Twin Research and Human Genetics
Volume 16 ▌ Number 2 ▌ pp. 535–543 ▌ © The Authors 2013 ▌ doi:10.1017/thg.2013.9

Genetic and Environmental Influences on Longitudinal Changes in Leisure-Time Physical Activity From Adolescence to Young Adulthood

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The aim of this study was to estimate genetic and environmental influences on the longitudinal evolution of leisure-time physical activity habits from adolescence to young adulthood. Data were gathered at four time points, at mean ages 16.2, 17.1, 18.6, and 24.5 years. At baseline, the sample comprised 5,216 monozygotic and dizygotic twins, born 1975–1979, and, at the last follow-up point, of 4,531 monozygotic and dizygotic twins. Physical activity volume was assessed as frequency of leisure-time physical activity and participants were categorized into three groups: inactive, moderately active, and active. Genetic and environmental influences were estimated using a multivariate, longitudinal Cholesky decomposition with a 'multifactorial liability threshold' approach. The results suggest that, in both sexes the heritability of leisure-time physical activity remained moderate (~43–52%) during adolescence, declining to ~30% in young adulthood. Shared environmental influences increased from adolescence (~18–26%) to young adulthood (43% in men and 49% in women). Specific environmental influences remained relatively stable during the total follow-up (~20–30%). New genetic, shared, and specific environmental influences at every follow-up point were suggested by the low correlations across occasions. In conclusion, the study demonstrated gender differences in genetic influences in the evolution of leisure-time physical activity habits from adolescence to young adulthood. However, shared environmental influences, especially in women, were crucial in explaining longitudinal changes in leisure-time physical activity. These outcomes emphasize the need of gender-specific measures to promote physical activity habits during young adulthood.

■ **Keywords:** twins, genetic analysis, leisure-time physical activity

It is known that complex behaviors are regulated by environmental, genetic, or biological aspects, and/or interaction between these. Leisure-time physical activity habits are the results of a complex interplay of both genetic and environmental influences. For some time now, attempts have been made to clarify the role of genetics and environmental factors in physical activity behavior. So far, large genetic epidemiological cross-sectional studies have reported various findings. Although a final consensus has not been reached, the results of these studies are important in the effort to induce people to be more physically active, and hence healthier.

Twin studies have clearly shown that genetic influences in large part explain individual differences in physical activity (Beunen & Thomis, 1999; Carlsson et al., 2006; Kaprio et al., 1981; Maia et al., 2002; Stubbe et al., 2006; Stubbe & de Geus, 2009). Among these studies, the heritability of physical activity behavior has ranged between 27% and 71%. However, in some studies environmental factors have been shown to exert the strongest influence on participation in physical activity (Duncan et al., 2008; Perusse et al., 1989). In addition to possible methodological issues, it is suggested that a significant proportion of the heterogeneity in these study results may derive from notable changes in the genetic

RECEIVED 23 October 2012; ACCEPTED 30 January 2013.

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contribution to this trait across the age range, meaning that not only environmental but also genetic factors vary over the lifespan.

During the transition from adolescence to young adulthood, many changes take place in health-related behavior. Physical activity level is one such example (Dumith et al., 2011; Kimm et al., 2002). Indeed, during adolescence and young adulthood notable changes appear to occur in the genetic contribution to physical activity. A shift between genetic and environmental influences has been reported during this period, although at different times in different studies, and in different directions. Stubbe et al. (2005) found that between the ages of 13 and 16 years genetic effects are not important, whereas after the age of 18 years genes largely explain individual differences in physical activity participation. In contrast, van der Aa et al. (2010) found that variation in adolescent physical activity behavior was mostly accounted for by genetic factors at the ages of 13–14 in boys and at ages 15–16 in girls. In young adulthood, after adolescence, it seems that the heritability estimate remains moderate (41%), with unique environmental factors accounting for most of the trait variance (Mustelin et al., 2012). Indeed, there is evidence that the genetic drive to engage in leisure-time physical activity is increasingly constrained by environmental factors as people get older (Vink et al., 2011).

Most studies have examined genetic and environmental influences on physical activity cross-sectionally. Cross-sectional studies may reflect true aging effects, but uncertainty remains as cohort effects may also account for such differences between age groups. Longitudinal studies enable investigation of age effects over the lifespan. Only a few studies investigating the genetic and environmental effects of longitudinal physical activity have been carried out. In a sample of healthy 18–60-year-old twins, genetic influences on leisure-time physical activity declined from baseline (44%) to follow-up (34%) during a 6-year follow-up (Aaltonen et al., 2010). Similarly, the heritability estimate of leisure-time physical activity declined during a 4-year follow-up among young Swedish males (Eriksson et al., 2006). A longitudinal animal study by Turner et al. (2005) also showed a high genetic influence on age-related changes in physical activity, although in the opposite direction, as at about 12 weeks of age in the mouse (late adolescence), the genetic influence on physical activity markedly increased.

