A Longitudinal Study on Genetic and Environmental Influences on Leisure Time Physical Activity in the Finnish Twin Cohort

Sari Aaltonen,[1] Alfredo Ortega-Alonso,[2] Urho M. Kujala,[3] and Jaakko Kaprio[4,5]

[1] *Department of Health Sciences, University of Jyväskylä, Finland*
[2] *Department of Public Health, University of Helsinki, Finland*
[3] *Department of Health Sciences, University of Jyväskylä, Finland*
[4] *Department of Public Health, University of Helsinki, Finland*
[5] *Department of Mental Health and Substance Abuse Services, National Institute for Health and Welfare, Helsinki, Finland*

The purpose of this study was to examine changes in the contribution of genetic and environmental influences to leisure time physical activity among male and female twins over a 6-year follow-up. At baseline the sample comprised 4,280 monozygotic and 9,276 dizygotic twin individuals, and at follow-up 4,383 monozygotic and 9,439 dizygotic twin individuals. Participants were aged 18–54 years at baseline. Genetic modeling results showed that genetic influences on leisure time physical activity declined from baseline (44%) to follow-up (34%). Most of the genetic influences identified at baseline were present at follow-up (r_g = 0.72). Specific environmental influences increased from baseline (56%) to follow-up (66%) while at follow-up new environmental time-specific influences were observed (r_e = 0.23). The model with sex differences showed a higher estimate of genetic influences for men than women both at baseline (men 47% vs. women 42%) and at follow-up (men 38% vs. women 31%). The additive genetic correlation for this phenotype was greater for men (r_g = 0.79) than women (r_g = 0.64). The specific environmental influences were corresponding; at baseline men 53% and women 56% and at follow-up men 62 % and women 69%. The environmental correlations between the two time points were similar for men (r_e= 0.21) and for women (r_e= 0.24). In conclusion, in a sample of healthy twins most of the genetic influences on leisure time physical activity expressed at baseline were present at 6 years of follow-up. New specific environmental factors underlying follow-up leisure time physical activity were observed.

Keywords: genetic influences; heritability; physical activity; twin study

At present considerable evidence exists on the benefits of physical activity, such as reduced prevalence of several diseases and reduced mortality (Kujala et al., 1998; Laaksonen et al., 2004; Morris et al., 1980; Paffenbarger et al., 1986; Physical Activity Guidelines Advisory Committee, 2008). However, part of the population still remains nearly completely sedentary (Martínez-González et al., 2001; Pratt et al., 1999). The fact that many different factors play a role in exercise behavior, presents a challenge for researchers interested in exploring the reasons for physical activity. Twin studies provide an opportunity to disentangle the effects of genes and environment.

A number of twin studies have shown that the genetic influences play an important role in explaining individual differences in exercise participation and leisure time 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). Genetic influences seem to be higher for vigorous activity compared to nonvigorous activity. The largest of these studies pooled data on leisure time exercise behavior from seven different countries (GenomEUtwin project), and found that heritability of exercise participation ranged from 48% to 71%, with the exception of Norwegian males 27% (Stubbe et al., 2006). However, in some studies, one of which is recent, environment factors have been shown to exert the strongest influence on physical activity participation (Duncan et al., 2008; Perusse et al., 1989). Although many studies have focused on the genetic determinations of leisure time physical activity, to date little is known about longitudinal changes (Stubbe et al., 2005).

As all the previous knowledge on the genetics of physical activity among adults has been based on cross-sectional data, it has not been possible to test the potential age dependency of genetic influences or examine changes in genetic and environmental influences on physical activity. Although, there are indications that physical activity level declines with age

Received 8 June, 2010; accepted 30 July, 2010.

Address for correspondence: Sari Aaltonen, Department of Health Sciences (Viv), P.O Box 35, FIN-40014 University of Jyväskylä, Finland. E-mail: sari.s.aaltonen@jyu.fi

(Crespo et al., 1996; Sallis, 2000), few studies have demonstrated changing heritability estimates across the lifespan for exercise (Simonen et al., 2004) and sports participation (Stubbe et al., 2005). An animal study showed that genetic influences on physical activity changed with age in different groups of selectively bred mice, suggesting potential interactions between genetic background and age in explaining differences in physical activity behaviors (Turner et al., 2005).

