

Meghan Schreck,¹ Robert Althoff,¹ Charlotte Huppertz,^{2,3} Catherina van Beijsterveldt,^{2,3} Dorret Boomsma,^{2,3} James Hudziak,¹ Eco de Geus,^{2,3} and Meike Bartels^{2,3}

¹University of Vermont College of Medicine, ²Department of Biological Psychology, VU University Amsterdam, the Netherlands, ³EMGO+ Institute for Health and Care Research, VU University Medical Center, Amsterdam, the Netherlands

Introduction

Attention-Deficit Hyperactivity Disorder (ADHD) is the most common neurobehavioral disorder diagnosed in children (American Academy of Pediatrics, 2000). Recent review papers indicated that physical activity may positively impact the same behavioral, cognitive, and neurobiological domains implicated in ADHD (Archer & Kostrzewa, 2012; Gapin, Labban, & Etnier, 2011; Halperin & Healey, 2011).

ADHD and exercise behavior both have genetic and environmental influences. Family, twin, and, adoption studies provide evidence supporting significant genetic influences on ADHD with heritability ranging from 60-91% (Derks et al., 2008, Lehn et al., 2007, Thapar, Langley, Owen, & O'Donovan, 2007). In addition, up to 40% of the etiology of attention problems may be due to environmental factors (Lehn et al., 2007).

Studies investigating the determinants of voluntary exercise have traditionally focused on environmental factors (Sallis, Prochaska, & Taylor, 2000; Van der Horst, Paw, Twisk, & Van Mechelen, 2007). In recent years, an increasing amount of evidence supports the relative influence of genetic factors in contributing to individual differences in exercise behavior (De Moor et al., 2011; Stubbe, Boomsma, & De Geus, 2005; Van der Aa, de Geus, Van Beijsterveldt, Boomsma, & Bartels, 2010). However, the heritability of exercise behavior drastically changes over the lifespan with exercise participation predominately explained by shared environmental factors in children and genetic factors in adolescence (Huppertz, 2012; Van der Aa et al., 2010). In adulthood, the heritability of exercise behavior decreases to between 40% and 60% (Stubbe et al., 2005).

A bivariate behavioral genetic design can be used to determine whether the relation between exercise and attention problems is due to genes that influence both traits or due to environmental influences that act as a risk factor for both traits (Plomin et al., 2008). By exploring the underpinnings of this association at different ages in childhood, we can begin to understand the developmental course of the relation between attention problems and exercise behavior.

Methods

Participants
Children registered with the Netherlands Twin Registry (NTR), established by the Department of Biological Psychology at the Vrije Universiteit (VU) in Amsterdam (Bartels et al., 2007; Boomsma, de Geus, & Vink, 2006) were recruited for the present study. Consented parents reported on their twins and received paper surveys containing items about behavior, sport, lifestyle, and well-being.

A total of 12,830 twins (51% female) from 6,415 families, consisting of complete and incomplete twin pairs born between 1991 and 2000, participated in this ongoing study. The present sample was divided into six groups by sex and zygosity; 1,026 monozygotic males (MZM) and 1,201 females (MZF), 1,061 dizygotic males (DZM) and 1,007 females (DZFM), and 1,103 dizygotic opposite sex, male first-born twins (DOSMF) and 1,017 female first-born twins (DOSFM).

For both variables under investigation, Attention Problems (AP) and Leisure-Time Exercise Behavior (LTEB), participants were divided into three age groups: 7, 10, and 12 years. The age groups are not completely independent, because data from participants who returned surveys at more than one time point (e.g., twins returned a survey at age 7 and 10) were included.

Methods continued

Measures

Leisure-Time Exercise Behavior (LTEB)

Participants in each age group were asked to indicate what type(s) of regular leisure time exercise they were involved in within the past year. A list of 17 common individual and team based physical activities was provided to choose from and five open entry spaces were available for participants to fill in alternative activities. For each activity endorsed, participants reported how many months per year, weekly frequency, and the average duration of the activity in minutes.

The Compendium of Energy Expenditures for Youth activity was used to assign a MET (Metabolic Equivalent) score to each exercise activity (Ridley and Ainsworth, 2008). For each participant, a total weekly MET score was computed across all leisure exercise activities by summing the products of the frequency, duration, and MET score of each activity.

Attention Problems (AP)

Total AP was assessed through maternal parent reports using the Child Behavior Checklist (CBCL) (Achenbach & Rescorla, 2001). The AP scale on the CBCL consists of 11 items (e.g., "cannot sit still, restless or hyperactive," "cannot concentrate, pay attention for long," "impulsive or acts without thinking," etc.) rated on a 3-point scale (0= "not true"; 1= "somewhat true"; 2= "very true" or "often true"). A participant's total AP score equals the sum of scores for the 11 items, with a possible range of 0 to 22.

Results

Cross-twin within-trait correlations were calculated for AP and LTEB for each zygosity and age group. Monozygotic (MZ) and dizygotic (DZ) correlations were compared to approximate the relative genetic and environmental influences of each trait. Univariate statistical modeling using OpenMx (Neale, 2009) indicated that AP fit an ADE model, whereas LTEB fit an ACE model. Both models revealed quantitative sex differences for all age groups. Qualitative sex differences were observed for LTEB for 7- and 12-year-olds. The non-additive genetic and shared environmental correlations could not be estimated in a model with one trait observing an ADE model and the other an ACE model. Therefore, an AE bivariate model was estimated.

