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André F. Seabra a, Denisa M. Mendonça b, Harald H. H. Göring c, Martine A. Thomis d & José A. Maia e

a Research Centre in Physical Activity, Health and Leisure (CIAFEL), Faculty of Sport, University of Porto, Porto, Portugal
b Institute of Public Health (ISPUP), Institute of Biomedical Sciences Abel Salazar, University of Porto, Porto, Portugal
c Department of Genetics, Texas Biomedical Research Institute, San Antonio, TX, USA
d Faculty of Sport Sciences and Physical Education, Department of Biomedical Kinesiology, Katholieke Universiteit Leuven, Leuven, Belgium
e Research, Education, Innovation and Intervention in Sport (CIFI2D), Faculty of Sport, University of Porto, Porto, Portugal

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Genetic influences of sports participation in Portuguese families

ANDRÉ F. SEABRA1, DENISA M. MENDONÇA2, HARALD H. H. GÖRING3, MARTINE A. THOMIS4, & JOSÉ A. MAIA5

1Research Centre in Physical Activity, Health and Leisure (CLAFEL), Faculty of Sport, University of Porto, Porto, Portugal, 2Institute of Public Health (ISPUP), Institute of Biomedical Sciences Abel Salazar, University of Porto, Porto, Portugal, 3Department of Genetics, Texas Biomedical Research Institute, San Antonio, TX, USA, 4Faculty of Sport Sciences and Physical Education, Department of Biomedical Kinesiology, Katholieke Universiteit Leuven, Leuven, Belgium, 5Research, Education, Innovation and Intervention in Sport (CIFI2D), Faculty of Sport, University of Porto, Porto, Portugal

Abstract
To estimate familial aggregation and quantify the genetic and environmental contribution to the phenotypic variation on sports participation (SP) among Portuguese families. The sample consisted of 2375 nuclear families (parents and two offspring each) from different regions of Portugal with a total of 9500 subjects. SP assessment was based on a psychometrically established questionnaire. Phenotypes used were based on the participation in sports (yes/no), intensity of sport, weekly amount of time in SP and the proportion of the year in which a sport was regularly played. Familial correlations were calculated using family correlations (FCOR) in the SAGE software. Heritability was estimated using variance-components methods implemented in Sequential Oligogenic Linkage Analysis Routines (SOLAR) software. Subjects of the same generation tend to be more similar in their SP habits than the subjects of different generations. In all SP phenotypes studied, adjusted for the effects of multiple covariates, the proportion of phenotypic variance due to additive genetic factors ranged between 40% and 50%. The proportion of variance attributable to environmental factors ranged from 50% for the participation in sports to 60% for intensity of sport. In this large population-based family study, there was significant familial aggregation on SP. These results highlight that the variation on SP phenotypes have a significant genetic contribution although environmental factors are also important in the familial resemblance of SP.

Keywords: Sports participation, families, genetics, environment

Introduction
The challenge of understanding the excellence of sports performance evidenced by homo olympicus has occupied sports science researchers since the 1950s. A very thoughtful way of study has been based on family research, focusing in sports family lines, revisiting Francis Galton’s work about excellence and genius. Gedda (1955, 1960) was one of the first to show that elite athletes belong to certain families with a strong sport history, and suggested that sport participation (SP) is partially genetic and followed a familial pattern.

Genetic studies with Olympic athletes were conducted by De Garay, Levine, and Carter (1974) in the Mexico Olympics, and a similar research was made in the Montreal Olympic games (Chagnon, Allard, & Bouchard, 1984; Couture, Chagnon, Allard, & Bouchard, 1986). Four recent reviews (Bouchard, Malina, & Péрусse, 1997; de Vilhena e Santos, Katzmarzyk, Seabra, & Maia, 2012; Rankinen et al., 2006; Roth et al., 2012) presented a strong evidence that a variety of phenotypes related to sports performance may be determined, moderately to strongly, by genetic factors. A recent study has also concluded that physical activity levels were significantly lower in patients with mitochondrial mutations compared with controls (Apabhai et al., 2011) which represents an excellent illustration of the multifactorial and polygenic nature of complex traits such as physical activity levels and sedentary behaviours (Roth et al., 2012).
However, athletes are a sub-sample of the population, a highly selected group, whose phenotypic values and response to training are in the upper centiles compared to ‘ordinary people’. SP is a complex phenotype influenced by multiple genetic and non-genetic (environmental) factors, which shows an enormous inter-individual variability at the population level. In fact, the type of sports, intensity, duration and frequency of its practice is highly variable among individuals, within and between family members.