The main aim of our study was to examine changes in genetic and environmental factors on leisure time physical activity among male and female twins over a 6-year follow-up. Overall, increased knowledge of the persistent, longitudinal factors influencing leisure time physical activity may help us in developing effective means of physical activity counselling.

Subjects and Methods

Study Cohort

This research is part of the Finnish Twin Cohort study (Kaprio et al., 1990; Kaprio et al., 1978). The cohort was established in 1975 and it consists of same-sex twins born in Finland before 1958 and with both co-twins alive in 1967. Zygosity of twins was defined on the basis of a validated questionnaire according to whether people always confused the twins in childhood and how similar in appearance they were (Kaprio et al., 1978; Sarna et al., 1978). The data used in this study was collected at two time points, 1975 and 1981, through mail surveys. The mailed questionnaires also included items on body weight, body height, physical activity, occupation, alcohol use and smoking.

The current study consisted of 4 280 monozygotic (MZ) and 9 276 dizygotic (DZ) twin individuals at baseline in 1975 and 4 383 MZ and 9 439 DZ twin individuals at follow-up in 1981. Overall, there were 4 000 MZ and 8 660 DZ twin individuals participating both at baseline and at follow-up. We included subjects who were working in 1981, supplied complete questionnaire data on the intensity of their leisure time physical activity at both time points and were aged 24–60 years on January 1, 1982. These inclusion criteria were needed because occupational or work status may partly determine the levels of leisure activity. Because chronic disease may also restrict the ability to exercise, we excluded at baseline subjects with specific chronic diseases (e.g. diabetes) or who were receiving reimbursable medication for selected chronic diseases via procedures described elsewhere (Kujala et al., 2002).

Descriptive Variables and Assessment of Volume of Leisure Time Activity

The descriptive variables and assessment of leisure time physical activity volume were based on the questionnaire. Body mass index was calculated from body weight and height, which were elicited in the questionnaire. The volume of leisure activity in metabolic equivalent units (MET index) was based on a series of structured questions on leisure time physical activity (monthly frequency, mean duration and mean intensity of sessions) and physical activity during journeys to and from work. The index was calculated by assigning a multiple of resting metabolic rate (MET score) to each activity and by calculating the product of activity, defined as intensity × duration × frequency. The MET index was expressed as the sum score of leisure MET hours/day. Earlier analyses have shown high correlations between the leisure time physical activity questions we used and physical activity data obtained by interview (Waller et al., 2008).

Statistical Analyses

The normality of the data was tested by the Kolmogorov-Smirnov test, equality of means and variances by ANOVA, *t* test and Mann-Whitney U test using SPSS version 15.0 (SPSS Inc. Chigago, IL) and Stata version 10.0 (Stata Corp., College Station, TX), which allows controlling for the clustering of observations from twin pairs. Because the MET index variables showed non-normality, which may later compromise the genetic modeling procedures, they were normalized using rank transformation methods. To obtain preliminary information on the within-pair resemblance, we computed intraclass correlation coefficients (ICCs) separately for MZ and DZ twins.

The twin data were further analyzed following biometric methods (Neale, 1997), using maximum likelihood algorithms, and treating unobserved data as missing-at-random (Little & Rubin, 2002), including the individual's age as a definition variable. The significance of estimates and path coefficients were tested by removing them sequentially in different subsequent models. Their fit was compared against the fit of the unconstrained initial model in which a higher number of possible paths of relations and estimates were present. This comparison was done by applying likelihood-ratio tests (LRT) (Akaike, 1987), the Schwartz's Bayesian Information Criterion (BIC) (Schwartz, 1978) and the Deviance Information Criterion (DIC) (Spiegelhalter et al., 2002). Initially, smaller AIC, BIC or DIC values normally indicate a better fit to the data. When a discrepancy was noticed between these criteria, preference was given to the models achieving best fit with the BIC and DIC, as they are thought to perform better than the other criteria in more complex models with relatively large sample sizes (Markon, 2004). In addition, because the sample was composed of men and women, the significance of potential gender differences in the estimates were tested by comparing model fitting statistics from a model that constrains the A, C, and E parameters to be equal for men and women with models where these parameters are allowed to differ by gender.