Despite indications that the heritability of physical activity is affected by age, at least with respect to younger ages, the age spectrum remains somewhat unclear. Moreover, cross-sectional studies do not allow us to test the potential age dependency of genetic influences or examine changes in genetic and environmental influences on physical activity. It would be useful, therefore, to investigate the predictors of physical activity relative to health and physical activity behavior across important periods of the life course, such as adolescence and young adulthood, with the help of longitudinal genetic designs. Hence, the aim of this study was to estimate genetic and environmental influences on the longitudinal evolution of leisure-time physical activity habits from adolescence to young adulthood. Genetic and environmental determinants of stability and change of leisure-time physical activity are examined using quantitative genetic models.

Materials and Methods

Study Cohort

The participants for this study were identified from the FinnTwin16 Cohort, which is a prospective longitudinal study of Finnish twins born between October 1974 and December 1979. Originally, the twins were identified from the Central Population Registry of Finland (Kaprio et al., 2002). The twin cohort was initiated in 1991, and it is a nationwide study of health behaviors in twins and their families. An invitation to participate was sent to all twins within two months of their 16th birthday, of which 2,773 pairs agreed to participate. After baseline assessment, twins were surveyed again in tight age bands at mean ages 17.1, 18.6, and 24.5. The zygosity of the twins was determined through a validated questionnaire. A more detailed description of the FinnTwin16 study can be found elsewhere (Kaprio et al., 2002).

The potential study sample comprised 996 monozygotic (MZ) and 716 dizygotic (DZ) men, 877 MZ and 891 DZ women, and 1,853 DZ opposite-sex twin pairs. Altogether, 241 twins were excluded from the present study as it was not possible to determine their zygosity, and 311 persons because of pregnancy or a medical condition that could clearly prevent engagement in leisure-time physical activity (e.g., motor handicap, cerebral palsy or mental disability). The final study sample comprising those for whom leisure-time physical activity data were obtained across the different follow-ups is detailed in Table 1. The sample dropout was low: 73.8% of the participants had provided leisure-time physical activity data on all four occasions, 16.2% on three occasions, and only about 10% on at most two occasions.

The ethics committee of the Department of Public Health of the University of Helsinki (Finland), and the Institutional Review Board of Indiana University (United States of America) approved the study protocol. The Data Protection Board of Finland approved the maintenance of the study cohort database. The families were provided with information about the study at baseline and were given regular feedback during follow-up in the form of personal letters.

Sample Measurements

At each of the four data collection waves, participants answered a medical–social questionnaire with items on, for example, height, weight, health habits, attitudes (including current leisure-time physical activity habits), and social relationships, along with chronic disease and symptom checklists, and personality scales (Kaprio et al., 2002). All the survey items were self-reported. For the purpose of the

https://doi.org/10.1017/thg.2013.9 Published online by Cambridge University Press

TABLE 1

Participants' Characteristics at Each Measurement Wave

Physical activity group	Zygosity/sex	Baseline Mean age 16.2 years (*N* = 5,216) *N* (%)	Follow-up 1 Mean age 17.1 years (*N* = 4,949) *N* (%)	Follow-up 2 Mean age 18.6 years (*N* = 4,930) *N* (%)	Follow-up 3 Mean age 24.5 years (*N* = 4,531) *N* (%)
Inactive	MZ men	128 (2.5%)	112 (2.3%)	122 (2.5%)	125 (2.8%)
	MZ women	185 (3.5%)	149 (3.0%)	171 (3.5%)	130 (2.9%)
	DZ men	190 (3.6%)	166 (3.4%)	190 (3.9%)	182 (4.0%)
	DZ women	201 (3.9%)	165 (3.3%)	167 (3.4%)	151 (3.3%)
	DZ opp-sex men	213 (4.1%)	174 (3.5%)	193 (3.7%)	203 (4.5%)
	DZ opp-sex women	203 (3.9%)	159 (3.2%)	187 (3.8%)	146 (3.2%)
	Total (*N*)	1,120 (21.5%)	925 (18.7%)	1,030 (21.0%)	937 (20.7%)
Moderately active	MZ men	250 (4.8%)	208 (4.2%)	230 (4.7%)	244 (5.4%)
	MZ women	486 (9.3%)	479 (9.7%)	486 (9.9%)	477 (10.5%)
	DZ men	394 (7.6%)	372 (7.5%)	380 (7.7%)	377 (8.3%)
	DZ women	456 (8.7%)	459 (9.3%)	453 (9.2%)	417 (9.2%)
	DZ opp-sex men	414 (7.9%)	372 (7.5%)	388 (7.9%)	401 (8.9%)
	DZ opp-sex women	493 (9.5%)	508 (10.2%)	484 (9.9%)	466 (10.3%)
	Total (*N*)	2,493 (47.8%)	2,398 (48.5%)	2,421 (49.2%)	2,382 52.5%)
Very active	MZ men	223 (4.3%)	224 (4.5%)	188 (3.8%)	140 (3.1%)
	MZ women	319 (6.1%)	339 (6.9%)	303 (6.1%)	256 (5.7%)
	DZ men	306 (5.9%)	299 (6.0%)	256 (5.1%)	187 (4.1%)
	DZ women	217 (4.2%)	218 (4.4%)	227 (4.6%)	210 (4.6%)
	DZ opp-sex men	303 (5.8%)	310 (6.3%)	275 (5.6%)	192 (4.2%)
	DZ opp-sex women	235 (4.5%)	236 (4.8%)	230 (4.7%)	227 (5.0%)
	Total (*N*)	1,603 (30.7%)	1,626 (32.8%)	1,479 (29.8%)	1,212 (26.8%)

