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Genetic analysis of self-reported physical activity and adiposity: The Southwest Ohio Family Study

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Abstract

Objective—Physical inactivity poses a major risk for obesity and chronic disease, and is influenced by both genetic and environmental factors. However, the genetic association between physical activity (PA) level and obesity is not well characterized. Our aims were to: (i) estimate the extent of additive genetic influences on physical activity while adjusting for household effects; and (ii) determine whether physical activity and adiposity measures share common genetic effects.

Subjects—The sample included 521 (42% male) adult relatives, 18–86 years of age, from five large families in the Southwest Ohio Family Study.

Design—Sport, leisure and work PA were self-reported (Baecke Questionnaire of Habitual Physical Activity). Total body and trunk adiposity, including percentage body fat (%BF), were measured using dual-energy X-ray absorptiometry. Abdominal visceral and subcutaneous adipose tissue mass were measured using MRI.

Results—Heritabilities for adiposity and PA traits, and the genetic, household and environmental correlations among them, were estimated using maximum likelihood variance components methods. Significant genetic effects ($P < 0.05$) were found for sport ($h^2 = 0.26$) and leisure PA ($h^2 = 0.17$). Significant ($P < 0.05$) household effects existed for leisure PA ($c^2 = 0.25$). Sport PA had a negative genetic correlation with central adiposity measurements adjusted for height ($\rho_G > |-0.40|$). Sport and leisure PA had negative genetic correlations with %BF ($\rho_G > |-0.46|$).

Conclusions—The results suggest that the association of sport and leisure PA with lower adiposity is due, in part, to a common genetic inheritance of both reduced adiposity and the predisposition to engage in more physical activity.

Keywords

Genetics; Physical activity; Obesity; Visceral adiposity

Low physical activity (PA) is an important and modifiable risk factor for CVD and obesity (1,2). Like other CVD risk factors, there is some evidence that PA is influenced by genetic factors. Heritability estimates for PA from studies of twins range from 30% to 83% (3–7). In

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studies examining nuclear and extended families these estimates are typically lower, ranging from 9% to 57% (8–10).

Despite studies showing genetic effects on PA, associations between PA and obesity have not been examined at the genetic level. In many genetic studies of PA, obesity (typically BMI) is treated as a covariate, or the effect of obesity is regressed out at the phenotypic level. Conversely, many genetic studies of obesity (justifiably) include PA as a covariate (8,9). Interestingly, candidate gene association studies and quantitative trait linkage studies of obesity and PA traits have identified several chromosomal regions in common (7,10–17). For example, quantitative trait loci related to obesity on chromosomes 11q23 and 18q22 (11,13,15,17) have also been implicated in genetic linkage analyses of PA (7,10,12,14,16). Identifying common genetic pathways between PA and obesity would provide insight into how and why PA is related to obesity and diabetes, and may lead to better strategies for weight maintenance and improving cardiovascular health (18–20).

Few studies to date have directly examined the genetic relationship between adiposity and PA. In a study of Finnish twin males (5), percentage body fat (%BF) was associated with PA at the genetic level. However, there were several limitations to that study, chief among them being that the results could not be extended to singletons, who comprise the majority of births. Instead, examining PA levels in adults from large extended families who live in many different households allows for the separate influences of genetic and shared household effects on PA level to be estimated. Using different domains of PA (e.g. sport, leisure and work activity) as they relate to adiposity may also help to resolve conflicting results concerning the inheritance of PA levels and its relationship to obesity. Moreover, including MRI measures of visceral adipose tissue, which may be more closely related to PA levels than total adiposity (21), may help in investigations of obesity and PA. Using an extended pedigree design, the purposes of the present study were to: (i) to examine to what extent genetic and household effects influence PA; and (ii) determine the extent to which PA and adiposity traits share genetic and common household effects.

Materials and methods

The Southwest Ohio Family Study

The Southwest Ohio Family Study (22–24) is a genetic epidemiological study of CVD risk factors, and comprises five large multi-generational families (one predominantly African American and four predominantly European American) from the Dayton, Ohio area. Participants were originally recruited for a National Institutes of Health-funded genetic study of blood pressure and CVD risk in the late 1970s. Initially, each family was ascertained on a single male proband aged 35–58 years with essential hypertension. Family members of these five men were recruited for participation regardless of hypertensive status. Given the large family size and the small number of probands, adjustment for ascertainment was not performed. The current phase of data collection began in 2003 and includes numerous measures of CVD risk, including self-reported PA. Data collected during the current phase of the study were analysed for the present paper. The Southwest Ohio Family Study was approved by the Institutional Review Board of Wright State University and informed consent was obtained from the participants.

Study sample

The study sample consisted of a subset of 521 (219 males, 302 females) adults from the Southwest Ohio Family Study whose visits occurred between January 2003 and November 2006. Participants ranging in age from 18 to 86 years had data for PA, adiposity traits measured through MRI and dual-energy X-ray absorptiometry (DEXA), and covariates such as tobacco

use. Of the 521 participants, 423 were of European descent, while ninety-eight were African American.

Pedigree and household structure

The five families in the study contained between forty-seven and 188 individuals, with a mean family size of 104 and median family size of ninety. In all, there were a total of 18 295 relative pairings of varying degrees of relationship to one another that contributed familial information to the analyses. There were 652 first-degree relative pairs, 741 second-degree relative pairs, 1369 third-degree relative pairs and 2723 fourth-degree relatives. The remaining 12 810 relative pairs were fifth-degree relatives or higher. Among these pair-wise relationships, at least four generations were represented. Current residential address was used to define households for the determination of shared household effects.

Physical activity

PA was assessed using the Baecke Questionnaire of Habitual Physical Activity(25). The Baecke questionnaire is a sixteen-question form that delineates PA into work (work PA), sport (sport PA) and non-sports leisure (leisure PA) indices. Each activity consists of questions scored mostly on a 5-point, sometimes a 3-point, Likert scale with higher scores indicating greater PA levels. Questions related to sitting, standing, walking, lifting, sweating during work hours and occupation are incorporated into the work index, while questions related to sport PA include the type of sport, how often participants engage in it and how much they sweat while they do so. Leisure PA is based on questions regarding mode of transportation to school and work, television watching, walking and cycling. Total activity is the sum of the three indices (25).