Not many studies have addressed the issue of identification of multiple facets of the SP phenotype. The available information is mostly related to twins research (Beunen & Thomis, 1999; Boomsma, van den Bree, Orlebeke, & Molenaar, 1989; Eriksson, Rasmussen, & Tynelius, 2006; Koopmans, van Doornen, & Boomsma, 1994; Maia, Thomis, & Beunen, 2002; Stubb et al., 2006) and only one study looked at familial clustering in exercise participation of different intensities (Perusse, Tremblay, Leblanc, & Bouchard, 1989). In summary, heritability estimates of SP are highly variable – from 35% to 77% of the total variation. Intensity, duration and frequency of SP are relevant aspects of a complex phenotype reflecting a highly plastic and multifaceted behaviour that has implications in healthy living at the population level. We have no data available to address the issue of searching for their putative genes, however, this issue motivated this study to investigate if 1. familial aggregation is evident in these phenotypes, and 2. to quantify the genetic and environmental contribution to phenotypic variation in SP in Portuguese nuclear families.

**Methods**

**Participants**

The sample consisted of 2375 nuclear families (i.e. father, mother and two siblings >10 years of age) from different municipalities in the northern part of Portugal (Porto, Viseu, Vila Real and Bragança) with a total of 9500 subjects. Nuclear families were drawn from a research project aimed to identify the relevant determinants of physical activity within the Portuguese population. Children and adolescents were approached to freely participate in the study. Those who had one or more siblings were asked to enrol themselves as well as their parents. Since, the segment of families with three or more children over 10 years of age is very small in the Portuguese population (National Statistic Institute, 2001), our sample comprises nuclear families with only two siblings. The decision to set a cut-off age at 10 years is related to reliability and validity experiences with the Baecke, Burema, and Frijters (1982) questionnaire done in Portugal in previous research (Seabra et al., 2008; Seabra, Mendonca, Thomis, Peters, & Maia, 2008). Furthermore, reading and comprehensive abilities may be insufficient below 10 years to understand the instrument (Maia & Lopes, 2003). This project was approved by the research committee of the Faculty of Sport of the University of Porto and by school authorities. Parents and children also provided informed consent.

**Phenotypes and covariates**

**Sport participation (SP)**

SP was estimated with the Baecke et al. (1982) protocol, a reliable and valid instrument to estimate different facets of physical activity (Montoye, Kemper, Saris, & Washburn, 1996; Philippaerts & Lefevre, 1998). The questionnaire has been reliably used in several samples of the Portuguese population aged 10–18 years, where intraclass correlations for estimated physical activity ranged from 0.80 to 0.87 (Vasconcelos & Maia, 2001). The Baecke questionnaire consists largely of 16 items that are designed to assess different categories of physical activity (work/school, sport and leisure). The items require Likert-type responses ranging from 1 to 5. For this study, four different phenotypes were calculated from the responses to the most frequently played sport.

The first one was a binary variable regarding participation in sports: ‘Do you play sport? – yes/no’. SP was defined as all practice of formal sports in both schools and private clubs. School sports in Portugal are voluntary programmes offered during free/discretionary time and have defined competitive seasons. Information on formal physical education activities was not utilised. The second was about the intensity of sport and is based on the following question: ‘Which sport do you play most frequently?’ Intensity of SP was estimated after Durnin and Passmore (1967) where energy expenditure was divided into three categories: low (0.76 Mjoules h⁻¹), for example, bowling, golf, sailing; medium (1.26 Mjoules h⁻¹), for example, badminton, cycling, dance, swimming; and high (1.76 Mjoules h⁻¹), for example, boxing, basketball, soccer. The third was about the weekly amount of time this sport is played: ‘How many hours a week?’ The amount of time per week was divided into five categories: <1 hour, 1–2 hours, 2–3 hours, 3–4 hours and >4 hours. The fourth phenotype was about the proportion of the year in which the sport was regularly played: ‘How many months a year?’ This phenotype was calculated according to five monthly fractions: <1 month, 1–3 months, 4–6 months, 7–9 months and >9 months.
**Social economic status (SES)**

SES was assessed by questions asking parents about their jobs as suggested by Kunst, Bos, and Mackenbach (2001). The occupation was categorised into four levels: students, low (semi-skilled and unskilled manual workers), medium (intermediate skilled workers) and high (professionals).

