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Genetic Analysis of Anger: Genetic Dominance or Competitive Sibling Interaction

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The knowledge of the causes and development of anger is still scarce. Previous studies on the sources of variance on Type A Behavior Pattern (TABP) related measures found variable heritability estimates ranging from 0.12 to 0.68, and large differences between MZ and DZ correlations. Some authors considered dominance genetic effects, competitive sibling interaction and sex differences as possible mechanisms to explain the results, but most studies lacked power. The present study uses a large sample of more than 2500 families, with longitudinal data from MZ and DZ pairs as well as their parents, to disentangle the sources of variance on anger. Model Fitting results showed that the sources of variance differ across sexes. For males 23% of the variance is due to additive genetic effects, and 26% to dominance genetic effects. For females 34% of the variance is due to additive genetic effects, and no dominance effects are found. There was no consistent evidence to confirm the presence of competitive sibling interaction as an alternative explanation for the low correlations in DZ males. The focus of research on the prediction of coronary heart disease (CHD) risk through psychological characteristics has recently changed from the multidimensional TABP to its emotional component: Anger. Understanding the sources of individual differences on anger can help to clarify the mechanisms that link it with CHD and its possible implications for treatment and prevention.

KEY WORDS: Anger; dominance; family; sex differences; sibling interaction; twins; Type A behavior.

INTRODUCTION

Anger is defined by Spielberger *et al.* (1983) as an emotional state that consists of feelings of variable intensity, from mild irritation or annoyance to intense fury and rage. The study of the sources of individual differences in anger has focused mainly on the contribution of psychological factors to the development of coronary heart disease (CHD). Friedman and Rosenman (1974) identified and defined the Type A Behavior Pattern (TABP) as a pool of characteristics that increase the risk of CHD. As a multidimensional construct, TABP comprises physical components, motivational and cognitive aspects, behavioral tendencies, attitudes and emotions; including loud voice, facial muscle tension, hostility, anger, aggressiveness, achievement motivation, competitiveness, alertness, work involvement, or necessity of environmental control. A number of studies showed a significant relationship between TABP and CHD (e.g. Matthews and Jennings, 1984; Obrist, 1976; Rosenman *et al.*, 1964; Zyzanski *et al.*, 1979). However it soon appeared that the findings were inconsistent and unstable and that TABP, as it was originally defined, could not be considered a reliable predictor of CHD (Matthews, 1988). Subsequent research has shown that, among the multiple elements encompassed in the TABP, only the emotional and attitudinal components such as anger, hostility and aggressiveness ('the AHA syndrome') contribute to

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the prediction of CHD incidence; and so the focus of research has changed from TABP to hostility, and to anger (Dembroski *et al*., 1989; Matthews, 1988; Palmero *et al*., 2001; Siegman and Smith, 1994).

Anger has been related to several phenomena in behavioral medicine and psychological research. High levels of trait anger and internal expression have been associated with increases in blood pressure and induced hypertension (Crane, 1981; Markovitz *et al*., 1991; Schneider *et al*., 1996). Some studies show positive correlations between external expression of anger and cardiovascular reactivity in irritated patients (Engebretson and Matthews, 1989; Siegman, 1994). Furthermore, high levels of anger have been found to be good predictors of risk to coronary disease is several studies (Atchison and Condon, 1993; Bishop and Quah, 1998; Chang *et al*., 2002; Eaker *et al*., 2004; Julkunen *et al*., 1994; Kawachi *et al*., 1996; Mendes de Leon, 1992; Williams *et al*., 2000). Others have found a positive relationship between the anger trait and anger held in and chronic symptoms suffered by patients of posttraumatic stress (Lasko *et al*., 1994; Tschannen *et al*., 1992). High levels of anger have also been associated with psychological disorders like anorexia and bulimia nervosa (Fassino *et al*., 2001), or borderline personality disorder (Nothmann, 1999). Finally, different patterns of anger expression have been studied in relation to socially relevant issues like criminal personality (Slaton *et al*., 2000), sexual offense (Dalton *et al*., 1998), aggressive behavior in adolescents (Peters, 1998), drug addiction (De Moja and Spielberger, 1997) or marital maltreatment (Barbour *et al*., 1998).