The sport PA index incorporates questions related to energy expenditure. The first question (question 9) of the sport PA index is an estimate of energy expenditure (EE) from sport activities (25,26). The estimated EE is then converted into a 5-point Likert score. This value and three additional sport PA-related questions (questions 10–12) are averaged to get the sport PA index, which ranges from 1 to 5(25).

The Baecke questionnaire has been repeatedly validated against accelerometer measures of activity EE(27,28), doubly labelled water measures of total EE(29,30) and other activity questionnaires(31,32). The strongest correlation between EE measured through doubly labelled water and Baecke questionnaire sub-indices was for sport PA ($r = 0.55$)(29). The instrument is also reported to be highly reliable, both over short- and long-term periods, with correlations ranging from 0.86–0.95 for a 1-month period(33) to 0.65–0.84 for an 11-month period(34).

Adiposity measurements and other covariates

Abdominal adipose tissue was assessed using MRI. MR images were obtained using a Magnetom Vision 1.5 Tesla whole-body scanner (Siemens, Mississauga, Canada). Contiguous, 1 cm thick axial images were acquired across the entire abdominal region (T9–S1). Adipose tissue areas within each image were identified and tagged by trained technicians using Slice-O-Matic software version 4.2 (Tomovision Inc., Montreal, Canada). Adipose tissue areas were summed across all images to obtain volumes and then converted into abdominal visceral (VAT) and subcutaneous (SAT) adipose tissue mass (kg) by multiplying the volumes by the density of adipose tissue (0.916). More details regarding the MRI protocols have been given elsewhere (35,36).

Weight (kg) and height (cm) were measured using standard protocols(37). BMI was calculated as weight (kg)/height² (m²). Total body fat mass (BFM, kg) and trunk fat mass (TFM, kg) were

measured using a Hologic 4500E DEXA instrument (Hologic Inc., Waltham, MA, USA). Total %BF was calculated as $\text{BFM}/\text{weight} \times 100$. BFM, TFM, VAT and SAT were also adjusted for height by dividing by height^2 (m^2), to obtain fat mass index (FMI), trunk fat index (TFMI), visceral fat index (VFI) and subcutaneous fat index (SFI), respectively. Height^2 was used so that the indices were on the same scale as BMI(38).

Covariates included age, sex, self-reported race, current smoking status and educational attainment. Current smokers were individuals who were smoking a pipe, cigars or cigarettes at the time of their visit. Males, African Americans, non-smokers and participants with less than a university education were treated as the reference groups.

Statistical analysis

PA and adiposity traits were treated as continuous variables and all dependent variables were normalized by either log or square root transformations where necessary. Phenotypic partial correlations adjusting for age, sex, age^2 , age-by-sex, age^2 -by-sex, race, race-by-sex, smoking and university education(35,39–41) were estimated between measures of PA and adiposity traits using the Statistical Analysis System statistical software package version 9.1 (SAS Institute, Cary, NC, USA).

Quantitative genetic analysis

Quantitative traits are influenced by sources that may be genetic, environmental and/or interactions between the two. Genetic epidemiological methods are used to determine and characterize the effects of (many) genes influencing variation in complex phenotypes. The heritability of a trait is simply the proportion of the phenotypic variation attributable to additive genetic variation and is given by $h^2 = \sigma_G^2 / \sigma_P^2$, where σ_G^2 is the variance due to additive effects of genes and σ_P^2 is the phenotypic variance(42,43). This relationship is derived from the same variance components methods used by Fisher(44) to develop linear regression and analysis of variance methods. Heritabilities are squared correlation coefficients (R^2)(45).

Maximum likelihood variance decomposition methods were used to estimate heritability (h^2) and household effects (c^2) for each study trait using procedures implemented in SOLAR version 4 (Southwest Foundation for Biomedical Research, San Antonio, TX, USA)(46). Heritabilities were estimated for each PA trait (i.e. sport, leisure, work PA) and for the obesity-related traits that represented overall adiposity (weight, BMI, BFM, %BF) and central adiposity (TFM, VAT, SAT). For continuously distributed quantitative traits, the covariance matrix for a pedigree is given by:

$$\mathbf{\Omega} = 2\mathbf{\Phi}\sigma_G^2 + \mathbf{H}\sigma_C^2 + \mathbf{I}\sigma_E^2,$$

where $\mathbf{\Phi}$ is the $n \times n$ matrix of kinship coefficients that structures σ_G^2 ; \mathbf{H} is a matrix that structures σ_C^2 , the variance due to common household effects; and \mathbf{I} is an identity matrix of order n that serves as the structuring matrix for σ_E^2 , the variance due to residual environmental factors (46).

The trait mean and mean effects of age, sex, age^2 , age-by-sex, age^2 -by-sex, race, race-by-sex, smoking and education were also estimated for each trait. Significance of h^2 and c^2 ($P < 0.05$) was determined using the likelihood ratio test(47) by comparing the log-likelihood of the general model, where h^2 and c^2 were estimated, against models where either h^2 or c^2 was fixed to zero. Covariates were selected to determine the most parsimonious models and were tested

for significance ($P < 0.05$) in a similar manner. Only significant covariates were retained in the model and used in the bivariate analyses.

Bivariate extensions(48,49) of the univariate quantitative genetic procedure were used to obtain estimates of the additive genetic (ρ_G) correlation as well as the common household correlation (ρ_C) between the PA traits and obesity traits. The phenotypic covariance between two individuals for two traits is given by a 2×2 covariance matrix whose elements are defined as:

$$\Omega_{ab} = 2\Phi\rho_G\sigma_{Ga}\sigma_{Gb} + \mathbf{H}\rho_C\sigma_{Ca}\sigma_{Cb} + \mathbf{I}\rho_E\sigma_{Ea}\sigma_{Eb},$$

where ρ_G is the additive genetic correlation between traits a and b , ρ_C is the common environmental (household) correlation between the two traits, and ρ_E is the unmeasured environmental correlation between the two traits(48,50). In the bivariate analyses, only significant ($P < 0.05$) covariates in the univariate analysis were included in the model. For the bivariate analysis, if both traits did not have significant household effects, the c^2 terms and ρ_C were not analysed. If one of the two traits had a significant c^2 term, each of the c^2 terms was estimated, but ρ_C was fixed to zero. If both traits had significant household effects, then all parameters were estimated.