Results

The sample characteristics and pairwise correlations are shown in Table 1. Among the participants the average volume of leisure time physical activity increased from baseline (2.9 ± 3.3) to follow-up (3.1 ± 3.3) (p <.001). However, their body mass index (BMI)

https://doi.org/10.1375/twin.13.5.475 Published online by Cambridge University Press

also increased from baseline (22.6 ± 3.0) to follow-up (23.3 ± 3.2) ($p < .001$). At all times the MZ twins resembled each other more than did DZ twins, which is indicative of the greater importance of genetic influences on the physical activity phenotype.

This observation was later confirmed in the results of the longitudinal bivariate Cholesky decomposition (Table 2). The genetic modeling was started with the full ACE model. After dropping the weakest parameters, the model with the best fit to the data according to the DIC contained additive genetic (A) and specific environmental (E) influences with equal estimates in men and in women. Because the model with gender differences may also provide the best fit according to the AIC and BIC, it is preferable to report the results from both models, with and without gender differences in the parameter estimates (Markon, 2004).

Accordingly, in the overall sample model (with no gender differences) the genetic influences on physical activity declined from 44% of total variance at baseline to 34% at follow-up (Figure 1). The remaining variance at each time point was explained by environmental influences. The high genetic correlation between the time points (r_g = 0.72) suggested that an important amount of the additive genetic influences at baseline was still present at follow-up. Conversely, the environmental correlation between the two time points was modest (r_e= 0.23). The longitudinal phenotypic correlation between the baseline and follow-up measures was moderate (r_p = 0.42), with 67% due to longitudinal additive genetic influences.

In the model allowing gender differences, we observed a similar pattern of declining genetic influences from baseline to follow-up in both men (from 47% to 38%) and women (from 42% to 31%). The remaining percentages at each time were accounted for by environmental influences. The additive genetic correlation for leisure time physical activity was greater for men (r_g = 0.79) than for women (r_g = 0.64) (Figure 2). The environmental correlation between the two time points did not differ substantially between the sexes (men r_e = 0.21 vs. women r_e = 0.24). The longitudinal phenotypic correlation in men was r_p=0.45 of which 74% was due to longitudinal additive genetic influences, while in women the longitudinal phenotypic correlation was r_p = 0.38 of which 60% was due to longitudinal additive genetic influences.

Our results indicated that the drop detected in the heritability of leisure time physical activity was produced by a decline in the genetic variance and an increase in the environmental variance, with no substantial change in

Table 1

Baseline and Follow-Up Characteristics

Variable	Baseline 1975			Follow-up 1981		
	N	Mean ± *SD*	ICC (95% CI)	*N*	Mean ± *SD*	ICC (95% CI)
Age, years	13,556	29.6 ± 9.0		13,822	35.6 ± 9.1	
MZ	4,280	29.4 ± 9.0		4,383	35.4 ± 9.0	
DZ	9,276	29.8 ± 9.1		9,439	35.7 ± 9.1	
BMI, kg/m^2	13,456	22.6 ± 3.0		13,728	23.3 ± 3.2	
MZ	4,250	22.3 ± 3.0	0.79 (0.77–0.80)	4,360	23.1 ± 3.1	0.78 (0.76–0.80)
DZ	9,206	22.7 ± 3.0	0.52 (0.50–0.55)	9,368	23.4 ± 3.2	0.47 (0.44–0.50)
MET hours/day	13,556	2.9 ± 3.3		13,822	3.1 ± 3.3	
MZ	4,280	3.1 ± 3.6	0.54 (0.51–0.58)	4,383	3.1 ± 3.3	0.43 (0.39–0.47)
DZ	9,276	2.8 ± 3.1	0.24 (0.20–0.27)	9,439	3.1 ± 3.3	0.15 (0.11–0.18)

Note: MZ = monozygotic; DZ = dizygotic; SD = standard deviation; ICC = intraclass correlation coefficients; CI = confidence intervals.