The present study is intended to explore the extent to which environmental and genetic factors underlie variation in trait anger. Trait anger is conceptualized as the frequency with which an individual experiences the emotional state of anger over time and in response to a variety of situations (Eckhardt *et al*., 2004). Sluyter *et al*. (2000) studied the genetics of trait anger as a component of the AHA syndrome, and its relation with testosterone. Additive genetic effects explained 25% of the variance of anger, all common to other measured traits such as TABP, irritability or hostility. Among the nine personality traits considered by the authors in an independent pathway model to define the AHA syndrome, trait anger and indirect hostility showed the lowest heritability estimates (respectively 0.25, CI = 0.07, 0.50 for anger and 0.23 CI = 0.04, 0.46 for hostility). Both anger and hostility showed quite low DZ correlations (0.07 and −0.03). Non-additive genetic effects were not included in the model, in spite of its possible relevance suggested by the large difference between the MZ and DZ correlations.

Carmelli *et al*. (1988) considered thirteen different variables in a study of the genetic and environmental influences on TABP. They found low DZ correlations ranging from −0.09 to 0.32. Only 4 out of 13 DZ correlations were significantly different from zero. Four were measures of anger (anger-in, anger-discuss, anger-symptoms and Framingham anger), among which only anger-symptoms showed a DZ correlation significantly different from zero. The heritability estimate for anger was 0.36, but no distinction was made between additive and non-additive genetic effects.

Other studies about the sources of variance on TABP obtained similar results (Duffy *et al*., 1994; Finkel *et al*., 1998; Koskenvuo *et al*., 1981; Matthews and Krantz, 1976; Meininger et al., 1988; Pedersen *et al*., 1989; Rahe *et al*., 1978), with DZ correlations close to zero and heritability estimates in the order of 0.40 (ranging from 0.12 to 0.68). The heritability was larger for younger samples (Koskenvuo *et al*., 1981; Meininger *et al*., 1988).

Loehlin (1986) noticed the same pattern of low DZ correlations for the Thurstone Temperament Schedule. Loehlin proposed three mechanisms that could produce DZ correlations markedly lower than half the MZ correlations (Loehlin, 1986, p. 66):

First, *MZ twin environments may be relatively more similar than DZ twin environments*. This could happen through a gene–environment correlation process, but that would appear in genetic models as genetic variance, and would still predict larger DZ correlations. Other authors have suggested that the DZ environment could be less similar than the MZ environment beyond the genetic indirect effects, suggesting some violation of the equal environments assumption (EEA) (Meininger *et al*., 1988; Rahe *et al*., 1978). Some studies have found that sociodemographic factors like education, occupation, health behaviour, or social support are significantly related with TABP related measures (Carmelli *et al*., 1988; Koskenvuo *et al*., 1981; Raynor *et al*., 2002). They also find that MZ twins tend to be more similar in those variables than DZ twins. Nevertheless, partial correlations between those sociodemographic variables and twin resemblance for TABP related traits, controlling for zygosity, tend to be small and non-significant (Raynor *et al*., 2002). Furthermore, adjusting the heritability estimates by those covariates, barely changes the results (Carmelli *et al*., 1988).

Secondly, the presence of genetic dominance and epistasis could also account for DZ correlations lower than half the MZ correlations. Most of the above studies did not test for the presence of non-additive genetic effects. Three studies (Duffy et al., 1994; Sims *et al*., 1991; Tambs *et al*., 1992) found non-significant dominance genetic effects, and one (Pedersen et al., 1989) found that actually most of the genetic variance was non-additive. It must be noted that detecting dominance requires large samples, and preferably including pairs of varying genetic relatedness—e.g. twins reared together and twins reared apart, full siblings, half siblings, and step-siblings-(Posthuma and Boomsma, 2000; Rietveld *et al*., 2003). Only the study by Pedersen and colleagues fulfilled some of those conditions.