Significance of genetic, household and random environmental correlations was evaluated using the likelihood ratio test. Shared genetic or pleiotropic effects (i.e. $|\rho_G| > 0$) and household effects (i.e. $|\rho_C| > 0$) each were tested by comparing the log-likelihood of the general model to nested models where ρ_G and ρ_C were fixed to zero respectively. Models positing completely shared genetic effects (i.e. $|\rho_G| = 1$, or complete pleiotropy) and completely shared household effects (i.e. $|\rho_C| = 1$) were tested in a similar manner. Each was tested by fixing ρ_G and/or ρ_C to (positive or negative) 1 respectively, and comparing the log-likelihoods of the nested model to the general model. Genetic correlations that are statistically different from 0 and 1 (incomplete pleiotropy, $0 < |\rho_G| < 1$) indicate that some of the genetic effects are shared by both traits, but that each trait also has unique genetic effects influencing their phenotypic variation.

Results

Table 1 shows the descriptive statistics for the study sample. The mean age of the participants was 45 (SD 15.3) years. On average the participants were overweight (i.e. $\text{BMI} \geq 25 \text{ kg/m}^2$) with participants having a mean BMI of 28.6 (SD 6.2) kg/m^2 .

Additive genetic and household effects

Table 2 shows the estimated parameters from the univariate analysis from the final model. Sport, leisure and total PA were significantly ($P < 0.05$) influenced by additive genetic effects (h^2), but only leisure PA was significantly influenced by a common household environment (c^2). Work PA was influenced neither by additive genetic effects nor by common household effects. Height was significantly influenced by additive genetic ($h^2 = 0.82$) and household effects ($c^2 = 0.11$). Adiposity traits were significantly heritable ($h^2 = 0.35$ to 0.47), but were not significantly influenced by common household effects.

Combined covariate effects accounted for between 0% and 20.6% of the phenotypic variation in PA. Generally, covariate effects accounted for much more of the variation in the adiposity traits and height (13.3% to 61.6%) than for the variation in PA levels (Table 2). Leisure PA was not significantly influenced by any of the covariates analysed in the present study. Smokers had significantly lower sport PA than non-smokers. Higher education levels were significantly associated with lower work and higher sport PA. Males and younger individuals tended to have significantly higher sport PA than females and older participants, respectively.

Age influenced all adiposity traits, with increased age leading to higher levels of adiposity. Females had lower VAT and VFI (VAT adjusted by height²) than males but had higher levels of adiposity for the rest of the traits examined. Higher education was associated with lower adiposity levels. African Americans had lower VAT levels. For all adiposity variables, there was a significant race-by-sex interaction; thus the effect of race on adiposity traits is not consistent across the sexes. For example, white males had the highest levels of VAT. Among females, there was no race difference. These results have also been observed elsewhere(35, 51).

Phenotypic correlations

Partial phenotypic correlations adjusting for the same final covariates from the univariate analysis indicated that work PA was not associated with any of the adiposity traits (Table 3). Correlations between leisure PA and adiposity were significant ($P<0.05$) but weak, ranging from $r = -0.11$ to -0.16 , while sport PA was moderately and negatively associated with all adiposity traits examined ($r = -0.23$ to -0.32).

Genetic, household and environmental correlations

Table 4 shows the additive genetic correlations, the common household correlations and the random environmental correlations from the bivariate genetic analysis. Because work PA did not have a significant heritable component, it was not included in this analysis. Total PA also was not examined in the bivariate analyses because the different PA components (sport, leisure, work) had significantly different genetic and household effects and the underlying genetic effects from these sub-domains would be obscured by results for total PA.

Significant ($P<0.05$) negative genetic correlations (ρ_G) were found between sport PA and % BF ($\rho_G = -0.46$), VAT ($\rho_G = -0.46$), TFMI ($\rho_G = -0.42$), VFI ($\rho_G = -0.47$) and SFI ($\rho_G = -0.40$). All of these genetic correlations were significantly different from -1 , indicating shared and some unique genetic influences between sport PA and %BF, TFMI, VAT, VFI and SFI. None of the genetic correlations between leisure PA and adiposity traits was significant except for %BF ($\rho_G = -0.66$), which was not statistically different from -1 , indicating completely shared genetic effects. None of the estimated household correlations (ρ_C) or any of the random, unmeasured, environmental correlations (ρ_E) between PA and adiposity traits was significant.

Discussion

While habitual PA is certainly modifiable to some extent, numerous studies including ours demonstrate that it is also significantly heritable. Our results indicate that sport and leisure PA are significantly influenced by genetic components. Our h^2 estimates for PA (0.17–0.26) are lower than those found among twin studies, which range from 0.30 to 0.85(3–5,7), but they are within the range of estimates derived from studies of non-twin relatives, which range from 0.09 to 0.57(8–10). Not only have others also found significant genetic effects using the same index(3) and other ‘habitual’ activity instruments(9), but the similarities in h^2 exist despite the fact that the research protocols of these studies differ from ours(8–10), such as adjusting for BMI(8,9) or examining different age ranges(10).

While confounding of genetic effects by within-family transmission of behavioural norms is an issue in quantitative genetic studies, we specifically modelled and tested for household effects in our study, so that the estimates of heritability have been adjusted for household environment. Furthermore, the extended pedigree design of our study means that the related individuals reside in different households and are exposed to varying environments across multiple generations, so that the independent environmental and genetic sources of variance on PA can be estimated with greater confidence. With regard to household environment we

found significant household effects on leisure PA, indicating that walking to work, engaging in gardening, housework and other kinds of non-sport, and non-work time PA are significantly influenced by one's spouse or other shared household influences. Estimates of the magnitude of c^2 on PA are inconsistent across previous studies. In one study, significant household effects were found for females(6) but not for males(4,6) and in another, household effects were observed for adolescents but not in adults(5). Using the same Baecke questionnaire sport and leisure PA indices, Maia *et al.*(3) found significant c^2 among both male and female twins for sport PA, but only among female twins for leisure PA. Among family studies, significant c^2 have been found for exercise participation, but not habitual exercise(9). Mitchell *et al.*(8) did not find significant c^2 for PA but did find that spousal correlations were stronger than cross-generation correlations, indirectly indicating the presence of household effects. These inconsistencies are not surprising as potential household effects may be heterogeneous and may represent unmeasured factors such as diet, attitudes towards PA, family composition, neighbourhood characteristics and cultural background.

