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Genetic determinants of sports participation and daily physical activity

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OBJECTIVE: The purpose is to review the existing literature on genetic determinants of sports participation, daily physical activity (PA) resting metabolic rate (RMR) and activity as a temperamental trait.
DESIGN: A synthesis will be given of the published material on this topic with special focus on twin and family data, and association and linkage studies.
MEASUREMENTS: Self reported sports participation, daily PA, RMR and activity as a temperamental trait.
ANALYSIS: Transmission and heritability coefficients calculated from twin and family data will be reported.
RESULTS: The reported heritability coefficients for sports participation vary between 0.35 – 0.83, and those for daily PA between 0.29 – 0.62. If one of the parents or co-twins is active in sports, it is more likely that the child or co-twin is also active in sports (odds ratios (ORs) vary from 1.2 – 5.8). Twin and parent-child correlations for RMR also indicate a moderate genetic effect. At present, only a linkage between RMR and uncoupling protein 2 markers has been demonstrated.
CONCLUSION: The genetic determination of sports participation, daily PA and RMR, varies from low to moderately high, and only between the uncoupling protein 2 genetic marker and RMR has a linkage has been demonstrated.

Keywords: physical activity; resting metabolic rate; family and twin studies; heritability; genetic variation; environmental variation; linkage; sports participation; temperament

Introduction

Since at one side of the energy balance equation, energy expenditure (EE) is the parameter of interest, it seems indicated to study the genetic determinants of this parameter. At the extremes of the EE distribution, athletes are found who compete in ultra long distance runs and super triathlons or cyclists who participate in the Tour de France. During their training process and competition, they expend enormous amounts of energy (see for example Westerterp *et al*[1]). A legitimate question then arises, as to whether the inter-individual variation in EE is partly explained by genetic factors. EE is a complex phenotype, comprising of several factors: basal (BMR) and resting metabolic rate (RMR), thermic effect of food and EE in a variety of activities such as sports activities. Children have added energy costs associated with normal growth and maturation.

The purpose of this contribution, is to review the available literature on twin and family studies on genetic determinants of sports participation, daily physical activity (PA), RMR and activity as a temperamental trait. Furthermore, the limited studies investigating association and linkage between genetic markers and the activity phenotypes will also be considered.

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Before reviewing the existing data, the analytic strategies used to decipher genetic determinants will be briefly discussed.

Methodological issues to study human variation in daily PA

Participation in daily physical activities is mostly assessed by measurements on a continuous scale. The distribution of daily PA in a population shows a Gaussian or skewed distribution, which is typical for quantitative, multifactorial phenotypes that are influenced by both multiple genes (polygenic) and environmental factors. The search for the genetic basis of daily PA in humans can be studied by two basic approaches: the unmeasured genotype (or top-down) and the measured genotype (or bottom up approach).[2,3]

When the genotype is not available, inferences of the genetic influences are made from the phenotype, which is mostly based on the statistical analysis of the distributions in PA measures in related individuals and families. The studied populations are twins (monozygotic (MZ) and dizygotic (DZ)), families or twin/sibling adoption studies, and the selection of the samples determines which contributing factors can be estimated. Parameters that are estimated in these epidemiological studies are familial aggregation and heritability, and if more sophisticated statistical analyses are made, contributions of environmental

factors, measurement of specific twin environment, assortative mating, etc. can be obtained. Estimating relative risk or determination of the lambda coefficient can estimate the level of familial aggregation. This coefficient expresses the increased risk for a relative of a very active (or non-active) person to be very active (or non-active) himself, compared to the overall prevalence of being active (or non-active) in the population. This measure of relative risk is used for diseases or in case of daily PA, for a high *vs* low categorization. The presence of familial aggregation in a quantitative measurement of PA can be tested using a one-way analysis of variance, in which the variance within families is compared to the variance between families. Familial resemblance is evident when a significantly higher between-family than within-family variance (significant F-ratio) is found. Intra-class correlations between family members (father–daughter, father–son, mother–daughter, mother–son, sister–sister, brother–brother, sister–brother, midparent–offspring, spouses) provide insights about the relative importance of genetic and environmental factors in the daily PA phenotype. This is further shown in Figure 1. Taking into account several assumptions, Figure 1 is indicative of the relation between the degree of genetic relatedness and the observed similarities in these groups. When the observed similarity in a daily PA phenotype, expressed as an intra-pair correlation, is 1.0 in pairs sharing all their genetic material (MZ twins), 0.5 in pairs sharing on average 50% of their genes. (DZ twins, parent–offspring, sibs), 0.25 in pairs sharing 25% of their genes (grandparent–grandchildren), and no correlation in random pairs, a line of identity is observed when drawing a line between these points (Figure 1A). In this case, all of the observed variance would be caused by additive genetic factors (variance due to the action of several genes, with co-dominant allelic effects).