Note: inactive = twice per month or less often; moderately active = regularly, one to three times per week; very active = regularly, more than three times per week; MZ = monozygotic; DZ = dizygotic.

present investigation, we analyzed answers to the following question: 'How often do you exercise or do sports during your leisure-time?' The possible answers were: (1) *not at all*, (2) *less than once a month*, (3) *one or two times a month*, (4) *about once a week*, (5) *two or three times a week*, (6) *four or five times a week*, and (7) *just about every day*. The participants' answers were further recoded as follows: (1) inactive, if exercising less than once a week; (2) moderately active, if exercising one to three times per week; and (3) very active, if exercising four or more times per week. The item was asked in exactly the same form at all time points. Participants who were native Swedish speakers received a questionnaire in Swedish; Finland has two official languages, Finnish and Swedish. Some 6% of the population has Swedish as their mother tongue. Earlier analyses have shown a high correlation between the very similar leisure-time physical activity question we used and physical activity data obtained by a detailed interview (Waller et al., 2008).

Statistical Analyses

The twin data were analyzed by fitting a series of statistical models to the raw categorical data, utilizing biometric methods in a 'multifactorial liability threshold' approach (Neale & Cardon, 1992). Accordingly, it is assumed that the underlying liability to the categories in the phenotype of interest is normally distributed and holds different thresholds generally defining the *z* value within the liability distribution separating the different categories. In our case, two thresholds were assumed, as we defined three different leisure-time physical activity categories.

In quantitative genetic analyses, variation in the underlying liability to each of the categories within the phenotype is normally modeled as the results from a combination of three different sources of influence: additive genetic influences (labeled as A), reflecting the sum of the additive allelic effects of many segregating genes; shared environmental influences (labeled as C), reflecting the effects of environmental factors shared by the co-twins in a pair; and specific environmental influences (labeled as E), reflecting environmental experiences and exposures unique to each person. Since MZ twins share 100% of their genes whereas DZ twins, on average, share 50% of their segregating genes, higher within-pair resemblance in MZ than in DZ twins is considered a sign of genetic influences underlying the phenotype of interest. Shared environment between siblings is assumed to contribute equally to the similarity within both MZ and DZ pairs. Within-pair differences are considered to result from individual-specific environmental factors. In biometric modeling methods, A, C, and E are estimated on the basis of the information available on the twin and co-twin covariance structure and comparison of observed and expected variance–covariance matrices.

In the present study, preliminary information on the within-pair resemblances was obtained by estimating polychoric correlation coefficients in the MZ and DZ twin pairs. Subsequently, biometric model fitting analyses were started by computing a series of univariate models to determine whether A, C, and E conclusively influenced leisure-time physical activity at each time point. Finally, a series of longitudinal Cholesky decompositions were fitted in order to evaluate the stability and change in genetic and environmental influences across the ~8.5 follow-up period.