To date, most genetic studies have treated measures of obesity as covariates when investigating PA, or have treated PA as a covariate when studying obesity measures. We found only a few studies that directly attempted to identify genes with pleiotropic effects on both obesity and PA(5,10,52). In a study based on Finnish twins, shared additive genetic effects between lifetime exercise and %BF(5) were estimated at -1 , indicating complete negative pleiotropy, where the gene(s) that increase activity levels also decrease %BF levels. In that study, the genetic correlation between BMI and exercise was not reported because their statistical genetic models did not attain a reasonable model fit. These findings are similar to our results. We found significant additive genetic effects between sport and leisure PA and %BF, but not with BMI. One study reported a significant genetic correlation between PA and BMI(52). In that study of adolescent siblings the genetic correlation between BMI Z score and physical inactivity (i.e. television watching) was 0.10(52). In our analyses, we found negative genetic correlations between measures of abdominal adiposity (VAT, TFMI, VFI, SFI) and sport PA. Thus, our data suggest that the established association between PA and lower total and abdominal adiposity is due, in part, to a jointly inherited genetic mechanism or pathway, although this mechanism has yet to be identified. This has important public health implications in that, at the population level, obese individuals in need of increased PA may carry a genetic disposition for lower PA and therefore modifying therapeutic methods and targeting public health awareness to this group is essential.

We observed that sport and leisure PA had slightly different relationships with adiposity, which may relate to differences in the relationship of sport and leisure PA to total EE. In validity studies, sport PA and EE are moderately correlated ($r = 0.55$) while leisure PA is more weakly correlated ($r = 0.21$) with EE(28). Alternatively, greater sport PA may also reflect greater opportunities to exercise, lower limitations related to higher socio-economic status and resources, lower physical disability, and/or better recall associated with vigorous activity such as sport PA(53,54).

The significant genetic correlation between sport PA and measures of central adiposity, but not with more general measures of fat such as BMI, may reflect tissue differences in metabolic response to exercise. In exercise trials, weight (BMI) loss may not always be attained but VAT and SAT loss does occur(21,55). Reductions in total abdominal fat (VAT+SAT) are greater than fat loss in lower limbs(21). As abdominal tissue is more metabolically active than limb adipose tissue(21,56), the shared genetic influence between abdominal fat and PA may point to genetic pathways linking PA to tissue-specific glucose or fat metabolism. Moreover, our findings of stronger genetic correlations using central adiposity adjusted for size (i.e. height²) indicates that body size adjustment is necessary in order to reveal the underlying relationship between sport PA and central adiposity, as taller individuals tend to be absolutely

larger in all aspects but may have relatively less fat in critical visceral regions than shorter individuals (i.e. they are able to distribute more of their adipose tissue in the vertical plane).

Our study has a number of strengths including the use of an extended family study design, the incorporation of common household effects, and the use of criterion measures of central adiposity. To our knowledge, the present study is the first to directly estimate common genetic effects between PA and adiposity using DEXA and MRI to quantify adiposity. In our analyses, we did not perform the regression of PA on BMI or use BMI as a covariate as others have (8, 9). When performing regression on adiposity levels at the phenotypic level, information pertaining to the common genetic pathways between PA and adipose tissue, as suggested by our findings, may be lost. Studies that do not take account of the heritability of PA and its common genetic influences with adiposity may actually underestimate the role of PA in chronic disease outcomes relative to that of obesity, because their joint contributions are not fully addressed.

One of the limitations of the present study is the use of self-reported PA. While the more intense PA level captured by the sport PA index accurately reflects sport activity records and aerobic fitness (32), it does not measure EE directly but rather self-reported participation in sport PA activity. Thus it is possible that our h^2 and c^2 estimates on self-reported PA might be lower or have greater standard errors than if more direct measurements were used. Further, other factors related to PA such as musculoskeletal health, physical disability, socio-economic status and neighbourhood factors, cardiopulmonary fitness level, or other behavioural, physiological or psychological traits influencing PA (57) are likely to be reflected in this measure. None the less, significant correlations (genetic and phenotypic) were observed between PA and body composition, while controlling for shared household effects and individual effects of education and smoking, which suggests that this measure is explanatory for familial risk of combined low activity and elevated adiposity. Another limitation to the study may be our measurement of household effects. Current household residence was used to identify common households and thus may not capture long-term cumulative household effects, particularly those that would take place during an individual's formative years.

In summary, our study indicates that sport and leisure PA are under additive genetic effects and that leisure PA is additionally influenced by household effects. Our novel finding is that the likelihood of engaging in sport and leisure PA is, at least in part, inherited jointly with adiposity level. As increasing PA at the individual and population levels is integral to the prevention and treatment of obesity and CVD risk, it is important to attempt to elucidate the genetic as well as the behavioural and environmental factors influencing it. Current and future work involves identification of specific chromosomal regions and genetic variants acting in the physiological pathway between PA and reductions in abdominal adipose tissue, weight loss or weight maintenance.

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References

1. Sesso HD, Paffenbarger RS Jr, Lee IM. Physical activity and coronary heart disease in men: the Harvard Alumni Health Study. *Circulation* 2000;102:975–980. [PubMed: 10961960]