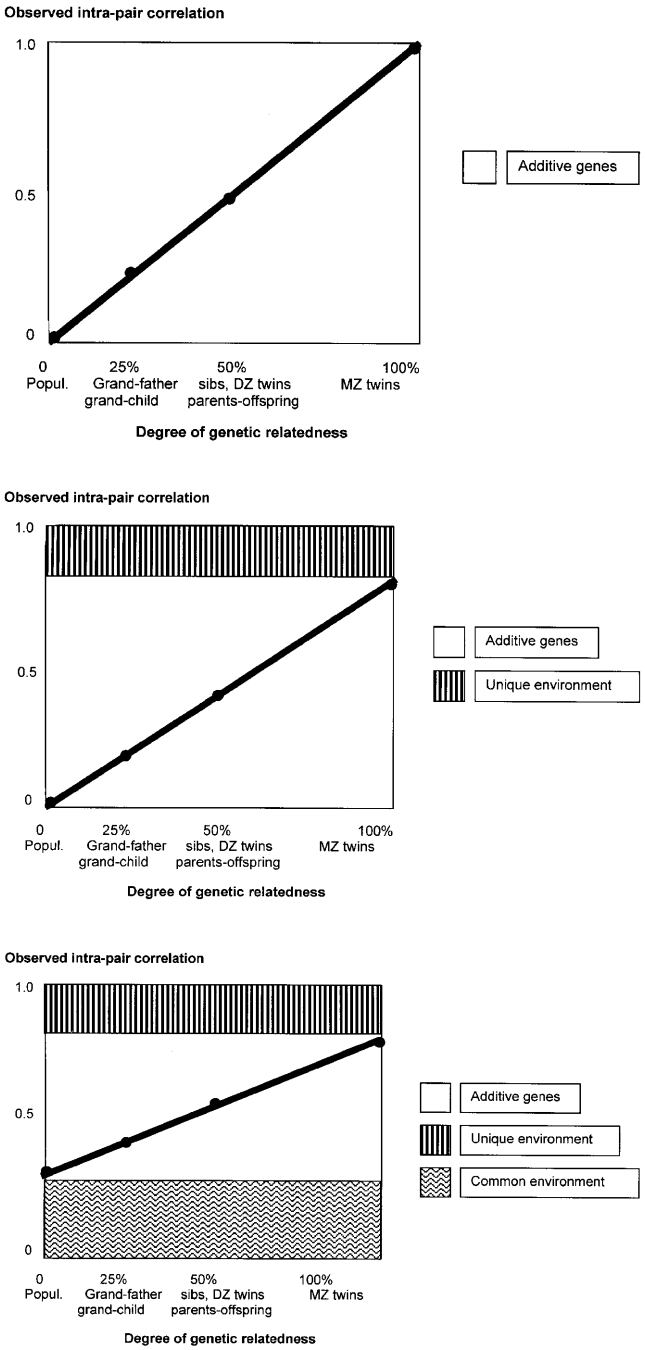

Figure 1 Visual representation (A, B, C) of the relationship between the degree of genetic relatedness and the observed intra-pair similarities. See text for further explanation Popul. = unrelated pairs in a population; DZ = dizygotic; MZ = monozygotic.

When no perfect similarity is found in MZ twins (for example, rMZ = 0.80), but the similarities in other pairs of relatives follow the ratios, according to the ratios in genetic relationship (in DZ, $r = 0.40$), in grandparents–grandchildren ($r = 0.20$) then, two factors will be causing the phenotype: additive genetic factors and environmental factors, contributing in a unique way to an individual (Figure 1B). In this case, additive genes would contribute about 80% to the variation and unique environmental factors for 20% of the variation. In the case when one finds a similarity in pairs which is higher than the expected similarity based on their genetic similarity, part of the variation in the phenotype will be contributed to by common environmental factors, which are shared by both members of the pairs under study (family environment) (Figure 1C). More sophisticated analyses of different familial covariances can be made using path analysis, in which several of the indicative measures (as mentioned above) can now be quantified by testing a hypothetical model to the observed familial variation/covariation matrices.[4,5]

Assessment of the heritability in daily PA is the next step after showing aggregation in families. In the basic genetic model, the total variation (V_{tot}) in a multifactorial trait like PA, is partitioned into genetic (V_G) and environmental (V_C, V_E) variation components ($V_{tot} = V_G + V_C + V_E$)[5]. Heritability ($h^2$) refers to the proportion of the total variation that can be attributed to genetic effects (V_G/V_{tot}). In a similar manner, the contribution of environmental factors shared by family members (common environmental factors, $c^2 = V_C/V_{tot}$) and the proportion of

environmental factors that act on an individual level, can be estimated ($e^2 = V_E/V_{tot}$). When using this additive model of sources of variation, several assumptions should be met: no interaction between gene action and environment (different genotypes all react equally to similar environmental factors), no gene×environment correlation (similar exposure of environments for different genotypes), no gene×gene interaction and no assortative mating. Probably influences on daily PA do not follow these assumptions. The heritability coefficient is further a population parameter and is specific for the studied population. Designs to estimate the heritability fall into twin studies, adoption studies and family studies. Several simple, but less accurate, formulas have been used to estimate the heritability coefficient in the past. More flexibility and accuracy in modelling different factors that influence the phenotype, can be achieved by using path analysis.[4,6] This method constructs correlational and causal paths from and between latent and observed variables, and expresses these relations in linear equations. Alternative models can be tested and the goodness of fit evaluated. An iterative procedure estimates the contributions of each causal path and calculates confidence intervals (CI) for all contributing factors.