All the models were fitted to the raw categorical data using maximum likelihood algorithms (allowing inclusion

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TABLE 2

Within-Pair Polychoric Correlations for Participants at Each Measurement Wave

Zygosity/sex	Baseline Mean age 16.2 years (*N* = 5,216) PCC (95% CI)	Follow-up 1 Mean age 17.1 years (*N* = 4,949) PCC (95% CI)	Follow-up 2 Mean age 18.6 years (*N* = 4,930) PCC (95% CI)	Follow-up 3 Mean age 24.5 years (*N* = 4,531) PCC (95% CI)
MZ men	0.72 (0.66–0.77)	0.71 (0.65–0.76)	0.69 (0.63–0.74)	0.79 (0.74–0.83)
MZ women	0.77 (0.74–0.81)	0.77 (0.73–0.80)	0.76 (0.72–0.80)	0.80 (0.77–0.83)
DZ men	0.48 (0.41–0.55)	0.48 (0.41–0.55)	0.51 (0.44–0.57)	0.64 (0.59–0.69)
DZ women	0.50 (0.43–0.57)	0.54 (0.47–0.60)	0.31 (0.23–0.39)	0.69 (0.64–0.73)
DZ opp-sex	0.24 (0.18–0.29)	0.23 (0.17–0.29)	0.25 (0.20–0.31)	0.58 (0.54–0.62)

Note: MZ = monozygotic; DZ = dizygotic; CI = confidence intervals; PCC = polychoric correlation.

also of co-twins without information on their birth partner at any of the data collection waves), and treating unobserved data as missing-at-random (Little & Rubin, 2002). The significances of estimates and path coefficients were tested by removing them sequentially in different submodels. The fit indices of these submodels were compared against the fit of the less constrained initial model by applying likelihood-ratio tests (LRT; Neale & Cardon, 1992) as well as the Akaike's Information Criterion (AIC; Akaike, 1987). LRT are based on the deviance variation, $-2\ln(L)$ and degrees of freedom, between an initial, less constrained model and a candidate, hypothetical model. In the case where the likelihood of the hypothetical submodel is statistically different ($p \leq .05$) from that of the initial model, the fit of the submodel is considered to be poorer and may be rejected. Estimating the AIC developed the fitting process further. This criterion was included because LRT tend to favor models with more estimated parameters. This may result in submodels offering very low p values ($p < .001$) even when minor deviations with respect to the initial model are present (Christensen et al., 2003). The AIC tends to disfavor models with more parameters and so balances model fit with model parsimony. Initially, smaller AIC values indicate a better fit to the data. Where a discrepancy was noticed between the LRT and the AIC, preference was given to models achieving the best fit with the AIC.

Sex differences were tested as follows. In a first step, potential sex differences in A, C, and E estimates were tested by comparing model fit statistics from a model that constrains the parameters to be equal for men and women with respect to models where the parameters were allowed to differ by sex. Afterward, potential sex-specific genetic effects underlying leisure-time physical activity were tested by comparing the model fit statistics from a model in which the genetic covariance between opposite-sex DZ twins was constrained to be equal (at a 0.5 level, the expected genetic correlation for full siblings) with those from a less constrained model where the genetic covariance between opposite-sex DZ twin was freely estimated. All the analyses were developed using the R-CRAN statistical software and utilizing the specialized packages 'psych' and 'OpenMx' (Boker et al., 2011; R Development Core Team, 2011; Revelle, 2011).

Results

Tables 1 and 2 show the characteristics and the correlations of the sample at each data collection wave. The mean ages (and standard deviation) of the participants at the four data collection waves were 16.2 (0.1), 17.1 (0.1), 18.6 (0.2), and 24.5 (0.9) years. At baseline, 30.7% of the participants (34.6% of the men and 27.6% of the women) were very active. However, the percentage of very active persons had decreased to 26.8% by young adulthood (25.3% of the men and 27.9% of the women). In contrast, the percentage of moderately active participants increased from 47.8% (43.5% of the men and 51.3% of the women) to 52.5% (49.8% of the men and 54.8% of the women) during the follow-up. The percentage of inactive participants remained relatively stable over the follow-up, except for a slight decrease observed at the mean age of 17.1 years.

The analyses of the polychoric correlations revealed that the MZ twins were more likely to have a similar leisure-time physical activity level than the DZ twins, suggesting the presence of A in the phenotype. The preliminary univariate biometric models confirmed that models with A, C, and E fitted to the data better than the other sub-models with fewer estimated parameters (Table 3). The modeling of the Cholesky decompositions corroborated the need to include A, C, and E in the final longitudinal model. Additional tests for sex differences revealed that constraining the genetic covariance for opposite-sex DZ to be the same as that for same-sex DZ twins did not present an improvement in model fit. This result suggested not only that the estimates of A, C, and E were different in men and women, but also that there were sex-specific genetic effects (Table 4).