Table 2

Results from Fitting a Bivariate Longitudinal Cholesky Decomposition

Model	-2LL	df	χ^2	Δdf	*p*	AIC	BIC	DIC
ACE: gender differences	74317.631	27356	—	—	—	19605.631	-44546.601	-62874.638
ACE: no gender differences	74347.149	27365	29.519	9	0.001	19617.149	-44558.723	-62892.789
ACE males – AE females	74318.197	27359	0.567	3	0.904	19600.197	-44555.278	-62885.325
AE males – ACE females	74317.631	27359	0.000	3	1.000	19599.631	-44555.562	-62885.608
AE: gender differences	74318.197	27362	0.567	6	0.997	19594.197	-44564.239	-62896.295
AE: no gender differences	74347.345	27368	29.715	12	0.003	19611.345	-44567.585	-62903.661
CE: gender differences	74422.111	27362	104.481	6	0.000	19698.111	-44512.281	-62844.338
CE: no gender differences	74450.719	27368	133.089	4	0.000	19714.719	-44515.898	-62851.974

https://doi.org/10.1375/twin.13.5.475 Published online by Cambridge University Press

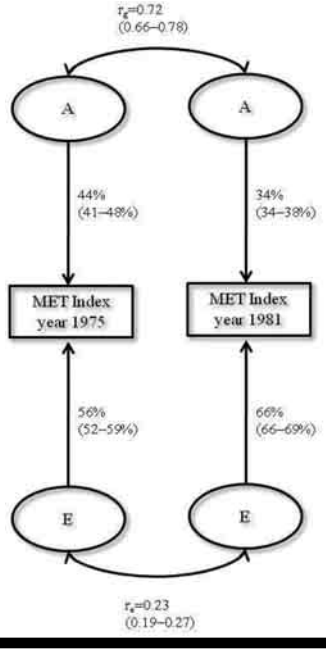

Figure 1

Summary models of the changes over 6 years in influences on leisure time physical activity. A refers to additive genetic and E to specific environmental influences. Estimates for each pathway to leisure time physical activity are shown with 95% confidence intervals (CI). Additive genetic and specific environmental correlations between the baseline and follow-up results are shown as curved arrows along with the estimates and 95% CI.

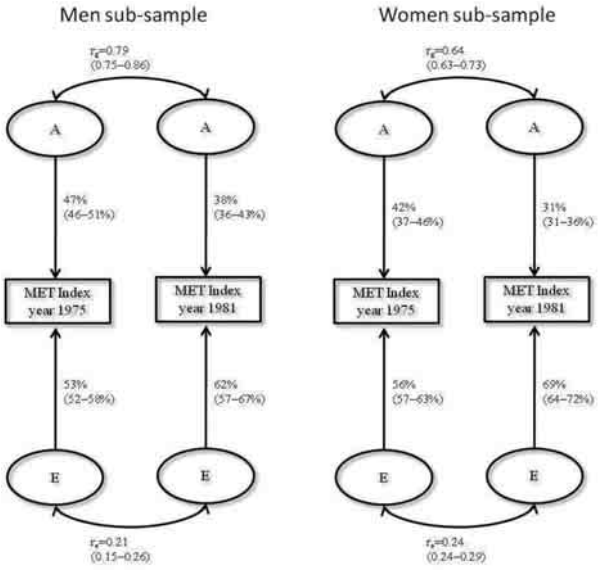

Figure 2

Summary models of the changes over 6 years in influences on leisure time physical activity among men and women. A refers to additive genetic and E to specific environmental influences. Estimates for each pathway to leisure time physical activity for both sexes are shown with 95% confidence intervals (CI). Additive genetic and specific environmental correlations between the baseline and follow-up results are shown as curved arrows along with the estimates and 95% CI.

Table 3

Raw Variance Estimates for Physical Activity (MET index) at Baseline and at Follow-Up

	Baseline		6-years follow-up	
	Men (95% CI)	Women (95% CI)	Men (95% CI)	Women (95% CI)
Total variance	1.02 (0.98–1.06)	0.97 (0.93–1.00)	1.02 (0.98–1.06)	0.96 (0.96–1.00)
Genetic variance	0.48 (0.43–0.53)	0.40 (0.35–0.45)	0.39 (0.33–0.45)	0.30 (0.26–0.35)
Environmental variance	0.54 (0.50–0.59)	0.56 (0.55–0.61)	0.63 (0.62–0.69)	0.67 (0.62–0.72)
Heritability	0.47 (0.46–0.51)	0.42 (0.37–0.46)	0.38 (0.36–0.43)	0.31 (0.31–0.36)

Note: CI = confidence intervals

the overall variance. These results were consistent in both men and women (Table 3).

