural transmission and fixed genetic transmission (see Fig. 1a):								
1.	0.41	0.66	0.17	0.73	0.48	0.48	0.74	0.35
2.	0.41	0.68	-	0.73	0.66	-	0.75	0.30
3.	0.41	0.41	0.54	0.74	0.41	0.54	0.74	0.5 ^a
4.	0.41	0.65	-	0.76	0.65	-	0.76	0.5 ^a
Models with estimated genetic transmission and no cultural transmission (see Fig. 1b):								
5.	0.41	0.72	0.00	0.69	0.64	0.20	0.75	0.29
6.	0.41	0.72	-	0.69	0.66	-	0.75	0.28
7.	0.41	0.55	0.37	0.74	0.55	0.37	0.74	0.5 ^a
8.	0.41	0.65	-	0.76	0.65	-	0.76	0.5 ^a
Best fitting constrained model:								
9.	0.41	0.64	-	0.77	0.64	-	0.77	0.5 ^a

Δ = Assortative mating path, a = additive genetic path, d = non-additive genetic path, e = non-shared environmental path, r_{A,FA,MO} = additive genetic correlation in opposite-sex pairs, FA = father, MO = Mother

^a These values were fixed in these models

generation specific shared environmental factors and 12% by unique environmental factors.

Discussion

We extended existing genetic models for the analysis of parent-offspring data to allow for differences in variance

decomposition across sex and generation. These models were fitted simultaneously to data on exercise behavior from adolescent twins and siblings and their parents, and adult twins and siblings. We explored to what extent the shared environment typically found for exercise participation in adolescents is composed of the effects of the parental phenotype on the offspring's exercise behavior (vertical cultural transmission), generation-specific

environmental factors (horizontal cultural transmission) and the effects of assortative mating. Firstly, data from adult twins and their spouses were analyzed to investigate whether the spouse correlation for exercise participation was best explained by social interaction, social homogamy or phenotypic assortment. To account for differences in genetic architecture between generations, data from an adult twin-sibling sample were analyzed simultaneously with data from parents and offspring to obtain an estimate of the heritability of exercise behavior in the parental generation.

The spouse correlation for exercise participation was high (0.41) and did not increase as a function of the duration of the relationship (ranging from less than 5 years to more than 15 years), indicating that social interaction between spouses is not a major explanation for assortment for exercise behavior. The correlations between twins with their co-twins' spouse were a function of zygosity and were higher in MZ than in DZ twins. These results suggest that phenotypic assortment is the best explanation for the spouse correlation. Phenotypic assortment would mean that individuals who are both exercisers may be more attracted to each other because they share similar interests (for example, participating in sports or being active). The observation of a positive spouse correlation is consistent with previous studies reporting significant spouse correlations for exercise behavior ranging from 0.16 to 0.60 (Aarnio et al. 1997; Boomsma et al. 1989; Perusse et al. 1989; Perusse et al. 1988; Seabra et al. 2008). We are the first to have tested for different explanations of this spouse correlation.

The best fitting parent-offspring model fitted included additive genetic and unique environmental influences for fathers and mothers, phenotypic assortment between parents, cultural transmission effects for sons and additive genetic and unique and shared environmental influences with quantitative and qualitative sex limitation in sons and daughters. In the parental generation, the heritability was estimated at 42%. This is in keeping with previous studies, using twins with a highly similar age range (de Geus et al. 2003) or slightly younger twins (19–40 years old) (Stubbe et al. 2006). In adolescent boys 42% of the variance in exercise participation was explained by additive genetic factors, 41% by generation specific shared environmental factors, 3% by vertical cultural transmission and 14% by unique environment. In girls, 36% of the variance was explained by genetic factors, 52% by generation specific shared environmental factors and 12% by unique environment. The sex differences in genetic architecture in the offspring are both quantitative and qualitative. We found evidence for cultural transmission in boys only, with a positive effect from fathers and a negative effect from mothers. In addition, the environmental factors shared by boys that do not come from the parental phenotypes also

differ from the environmental effects shared by girls from the same family. Thus, the effects of paternal exercise behavior were only significant in boys and the impact is minor compared with the influence of other environmental and genetic factors.