Teachers at each of the selected schools administered the questionnaires, which were completed in the school. They were trained in the administration of the questionnaire prior to the survey and had also written the guidelines for its administration. Students’ and parents’ questions and difficulties were answered.

**Statistical procedures**

Descriptive statistics (SPSS 20.0) summarised the physical characteristics and SP phenotypes for parents and offspring. Differences in the prevalence of SP among parents and offspring were analysed using logistic regression. Differences between parents and offspring in the intensity of sport, weekly amount of time and the proportion of the year were tested with Mann–Whitney U-test. Familial aggregation was calculated using familial correlations computed by the family correlations (FCOR) procedure in the SAGE software (S.A.G.E., 2007) for spouses, parent–offspring pairs, father–son pairs, father–daughters pairs, mother–son pairs, mother–daughters pairs, siblings, brother pairs, brother–sister pairs and sister pairs. The heritability of different phenotypes was calculated using the variance-components methods implemented in the Sequential Oligogenic Linkage Analysis Routines (SOLAR) software (Almasy & Blangero, 1998). The variance-components were estimated by maximum likelihood and the usual model follows classical quantitative genetic principles, in which the phenotypic variance ($\sigma^2_p$) is decomposed into additive components for additive genetic ($\sigma^2_A$) and non-genetic (i.e. environmental) effects ($\sigma^2_E$). The heritability of a phenotype is estimated as $h^2 = \sigma^2_A / \sigma^2_p$. As defined in this manner, $h^2$ refers specifically to ‘narrow-sense’ heritability (i.e. the proportion of phenotypic variance attributable to additive genetic effects), however, shared familial environmental effects might also contribute to this estimate. Significance testing is accomplished by a likelihood ratio test (Almasy & Blangero, 1998).

Covariates as age, sex, age$^2$, sex*age, sex*age$^2$, SES, sex*SES, and age*SES were included in the model. SOLAR only supports discrete traits with two possible values. Since intensity of sport, weekly amount of time and proportion of the year are discrete phenotypes although mark continuities, we let SOLAR consider them as quantitative continuous phenotypes (Almasy & Blangero, 1998).

**Results**

The prevalence of SP was higher in offspring than parents (OR: 6.4; 95%CI: 5.8–7.0) (Table I). A higher proportion of SP was found in fathers (OR: 2.0; 95%CI: 1.7–2.3) than mothers, and in sons compared to daughters (OR: 3.0; 95%CI: 2.6–3.4). For the other phenotypes, offspring had higher prevalence than parents (intensity of sport: $z = 19.33; p < 0.001$; weekly amount of time: $z = 9.28; p < 0.001$; proportion of the year: $z = 2.28; p = 0.02$).

Fathers had higher percentages than mothers for the intensity of sport ($z = 6.70; p < 0.001$) and for the proportion of the year ($z = 3.56; p < 0.001$). Sons had higher proportions than daughters (intensity of sport: $z = 16.54; p < 0.001$; weekly amount of time: $z = 12.44; p < 0.001$; proportion of the year: $z = 5.14; p < 0.001$).

Familial correlations between the various types of relative pairs are shown in Table II. The correlations ($0.02–0.52$) indicated a significant familial resemblance in almost all the SP phenotypes studied (except mother–daughter and father–daughter for intensity of sport and mother–son for proportion of the year). The results also show that subjects of the same generation tend to be more similar in their SP than subjects of different generations. Father–mother (0.12–0.48) and siblings (0.27–0.31) correlations were higher than parent–offspring (0.13–0.25). Furthermore, there are no large sex-specific differences between correlations reported among parents and offspring (father–son, father–daughter, mother–son and mother–daughter). There is some variation in the correlations in siblings by sex. Brother–brother and sister–sister correlations were much higher than brother–sister sets (except for the proportion of the year between brothers).

The proportions of the variance in the phenotypes that can be attributed to genetic effects are presented in Table III. After adjustments for the multiple covariates all heritability estimates were significant. The proportion of phenotypic variance due to additive genetic factors ranged from 40% to 50%. The remaining variance was attributable to unique environmental influences (i.e. not shared among the individuals) and random error. The estimated heritability was highest in SP (50%) and proportion of the year phenotypes (49%), then weekly amount of time (46%) and intensity of sport (40%). The residual kurtosis, after adjusting for covariates, was close to zero, indicating that the assumption of multivariate normality of the phenotypes was not violated.