Discussion

The most important results in the present study were that among healthy 18- to 60-year-old men and women genetic influences on leisure time physical activity were moderate at baseline and slightly declined over a 6-year follow-up. Baseline genetic influences were highly correlated with those at follow-up, suggesting the consistent expression of an important group of genes at both time points, but also that a relatively small proportion of new influences emerged with age.

As far as we know, a longitudinal genetic model of physical activity has not been examined in many earlier studies. Many cross-sectional twin studies have explored the genetic influences on physical activity but so far the results have been somewhat conflicting (Beunen & Thomis, 1999; Carlsson et al., 2006; Duncan et al., 2008; Kaprio et al., 1981; Maia et al., 2002; Perusse et al., 1989; Stubbe et al., 2006; Stubbe & de Geus, 2009). Our study provides new information on the longitudinal genetic and environmental factors influencing physical activity and in particular the fact that most of the genetic effects were sustained across time. In addition, our study suggested that a small proportion of these genes could switch off and others switch on with age.

Heritability has been shown to have an increasing influence on exercise from adolescence to adulthood (Simonen et al., 2004). A study among adolescents and young adults showed that younger twins participated more in sports than older twins (Stubbe et al., 2005). The same study also suggested that the influence of

https://doi.org/10.1375/twin.13.5.475 Published online by Cambridge University Press

genes on individual differences in sports participation gains in importance during late adolescence. The present longitudinal study among adults indicated that environmental influences on leisure time physical activity increase with age. The decline in the heritability of physical activity was produced by a fall in the genetic variance and an increase in the environmental variance. This would suggest a complex longitudinal mechanism in which genetic influences are mostly sustained while environmental influences gain in strength, but with highly inconsistent effects across time. This is supported by an animal model, which also showed that genetic background has a highly significant influence on physical activity level which in turn changes as a function of time (Turner et al., 2005). It seems that because the genetic effects tend to remain mostly stable, fluctuations in the overall physical activity level across early adolescence and again in adulthood are mostly determined by changes in environmental influences. Before adolescence, the shared family environmental influences seem to play an important role. In adolescence, environmental influences decline until adulthood when unique environmental influences began to gain in importance once again. Consequently, genetic effects peak in their contribution to physical activity in very young adulthood (Stubbe & de Geus, 2009).

The estimates of additive genetic influences for leisure time physical activity differed by gender being higher for men than for women both at baseline and at follow-up. The additive genetic correlation for this phenotype was also greater for men than for women. In this case, genetic influences seemed to be more important in keeping men physically active. There is also evidence suggesting that the genetic influences may play a more important role in men's leisure time physical activity. The genetic contribution may be higher for vigorous activity than for nonvigorous activity, as shown by several studies (Beunen & Thomis, 1999; Kaprio et al., 1981; Lauderdale et al., 1997), and it is known that men exercise more vigorously than women (Barnekow-Bergkvist et al., 1996).

On the other hand, comparison between studies is difficult because of the many differences in study parameters. The changing heritability estimates of physical activity and exercise participation may partially be explained by different sample sizes, the specific physical activity measurements used and different age ranges within samples. Different studies have also used different definitions of self-chosen physical activity. The most used ones of these are; daily physical activity, leisure time physical activity, sports participation and exercise participation. These definitions may assess slightly different aspects of self-chosen physical activity and may have an effect on the study results, mainly on the grounds that the genetic contribution may be different for different intensity levels of physical activity (Beunen & Thomis, 1999; Kaprio et al., 1981; Lauderdale et al., 1997).

The greatest strength of the present study is the use of a large data set that has been followed longitudinally. We excluded all the subjects with chronic diseases, which should have eliminated 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. Among the potential limitations of our study is the use of self-report questionnaire data to estimate physical activity level. 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 the measurement errors may also explain a small part of the results. In addition, in the present study the age range of the sample was wide. If we had divided the data set in different age groups, there may have been some age-specific results, but there are no natural ages at which to divide adults according to overall leisure physical activity.