Observation of the pattern of additive genetic, shared environmental, and specific environmental influences across the follow-ups revealed an interesting phenomenon. As described in Figure 1, in both men and women the heritability of leisure-time physical activity remained relatively stable during adolescence at ~43–52%, finally declining to ~30% in young adulthood. In contrast, shared environmental influences also showed relative stability during adolescence at ~18–26%, finally increasing to 43% in men and 49% in women in young adulthood. Specific environmental influences remained relatively stable at all the

https://doi.org/10.1017/thg.2013.9 Published online by Cambridge University Press

TABLE 3

Model Fitting Statistics Using Raw Data, Assuming Unequal Thresholds

	−2LL	*df*	ΔLL	Δ*df*	AIC	*P* value
Physical activity at mean age 16.2 years						
1. ACE model, allowing sex differences	8,437.78	4,300	—	—	−162.22	—
2. ACE model, same genes in men and women	8,443.60	4,301	5.82	1	−158.40	.02
3. ACE model, equating men and women	8,457.25	4,304	19.47	4	−150.75	<.001
4. AE model, allowing sex differences	8,446.69	4,302	8.91	2	−157.31	.01
5. AE model, same genes in men and women	8,455.78	4,303	18.01	3	−150.22	<.001
6. AE model, equating men and women	8,457.55	4,305	19.77	5	−152.45	<.001
7. CE model, allowing sex differences	8,538.30	4,303	100.52	3	−67.70	<.001
8. CE model, equating men and women	8,545.11	4,305	107.33	5	−64.89	<.001
Physical activity at mean age 17.1 years						
1. ACE model, allowing sex differences	7,906.50	4,093	—	—	−279.50	—
2. ACE model, same genes in men and women	7,911.79	4,094	5.29	1	−276.21	.02
3. ACE model, equating men and women	7,923.36	4,097	16.86	4	−270.64	<.001
4. AE model, allowing sex differences	7,919.94	4,095	13.44	2	−270.06	<.001
5. AE model, same genes in men and women	7,924.72	4,096	18.23	3	−267.28	<.001
6. AE model, equating men and women	7,925.81	4,098	19.31	5	−270.19	<.001
7. CE model, allowing sex differences	7,978.39	4,096	71.89	3	−213.61	<.001
8. CE model, equating men and women	7,986.03	4,098	79.54	5	−209.97	<.001
Physical activity at mean age 18.6 years						
1. ACE model, allowing sex differences	7,990.29	4,068	—	—	−145.71	—
2. ACE model, same genes in men and women	7,990.44	4,069	0.15	1	−147.56	.7
3. ACE model, equating men and women	8,004.20	4,072	13.92	4	−139.80	.01
4. AE model, allowing sex differences	7,998.11	4,070	7.82	2	−141.89	.02
5. AE model, same genes in men and women	8,003.67	4,071	13.38	3	−138.33	<.001
6. AE model, equating men and women	8,004.20	4,073	13.92	5	−141.80	.02
7. CE model, allowing sex differences	8,087.06	4,071	96.77	3	−54.94	<.001
8. CE model, equating men and women	8087.09	4,073	96.81	5	−58.91	<.001
Physical activity at mean age 24.5 years						
1. ACE model, allowing sex differences	6,567.04	3,742	—	—	−916.96	—
2. ACE model, same genes in men and women	6,567.91	3,743	0.86	1	−918.09	.35
3. ACE model, equating men and women	6,568.36	3,746	1.31	4	−923.64	.86
4. AE model, allowing sex differences	6,606.90	3,744	39.86	2	−881.10	<.001
5. AE model, same genes in men and women	6,617.94	3,745	50.89	3	−872.06	<.001
6. AE model, equating men and women	6,617.97	3,747	50.93	5	−876.03	<.001
7. CE model, allowing sex differences	6,580.99	3,745	13.95	3	−909.01	<.001
8. CE model, equating men and women	6,582.01	3,747	14.97	5	−911.99	.01

Note: LL = log-likelihood; *df* = degrees of freedom; ΔLL = log-likelihood difference (χ^2) between the initial model and fitted submodel; Δ*df* = increment in degrees of freedom with respect to the initial model; AIC = Akaike's Information Criterion.

TABLE 4

Multivariate (Longitudinal) Model Fitting Statistics

Model	−2LL	*df*	ΔLL	Δ*df*	AIC	*P* value
1. ACE model, allowing sex differences	33,778.53	19,498	—	—	−5,217.47	—
2. ACE model, same genes in men and women	33,820.48	19,502	41.95	4	−5,183.52	<.001
3. ACE model, equating men and women	33,912.34	19,532	133.81	34	−5,151.66	<.001
4. AE model, allowing sex differences	33,851.26	19,518	72.73	20	−5,184.74	<.001
5. AE model, same genes in men and women	34,045.29	19,522	266.76	24	−4,998.71	<.001
6. AE model, equating men and women	33,912.35	19,542	133.82	44	−5,171.65	<.001
7. CE model, allowing sex differences	34,283.43	19,522	504.90	24	−4,760.57	<.001
8. CE model, equating men and women	34,098.71	19,542	350.18	44	−4,985.29	<.001

Note: LL = log-likelihood; *df* = degrees of freedom; ΔLL = log-likelihood difference (χ^2) between the initial model and fitted submodel; Δdf = increment in degrees of freedom with respect to the initial model; AIC = Akaike's Information Criterion.

follow-ups, ranging between ∼20% and 30% in both men and women.