All these methods only provide a measure of importance of the role of genetics whereas more specific segregation models can give indications on the action of major genes, mode of inheritance etc. To identify specific genes that contribute to a multifactorial phenotype, one needs to go on with the measured genotype methods.

Two major strategies are available in humans to identify genes that explain variability in daily PA. A first strategy concerns association studies in which one studies the co-occurence of a specific polymorphic marker or a candidate gene and the mean level of PA in groups of carriers or non-carriers of the specific allele. If there is a significant association of an allele with a more active or less active phenotype, almost all carriers of this allele will have a high daily PA level, and only few of the carriers will show a low daily PA level. Positive association results should be carefully interpreted. Association can be positive, because the studied marker does cause the major gene effect, but it might also be that the marker is in linkage disequilibrium with the real gene causing the effect. Association might also be found due to chance or because of population heterogeneity (any allele most frequent in a subgroup of the population, will show association with all phenotypes studied in a mixed population). Association studies do not need genetically related subjects, for example, parents. However, family data can be included in the analyses.[7]

In linkage studies, parametric and non-parametric methods are available, to prove that a marker is in linkage with a gene causing the phenotype, under study. In the parametric method, one tests whether the recombination fraction between a marker and a causal gene is significantly < 0.5. This can be tested by a lod score, with a value of 3 (odds of 1000 to one, in favour of linkage) indicating significant linkage between a marker locus and a locus causing the phenotype. The mode of inheritance has to be known to perform this type of model-based analysis on family data. The sib-pair linkage method, based on the principle of Haseman and Elston,[8] is a non-parametric method. It is based on the relationship of the number of alleles shared at a marker locus (number of alleles shared by descent in pairs of sibs) and the squared differences in observed phenotypes. Evidence for linkage is found when the squared sib-pair trait differences decrease with an increase in the proportion of alleles shared at the marker locus. Multipoint linkage mapping, using all available markers at one time, will be applicable in the future and will allow mapping and identifying loci affecting PA phenotypes.[9]

Genetics of sports participation

The homo olympicus is the result of a superior genotype and a favourable milieu. This is a truism and was accepted as state of the art until the 1950s. Subsequently, studies by Gedda,[10,11] Grebe,[12,13] Grimm[14] and Jokl and Jokl,[15] presented more and more evidence that inheritance was a major determinant of athletic success. These studies, based on family studies of successful athletes, provided evidence that the success in sports followed a familial pattern. This led to the notion of isosportivation[11] (an index of familial aggregation of sports participation, that is, the number of family members that practise the same sport as the successful athlete). Gradually, twin and family studies were undertaken to study the genetic determinants of relevant biological traits that underly athletic success. A recent substantial review of these studies is given in Bouchard *et al.* [2] Although considerable variation is observed in the reported heritabilities, these studies give clear evidence that somatic dimensions, body composition and body type, are all under moderate to high genetic control. Skeletal dimensions, more so than muscle and fat tissue. Also a variety of performance characteristics, including maximal oxygen uptake, various strength measures, flexibility and speed, are all, to some extent, genetically determined. Most of the reported heritabilities from the larger samples, vary between $h^2 = 0.25$ and $h^2 = 0.75$.[2,16]

So far, in only a few studies, the heritability of sports participation has been considered.

In two studies on Dutch twins, variation in self-reported sports participation showed low ($h^2 = 0.35$) to moderately high ($h^2 = 0.77$) heritabilities with higher heritabilities for adolescent males (14–20 y) (Table 1).

In the Leuven Longitudinal Twin Study, data were available on the number of hours spent on sports each

Table 1 Heritabilities for sports participation[a]

Study	*Sample*	h^2
Boomsma *et al*[16]	90 pairs MZ + DZ parents 14–20 y	0.35 females 0.77 males
Koopmans *et al*[17]	1587 pairs MZ + DZ + parents	0.45

[a]sports participation from self reported questionnaires h^2 = heritability; MZ = monozygotic twins; DZ = dizygotic twins.

week within the year preceding data collection. The sports participation not only included organized sports in clubs or school sports, but also sports activities with friends and family members. Physical education classes were excluded since virtually all Flemish secondary schools have two hours of physical education per week. The sample of this study and methods of zygosity determination have been described elsewhere.[18] Results of univariate model-fitting analyses are presented on the logarithmically transformed variable, indicating sports participation in 10 min units of sport/week/year in twins aged 15 y (Log(USP 15)). A raw score of 18 (USP 15) indicates that a mean of 180 min of sports a week during a total year was reported. This score is the sum of all reported sports activities (for example, score 6 for swimming and score 12 for soccer). The goodness-of-fit of different models, each representing an alternative hypothesis, was tested. In the AE model, the total variation in Log(USP 15) was decomposed in additive genetic variation (A) and unique environmental factors (E), in the ACE model, also common environmental factors (C) were included in the model. In the ADE model, dominance genetic variation (D) was included and in the APE model, the effects of phenotypic interaction (P) were tested. The CE and E model are purely environmental models. Gender differences observed in the amount of sports activities in adolescence can result from difference in magnitude of the genetic and/or environmental influences, or by a different set of 'sports activities (susceptibility)' genes, acting in males and females. These gender heterogeneity models were tested in specific models, including the data of five types of twins (monozygotic male–male, female–female, dizygotic male–male, female–female, male–female). Neale and Cardon[5] provide a detailed description of univariate model fitting. Analyses were performed on raw data of logarithmically transformed scores (Log(USP 15)). The goodness-of-fit of the alternative models, was compared with a saturated model and the difference in log likelihood was tested for significance by Chi^2-ratio test. The Aikake's Information Criterion (AIC) was used to finally select the most parsimonious model. The contribution of each factor in the total variation of time spent at sports activities, was expressed as a percentage of the total variation. Confidence intervals (CI) were calculated for these estimates.