2. Lee IM, Paffenbarger RS Jr. Physical activity and stroke incidence: the Harvard Alumni Health Study. *Stroke* 1998;29:2049–2054. [PubMed: 9756580]
3. Maia JA, Thomis M, Beunen G. Genetic factors in physical activity levels: a twin study. *Am J Prev Med* 2002;23:87–91. [PubMed: 12133742]
4. Lauderdale DS, Fabsitz R, Meyer JM, Sholinsky P, Ramakrishnan V, Goldberg J. Familial determinants of moderate and intense physical activity: a twin study. *Med Sci Sports Exerc* 1997;29:1062–1068. [PubMed: 9268964]
5. Simonen R, Levälähti E, Kaprio J, Videman T, Battié MC. Multivariate genetic analysis of lifetime exercise and environmental factors. *Med Sci Sports Exerc* 2004;36:1559–1566. [PubMed: 15354038]
6. Beunen G, Thomis M. Genetic determinants of sports participation and daily physical activity. *Int J Obes Relat Metab Disord* 1999;23(Suppl 3):S55–S63. [PubMed: 10368004]
7. Wolfarth B, Bray MS, Hagberg JM, Pérusse L, Rauramaa R, Rivera MA, Roth SM, Rankinen T, Bouchard C. The human gene map for performance and health-related fitness phenotypes: the 2004 update. *Med Sci Sports Exerc* 2005;37:881–903. [PubMed: 15947712]
8. Mitchell BD, Rainwater DL, Hsueh WC, Kennedy AJ, Stern MP, Maccluer JW. Familial aggregation of nutrient intake and physical activity: results from the San Antonio Family Heart Study. *Ann Epidemiol* 2003;13:128–135. [PubMed: 12559672]
9. Pérusse L, Tremblay A, Leblanc C, Bouchard C. Genetic and environmental influences on level of habitual physical activity and exercise participation. *Am J Epidemiol* 1989;129:1012–1022. [PubMed: 2705422]
10. Cai G, Cole SA, Butte N, Bacino C, Diego V, Tan K, Goring HH, O’Rahilly S, Farooqi IS, Comuzzie AG. A quantitative trait locus on chromosome 18q for physical activity and dietary intake in Hispanic children. *Obesity* 2006;14:1596–1604. [PubMed: 17030971]
11. Chagnon YC, Chen WJ, Pérusse L, Chagnon M, Nadeau A, Wilkison WO, Bouchard C. Linkage and association studies between the melanocortin receptors 4 and 5 genes and obesity-related phenotypes in the Quebec Family Study. *Mol Med* 1997;3:663–673. [PubMed: 9392003]
12. Loos RJ, Rankinen T, Tremblay A, Pérusse L, Chagnon Y, Bouchard C. Melanocortin-4 receptor gene and physical activity in the Quebec Family Study. *Int J Obes (Lond)* 2005;29:420–428. [PubMed: 15597110]
13. Comings DE, Flanagan SD, Dietz G, Muhleman D, Knell E, Gysin R. The dopamine D2 receptor (DRD2) as a major gene in obesity and height. *Biochem Med Metab Biol* 1993;50:176–185. [PubMed: 8260195]
14. Simonen RL, Rankinen T, Pérusse L, Leon AS, Skinner JS, Wilmore JH, Rao DC, Bouchard C. A dopamine D2 receptor gene polymorphism and physical activity in two family studies. *Physiol Behav* 2003;78:751–757. [PubMed: 12782232]
15. Norman RA, Thompson DB, Foroud T, Garvey WT, Bennett PH, Bogardus C, Ravussin E. Genomewide search for genes influencing percent body fat in Pima Indians: suggestive linkage at chromosome 11q21–q22. Pima Diabetes Gene Group. *Am J Hum Genet* 1997;60:166–173. [PubMed: 8981960]
16. Rankinen T, Bray MS, Hagberg JM, Pérusse L, Roth SM, Wolfarth B, Bouchard C. The human gene map for performance and health-related fitness phenotypes: the 2005 update. *Med Sci Sports Exerc* 2006;38:1863–1888. [PubMed: 17095919]
17. Rankinen T, Zuberi A, Chagnon YC, Weisnagel SJ, Argyropoulos G, Walts B, Pérusse L, Bouchard C. The human obesity gene map: the 2005 update. *Obesity (Silver Spring)* 2006;14:529–644. [PubMed: 16741264]
18. Bassuk SS, Manson JE. Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *J Appl Physiol* 2005;99:1193–1204. [PubMed: 16103522]
19. Haskell WL, Lee IM, Pate PR, Powell KE, Blair SN, Franklin BA, Macera CA, Heath GW, Thompson PD, Bauman A. Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation* 2007;116:1081–1093. [PubMed: 17671237]
20. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of US adults. *Am J Epidemiol* 1997;146:214–222. [PubMed: 9247005]