Baseline genetic influences had a residual effect in the subsequent waves that tended to decrease with age. The genetic correlation (r_a) between the first and second waves was 0.78 for men and 0.67 for women, the corresponding estimates between the first and the last waves were ∼0.44 for both sexes. This suggests that only ∼19% of the genetic influences detected at the mean age of 16.2 years were present at the mean age of 24.5 years. Similarly, baseline shared environmental influences had a residual effect that tended to decrease with age. The shared environmental correlations (r_c) between the first and second waves were 0.76 for men and 0.81 for women, and the corresponding estimates between the first and the last waves were 0.57 for men and 0.41 for women. Finally, a parallel trend was observed for the specific environmental correlation (r_e): the values between the first and second waves were as high as 0.44 for men and 0.36 for women, and the corresponding estimates between the first and the last waves were 0.10 for men and 0.19 for women.

https://doi.org/10.1017/thg.2013.9 Published online by Cambridge University Press

https://doi.org/10.1017/thg.2013.9 Published online by Cambridge University Press

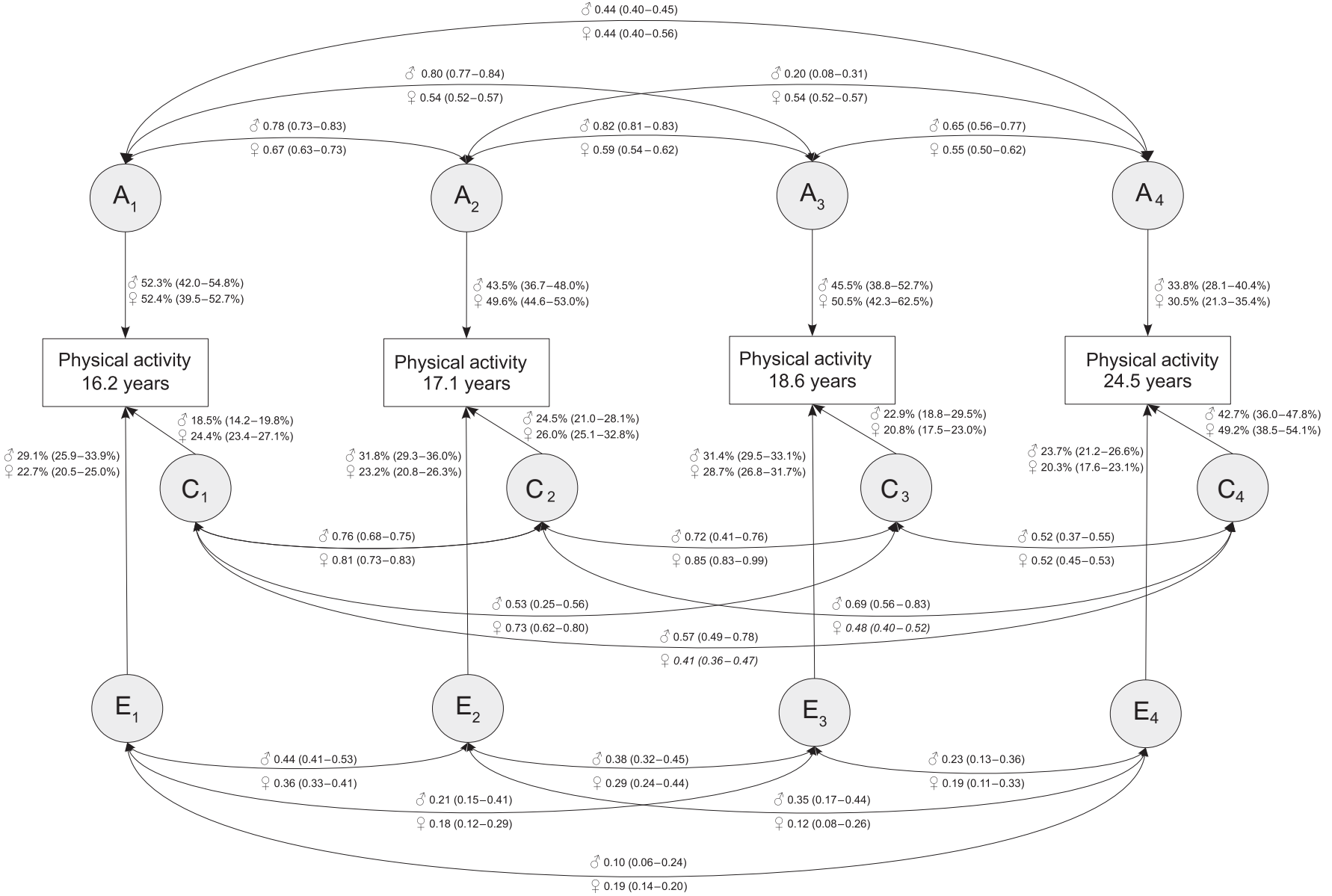

FIGURE 1

Summary of the best-fitting longitudinal Cholesky decomposition for leisure-time physical activity over a period between ages 16.2 and 24.5 years.