These findings are largely consistent with two previous studies in adolescent twins and their parents (Boomsma et al. 1989; Aarnio et al. 1997) with regard to the low parent-offspring correlation and moderate to large adolescent MZ and DZ twin correlations. The study from Aarnio et al. (1997), conducted in 3,254 twins of 16 years old and their parents, reported low parent-offspring correlations (0.05–0.10), high monozygotic (MZ) correlations (0.64–0.72) and moderate DZ correlations (0.22–0.45) for leisure time physical activity. The low parent-offspring correlations in this study also suggest that the influence of the parental phenotype on adolescent exercise behavior is small. In contrast, in a sample of 893 biological and adopted children (including twins) with a mean age of 15 years ($SD = 3.3$) and their parents Perusse et al. (1989) observed that exercise participation is influenced by a combination of vertical and horizontal cultural transmission. Vertical transmission effects explained 12% of the variance in exercise participation. It was not tested whether these effects were sex specific. Level of habitual physical activity, however, was not influenced by vertical cultural transmission. Both measures were based on a three-day activity diary, which reflects daily physical activity and may be different from our measure of past year's leisure time exercise behavior.

The low impact of vertical cultural transmission in boys and the complete absence of cultural transmission in girls does not necessarily mean that parents do not have any influence on their children's exercise behavior. Cultural transmission was modeled as phenotypic, that is, exercise behavior in the parents has a direct influence on the environment of their children. If specific parental influences that are unrelated to their own exercise behavior also have an impact on children's exercise behavior, these influences will be part of the residual shared environmental variance. Not much is known yet about parental influences such as attitudes toward children's exercise behavior, support, and actual facilitation of exercise behavior (pay for equipment, drive to the playing field etc.), although a review suggests that parental attitudes to exercise behavior are more strongly correlated to children's exercise behavior than the exercise behavior of the parents themselves (Gustafson and Rhodes 2006). Data on parental exercise behavior, attitudes and social support in twin families are needed to test the hypothesis that parental influences unrelated to the parents own exercise behavior are of importance for exercise behavior in their children, while taking into account the effects of assortative mating and genetic transmission.

Modeling cultural transmission from the full parental phenotype to offspring is only one of several possibilities as noted above. With respect to vertical transmission, it is possible that parents pass only certain (e.g., environmental) aspects of their phenotype to offspring. For example, parents might ‘pass on’ sports behavior to their children not directly through their own behavior, but indirectly, through non-genetically mediated aspects of their environment that are related to exercise participation (Keller et al. 2009).

A potential limitation of the present study is that we obtained heritability estimates and spouse correlations for exercise participation in the parental generation from twins who had a comparable age range as the parents, but who came from a younger birth cohort (about 10 years difference). This could inadvertently have introduced a birth cohort related difference in heritability. To examine this possibility, we compared heritability estimates in the adult twins to published values in adult twins that came from the same birth cohort as the parents of the adolescent twins (de Geus et al. 2003). Although the sample from this published study was smaller (213 adult twin pairs), the heritability of exercise was estimated at 41% (de Geus et al. 2003), a value highly comparable to that found in the larger sample of adult twins and siblings in the current study. Thus, at least for heritability estimates, birth cohort effects were small at best.

A second potential limitation of this study is that in the parent-offspring models we were unable to model the qualitative generation differences together with vertical cultural transmission. Therefore, the absence of cultural transmission in girls should be interpreted with caution. Also, the negative effect from mothers on sons may represent a combination of cultural transmission and qualitative differences across sex and generations rather than a true negative effect. It could be that there are positive cultural transmission effects of the parental exercise behavior on their daughters and of mothers on sons, but that they are confounded with the existence of qualitative generation differences, possibly in combination with qualitative sex differences. Extending the parent-offspring model with different types of relatives, such as second-degree relatives, or with data from adoptive families, may refine the explanations for the family resemblances in exercise behavior. Nonetheless, our main conclusion that the shared environmental variance typically found for exercise behavior in adolescent twin studies largely represents generation specific effects rather than vertical cultural transmission or assortative mating stands up.

Future research needs to include measures of parental attitudes and social support towards children’s exercise behavior to resolve whether parental influences through attitudes and support are of more importance than the actual exercise behavior of the parents. It also needs to

focus on the generation specific environmental factors on adolescent exercise behavior while taking into account sex differences (e.g. peer behavior), as these, together with genetic factors, appear to be the largest contributors to adolescent exercise behavior.