21. Lee S, Kuk JL, Davidson LE, Hudson R, Kilpatrick K, Graham TE, Ross R. Exercise without weight loss is an effective strategy for obesity reduction in obese individuals with and without type 2 diabetes. *J Appl Physiol* 2005;99:1220–1225. [PubMed: 15860689]
22. Siervogel RM, Frey MA, Kezdi P, Roche AF, Stanley EL. Blood pressure, electrolytes, and body size: their relationships in young relatives of men with essential hypertension. *Hypertension* 1980;2:83–92. [PubMed: 7399649]
23. Choh AC, Czerwinski SA, Lee M, Demerath EW, Wilson AF, Towne B, Siervogel RM. Quantitative genetic analysis of blood pressure response during the cold pressor test. *Am J Hypertens* 2005;18:1211–1217. [PubMed: 16182112]
24. Choh AC, Czerwinski SA, Lee M, Demerath EW, Cole SA, Wilson AF, Towne B, Siervogel RM. Quantitative genetic analysis of blood pressure reactivity to orthostatic tilt using principal components analysis. *J Hum Hypertens* 2006;20:281–289. [PubMed: 16437129]
25. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr* 1982;36:936–942. [PubMed: 7137077]
26. Durnin, JVGA.; Passmore, R. *Energy, Work and Leisure*. London: Heinemann; 1967.
27. Miller DJ, Freedson PS, Kline GM. Comparison of activity levels using the Caltrac accelerometer and five questionnaires. *Med Sci Sports Exerc* 1994;26:376–382. [PubMed: 8183104]
28. Philippaerts RM, Lefevre J, Delvaux K, Thomis M, Vanreusel B, Vanden Eynde B, Claessens AL, Lysens R, Beunen G. Associations between daily physical activity and physical fitness in Flemish males: a cross-sectional analysis. *Am J Hum Biol* 1999;11:587–597. [PubMed: 11533977]
29. Bonnefoy M, Normand S, Pachaiaudi C, Lacour JR, Laville M, Kostka T. Simultaneous validation of ten physical activity questionnaires in older men: a doubly labeled water study. *J Am Geriatr Soc* 2001;49:28–35. [PubMed: 11207839]
30. Philippaerts RM, Westerterp KR, Lefevre J. Doubly labelled water validation of three physical activity questionnaires. *Int J Sports Med* 1999;20:284–289. [PubMed: 10452224]
31. Pereira MA, FitzerGerald SJ, Gregg EW, Joswiak ML, Ryan WJ, Suminski RR, Utter AC, Zmuda JM. A collection of physical activity questionnaires for health-related research. *Med Sci Sports Exerc* 1997;29:S1–S205. [PubMed: 9243481]
32. Richardson MT, Ainsworth BE, Wu HC, Jacobs DR Jr, Leon AS. Ability of the Atherosclerosis Risk in Communities (ARIC)/Baecke Questionnaire to assess leisure-time physical activity. *Int J Epidemiol* 1995;24:685–693. [PubMed: 8550264]
33. Philippaerts RM, Lefevre J. Reliability and validity of three physical activity questionnaires in Flemish males. *Am J Epidemiol* 1998;147:982–990. [PubMed: 9596477]
34. Pols MA, Peeters PH, Bueno-De-Mesquita HB, Ocke MC, Wentink CA, Kemper HC, Collette HJ. Validity and repeatability of a modified Baecke questionnaire on physical activity. *Int J Epidemiol* 1995;24:381–388. [PubMed: 7635600]
35. Demerath EW, Shen W, Lee M, Choh AC, Czerwinski SA, Siervogel RM, Towne B. Approximation of total visceral adipose tissue with a single magnetic resonance image. *Am J Clin Nutr* 2007;85:362–368. [PubMed: 17284730]
36. Demerath EW, Ritter KJ, Couch WA, et al. Validity of a new automated software program for visceral adipose tissue estimation. *Int J Obes (Lond)* 2007;31:285–291. [PubMed: 16770332]
37. Lohman, TG.; Roche, AF.; Martorell, R. *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books; 1988.
38. VanItallie TB, Yang MU, Heymsfield SB, Funk RC, Boileau RA. Height-normalized indices of the body's fat-free mass and fat mass: potentially useful indicators of nutritional status. *Am J Clin Nutr* 1990;52:953–959. [PubMed: 2239792]
39. Elia M. Obesity in the elderly. *Obes Res* 2001;9(Suppl 4):244S–248S. [PubMed: 11707549]
40. Villareal DT, Apovian CM, Kushner RF, Klein S. Obesity in older adults: technical review and position statement of the American Society for Nutrition and NAASO, The Obesity Society. *Obes Res* 2005;13:1849–1863. [PubMed: 16339115]
41. Flegal KM, Troiano RP. Changes in the distribution of body mass index of adults and children in the US population. *Int J Obes Relat Metab Disord* 2000;24:807–818. [PubMed: 10918526]
42. Falconer, DS.; Mackay, TFC. *Introduction to Quantitative Genetics*. 4. Harlow: Longman; 1996.

43. Hartl, DL.; Clark, AG. Principles of Population Genetics. 3. Sunderland, MA: Sinauer Associates, Inc; 1997.
44. Fisher RA. The correlation between relatives on the supposition of Mendelian inheritance. *Trans R Soc Edinb* 1918;52:399–433.
45. Lynch, M.; Walsh, B. Genetics and Analysis of Quantitative Traits. Sunderland, MA: Sinauer Associates, Inc; 1998.
46. Almasy L, Blangero J. Multipoint quantitative trait linkage analysis in general pedigrees. *Am J Hum Genet* 1998;62:1198–1211. [PubMed: 9545414]
47. Edwards, AWF. Likelihood. Baltimore, MD: Johns Hopkins University Press; 1992. expanded ed
48. Almasy L, Dyer TD, Blangero J. Bivariate quantitative trait linkage analysis: pleiotropy versus coincident linkages. *Genet Epidemiol* 1997;14:953–958. [PubMed: 9433606]
49. Williams JT, Van Eerdewegh P, Almasy L, Blangero J. Joint multipoint linkage analysis of multivariate qualitative and quantitative traits. I. Likelihood formulation and simulation results. *Am J Hum Genet* 1999;65:1134–1147. [PubMed: 10486333]
50. Hopper JL, Mathews JD. Extensions to multivariate normal models for pedigree analysis. *Ann Hum Genet* 1982;46:373–383. [PubMed: 6961886]
51. Hoffman DJ, Wang Z, Gallagher D, Heymsfield SB. Comparison of visceral adipose tissue mass in adult African Americans and whites. *Obes Res* 2005;13:66–74. [PubMed: 15761164]
52. Nelson MC, Gordon-Larsen P, North KE, Adair LS. Body mass index gain, fast food, and physical activity: effects of shared environments over time. *Obesity* 2006;14:701–709. [PubMed: 16741273]
53. Sallis JF. Age-related decline in physical activity: a synthesis of human and animal studies. *Med Sci Sports Exerc* 2000;32:1598–1600. [PubMed: 10994911]
54. Jacobs DR Jr, Ainsworth BE, Hartman TJ, Leon AS. A simultaneous evaluation of 10 commonly used physical activity questionnaires. *Med Sci Sports Exerc* 1993;25:81–91. [PubMed: 8423759]
55. Ross R, Janssen I, Dawson J, Kungl AM, Kuk JL, Wong SL, Nguyen-Duy TB, Lee S, Kilpatrick K, Hudson R. Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial. *Obes Res* 2004;12:789–798. [PubMed: 15166299]
56. Wong S, Janssen I, Ross R. Abdominal adipose tissue distribution and metabolic risk. *Sports Med* 2003;33:709–726. [PubMed: 12895129]
57. Davis C, Kaptein S. Anorexia nervosa with excessive exercise: a phenotype with close links to obsessive-compulsive disorder. *Psychiatry Res* 2006;142:209–217. [PubMed: 16697469]

Descriptive statistics of the study sample: subset of adults from the Southwest Ohio Family Study whose visits occurred between January 2003 and November 2006