At the age of 15 y, boys spent on average about 4 hours (23.1 time units) in sports, while girls were only active in sports for about half that time (14.6 time units) (Student *t*-test: 181.0, $P < 0.01$) (Table 2). The total variation in the amount spent at sports activities was, however, equal in boys and girls. When comparing the five different intra-pair correlations, it is observed that the intra-pair correlations in girls, were much higher than in boys. The female DZ correlation was larger than half the female MZ correlation, suggesting that common environmental factors play an important role. The similarity in the opposite-sex pairs group ($r_{DZOS} = 0.23$) was much lower than in both same-sexed DZ twin groups. This low male-female correlation could, in part, be due to a different set of genes acting in males and females. However, in testing several different genetic heterogeneity models, a model including additive genetic factors, common environmental variation, and unique environmental variation, with a different magnitude of effect in males and females, gave the most parsimonious explanation of the data (Figure 2). Additive genetic factors did explain about 44% of the variation in female sports participation variability (95% CI: 21–91%), while common environmental factors explained about half of the observed variation (proportion of explained variance by common environmental factors, $c^2 = 0.54$, 95% CI: 0.06–0.77). Only a small part of the variation was explained by environmental factors that were unique to the individual members of female twin pairs (proportion of explained variance by unique environmental factors, $e^2 = 0.02$, 95% CI: 0.01–0.04). In males, the common environmental factor could be omitted from the model. The additive genetic factors explained about 83% of the total variance

Table 2 A. Mean and standard deviation (s.d.) of time units of sports participation (USP 15) in male and female twins aged 15 y. Normality test, F-ratio and *t*-test for gender differences. B. Pearson correlations for Log(USP 15) in twins aged 15 y

A		*n*	*Mean*	*s.d.*		*Normality*
USP 15	Boys	92	23.1	21.4		†
(units of 10 min/week/y)	Girls	91	14.6	18.4		†
B		MZm	MZf	DZm	DZf	DZos
Log(USP 15)	*n*	17	17	19	19	19
	r	0.66	0.98	0.62	0.71	0.23

†$P < 0.01$ for H_0: normally distributed data.
* $P < 0.01$ for H_0: USP 15 males = USP 15 females.
MZ = monozygotic twins; DZ = dizygotic twins; m = males; f = females; os = opposite sex; NS = not statistically significant.

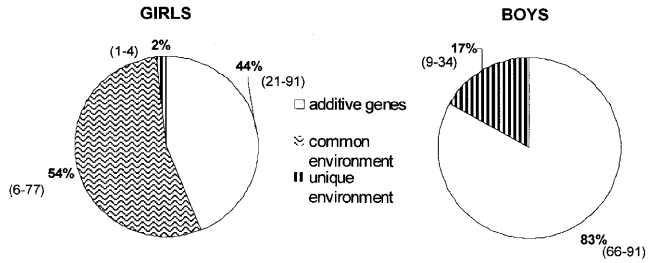

Figure 2 Components of variation in sports participation of girls and boys aged 15 y. 95% Confidence intervals (CI) shown in brackets. (Saturated model: 25 parameters; −2 times log likelihood: 773.5. Most parsimonious model: femaleACE/maleAE: 15 parameters; −2 times log likelihood: 785.4; diff $Chi^2 = 11.9$, probability 0.29, Akaike's Information Criterion = −8.1).

(95% CI: 0.66−0.91), while environmental factors unique to the individual explained the rest of the variance ($e^2 = 0.17$, 95% CI: 0.09−0.34).

Our findings suggest that in this sample we found no evidence for a different set of sports activity genes or sports participation-susceptibility genes acting in males and females aged 15 y. For girls at this age, the fact of being active or inactive in sports is largely influenced by familial environmental factors and genetic factors, while in boys of this age, we found a large genetic contribution to the time spent on sports activities. This could be due to the fact that in sports-related activities, in males especially, physical morphology and basic motor performance are important contributing factors at that age. These physical traits are under important genetic control,[18] and could, in part, explain this large genetic factor in males. In girls, sports participation could be to a larger part a social activity, and therefore more explicitly influenced by environmental factors, especially induced by family and peers. It needs to be elucidated further as to whether these observations are similar in different age groups.