Discussion

The present study among healthy 16–24-year-old men and women revealed a change in the pattern of genetic and environmental influences in the progress of leisure-time physical activity from adolescence to young adulthood. The relative role of genetic influences remained rather stable during adolescence and declined in young adulthood. Shared environmental influences, in turn, also showed relative stability during adolescence, but in contrast to genetic influences, increased markedly in young adulthood. Furthermore, new genetic, shared, and specific environmental influences emerged at each follow-up point, as the correlations across occasions were far from being even close to unity. In addition, the correlations decreased gradually with age, the phenomenon being more pronounced in the transition from late adolescence (mean age 18.5 years to young adulthood mean age 24.5 years).

Generally, adolescence and young adulthood are a period of multiple changes in health-related behaviors, as also emerged in the present study. The number of very active persons decreased during the 8-year follow-up, also confirming the earlier results of physical activity changes in adolescence and in young adulthood (Dumith et al., 2011; Kimm et al., 2002). Furthermore, this study produced results that corroborate the findings of much of the previous work in this field, suggesting that the heritability of physical activity behavior ranges between 27% and 71% (Beunen & Thomis, 1999; Carlsson et al., 2006; Kaprio et al., 1981; Maia et al., 2002; Stubbe et al., 2006; Stubbe & de Geus, 2009). In the present study, the heritability of leisure-time physical activity ranged between ∼30% and ∼52%. This lends important support to the idea that physical activity levels are moderately accounted for by genetic influences.

However, the studies of genetic influences on physical activity behavior during adolescence and young adulthood have shown discrepancies. Our results support previous cross-sectional findings suggesting that genetic effects are relatively more prominent during adolescence than in young adulthood (Mustelin et al., 2012; van der Aa et al., 2010; Vink et al., 2011). In the present study, genetic influences decreased as early as after the age of 18 years. Moreover, the present study seems to be the first one to emphasize not specific, but shared environmental effects in young adulthood. We also found a decrease both in genetic influences on physical activity and in the proportion of very active participants during the 8-year follow-up from adolescence to young adulthood. This may suggest a connection between these two factors. On the other hand, the decrease in physical activity observed during the follow-up may have other explanations. For example, it is a known fact that a low level of physical activity and obesity are related to each other; however, it has been suggested recently that obesity may be a driver of physical inactivity (Bauman et al., 2012). In general, people, including the present twins, gain in weight as they get older (Nooyens et al., 2009), which may explain the decrease in physical activity. Interestingly, in our study, the number of very active men and the number of inactive women decreased during the follow-up. Probably due to this phenomenon, sexes became more alike to each other, increasing the polychoric correlations for DZ opposite-sex twin pairs in adolescence. It should also be borne in mind that the idea that environmental influences may have more effect on leisure-time physical activity as people get older is not a finding from genetic studies alone. Several life events may decrease leisure-time physical activity behavior (Engberg et al., 2012), and it is generally known that major life transitions such as moving out of the parental home, starting work, continuing to tertiary education, and the formation of new interpersonal relationships are very common in young adulthood. Such changes have not been analyzed in the present article.

Including the present study, longitudinal genetic models have been examined in only a few studies (Aaltonen et al., 2010; Eriksson et al., 2006; Turner et al., 2005). However, the findings on the genetic influences in the progress of physical activity in these studies are largely consistent with each other: they all reveal that genetic influences on physical activity change with age. In particular, our findings corroborate the suggestion of Eriksson et al. (2006) that the heritability estimate of leisure-time physical activity is reduced in young adulthood. However, some contradictory findings also emerged between the present study and earlier longitudinal studies. The present study provides information on the fact that only a small proportion of the genetic influences detected at baseline were present at the last follow-up point, while most of the genetic effects were sustained across time among the older adult twins aged 18–60 in our earlier study (Aaltonen et al., 2010). Our earlier study covered the time period of 1975 to 1990, while the present study data were collected between 1991 and 2002, so changes in society over 30 years may also affect the comparability of the two studies even within a single country.