Secondly, *sibling interaction or contrast effects* occur when a high-scoring sibling influences the behaviour of the other inhibiting his development, and thus incrementing the within pair difference for a given trait (Eaves, 1976). The within pair difference can also be increased when the twins exaggerate their differences using each other as a reference to define themselves. Two studies have found significant sibling interaction effects on TABP measures (Duffy *et al*., 1994; Sims *et al*., 1991). However, in order to reliably detect a relatively large interaction effect (−0.20) with the classic twin design, at least 300 twin pairs are necessary (Rietveld *et al*., 2003). Neither Duffy's or Sims' studies fulfilled this condition and thus, in their results, additive and dominance genetic effects and, dominance and sibling interaction may be confounded. To increase the power of the study, Sims *et al*. (1991) included parent–offspring data, where the sibling interaction parameters became small and non-significant. In this vein, other authors have suggested that if competitive sibling interaction were to explain the low DZ correlations, then DZ twin pairs reared apart should be more similar than DZ twin pairs reared together, as they do not interact or compare to each other. But in most cases, correlations for DZ twins reared apart are equal or lower than correlations for DZ reared together (Pedersen *et al*., 1989; Tellegen *et al*., 1988).

The present study is intended to disentangle the sources of variance of anger trait as a relevant component of the TABP. As other authors have already suggested, the scope of the research on TABP as a multidimensional concept must be better directed to the study of each of its components separately for clearer results and understanding of the mechanisms linking personality and CHD (Eysenck and Fulker, 1983; Palmero *et al*., 2001). Some of the limitations of the previous studies are surpassed by the study design and methodology applied in the current paper: a large sample, a repeated measures design and the inclusion of data on parents of twins increase the power to detect stable and replicable effects. The combination of twin and parental data increases the power to distinguish between additive and dominance genetic effects, and between dominance and sibling interaction effects.

The focus of this article will be on the clarification of previous contradictory results concerning the presence of dominance genetic effects and/or competitive sibling interaction, as well as sex differences on the sources of individual differences in anger, as a relevant component of TABP. For that purpose, longitudinal data will be used as an instrument of replication, and all parameters will be simultaneously estimated using the data from the two surveys. Based on the results of previous studies it is expected that additive genetic effects explain a significant portion of the variance, and that either sibling interaction or dominance genetic effects account for the large difference between MZ and DZ twin correlations.

METHOD

Participants and Procedure

Participants were registered by the Netherlands Twin Registry (NTR), kept by the Department of Biological Psychology at the Vrije Universiteit in Amsterdam. They are part of the adolescent and adult cohort that was recruited through the city councils in 1990–1991 and in 1992–1993. They participate in longitudinal survey studies roughly every 2 years. The data analyzed here were collected in the 1991 and 1993 surveys. The questionnaires were sent to the families and returned by mail (Boomsma *et al*., 2002).

The complete sample consists of 2664 families for which data on the phenotypes of the twins and their parents were collected: 438 MZM (Monozygotic Males), 401 DZM (Dizygotic Males), 612 MZF (Monozygotic Females), 454 DZF (Dizygotic Females), 759 DOS (Dizygotic Opposite sex). 750 complete families (both twins and both parents) participated both in 1991 and 1993, 514 did so only in 1991, and 764 participated only in 1993. For the remaining families, some data were missing randomly e.g. only one twin participated, data for one twin is missing in 1 year, the mother but not the father participated (the randomly incomplete families comprise

5% of the twins and 20% of the parents). Complete and incomplete families were used in the analyses.

The mean age of the twins was 17.68 in 1991 (SD = 2.23, range = 12–25 years) and 17.76 in 1993 (SD = 3.06, range = 12–25 years). The mean ages of fathers and mothers were 47.77 (SD = 5.62, range = 35–71 years) and 45.64 (SD = 5.15, range = 33–63 years) respectively in 1991, and 48.07 (SD = 5.52, range = 29–73 years) and 45.95 (SD = 5.09, range = 32–63 years) respectively in 1993. The SES distribution for this sample is 22.1% of low, 43.8% of middle and 34.1% of high SES (Boomsma et al., 2002). The religious background of the families is comparable to the Dutch population (Boomsma *et al.*, 1999).