**Discussion**

To our knowledge, this study is the first large population-based family study that attempted to
estimate the degree of familial clustering in different phenotypes related to SP. In this study, the heritability estimates among Portuguese nuclear families ranged from 40% to 50%. These data confirm that genetic factors contribute to the familial aggregation of the SP and its components.

Comparisons of our results to other studies are difficult because they include a wide range of populations, sample sizes, study designs (twins and family studies), data analytical methods and phenotypes (Beunen & Thomis, 1999; Boomsma et al., 1989; Eriksson et al., 2006; Koopmans et al., 1994; Maia et al., 2002; Perusse et al., 1989; Roth et al., 2012; Stubbe et al., 2006). Although phenotypes in these studies are related to SP, no previous studies have reported heritability estimates for intensity, weekly amount of time and proportion of the year. Thus, despite the discrepancies between studies, our

Table I. Descriptive statistics of physical characteristics and sport participation for parents and offspring

<table>
<thead>
<tr>
<th>Variable</th>
<th>Father (n=2375)</th>
<th>Mother (n=2375)</th>
<th>Sons (n=2425)</th>
<th>Daughters (n=2325)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>45.45 ± 5.84</td>
<td>42.92 ± 5.47</td>
<td>16.15 ± 4.03</td>
<td>16.01 ± 3.98</td>
</tr>
<tr>
<td>Social economic status (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Students</td>
<td>–</td>
<td>–</td>
<td>86.4</td>
<td>91.5</td>
</tr>
<tr>
<td>Low skilled</td>
<td>79.2</td>
<td>82.1</td>
<td>11.8</td>
<td>6.5</td>
</tr>
<tr>
<td>Intermediate skilled</td>
<td>12.4</td>
<td>8.2</td>
<td>0.9</td>
<td>0.6</td>
</tr>
<tr>
<td>High skilled</td>
<td>8.4</td>
<td>9.7</td>
<td>0.9</td>
<td>1.4</td>
</tr>
<tr>
<td>Sport participation (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>18.7</td>
<td>9.9</td>
<td>1.9</td>
<td>2.2</td>
</tr>
<tr>
<td>Medium</td>
<td>40.9</td>
<td>87.8</td>
<td>21.8</td>
<td>54.7</td>
</tr>
<tr>
<td>High</td>
<td>41.3</td>
<td>2.3</td>
<td>76.3</td>
<td>43.0</td>
</tr>
<tr>
<td>Weekly amount of time (hours) (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1 hour</td>
<td>14.5</td>
<td>11.1</td>
<td>7.0</td>
<td>9.7</td>
</tr>
<tr>
<td>1–2 hours</td>
<td>33.5</td>
<td>39.7</td>
<td>19.4</td>
<td>36.9</td>
</tr>
<tr>
<td>2–3 hours</td>
<td>22.4</td>
<td>27.1</td>
<td>20.7</td>
<td>25.7</td>
</tr>
<tr>
<td>3–4 hours</td>
<td>13.2</td>
<td>12.2</td>
<td>16.5</td>
<td>11.1</td>
</tr>
<tr>
<td>&gt; 4 hours</td>
<td>16.4</td>
<td>9.9</td>
<td>36.3</td>
<td>16.6</td>
</tr>
<tr>
<td>Proportion of the year (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1 month</td>
<td>2.2</td>
<td>1.9</td>
<td>1.5</td>
<td>2.7</td>
</tr>
<tr>
<td>1–3 months</td>
<td>9.3</td>
<td>3.8</td>
<td>4.7</td>
<td>6.0</td>
</tr>
<tr>
<td>4–6 months</td>
<td>21.1</td>
<td>11.9</td>
<td>12.0</td>
<td>18.1</td>
</tr>
<tr>
<td>7–9 months</td>
<td>18.5</td>
<td>23.1</td>
<td>22.7</td>
<td>23.4</td>
</tr>
<tr>
<td>&gt; 9 months</td>
<td>48.9</td>
<td>59.2</td>
<td>59.1</td>
<td>49.8</td>
</tr>
</tbody>
</table>

*aFor all the phenotypes, there are significant higher prevalence’s in offspring than parents, in fathers than mothers (except weekly amount of time), and in sons compared to daughters.