In conclusion, in healthy men and women genetic influences on leisure time physical activity at baseline were moderate. These influences declined somewhat within six-year follow-up. Genetic influences at baseline were nevertheless highly correlated with those at follow-up suggesting both the consistent expression of an important group of genes at both time points, and also that a relatively small proportion of a new influences emerged with age. Therefore, as some genetic influences appear to be partially age-specific, future genetic studies would need to concentrate more precisely on the age of their sample when investigating genetic variants mediating longitudinal leisure time physical activity. Clinically, understanding the relative role of stable genetic and changing environmental influences on physical activity is a key to better focused health promotion.

Acknowledgments

The study was supported by the Finnish Ministry of Education (S.A and U.M.K.), the Juho Vainio Foundation (S.A.) and the Yrjö Jahnsson Foundation (S.A). A.O-A. has been partially supported by the Genodisc project, which has received funding from the European Community's Seventh Framework Programme (FP7, 2007-2013) under grant agreement no. HEALTH-F2-2008-201626. The Finnish Twin Cohort is supported by the Academy of Finland Centre of Excellence in Complex Disease.

References

Akaike, H. (1987). Factor-analysis and AIC. *Psychometrika, 52*, 317–332.

Barnekow-Bergkvist, M., Hedberg, G., Janlert, U., & Jansson, E. (1996). Physical activity pattern in men and women at the ages of 16 and 34 and development of physical activity from adolescence to adulthood.

https://doi.org/10.1375/twin.13.5.475 Published online by Cambridge University Press

Scandinavian Journal of Medicine and Science in Sports, 6, 359–370.

Beunen, G. & Thomis, M. (1999). Genetic determinants of sports participation and daily physical activity. *International Journal of Obesity and Related Metabolic Disorders, 23*, 55–63.

Carlsson, S., Andersson, T., Lichtenstein, P., Michaëlsson, K., & Ahlbom, A. (2006). Genetic effects on physical activity: Results from the Swedish twin registry. *Medicine and Science in Sports and Exercise, 38*, 1396–1401.

Crespo, C. J., Keteyian, S. J., Heath, G. W., & Sempos, C. T. (1996). Leisure-time physical activity among US adults. results from the Third National Health and Nutrition Examination Survey. *Archives of Internal Medicine, 156*, 93–98.

Duncan, G.E., Goldberg, J., Noonan, C., Moudon, A.V., Hurvitz, P., & Buchwald D. (2008). Unique environmental effects on physical activity participation: A twin study. *PLoS ONE, 3*, e2019.

Kaprio, J., Koskenvuo, M., & Rose, R. J. (1990). Population-based twin registries: Illustrative applications in genetic epidemiology and behavioral genetics from the Finnish twin cohort study. *Acta Geneticae Medicae et Gemellologicae (Roma), 39*, 427–439.

Kaprio, J., Koskenvuo, M., & Sarna, S. (1981). Cigarette smoking, use of alcohol, and leisure-time physical activity among same-sexed adult male twins. *Progress in Clinical and Biological Research, 69*, 37–46.

Kaprio, J., Sarna, S., Koskenvuo, M., & Rantasalo, I. (1978). The Finnish twin registry: Formation and compilation, questionnaire study, zygosity determination procedures, and research program. *Progress in Clinical and Biological Research, 24*, 179–184.

Kujala, U. M., Kaprio, J., & Koskenvuo, M. (2002). Modifiable risk factors as predictors of all-cause mortality: The roles of genetics and childhood environment. *American Journal of Epidemiology, 156*, 985–993.

Kujala, U. M., Kaprio, J., Sarna, S., & Koskenvuo, M. (1998). Relationship of leisure-time physical activity and mortality: The Finnish twin cohort. *Journal of American Medical Association, 279*, 440–444.

Laaksonen, D. E., Niskanen, L., Lakka, H. M., Lakka, T. A., & Uusitupa, M. (2004). Epidemiology and treatment of the metabolic syndrome. *Annals of Medicine, 36*, 332–346.

Lauderdale, D. S., Fabsitz, R., Meyer, J. M., Sholinsky, P., Ramakrishnan, V., & Goldberg, J. (1997). Familial determinants of moderate and intense physical activity: A twin study. *Medicine and Science in Sports and Exercise, 29*, 1062–1068.

Little, R. J. A & Rubin, D .B. (2002). *Statistical analysis with missing data* (2nd ed.). New York: John Wiley.

Maia, J. A. R., Thomis, M., & Beunen, G. (2002). Genetic factors in physical activity levels: A twin study. *American Journal of Preventive Medicine, 23*, 87–91.