At each age group, phenotypic correlations were computed across gender and zygosity and separately for boys and girls (Table 1). Within-twin cross-trait correlations were computed for AP and LTEB for each zygosity and age group to test whether the association between AP and LTEB shared etiological influences. Cross-twin cross-trait correlations were calculated to test whether the etiological relation between AP and LTEB was familial. MZ and DZ cross-twin cross-trait correlations were compared to infer whether the association was due to genetic factors. The within-twin and cross-twin cross trait correlations are shown in Table 2.

Table 1. Phenotypic correlations between AP and LTEB for ages 7, 10, and 12.

	Total	Boys	Girls
Age 7	-0.03 (3885)	-0.08** (1932)	-0.05* (1953)
Age 10	-0.04* (3454)	-0.07** (1693)	-0.09* (1761)
Age 12	-0.06** (8536)	-0.11** (4171)	-0.06* (4365)

**p<.01; *p<.05

Results continued

Table 2. Within-twin cross-trait and cross-twin cross-trait correlations between AP and LTEB for ages 7, 10, and 12

	Twin 1 AP						Twin 2 AP					
	MZM	DZM	MZF	DZF	DOSM F	DOSF M	MZM	DZM	MZF	DZF	DOSM F	DOSF M
Age 7												
Twin 1 LTEB	-0.09 (298)	-0.003 (340)	-0.08 (350)	-0.02 (301)	-0.09 (346)	-0.07 (314)	-0.07 (297)	-0.12* (340)	-0.08 (349)	-0.02 (299)	-0.02 (342)	-0.01 (312)
Twin 2 LTEB	-0.09 (297)	-0.02 (341)	-0.07 (348)	0.03 (301)	-0.08 (346)	-0.11 (313)	-.08 (296)	-0.13* (341)	-0.12* (347)	0.02 (299)	-0.04 (342)	-0.07 (311)
Age 10												
Twin 1 LTEB	0.04 (282)	-0.15* (296)	-0.07 (329)	-0.20** (282)	-0.13* (262)	-0.02 (279)	0.04 (280)	0.001 (295)	-0.06 (330)	-0.03 (280)	-0.11 (260)	-0.04 (279)
Twin 2 LTEB	0.04 (284)	-0.11 (294)	-0.11 (328)	-0.08 (278)	-0.04 (266)	.01 (280)	-.05 (282)	0.08 (293)	-0.08 (329)	-0.04 (276)	-0.15* (264)	-0.17** (278)
Age 12												
Twin 1 LTEB	-0.09* (714)	-0.10* (674)	-0.05 (816)	-0.09* (666)	-0.12** (731)	-0.01 (672)	-0.08* (709)	-0.12** (670)	-0.08* (812)	-0.02 (663)	-0.05 (726)	-0.05 (669)
Twin 2 LTEB	-0.12** (721)	-0.03 (678)	-0.07* (818)	-0.07 (664)	-0.07* (740)	-0.02 (665)	-0.10** (627)	-0.12** (674)	-0.10** (814)	-0.06 (662)	-0.07 (735)	-0.13** (662)

**p<.01; *p<.05

It is possible that a low or no phenotypic correlation can exist when there is a high genetic correlation and negative nonshared environmental correlation between two traits or vice versa (Purcell, 2007). Genetic and nonshared environmental correlations were computed for each age group (Tables 3 and 4).

Results suggest that the low phenotypic correlation between AP and LTEB in 10-year-old boys appears to be explained by nonshared environmental factors; whereas, in 10-year old girls, the relation appears to be explained by genetic factors. At age 12, the low phenotypic correlation between AP and LTEB appears to be explained by genetic factors in both boys and girls. In addition, the genetic and nonshared environmental correlations are negative, suggesting that the genetic and environmental factors that increase attention problems may decrease leisure-time exercise behavior.

Tables 3 and 4. Boys and girls genetic and nonshared environmental correlations between AP and LTEB at ages 7, 10, and 12

	Boys	
	R _A	R _E
Age 7	-0.05 (-0.11; 0.01)	-0.06 (-0.16; 0.04)
Age 10	-0.02 (-0.09; 0.05)	-0.20 (-0.31; -0.08)
Age 12	-0.13 (-0.17; -0.08)	0.02 (-0.05; 0.10)
	Girls	
	R _A	R _E
Age 7	-0.04 (-0.10; 0.03)	-0.08 (-0.18; 0.03)
Age 10	-0.12 (-0.19; -0.05)	-0.05 (-0.06; 0.16)
Age 12	-0.07 (-0.12; -0.03)	0.01 (-0.06; 0.08)

Conclusions

There are very low associations between attention problems and leisure-time exercise behavior in children ages 7, 10, and 12. Further analyses investigating a curvilinear relation between AP and LTEB is warranted, as well as exploration of the association at later stages in development.

Please contact Meghan Schreck at mschreck@uvm.edu for a list of references or with any questions or concerns.