Table 1

Variable	n	Mean or %	SD or n	Minimum	Maximum
Age (years)	521	45.23	15.34	18.03	85.50
Sex (% female) [†]	521	58.0	302	-	-
Race (% white) [†]	521	81.2	423	-	-
Smoking (% current smoker) [†]	521	31.9	166	-	-
Education (% university) [†]	521	21.5	112	-	-
Sport PA	519	2.31	0.75	1	4.75
Leisure PA	518	2.52	0.66	1	4.25
Work PA	517	2.82	0.66	1.38	4.62
Total PA	512	7.66	1.34	3.63	11.50
Weight (kg)	521	82.24	18.94	44.90	158.40
Height (cm)	521	169.53	10.08	143.70	206.60
BMI (kg/m ²)	521	28.61	6.22	17.17	57.28
BFM (kg)	490	24.91	9.81	4.34	56.51
%BF	490	30.64	9.34	7.62	51.72
TFM (kg)	490	12.27	5.72	1.63	29.37
VAT (kg)	521	3.00	2.20	0.16	11.91
SAT (kg)	521	6.23	4.22	0.52	29.19
BFMI (kg/m ²)	490	8.89	3.93	1.46	22.85
TFMI (kg/m ²)	490	4.37	2.20	0.55	11.68
VFI (kg/m ²)	521	1.03	0.72	0.06	4.06
SFI (kg/m ²)	521	2.22	1.56	0.18	10.44

PA, physical activity; BFM, total body fat mass; %BF, percentage body fat; TFM, trunk fat mass; VAT, visceral adipose tissue; SAT, subcutaneous abdominal adipose tissue; BFMI, fatmass index; TFMI, trunk mass index; VFI, visceral fat index; SFI, subcutaneous fat index. Data are presented as means with their standard deviation for continuous variables or as percentages and number of individuals for categorical variables (indicated by [†]).

Table 2
Final univariate estimates of heritability, trait means and covariate effects for physical activity and body composition traits, and their standard errors: subset of adults from the Southwest Ohio Family Study whose visits occurred between January 2003 and November 2006

	<i>n</i>	<i>h</i> ²	SE	<i>c</i> ²	SE	<i>μ</i>	SE	<i>σ</i> _P	SE	<i>β</i> _{age²}	SE	<i>β</i> _{sex}	SE	<i>β</i> _{age² × sex}	SE	<i>β</i> _{race}	SE	<i>β</i> _{race × sex}	SE	<i>β</i> _{education}	SE	<i>β</i> _{smoking}	SE	% of variation due to covariate effects	
Leisure PA	518	0.17 ^{**}	0.09	0.25 ^{**}	0.08	2.51 ^{***}	0.04	0.66 ^{***}	0.02	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.0	
Sport PA	519	0.26 ^{**}	0.10	0.13	0.09	2.56 ^{***}	0.06	0.66 ^{***}	0.02	-7.75 ^{***}	1.94	-0.54 ^{***}	0.06	-	-	-	-	-	-	-	0.28 ^{***}	0.07	-0.16 [*]	0.07	20.6
Work PA	517	0.13	0.09	0.13	0.09	2.93 ^{***}	0.04	0.64 ^{***}	0.02	-	-	-	-	-	-	-	-	-	-	-	-0.31 ^{***}	0.06	-	5.2	
Total PA	512	0.29 ^{**}	0.11	0.13	0.09	7.93 ^{***}	0.10	1.30 ^{***}	0.04	-12.16 [*]	3.70	-0.50 ^{**}	0.11	-	-	-	-	-	-	-	-	-	-	5.8	
Weight [‡] (kg)	521	0.47 ^{***}	0.10	0.07	0.09	4.50 ^{***}	0.02	0.20 ^{***}	0.01	2.62 ^{**}	0.56	-	-	-	-	-	-	-	-	-	-	-	-	19.4	
Height (cm)	521	0.82 ^{***}	0.07	0.11 [*]	0.06	178.28 ^{***}	0.60	6.35 ^{***}	0.22	-	-	-14.17 ^{***}	0.49	-	-	-	-	-	-	-	1.39 [*]	0.56	-	-	60.8
BMI [‡] (kg/m ²)	521	0.35 ^{**}	0.11	0.13	0.09	3.36 ^{***}	0.02	0.19 ^{***}	0.01	3.45 ^{***}	0.54	0.14 ^{**}	0.03	-	-	-	-	-	-	-	-0.15 ^{***}	0.03	-0.05 ^{**}	0.02	13.3
BFM (kg)	490	0.35 ^{***}	0.10	0.09	0.08	22.33 ^{***}	0.84	8.46 ^{***}	0.28	194.17 ^{***}	24.47	11.53 ^{***}	1.42	-	-	-	-	-	-	-	-	-	-	25.8	
%BF [§]	490	0.38 ^{***}	0.10	0.08	0.08	4.83 ^{***}	0.05	0.54 ^{***}	0.02	15.78 ^{***}	1.59	1.45 ^{***}	0.09	-	-	-	-	-	-	-	-	-	-	61.6	
TFM (kg)	490	0.35 ^{***}	0.10	0.12	0.08	12.11 ^{***}	0.50	5.06 ^{***}	0.17	141.70 ^{***}	14.66	4.35 ^{***}	0.85	-	-	-	-	-	-	-	-	-	-	21.3	
VAT [‡] (kg)	521	0.36 ^{***}	0.10	0.13	0.09	1.12 ^{***}	0.12	0.66 ^{***}	0.02	27.09 ^{***}	1.87	-0.47 ^{**}	0.14	-	-	-	-	-	-	-	0.49 ^{**}	0.20	0.41 ^{**}	0.13	41.6
SAT [‡] (kg)	521	0.44 ^{***}	0.11	0.08	0.09	1.58 ^{***}	0.06	0.59 ^{***}	0.02	10.50 ^{***}	1.65	0.56 ^{***}	0.09	-	-	-	-	-	-	-	-	-	-	17.6	
BFMI [‡] (kg/m ²)	490	0.37 ^{***}	0.10	0.09	0.09	1.90 ^{***}	0.04	0.36 ^{***}	0.01	9.58 ^{***}	1.05	0.62 ^{***}	0.06	-	-	-	-	-	-	-	-	-	-	41.3	
TFMI [‡] (kg/m ²)	490	0.41 ^{***}	0.10	0.12	0.08	1.22 ^{***}	0.05	0.46 ^{***}	0.02	15.37 ^{***}	1.32	0.47 ^{***}	0.08	-	-	-	-	-	-	-	-	-	-	31.3	
VFI [‡] (kg/m ²)	521	0.36 ^{***}	0.10	0.13	0.08	-0.02 ^{***}	0.13	0.66 ^{***}	0.02	28.00 ^{***}	1.87	-0.30 [*]	0.14	-	-	-	-	-	-	-	0.51 ^{**}	0.20	0.39 ^{**}	0.13	38.2
SFI [‡] (kg/m ²)	521	0.44 ^{***}	0.11	0.10	0.09	0.42 ^{***}	0.06	0.59 ^{***}	0.02	11.44 ^{***}	1.65	0.74 ^{***}	0.09	-	-	-	-	-	-	-	-	-	-	25.1	