The similarity in age in both surveys is due to those families who participated in either 1991 or 1993 but not in both. Appendix A summarizes the information about the age of parents and twins by survey participation. The table shows a tendency for the families that joined in 1993 for the first time to be a bit younger compared to those who where participating for the second time.

Zygosity for 726 same sex pairs was based on DNA polymorphisms, and for the remaining pairs zygosity was assigned by discrimination analysis using questionnaire items (see Boomsma et al., 2002 for further details). The correspondence between DNA and questionnaire based zygosity was 97%.

Measures

The Dutch adaptation of Spielberger's State-Trait Anger Scale (STAS) (Spielberger *et al.*, 1983; van der Ploeg *et al.*, 1982) was used to measure anger trait. The Trait Anger scale is designed to assess the frequency an individual experiences state anger over time and in response to a variety of situations. Reliability measured by the alpha coefficient was 0.86. The STAS is considered a strong measure of anger, based on a solid theoretical model, with excellent psychometric properties across several normative groups. It has shown good discriminant and convergent validity, as well as clinical utility, and it has been administered across a wide range of subject populations and psychological domains (Eckhardt *et al.*, 2004).

ANALYSES

Genetic analyses were conducted using structural equation modelling (SEM) as it permits the simultaneous analysis of multiple groups, and the possibility of imposing parameter constraints across groups. The statistical software package Mx was used (Neale *et al.*, 2003). Full information Maximum Likelihood estimation (FIML) was used to fit the models. Twice the negative log-likelihood (−2LL) of the data for each observation is calculated, and parameter estimates are produced that maximize the likelihood of the raw data. Submodels were compared using a likelihood ratio test computed by subtracting −2LL for the restricted nested model from that for the baseline model ($\chi^2 = (-2LL_0) - (-2LL_1)$). The resulting test statistic has a χ^2-distribution with degrees of freedom equal to the difference of the degrees of freedom between the two models. The fit of the genetic models is evaluated against the fit of a saturated model, were the covariance matrix and the mean structures are computed without any restriction. Besides the χ^2 test statistic, the Akaike's information criterion (AIC) and the root mean squared error of approximation (RMSEA) are used to evaluate the general fit of the models (the difference against the saturated model). The AIC compares the models on the basis of parsimony, taking jointly into account the χ^2 and the degrees of freedom (Jöreskog, 1993). The lower the AIC, the better the fit of the model to the data and the more parsimonious it is. The likelihood ratio test is performed under the assumption that the model holds exactly in the population (Loehlin, 2004). As a consequence, models that hold approximately in the population will always be rejected in large samples. The RMSEA is a measure of closeness of fit, and provides a measure of discrepancy per degree of freedom. A value of 0.05 indicates a close fit, and values up to 0.08 represent reasonable errors of approximation in the population (Jöreskog, 1993). Besides the estimation of the covariance structure, the mean structure is included as well in all models.

An alpha level of 0.01 was chosen. Given the power provided by a large sample size; this conservative criterion prevents interpreting slight differences between the models as relevant effects.

According to Neale and Cardon (1992), pooling across sexes is inappropriate unless it is known that there are no sex differences in means, variances or twin pair covariances. For this reason, zygosity groups will be separated by sex and possible sex differences are checked in a saturated model. Furthermore, age is considered as a covariate in the model for the means, with different *regression coefficients* for the parental and offspring generations. This way, age

effects on anger are regressed out, and the covariance structure is fitted on the residuals.

RESULTS

The saturated model

First, a saturated model is fitted in which variances, covariances and means are estimated without any constraint. The likelihood associated with the unconstrained saturated model was −2LL(DF) = 78542.056 (13271). The estimation of a saturated model also allows for a thoughtful exploration of the descriptive data through a progressive imposition of constraints on the means and variances, testing the effect on the likelihood of the model through the χ^2 statistic. The results of the saturated model guide the selection of the most appropriate genetic model to be fitted.