Daily PA

Organised sports participation is for the majority of the adult population only a fraction of their physical activities, and this is probably more so for the obese. It is thus of interest to consider physical activities in a broader sense, encompassing activities during which the body or large parts of the body are moved, resulting in an increase in EE. A variety of techniques have been used to quantify daily physical activities and in epidemiological studies self-reported activities are most often used. This is also the case for the family and twin studies looking at genetics of daily PA. The family correlations and heritability coefficients, reported in Table 3, indicate a small ($h^2 = 0.29$) to moderately high ($h^2 = 0.62$) genetic contribution to the explanation of inter-individual variation in daily PA. It is surprising that such large differences in heritabilities are to be found. The Canadian Fitness Survey[20] and the Québec Family Study,[22] as well as the Finnish Twin Registry,[19] are all comprised of very large samples and data are analysed using up-to-date analytical strategies. A possible explanation can be found in the recording of daily PA. In the Finnish study,[19] information on intensity and duration of leisure time PA, years of participation in leisure time PA, PA during working hours, and a subjective rating of one's own PA level, were obtained from questionnaires. For all these daily PA-characteristics, an intercorrelation matrix was calculated, and subsequently a PA-factor was extracted and individual PA-factor scores were calculated. After adjusting for age, these PA factor scores were then used to calculate intra pair correlations. In the Canada Fitness Survey,[20] information on frequency, duration, and intensity of PA performed on a daily, weekly, monthly and annual basis, was used to estimate average daily EE (expressed per kilogram body weight) and this daily average EE was used in the data analysis. Note that

Table 3 Heritabilities for daily physical activity (PA)[a]

Study	*Sample*	*Indic. Genetic. Det.*
Kaprio *et al*[19]	Finnish Twin Registry 1537 MZ+ 3057 DZ males +18 y	$h^2 = 0.62$ age adjusted
Pérusse *et al*[20]	Canadian Fitness Study 18 073 subjects	$r = 0.28$ spouses $r = 0.12$ Parent−offspring $r = 0.21$ sibs
Sallis *et al*[21]	95 Anglo-American + 111 Mexican-American families	hard PA $r = 0.48$ Mother−Daughter kcal/d $r = 0.45$ sibs $r = 0.38$ M-D adj. for Body mass index more sign. corr. in Mexican-American
Pérusse *et al*[22]	Québèc Family Study 1610 subjects 375 families	daily PA $h^2 = 0.29$ sports part. $b = 0.12$ (b = cultural transmission)

[a]Daily PA from self-reported questionnaires
h^2 = heritability; MZ = monozygotic twins; DZ = dizygotic twins; M-D = BMI = body mass index.

EE is standardised for body mass. In the Québec Family Study,[22] habitual PA and participation in moderate PA and participation in moderate to vigorous PA were obtained from a three-day activity record. Activity records were obtained for over 15 min periods, and expressed in a scale in which each score represented approximately a multiple of the RMR. The scores for each 15 min period were added for one day and totalled over the three days.

For the moderate to vigorous activities, the average number of periods in the highest categories of the rating scale was used. These data were subsequently adjusted for the effects of age, gender, physical fitness, body mass index (BMI) and socio-economic status (SES).

Thus, it is likely that the different estimations of daily PA and certainly also the adjustments for body mass and other characteristics in the Québec Family Study[22] and Canadian Fitness Survey[20] result in lower estimates of heritability than in the Finnish twin study.[19]

It can be argued that these adjustments are probably not the best way to analyse the daily PA data. Using structural equation modelling, it is possible to incorporate in the same multivariate analysis several phenotypes, and to investigate if the same genetic or environmental latent factors determine the variation in the different phenotypes. It is indeed probable that, for example, daily PA and certain fitness phenotypes are, to a certain extent, under the control of the same genetic or environmental factor(s).

Another explanation for the moderately high heritability in the Finnish twins[19] is that the heritability of daily PA is higher in males. This corresponds to what was observed for sports participation in the Leuven Longitudinal Twin Study.

Based on data from the Framingham Study, Moore *et al*[23] observed that children aged 4–7 y ($n = 100$), were more likely to be active when the father was active (odds ratio (OR) = 3.5) than when the mother was active (OR = 2.0). When both parents were active, the OR was considerably higher (OR = 5.8). Similarly, Lauderdale *et al*[24] found, in a study of adult male twins ($n = 3344$) aged 33–51 y, that the OR of one twin engaging in an activity when his co-twin does, was significant and ranged from 2.9–4.6 for intensive activities and between 1.4–1.9 for moderate activities. Genes may influence regular participation in specific intense exercises more than moderate activity, such as walking for exercise.

RMR and free living EE

Since in adults RMR comprises, on average, 70% of the total EE, it seems indicated, in the context of this review, to verify the genetic determinants of this phenotype.

Bogardus *et al*[25] performed a study with 130 non-diabetic healthy adult south-western (Pima) American Indians (74 men and 56 women) from 54 families. RMR was measured by indirect calorimetry. They found that 83% of the interindividual variance in RMR was accounted for by three covariates: fat free mass, age and gender–and fat free mass was the most important covariate ($r = 0.91$). Family membership explained an additional 11% of the variance (6% explained by other factors) in RMR. Thus, RMR, adjusted for fat free mass, age and gender, is a familial trait, suggesting that genetic and/or familial factors (independent of age, gender and fat free mass) explain part of the individual differences in RMR.