Table II. Familial correlations of sport participation phenotypes

<table>
<thead>
<tr>
<th>Relationship</th>
<th>No. of pairs</th>
<th>Sport participation</th>
<th>Intensity of sport</th>
<th>Weekly amount of time</th>
<th>Proportion of the year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father–mother</td>
<td>2375</td>
<td>0.23 (0.19, 0.27)*</td>
<td>0.12 (–0.06, 0.30)</td>
<td>0.46 (0.32, 0.60)*</td>
<td>0.48 (0.34, 0.62)*</td>
</tr>
<tr>
<td>Parent–offspring</td>
<td>9500</td>
<td>0.15 (0.13, 0.17)*</td>
<td>0.13 (0.07, 0.19)*</td>
<td>0.19 (0.11, 0.27)*</td>
<td>0.25 (0.17, 0.33)*</td>
</tr>
<tr>
<td>Father–son</td>
<td>2363</td>
<td>0.16 (0.12, 0.20)*</td>
<td>0.15 (0.05, 0.25)*</td>
<td>0.14 (0.04, 0.24)*</td>
<td>0.22 (0.12, 0.32)*</td>
</tr>
<tr>
<td>Father–daughter</td>
<td>2387</td>
<td>0.14 (0.10, 0.18)*</td>
<td>0.10 (–0.04, 0.24)</td>
<td>0.23 (0.09, 0.37)*</td>
<td>0.40 (0.28, 0.52)*</td>
</tr>
<tr>
<td>Mother–son</td>
<td>2363</td>
<td>0.11 (0.07, 0.15)*</td>
<td>0.15 (0.01, 0.29)*</td>
<td>0.21 (0.07, 0.35)*</td>
<td>0.08 (–0.06, 0.22)</td>
</tr>
<tr>
<td>Mother–daughter</td>
<td>2387</td>
<td>0.19 (0.15, 0.23)*</td>
<td>0.02 (–0.14, 0.18)</td>
<td>0.22 (0.06, 0.38)*</td>
<td>0.21 (0.05, 0.37)*</td>
</tr>
<tr>
<td>Siblings</td>
<td>2375</td>
<td>0.27 (0.23, 0.31)*</td>
<td>0.31 (0.25, 0.37)*</td>
<td>0.31 (0.25, 0.37)*</td>
<td>0.29 (0.23, 0.35)*</td>
</tr>
<tr>
<td>Brothers</td>
<td>598</td>
<td>0.29 (0.21, 0.37)*</td>
<td>0.51 (0.43, 0.59)*</td>
<td>0.37 (0.27, 0.47)*</td>
<td>0.22 (0.12, 0.32)*</td>
</tr>
<tr>
<td>Brother–sister</td>
<td>1167</td>
<td>0.23 (0.17, 0.29)*</td>
<td>0.18 (0.08, 0.28)*</td>
<td>0.22 (0.12, 0.32)*</td>
<td>0.27 (0.17, 0.37)*</td>
</tr>
<tr>
<td>Sisters</td>
<td>610</td>
<td>0.31 (0.23, 0.39)*</td>
<td>0.39 (0.23, 0.55)*</td>
<td>0.52 (0.38, 0.66)*</td>
<td>0.52 (0.38, 0.66)*</td>
</tr>
</tbody>
</table>

*p < 0.05.

Table III. Upper-limit heritabilities (h² ± SE) of sport participation phenotypes

<table>
<thead>
<tr>
<th>Phenotypes</th>
<th>Polygenic model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sport participation</td>
<td>0.50 ± 0.03</td>
</tr>
<tr>
<td>Intensity of sport</td>
<td>0.40 ± 0.04</td>
</tr>
<tr>
<td>Weekly amount of time</td>
<td>0.46 ± 0.05</td>
</tr>
<tr>
<td>Proportion of the year</td>
<td>0.49 ± 0.05</td>
</tr>
</tbody>
</table>
results are perhaps most directly comparable with studies which estimate the heritability of SP in a broad sense.

Heritability estimates from twins studies (Beunen & Thomis, 1999; Boomsma et al., 1989; De Moor, Stubbe, Boomsma, & De Geus, 2007; Koopmans et al., 1994; Maia et al., 2002; Stubbe, Boomsma, & De Geus, 2005; Stubbe et al., 2006) ranged from 31% to 77% and clearly indicated that elements of the genome were important and account for a substantial portion of the variation of SP at the population level.