*h*², additive genetic effects; *c*², household effects; *μ*, mean effects; *σ*_P, standard deviation; *β*_{age}, age effect (positive sex effects correspond to females); *β*_{age²}, age-squared effect; *β*_{age² × sex}, age-by-sex effect; *β*_{age² × sex}, age-squared-by-sex effect; *β*_{sex}, sex effect (positive race effects correspond to whites); *β*_{education}, education effect (positive education effects correspond to those with university education); *β*_{smoking}, smoking effect (positive smoking effects correspond to smokers); PA, physical activity; BFM, total body fat mass; %BF, percentage body fat; TFM, trunk fat mass; VAT, visceral adipose tissue; SAT, subcutaneous abdominal adipose tissue; BFMI, fat mass index; TFMI, trunk mass index; VFI, visceral fat index; SFI, subcutaneous fat index.

Only significant parameters are shown for covariates:

* *P*<0.05,

** *P*<0.01,

*** *P*<0.0001.

[‡] Value ×10⁻³.

[‡] Variables were log-transformed.

[§] Variables were square-root-transformed.

Table 3

Phenotypic correlations among physical activity scores and body composition traits adjusted for all covariates: subset of adults from the Southwest Ohio Family Study whose visits occurred between January 2003 and November 2006

	Sport PA		Leisure PA		Work PA	
	r	n	r	n	r	n
Weight [‡] (kg)	-0.17**	519	-0.08	518	-0.008	517
Height (cm)	0.16**	519	0.05	518	-0.06	517
BMI [‡] (kg/m ²)	-0.23***	519	-0.11*	518	0.01	517
BFM (kg)	-0.25***	488	-0.14**	489	0.0003	487
%BF [‡]	-0.32***	488	-0.15**	489	-0.009	487
TFM (kg)	-0.26***	488	-0.13**	489	0.008	487
VAT [‡] (kg)	-0.25***	519	-0.16**	518	-0.006	517
SAT [‡] (kg)	-0.26***	519	-0.13**	518	0.009	517
BFMI [‡] (kg/m ²)	-0.27***	488	-0.13**	489	-0.004	487
TFMI [‡] (kg/m ²)	-0.26***	488	-0.13**	489	-0.001	487
VFI [‡] (kg/m ²)	-0.26***	519	-0.16**	518	-0.0001	517
SFI [‡] (kg/m ²)	-0.28***	519	-0.13**	518	-0.02	517

PA, physical activity; r, phenotypic correlation; BFM, total body fat mass; %BF, percentage body fat; TFM, trunk fat mass; VAT, visceral adipose tissue; SAT, subcutaneous abdominal adipose tissue; BFMI, fat mass index; TFMI, trunk mass index; VFI, visceral fat index; SFI, subcutaneous fat index. Significant correlations:

* $P < 0.05$,

** $P < 0.01$,

*** $P < 0.0001$.

[‡] Variables were log-transformed.

[‡] Variables were square-root-transformed.

Table 4

Bivariate analyses of physical activity and body composition traits: subset of adults from the Southwest Ohio Family Study whose visits occurred between January 2003 and November 2006

	Sport PA				Leisure PA						
	ρ_G	SE	ρ_C	SE	ρ_E	SE	ρ_C	SE	ρ_E	SE	
Weight [†] (kg)	-0.17	0.21	§		-0.10	0.11	-0.11	0.24	[0]//	-0.06	0.13
Height (cm)	0.14	0.17	[0]//		0.07	0.27	0.27	0.20	-0.48	0.40	0.35
BMI [†] (kg/m ²)	-0.29	0.22	§		-0.10	0.10	-0.23	0.25	[0]//	-0.02	0.13
BFM (kg)	-0.28	0.23	§		-0.16	0.10	-0.40	0.29	[0]//	-0.01	0.11
%BF [‡]	-0.46*	0.20	§		-0.18	0.10	-0.66* [¶]	0.32	[0]//	0.02	0.11
TFM (kg)	-0.36	0.22	§		-0.13	0.10	-0.38	0.28	[0]//	-0.04	0.12
VAT [†] (kg)	-0.46*	0.20	§		-0.04	0.10	-0.23	0.25	[0]//	-0.16	0.11
SAT [†] (kg)	-0.38	0.19	§		-0.12	0.11	-0.33	0.23	[0]//	-0.04	0.12
BFMI [†] (kg/m ²)	-0.39	0.22	§		-0.12	0.10	-0.49	0.29	[0]//	0.01	0.11
TFMI [†] (kg/m ²)	-0.42*	0.20	§		-0.09	0.10	-0.38	0.27	[0]//	-0.02	0.12
VFI [†] (kg/m ²)	-0.47*	0.20	§		-0.04	0.10	-0.26	0.25	[0]//	-0.14	0.12
SFI [†] (kg/m ²)	-0.40*	0.19	§		-0.12	0.11	-0.41	0.24	[0]//	-0.03	0.12

PA, physical activity; ρ_G , genetic correlation; ρ_C , household correlation; ρ_E , random environmental residual correlation; BFM, total body fat mass; %BF, percentage body fat; TFM, trunk fat mass; VAT, visceral adipose tissue; SAT, subcutaneous abdominal adipose tissue; BFMI, fat mass index; TFMI, trunk mass index; VFI, visceral fat index; SFI, subcutaneous fat index.

* Significantly different from 0, i.e. shared genetic, household or environmental effects ($P < 0.05$).

[†] Variables were log-transformed.

[‡] Variables were square-root-transformed.

§ No household terms were modelled because neither trait had a significant c^2 (i.e. both c^2 terms and ρ_C were fixed to zero).

// Fixed at zero because one trait did not have significant household effects (c^2).

[¶] Significantly different from 0 and not significantly different from 1 ($P < 0.05$).