Few attempts have been made to quantify the role of genetic factors in RMR. In publications from Fontaine *et al*[26] and Bouchard *et al,*[27] genetic contributions to RMR are estimated. RMR was obtained by indirect calorimetry for 39 twin pairs in the first study[26] and 58 twin pairs and 31 parent-child pairs in the second study.[27] Heritability estimates based on twice the difference between MZ and DZ correlations, and twice the parent-child correlations, suggest that about 40% of the variance in RMR, after adjustment for age, gender, body mass and body composition, may be heritable.[27] More recently, Bouchard *et al*[2] reported the familial aggregation of RMR in 121 nuclear families of the Québec Family Study. About 55% of the variance in RMR, after adjustment for age, gender, fat free mass and fat mass, could be attributed to both genetic and common familial environment factors.

To our knowledge, only Goran[28] investigated the genetic determination of total energy expenditure (TEE). Using doubly labelled water, total 'free-living' EE was examined in 37 young (aged 5–9 y) sib-pairs (including 5 DZ pairs, but no MZ pairs). The sib-pair correlation for RMR, adjusted for fat free mass, was $r = 0.41$, suggesting a relatively high familial aggregation.

Interestingly, when total 'free-living' EE was adjusted for RMR, the sib-pair correlation was still $r = 0.33$, suggesting that there is an independent effect for 'free-living' EE. This confirms what has been observed for sports activity and leisure time physical activity as discussed herein.

Activity as a temperamental trait

Developmentally oriented research on personality has a long history and a small but growing number of findings relating genetics to developmental processes have emerged.[29] Attempts to construct a useful theoretical foundation for the study of temperament have proliferated since the mid-nineteen seventies. In short, temperament may be conceptualized as the foundation for later personality.[30] At present, it is characterized as

heritable, physiologically based and relatively constant over time.[31] Of interest for this review is that 'activity' is one of the components of temperament. In this context, 'activity' as a temperamental trait refers to total energy output[32] and is defined as the amount of energy expended in bodily movements. Temperament studies are mostly restricted to infancy and childhood, and traits are assessed by parental report questionnaires, parental interviews, observer ratings of behaviour in the home and laboratory, and objective behavioural measures such as frequency counts.

A selection of twin studies is summarized in Table 4. Correlations in MZ twins and DZ twins are reported together with heritability estimates. Heritability estimates vary from low ($h^2 = 0.36$) to moderately high ($h^2 = 0.73$) and seem to increase with age. Due to the small number of longitudinal studies and their diversity, firm conclusions regarding genetic influence on the developmental course of personality traits are difficult to draw. It should also be noted that the evidence of genetic influence on temperament in childhood largely comes from twin studies using parental ratings. However, the first parent-offspring and sibling adoption study of parental ratings of temperament found no evidence for genetic influence from the ages of 1–7 y.[38]

More recently,[39] teacher and tester ratings of temperament were investigated based on the Colorado Adoption Project. In this project, adopted children were separated from their biological mothers at an average age of 4 d and were placed in their adoptive homes at an average age of 28 d. Non-adoptive families were matched to adoptive families for gender and age of the children, as well as for number of children in the family and father's educational and occupational status.[39] For teacher ratings, the number of adoptive and non-adoptive siblings was 68 and 77, respectively. For tester ratings they were, 82 and 99, respectively. The analyses, using structural equation modelling, were carried out on the observations made at the age of 7 y. Besides univariate analyses, bivariate analyses were carried out to test whether the same factors determine the teacher and tester ratings. The model included latent additive genetic influences on teacher and tester ratings, shared environmental factors and unique environmental influences. The uni- and bivariate analyses suggest significant genetic influences on teacher and tester ratings. The bivariate model yields heritability estimates of $h^2 = 0.74$ (0.86^2) and $h^2 = 0.27$ ($0.35^2 + 0.38^2$) for teacher and tester ratings of activity, respectively. No significant shared environment influences were found for either measure. Despite the power of the adoptive sibling design for detecting shared environmental influences, the results for teacher and tester ratings of activity confirm the results of twin research in suggesting that there is no significant shared environmental effect for activity ratings, but a significant genetic effect.

Table 4 Selected correlations and heritabilities for activity. Summarized from Buss *et al*[33] Goldsmith and Gottesman,[34] Goldsmith and Campos,[35] Matheney[36] and Matheny and Dolan[37]

	MZ	*DZ*	h^2
3 months	0.30	0.33	
9 months	0.75	0.57	0.36
12 months	0.33	0.28	
24 months	0.58	0.14	
> 55 months	0.73 male	0.00 male	0.73
	0.70 female	0.00 female	0.70
7 y	0.55	0.22	
7 – 10 y	0.66	0.19	0.66

MZ = monozygotic twins; DZ = dizygotic twins; h^2 = heritability.

Genetic markers of RMR

Few studies have considered the role of specific genes in the observed variation in RMR.[40–43] Not surprisingly, all the studies reported, stem from the group with Bouchard in Laval. In the four reports,[40–43] subsamples of pedigrees from the Québec Family Study are used in the linkage analysis. The selection of polymorphic markers is hypothesis driven and based on the present knowledge of physiological, biochemical and metabolic processes that underlie RMR.