Constraints on the mean structure showed that there are significant differences in the means across zygosity groups, time point, and parents and offspring. Differences across zygosity groups are not conspicuous—all the means of the twin sample were within the range of 22.65–27.36—and did not show any clear pattern. There is a slight tendency for the means to be smaller in 1993 than in 1991, but still the largest difference is 0.92. However, there is a remarkable difference between parents and offspring where the former score lower (within the range of 13.97–17.14) than the latter. These differences were taken into account in the mean part. The genetic models include different means for 1991 and 1993, across zygosity groups, and for parents and offspring. The saturated model against which the genetic models are compared includes these restrictions, so that the mean structure does not affect the selection of the models.

The age regression effects on the means were significantly different from zero for twins ($\chi^2 = 53.46$, $\Delta DF = 1$, $p < 0.001$) and parents ($\chi^2 = 21.82$, $\Delta DF = 1$, $p < 0.001$). The unstandardized age effect is −0.19 for the twins and 0.05 for the parents. Standardizing the effects using the SD of anger and age in both groups, age explains 8% of the variance of anger in the offspring generation and 6% of the variance on the parental generation. The direction of the effects appears to change so that during adolescence and young adulthood anger tends to decrease with age, while during the adult years there appears to be a slight increase of anger with aging.

Differences of variance were found across zygosity groups ($\chi^2 = 47.38$, $\Delta DF = 16$, $p < 0.001$). There is a slight tendency for the same sex DZ twins to show the largest values, followed by the OS twins. MZ pairs show the smallest values. If the variances are constrained separately for males and females, the differences only remain significant in the male sample. Further constraints show that the differences are due to two specific comparisons: DZM in 1991 show a larger variance than OSM, and DZM and OSM in 1993 show a larger variance than MZM. Taking a closer look at the variances, it is clear that the three differences are due to two isolated low variances in the data when compared to the variances of the complete sample (24.59 in 1991 and 23.43 in 1993): that of first born MZM in 1993 (19.37), and that of OS second born in 1991 (16.82). Differences in the variance across zygosity groups can be an indicator of sibling interaction effects (Rietveld *et al.*, 2003), but the differences must show a consistent pattern of larger variances for DZ twins compared to the MZ twins, whereas the present results do not appear to indicate such consistent pattern.

There were also significant differences in the variance between males and females ($\chi^2 = 42.08$, $\Delta DF = 12$, $p < 0.001$). These differences were due to those comparisons that involved the two outlier variances (MZM93 vs. MZF93, and OSM91 vs. OSF91).

There is a significant difference in the variance between parents and offspring ($\chi^2 = 133.10$, $\Delta DF = 12$, $p < 0.001$). The parental generation shows a smaller variance (21.88 in 1991 and 19.46 in 1993) than the offspring generation (26.96 in 1991 and 26.63 in 1993). This difference was incorporated in the genetic models by a scalar parameter (see Fig. 1).

Comparisons between first and second born twins yielded 3 out of 12 significant differences for MZM93—1st born larger than 2nd—DZF91 and OSMF91—2nd born larger than 1st—. There was not a clear or consistent pattern, and this small number of differences is consistent with chance fluctuation.

The saturated model against which the genetic models are tested, that comprises the restrictions on the means, and includes two age regression parameters shows the following fit: −2LL(DF) = 78574.99(13285).

Based on the constrained saturated model, Mx was used to estimate the familial correlations (and confidence intervals) shown in Table I.