Acknowledgments We acknowledge financial support from the Netherlands Organization for Scientific Research (NWO) (NWO-904-61-193, NWO-311-60-008, NWO-480-04-004 and NWO-575-25-006, Spinozapremie SPI 56-464-14192) and by NIH DA018673.

References

- Aarnio M, Winter T, Kujala UM, Kaprio J (1997) Familial aggregation of leisure-time physical activity—a three generation study. *Int J Sports Med* 18:549–556
- Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, Strath SJ et al (2000) Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* 32:S498–S516
- Albright A, Franz M, Hornsby G, Kriska A, Marrero D, Ullrich I et al (2000) Exercise and type 2 diabetes. *Med Sci Sports Exerc* 32:1345–1360
- Berlin JA, Colditz GA (1990) A meta-analysis of physical activity in the prevention of coronary heart disease. *Am J Epidemiol* 132:612–628
- Beunen G, Thomis M (1999) Genetic determinants of sports participation and daily physical activity. *Int J Obes Relat Metab Disord* 23:S55–S63
- Boomsma DI, Molenaar PCM (1987) Constrained maximum-likelihood analysis of familial resemblance of twins and their parents. *Acta Genet Med Gemellol* 36:29–39
- Boomsma DI, Vandenberg MBM, Orlebeke JF, Molenaar PCM (1989) Resemblances of parents and twins in sports participation and heart-rate. *Behav Genet* 19:123–141
- Boomsma DI, Vink JM, van Beijsterveldt TC, de Geus EJ, Beem AL, Mulder EJ et al (2002) Netherlands Twin Register: a focus on longitudinal research. *Twin Res* 5:401–406
- Boomsma DI, de Geus EJC, Vink JM, Stubbe JH, Distel MA, Hottenga JJ et al (2006) Netherlands Twin Register: from twins to twin families. *Twin Res Hum Genet* 9:849–857
- Carlsson S, Andersson T, Lichtenstein P, Michaelsson K, Ahlbom A (2006) Genetic effects on physical activity: results from the Swedish twin registry. *Med Sci Sports Exerc* 38:1396–1401
- Cloninger CR (1980) Interpretation of intrinsic and extrinsic structural relations by path analysis: theory and applications to assortative mating. *Genet Res* 36:133–145
- de Geus EJC, Boomsma DI, Snieder H (2003) Genetic correlation of exercise with heart rate and respiratory sinus arrhythmia. *Med Sci Sports Exerc* 35:1287–1295
- Eaves LJ, Last KA, Young PA, Martin NG (1978) Model-fitting approaches to the analysis of human behavior. *Heredity* 41:249–320
- Eaves LJ, Silberg JL, Maes HH (2005) Revisiting the children of twins: can they be used to resolve the environmental effects of dyadic parental treatment on child behavior? *Twin Res Hum Genet* 8:283–290
- Eriksson M, Rasmussen F, Tynelius P (2006) Genetic factors in physical activity and the equal environment assumption—the Swedish young male twins study. *Behav Genet* 36:238–247
- Falconer DS, Mackay TFC (1996) Introduction to quantitative genetics, 4th edn. Pearson Education Limited, Essex