Five restriction fragment polymorphisms at three Na,K-ATPase genes (Alpha1, Alpha2 and Beta), were not linked with RMR (adjusted for age, fat mass and fat free mass).[40] Similarly, no evidence of linkage was found between a BclI restriction fragment length polymorphism of 8.3 kb and 4.5 kb in length, expressed in the brown adipose tissue uncoupling protein 1 (UCP1) gene and RMR adjusted for age, fat mass and fat free mass.[41] A missense Tryptophan 64$_{\rightarrow}$ Arginine mutation of the Beta 3 adrenergic receptor gene showed evidence of a weak linkage ($P = 0.04$) with RMR adjusted for age, fat mass and fat free mass.[42] Moreover, three markers (D11S911, D11S916 and D11S1321) encompassing the uncoupling protein 2 (UCP2) locus and spanning a 5 cM region on 11q13 are linked to RMR adjusted for the conventional concomitants.[43] Hence, UCP2 may be one of the biochemical mechanisms potentially involved in the regulation of RMR.

Conclusion

There are a limited number of twin and family studies looking for the genetic determinants of sports participation, daily PA, RMR and 'activity' as a temperamental trait. Considering the variability in the phenotypes under investigation, and the differences in study design and analytical methodology, it is not surprising that a wide range of heritabilities are reported (from $h^2 = 0.29$ to $h^2 = 0.83$).

It is of interest to note that adjustments for concomitant factors, such as age, gender, fat free mass and fat mass, may probably explain the low heritabilities observed in some studies. In our opinion, it can be questioned if these adjustments are the most optimal strategy to decipher the underlying genetic determination. Also, activity as a temperamental trait is under genetic control and no evidence is found for shared environmental factors. Children tend to be active partly because genetic factors underly this engagement in more active lifestyles. The limited amount of linkage studies thus far undertaken demonstrated that three markers encompassing the UCP2 locus on 11q13 are linked to resting EE in adult humans.