In the Quebec Family Study (QFS), comprising 325 families from Quebec/Canada, an estimate of heritability for the exercise participation has been reported (Perusse et al., 1989). Exercise participation was assessed using a three-day activity diary and each subject was asked to note, on a scale from 1 to 9 the energy expenditure of the dominant activity for each 15-minute period. The number of periods corresponding to activities rated from 6 to 9 (greater than or equal to five metabolic equivalent [MET]) was used as the indicator of exercise participation. In opposition to our study, in this report, no genetic effect was observed for exercise participation and the estimated transmissible variance was entirely of cultural origin (12%). The presence of cultural inheritance in exercise participation suggested that the children could acquire and share with their parents adequate behaviours favouring adherence to exercise participation and involvement. It was also shown that 88% of exercise participation was accounted for by non-transmissible environmental factors. This strong contribution of environmental factors was an indication that changes in exercise intensity due to SP at the population level could be achieved with appropriate intervention programmes.

Among the phenotypes analysed, SP assessed by the question ‘Do you play sport?’ had the highest heritability estimation which suggest that the practice of sport may be more influenced by genetic factors than the others phenotypes. Our heritability estimates for the SP phenotype was consistent with the previous twins studies (De Moor et al., 2007; Koopmans et al., 1994; Stubbe et al., 2006). The study of Koopmans et al. (1994) analysed twins and their parents to estimate parent–offspring heritability. They used a question to measure SP ‘Do you participate in sports regularly?’ The heritability estimate was comparable to our finding (48%). In another study, De Moor et al. (2007) investigated the genetic influence of exercise participation using the question ‘Do you exercise sport?’ and genetic factors were reported to contribute to 54% of the variability. In the GenomeEUtwin (Stubbe et al., 2006) project on exercise participation in leisure time, it was reported a median heritability of exercise participation of 62%. Although the heritability estimation of SP varied somewhat among studied populations, taken together, all these results provide an empirical evidence that SP has a significant genetic component and merits a search for the genes involved in its aetiology.

It has been suggested that the influence of shared environmental factors (i.e. those exposures which are shared among related individuals, such as typically their common diet) on the variation of SP was higher in comparison to unique environmental factors (i.e. those factors that act uniquely on one individual or another, such as the influence of separate circles of friends, or accidents impacting a particular individual but not the entire family, etc.) (Beunen & Thomis, 1999; Koopmans et al., 1994; Stubbe et al., 2005). As a result, it was then proposed that the design of efficient programmes to increase the prevalence of SP led to include the family and peers. In fact, the family has been unanimously considered a critical and decisive factor in the psychological and social well-being of offspring due to its capacity of shaping behaviours mostly related to healthy and active living (Duncan, Duncan, & Strycker, 2005).

According to these authors, social support from peers to SP in children and adolescents can be seen as an act of social integration (participating together in the activity), in emotional (encouragement) and instrumental aspects (sharing equipment and means of transportation) devoted to enjoyable exercise participation. Contrary to this, other authors showed a greater importance of unique environmental effects (Boomsma et al., 1989; De Moor et al., 2007; Maia et al., 2002; Stubbe et al., 2006). It seems that unique characteristics of individuals as well as their unshared behaviours with parents and siblings, linked with random life events may also explain the variation found at the population level in their SP. Unique environmental effects are those effects from the environment that are different between members of a family (different friends at school or sports organisations, nutritional choices different from ‘family menu’, sports injuries, etc.) and do not contribute to similarities between family members.

In this study, we also investigated how the correlations between members within families varied for different relationships and for genetically unrelated members. Familial correlations between family members were mostly greater than zero suggesting a significant familial similarity in SP, its intensity, duration and frequency. However, individuals of the same generation, genetically related as siblings, or genetically unrelated as parents, had more similar SP phenotypes than those from different generations (except for the intensity of the sport). This trend suggests that environmental factors and lifestyles...
shared by persons of the same generation play a significant role in the phenotypic variance of SP. In fact, for phenotypes whose variation is attributable to genetic effects, parental correlations should be very low and close to zero. Many of the parental correlations found were greater than zero and in some cases were higher in magnitude than the parents–offspring correlations (except for the intensity of sport). Since no parents were related by blood, any significant correlation of these SP phenotypes reflects environmental factors to which the couples are continuously exposed or parental similarities that already existed at the time the partners choose each other. Social homogamy and/or assortative mating effects may be contributing factors to parental concordance of SP. Social homogamy, or the sharing of the same or a similar household environment, usually implies the sharing of many aspects of lifestyle. Thus, individuals who cohabit should show concordance in SP, and similarities would increase by years of cohabitation. However, although husband and wife are not genetically related, concordance in SP may also be due to assortative mating. It has been suggested that individuals have the tendency to choose a marital partner who has similar phenotypic traits, but also similar behaviours and lifestyle characteristics. Previous studies have reported significant and positive parental correlations, suggesting that assortative mating may explain similarity in SP of couples (Boomsma et al., 1989; Willemsen, Vink, & Boomsma, 2003). Assortative mating for heritable traits induce genetic relatedness between parents for the genetic factors underlying the specific SP phenotype and might inflate $h^2$ estimates when estimated in the offspring generation. Organisational-related aspects of SP, for example, frequency and time spent during the year might be more related to environmentally induced similarity (social homogamy), while the intensity of SP, related to the nature of the sport might be less.