Markon, K. E. &. Krueger, R. F. (2004). An empirical comparison of information-theoretic selection criteria for multivariate behavior genetic models. *Behavior Genetics, 34*, 593–610.

Martínez-González, M. A., Varo, J. J., Santos, J. L., De Irala, J., Gibney, M., Kearney, J., & Martínez, J. A. (2001). Prevalence of physical activity during leisure time in the European Union. *Medicine and Science in Sports and Exercise, 33*, 1142–1146.

Morris, J. N., Everitt, M. G., Pollard, R., Chave, S. P., & Semmence, A. M. (1980). Vigorous exercise in leisure-time: Protection against coronary heart disease. *Lancet, 2*, 1207–1210.

Neale, M. C., (1997). *Mx: Statistical modeling* (4th ed.). Richmond, V. A. Department of Psychiatry, Medical College of Virginia, Virginia Commonwealth University.

Paffenbarger, R. S. Jr, Hyde, R. T., Wing, A. L., & Hsieh, C. C. (1986). Physical activity, all-cause mortality, and longevity of college alumni. *The New England Journal of Medicine, 314*, 605–613.

Perusse, L., Tremblay, A., Leblanc, C., & Bouhard, C. (1989). Genetic and environmental influences on level of habitual physical activity and exercise participation. *American Journal of Epidemiology, 129*, 1012–1022.

Physical Activity Guidelines Advisory Committee. (2008). *Physical Activity Guidelines Advisory Committee Report, 2008*. Department of Health and Human Services, Washington, DC, U.S.

Pratt, M., Macera, C. A., & Blanton, C. (1999). Levels of physical activity and inactivity in children and adults in the United States: Current evidence and research issues. *Medicine and Science in Sports and Exercise, 31*, 526–33.

Sallis, J. F. (2000). Age-related decline in physical activity: A synthesis of human and animal studies. *Medicine and Science in Sports and Exercise, 32*, 1598–1600.

Sarna, S., Kaprio, J., Sistonen, P., & Koskenvuo, M. (1978). Diagnosis of twin zygosity by mailed questionnaire. *Human Heredity, 28*, 241–254.

Schwartz, G. (1978). Estimating the dimension of a model. *Annals of Statistics, 6*, 461–464.

Simonen, R., Levälahti, E., Kaprio, J., Videman, T., & Battié, M. C. (2004). Multivariate genetic analysis of lifetime exercise and environmental factors. *Medicine and Science in Sports and Exercise, 36*, 1559–1566.

Spiegelhalter, D. J., Best, N. G., Carlin, B. P., & van der Linde, A. (2002). Bayesian measures of model complexity and fit. *Journal of the Royal Statistical Society: Series B, 64*, 583–616.

Stubbe, J. H., Boomsma, D. I., Vink, J. M., Cornes, B. K., Martin, N. G., Skytthe, A., Kyvik, K. O., Rose, R. J., Kujala, U. M., Kaprio, J., Harris, J. R., Pedersen, N. L., Hunkin, J., Spector, T. D., & de Geus, E. J. (2006). Genetic influences on exercise participation in 37,051 twin pairs from seven countries. *PLoS ONE, 20*, e22.

https://doi.org/10.1375/twin.13.5.475 Published online by Cambridge University Press

Stubbe, J. H., Boomsma, D. I., & de Geus, E. J. C. (2005). Sports participation during adolescence: A shift from environmental to genetic factors. *Medicine and Science in Sports and Exercise, 37*, 563–570.

Stubbe, J. H., & de Geus, E. J. C. (2009). Genetics of exercise behavior. In Yong-Kyu, K. (Ed.), *Handbook of Behavior Genetics* (pp. 343–358). New York: Springer.

Turner, M. J., Kleeberger, S. R., & Lightfoot, J. T. (2005). Influence of genetic background on daily running-wheel activity differs with aging. *Physiological Genomics, 22*, 76–85.

Waller, K., Kaprio, J., & Kujala, U. M. (2008). Associations between long-term physical activity, waist circumference and weight gain: A 30-year longitudinal twin study. *International Journal of Obesity, 32*, 353–361.

https://doi.org/10.1375/twin.13.5.475 Published online by Cambridge University Press