- Fulker DW (1989) Extensions of the classical twin method. In: Weir BS, Eisen EJ, Goodman MM, Namkoong G (eds) *Proceedings of the second international conference on quantitative genetics*. Sinauer Associates Inc, Sunderland MA, pp 395–406
- Gustafson SL, Rhodes RE (2006) Parental correlates of physical activity in children and early adolescents. *Sports Med* 36:79–97
- Heath AC, Eaves LJ (1985) Resolving the effects of phenotype and social background on mate selection. *Behav Genet* 15:15–30
- Heath AC, Kendler KS, Eaves LJ, Markell D (1985) The resolution of cultural and biological inheritance—informativeness of different relationships. *Behav Genet* 15:439–465
- Kaplan GA, Strawbridge WJ, Cohen RD, Hungerford LR (1996) Natural history of leisure-time physical activity and its correlates: associations with mortality from all causes and cardiovascular disease over 28 years. *Am J Epidemiol* 144:793–797
- Keller MC, Medland SE, Duncan LE, Hatemi PK, Neale MC, Maes HH et al (2009) Modeling extended twin family data I: description of the Cascade model 3. *Twin Res Hum Genet* 12:8–18
- King AC, Blair SN, Bild DE, Dishman RK, Dubbert PM, Marcus BH et al (1992) Determinants of physical activity and interventions in adults. *Med Sci Sports Exerc* 24:S221–S236
- Kujala UM, Kaprio J, Koskenvuo M (2002) Modifiable risk factors as predictors of all-cause mortality: the roles of genetics and childhood environment. *Am J Epidemiol* 156:985–993
- Lauderdale DS, Fabsitz R, Meyer JM, Sholinsky P, Ramakrishnan V, Goldberg J (1997) Familial determinants of moderate and intense physical activity: a twin study. *Med Sci Sports Exerc* 29:1062–1068
- Maes HH, Neale MC, Medland SE, Keller MC, Martin NG, Heath AC et al (2009) Flexible Mx specification of various extended twin kinship designs. *Twin Res Hum Genet* 12:26–34
- Maia JAR, Thomis M, Beunen G (2002) Genetic factors in physical activity levels—a twin study. *Am J Prev Med* 23:87–91
- Martinez-Gonzalez MA, Martinez JA, Hu FB, Gibney MJ, Kearney J (1999) Physical inactivity, sedentary lifestyle and obesity in the European Union. *Int J Obes Relat Metab Disord* 23:1192–1201
- Martinez-Gonzalez MA, Varo JJ, Santos JL, de Irala J, Gibney M, Kearney J et al (2001) Prevalence of physical activity during leisure time in the European Union. *Med Sci Sports Exerc* 33:1142–1146
- Neale MC, Cardon LR (1992) *Methodology for genetic studies of twins and families*. Kluwer Academic Publishers, Dordrecht
- Neale MC, Walters EE, Eaves LJ, Maes HH, Kendler KS (1994) Multivariate genetic-analysis of twin-family data on fears—Mx models. *Behav Genet* 24:119–139
- Neale MC, Boker SM, Xie G, Maes HH (2006) *Mx: statistical modeling*. Department of Psychiatry, Virginia Commonwealth University, Richmond, Virginia
- Perusse L, Leblanc C, Bouchard C (1988) Familial resemblance in lifestyle components—results from the Canada fitness survey. *Can J Public Health-Revue Canadienne de Sante Publique* 79:201–205
- Perusse L, Tremblay A, Leblanc C, Bouchard C (1989) Genetic and environmental-influences on level of habitual physical-activity and exercise participation. *Am J Epidemiol* 129:1012–1022
- Phillips K, Fulker DW (1989) Quantitative genetic analysis of longitudinal trends in adoption designs with application to IQ in the Colorado Adoption Project. *Behav Genet* 19:621–658
- Reynolds CA, Barlow T, Pedersen NL (2006) Alcohol, tobacco and caffeine use: spouse similarity processes. *Behav Genet* 36:201–215
- Sallis JF, Prochaska JJ, Taylor WC (2000) A review of correlates of physical activity of children and adolescents. *Med Sci Sports Exerc* 32:963–975
- Seabra AF, Mendonca DM, Goring HHH, Thomis MA, Maia JA (2008) Genetic and environmental factors in familial clustering in physical activity 1. *Eur J Epidemiol* 23:205–211
- Stephoe A, Wardle J, Fuller R, Holte A, Justo J, Sanderman R et al (1997) Leisure-time physical exercise: prevalence, attitudinal correlates, and behavioral correlates among young Europeans from 21 countries. *Prev Med* 26:845–854
- Stubbe JH, Boomsma DI, de Geus EJC (2005) Sports participation during adolescence: a shift from environmental to genetic factors. *Med Sci Sports Exerc* 37:563–570
- Stubbe JH, Boomsma DI, Vink JM, Cornes BK, Martin NG, Skytthe A et al (2006) Genetic influences on exercise participation: a comparative study in adult twin samples from seven countries. *PLoS ONE* 1:e22. doi:10.1371/journal.pone.0000022-http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0000022
- van Leeuwen M, van den Berg SM, Boomsma DI (2008) A twin-family study of general IQ. *Learn Individ Differ* 18:76–88
- Willemsen G, Posthuma D, Boomsma DI (2005) Environmental factors determine where the Dutch live: results from the Netherlands Twin Register. *Twin Res Hum Genet* 8:312–317