Among siblings, all correlation values were larger than the correlations between parents–offspring. This finding suggest that there is a greater sharing of environmental correlates between siblings than between parents–offspring, or that dominant as well as age-specific additive genetic effects may be involved. In fact, all children and adolescents recruited into this study were biological offspring and reared together.

In this study, no significant differences between sex-specific and non-sex-specific parents–offspring were found for most of the SP phenotypes. On the opposite, among siblings, larger similarities were found for same-sex siblings than for the opposite-sex siblings. This finding is consistent with a previous study (Wold & Andersen, 1992) showing that girls tend to identify with and imitate the SP of their sisters, while boys appear to be more influenced by their brothers. Probably girls and boys are more sensitive to indirect and direct messages that persuade them to conform to behavioural sexual stereotypes. Lower opposite-sex similarities in SP compared to same-sex correlations might also relate to sex-specific genetic contributions, however, there was no sex-specific estimation of $h^2$ in this study, as all phenotypes were covaried for gender and age effects.

Methods for the estimation of heritability make certain simplifying assumptions, which likely are not entirely realistic and which therefore may bias the estimated effects of genes in the aggregate, and this study is no exception. We here have estimated the heritability using a variance-components approach in which we portioned the phenotypic variance only into additive autosomal genetic effects, which are shared among relatives based on their overall (autosomal) genetic similarity, and environmental effects unique to the study participants or random error (such as error in phenotype assessment). We did not model mitochondrial inheritance, X- and Y-chromosomal inheritance, co-exposure to environmental factors among related individuals (i.e. shared environment), and more complicated factors such as gene-by-environment interactions or epigenetics. It is possible that some of the actual effects of shared environmental exposures in families (such as their shared diets) may have led to elevated heritability estimates. Similarly, to the degree that genes and environment interact, some of these interaction effects may here also have been attributed to the impact of genetic variation alone. Likewise, to the degree that epigenetic factors are passed on from parents to offspring, such similarity in epigenetic profiles among relatives may also bias heritability estimates upwards. Nuclear families are not as informative for disentangling the contributions of some of these types of effects as other study designs such as extended multigenerational families, and this limitation of our study should be kept in mind and the numerical heritability estimates should not be taken as overly precise measurement. They are estimates potentially subject to substantial biases.

This study has several potential limitations. First, we used questionnaires which may yield different SP estimates compared to other studies which may have used more objective instruments. Second, the data collected via questionnaires are prone to errors – in particular, recall bias – as family members were requested to recall information about them. Third, there are limitations in sampling design because we used only nuclear families. These sampling designs, compared with large pedigree structure provides lower genetic and environmental information within families. Fourth, the heritability estimates might be
influenced by shared environmental factors because the variance component approach did not account for these factors among family members. Therefore, the reported $h^2$ estimates should be interpreted as upper-limit heritabilities. It is also important to keep in mind that heritability may be underestimated because of gene–environment interactions or over-estimated due to genotype–environment correlation induced by parental similarity (due to assortative mating).

Conclusions

In summary, present results provide evidence of a significant familial aggregation of different SP phenotypes in Portuguese nuclear families. Although heritability estimates vary between studies, these data reinforce the general consensus that a moderate proportion of the variability of SP phenotypes was explained by genetic factors (40–50%). Further studies with more extended families may help disentangle more precisely genetic and environmental effects. In addition, research with genetic linkage and candidate gene association are required to identify specific genetic variants associated with those phenotypes. Present results also demonstrated the substantial shared and non-shared environmental influence in the variability of SP (50–60%) which points out the likely success of both individual and family-based lifestyle interventions in the promotion of SP.

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References