References

1 Westerterp KR, Saris WHM, van Es M, ten Hoor F. Use of the doubly-labeled water technique in humans during heavy sustained exercise. *J Appl Physiol* 1986; **61**: 2162–2167.
2 Bouchard C, Malina RM, Pérusse L. *Genetics of fitness and physical performance.* Human Kinetics: Champaign, 1997, pp 400.
3 Sing CF, Boerwinkle EA. Genetic architecture of inter-individual variability in apolipoprotein, lipoprotein and lipid phenotypes. In: Bock G, Collins GM (eds). *Molecular approaches to human polygenic disease.* Wiley: New York, 1987, pp 99–127.
4 Eaves LJ, Last KA, Young PA, Martin NG. Model-fitting approaches to the analysis of human behavior. *Heredity* 1978; **41**: 249–320.
5 Neale MC, Cardon L. *Methodology for genetic studies of twins and families.* Kluwer Academic: Dordrecht, 1992, pp 496.
6 Jinks JL, Fulker DW. Comparison of the biometrical genetical, MAVA and classical approaches to the analysis of human behavior. *Psychol Bull* 1970; **73**: 311–349.
7 George VT, Elston RC. Testing the association between polymorphic markers and quantitative traits in pedigrees. *Genet Epidemiol* 1987; **4**: 193–201.
8 Haseman JK, Elston RC. The investigation of linkage between a quantitative trait and a marker locus. *Behav Genet* 1972; **2**: 3–19.
9 Todorov AA, Siegmund KD, Gu C, Heath AC, Rao DC. Multivariate linkage analysis with structural relations. *Am J Hum Genet* 1997; **61**: A46 (239).
10 Gedda L. La valutazione genetica dell'atleta. *Acta Genet Gemell* 1955; **4**: 249–260.
11 Gedda L. Sport and genetics. A study on twins (351 pairs). *Acta Genet Gemell* 1960; **9**: 387–405.
12 Grebe H. Sport bei Zwillingen. *Acta Genet Gemell* 1955; **4**: 275–295.
13 Grebe H. Sportfamilien. *Acta Genet Gemell* 1956; **5**: 318–326.
14 Grimm H. Zur Frage nach den Erbfaktoren für sportliche Liestungsfähigkeit. In: *XVI Weltkongress für Sportmedizin, Hannover 1996. Kongressbericht.* Deutscher Ärzte Verlag: Köln, 1966, pp 530–634.
15 Jokl E, Jokl P. *The physiological basis of athletic records.* Thomas: Springfield, 1968.
16 Boomsma DI, van den Bree MBM, Orlebeke JF, Molenaar PCM. Resemblances of parents and twins in sports participation and heart rate. *Behav Genet* 1989; **19**: 123–141.
17 Koopmans JR, Van Doomen LJP, Boomsma DI. Smoking and sports in participation. In: Goldbourt U, de Faire U, Berg K (eds). *Genetic Factors in Coronary Heart Disease.* Kluwer Academic: Dordrecht, 1994, pp 217–235.
18 Maes HM, Beunen GP, Vlietinck RF, Neale MC, Thomis M, Vanden Eynde B, Lysens R, Simons J, Derom C, Derom R. Inheritance of physical fitness in 10-year-old twins and their parents. *Med Sci Sports Exerc* 1996; **28**: 1479–1491.
19 Kaprio JM, Koskenvuo M, Sarna S. Cigarette smoking, use of alcohol and leisure-time activity among same-sexed adult male twins. In: Gedda L, Parisi P, Nance WE (eds). *Progress in clinical and biological research. Twin research 3: epidemiological and clinical studies.* Alan R Liss: New York, 1981, pp 37–46.
20 Pérusse L, Leblanc C, Bouchard C. Inter-generation transmission of physical fitness in the Canadian population. *Can J Sport Sci* 1988; **13**: 8–14.
21 Sallis JF, Patterson TL, Buono MJ, Atkins CJ, Nader PR. Aggregation of physical activity habits in Mexican-American and Anglo families. *J Behav Med* 1988; **11**: 31–41.
22 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.
23 Moore LL, Lombardi DA, White MJ, Campbell JL, Oliveria SA, Ellison SA. Influence of parents' physical activity levels on young children. *J Pediatr* 1991; **118**: 215–219.
24 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.
25 Bogardus C, Lillioja S, Ravussin E, Abbott W, Zawadzki J, Young A, Knowler WC, Jacobowitz R, Molli PP. Familial dependence of the resting metabolic rate. *New Engl J Med* 1986; **315**: 96–100.
26 Fontaine E, Savard R, Tremblay A, Després JP, Poehlman E, Bouchard C. Resting metabolic rate in monozygotic and dizygotic twins. *Acta Genet Med Gemellol* 1985; **34**: 41–47.
27 Bouchard C, Tremblay A, Nadeau A, Després JP, Thériault G, Boulay MR, Lortie G, Leblanc C, Fournier G. Genetic effect in resting and exercise metabolic rates. *Metabolism* 1989; **38**: 364–370.
28 Goran Ml. Genetic influences on human energy expenditure and substrate ultilization. *Behav Genet* 1997; **27**: 389–399.
29 Goldsmith HH. Genetic influences on personality from infancy to adulthood. *Child Dev* 1983, **54**: 331–355.
30 Willeman L. *The psychology of individual and group differences.* Freeman: San Francisco, 1979.
31 Lengua LJ, West SG, Sandler IN. Temperament as a predictor of symptomatology in children: addressing contamination of measures. *Child Dev* 1998; **69**: 164–181.
32 Buss AH, Plomin R. *A temperament theory of personality development.* Wiley: New York, 1975.
33 Buss AH, Plomin R, Willerman L. The inheritance of temperaments. *J Pers* 1973; **41**: 513–524.
34 Goldsmith HH, Gottesman I. I. Origins of variation in behavioral style: a longitudinal study of temperament in young twins. *Child Dev* 1981, **52**: 91–103.
35 Goldsmith HH, Campos JJ. Toward a theory of infant temperament. In: Emde RN, Harmon HJ (eds). *The development of attachment and affiliative systems.* Plenum: New York, 1982.
36 Matheny AP. Bayley's Infant Behavior Record: behavioral components and twin analyses. *Child Dev* 1980; **51**: 466–475.
37 Matheny AP, Dolan AB. A twin study of personality and temperament during middle childhood. *J Res Pers* 1980; **14**: 224–234.
38 Plomin R, Coon H, Carey G, DeFries JC, Fulker DW. Parent-offspring and sibling adoption analyses of parental ratings of temperament in infancy and childhood. *J Pers* 1991; **59**: 705–732.
39 Schmitz S, Saudino KJ, Plomin R, Fulker DW, DeFries JC. Genetic and environmental influences on temperament in middle childhood: analyses of teacher and tester ratings. *Child Dev* 1996; **67**: 409–422.

40 Dériaz O, Dionne F, Pérusse L, Tremblay A, Vohl M-C, Côté G, Bouchard C. DNA variation in the genes of the Na,K-adenosine triphosphatase and its relation with resting metabolic rate, respiratory quotient and body fat. *J Clin Invest* 1994; **93**: 838–843.

41 Oppert J-M, Vohl M-C, Chagnon M, Dionne F, Cassard-Doulcier A-M, Ricquier D, Pérusse L, Bouchard C. DNA polymorphism in the uncoupling protein (UCP) gene and human body fat. *Int J Obes* 1994; **18**: 526–531.

42 Gagnon J, Mauriège P, Roy S, Sjöström D, Chagnon YC, Dionne FT, Oppert J-M, Pérusse L, Sjöström L, Bouchard C. The Trp64Arg mutation of the β3 adrenergic receptor gene has no effect on obesity phenotypes in the Québec Family Study and Swedish Obese Subjects Cohorts. *J Clin Invest* 1996; **98**: 2086–2093.

43 Bouchard C, Pérusse L, Chagnon YC, Warden C, Ricquier D. Linkage between markers in the vicinity of the uncoupling protein 2 gene and resting metabolic rate in humans. *Hum Mol Genet* 1997; **6**: 1887–1889.