Comparison between studies may be difficult because of the many differences in study designs. The discrepancies between heritability estimates of physical activity may partially be explained by different samples: both human and animal studies have been conducted, sample sizes vary widely, and samples are comprised of different age groups, sexes, and ethnic groups. In addition, studies differ in the methods used to capture physical activity, in the type of activities studied, and in definitions of physical activity, such as daily physical activity, leisure-time physical activity, sports participation, and exercise participation. The terms 'physical activity' and 'exercise' are often used interchangeably, even though the 'exercise' is a subcategory of 'physical activity'. These definitions may assess slightly different aspects of self-chosen physical activity and may have an effect on the study results. Concerning the present study, it is also important to understand that in Finnish, which is the original language

https://doi.org/10.1017/thg.2013.9 Published online by Cambridge University Press

of the physical activity question, the word 'liikunta' used in our questionnaire corresponds as much to physical activity as to exercise. Furthermore, the possibility of genetic differences between populations investigated should be noted.

A key strength of the present study is the use of a longitudinal design. Longitudinal studies are useful for investigating the predictors of physical activity as they may capture a true aging effect (Vink et al., 2011). Although many previous studies have examined genetic and environmental influences on physical activity, longitudinal data have been used on only a few occasions (Aaltonen et al., 2010; Eriksson et al., 2006; Turner et al., 2005). In the present study, the longitudinal evolution of leisure-time physical activity habits from adolescence to young adulthood provided new information on genetic and environmental influences during a specific part of the life course. Indeed, because we had data on specific age groups, we had a unique opportunity to investigate age-specific influences on change in leisure-time physical activity. A further strength of this study is the adequate size of the study sample. We also excluded all subjects with overt chronic diseases, which should have minimized the possibility of the influence on diseases on the level of physical activity reported by the subjects. Thus, our results can be generalized only to healthy people. The main limitation of the present study is related to the outcome measure. Physical activity is a complex trait, and only the assessment of the physical activity frequency, as in the present study, is not the most optimal way to measure the physical activity behavior. However, in our study the physical activity frequency was the only variable available longitudinally to assess physical activity. Potential limitations of self-reported estimates of leisure-time physical activity are that they may be unreliable and lack validity. Although the validity of our questionnaire has been demonstrated (Kaprio et al., 1978; Kujala et al., 1998; Sarna et al., 1978; Waller et al., 2008), the possibility of errors cannot be avoided when using such a non-objective instrument. All self-reports are prone to various reporting biases, which mean that measurement errors may also explain a small part of the results. In genetic models, measurement error is subsumed into the unique environmental component of variance.

In conclusion, the results of the study suggest that for both men and women, genetic influences are rather stable during adolescence (16–18 years). They seem to be slightly greater for women than for men, and tend to decrease by young adulthood in both sexes. In contrast, shared environmental influences had increased by young adulthood, especially in women. Altogether, these outcomes indicate that variations in environmental factors are the key element to understanding the observed deterioration in physical activity levels. There also seems to be a need for gender-specific measures to promote physically active leisure time in young adulthood. A prerequisite of better focused health promotion is an understanding of the role of increasing environmental influences and the role of targeted physical activity-promoting measures during this period of life. Public health promoters and health policy-makers should see the transitional period from adolescence to young adulthood as a strategic point to stimulate leisure-time physical activity that would lead also to an active lifestyle in later adulthood.

Clinically, physical activity-promoting measures may even be more important for women than for men, because of the greater role of environmental influences in women. This greater role may partly be connected with the fact that emerging adulthood brings with it different role expectations for women than for men. Although egalitarian gender role attitudes provide models for negotiating family and work, family responsibilities and child rearing nevertheless continue to be performed mainly by women (Davis & Greenstein, 2009). This may partially explain the differences between physical activity habits among men and women. Overall, our study contributes evidence for age-specific genetic and environmental influences. However, more research is needed and future genetic studies should consider more precisely the age of the sample when investigating genetic variants mediating longitudinal leisure-time physical activity.

Acknowledgments

The study was supported by the Finnish Ministry of Education (SA and UMK), Tekes — the Finnish Funding Agency for Technology and Innovation (grant number 1104/10; UMK), the Juho Vainio Foundation (SA), and the Yrjö Jahnsson Foundation (SA). JK has been supported by the Academy of Finland Center of Excellence in Complex Disease Genetics (grant numbers 213506 and 129680). AO-A has been supported by the Finland Distinguished Professor Programme of the Academy of Finland (grant number 139904). Data collection of the Finntwin16 study has been supported by the U.S. National Institute of Alcohol Abuse and Alcoholism (grants AA-12502, AA-00145, and AA-09203 to R. J. Rose) and the Academy of Finland (grants 100499, 205585, and 141054 to JK).

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