The MZ correlations are more than twice the DZ correlations. Only one DZ correlation, the one for OSMF in 1993, is larger than zero ($p < 0.01$). This

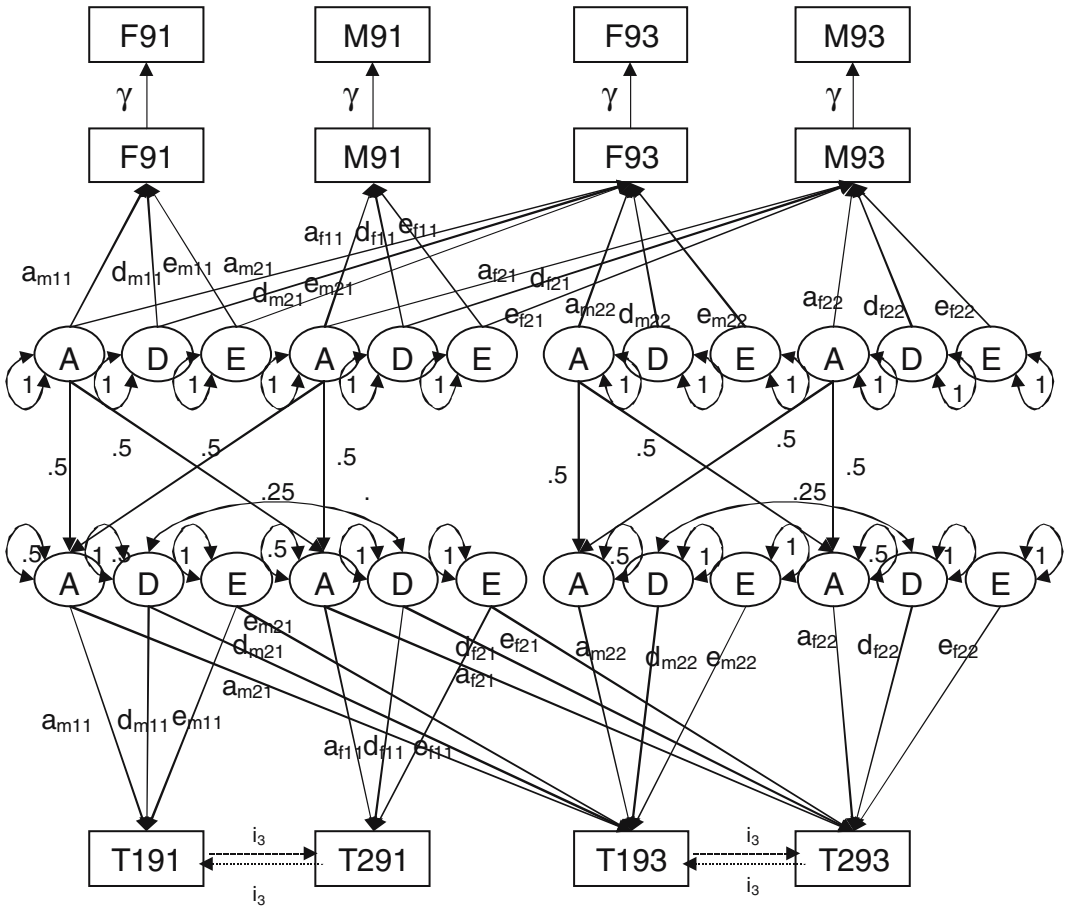

Fig. 1. Parent–offspring genetic ADEi model. The figure represents an opposite sex DZ pair where the first born is a male and the second born is a female. T1—first born, T2—second born, M—mother, F—father. 91 and 93 indicate the surveys from 1991 and 1993.γ-represents the scalar parameter to account for the difference in variance in the parental generation. Path coefficients with the subscript m are those for males, and the subscript f is for female parameters. The arrows connecting the twins represent the sibling interaction parameter, i_3 in the diagram is the sibling interaction for opposite sex twin pairs; i_1 would be the interaction parameter for males, and i_2 for females.

Table I. Twin, Parent–Offspring and Spouse Correlations for Anger, within Time in 1991 and 1993 and across Time

	1991	1993	Cross time
MZM	0.452(0.315, 0.571)	0.451(0.321, 0.563)	0.324(0.140, 0.476)
DZM	0.139(−0.021, 0.294)	0.003(−0.115, 0.157)	0.076(−0.125, 0.269)
MZF	0.400(0.284, 0.504)	0.417(0.313, 0.434)	0.356(0.210, 0.483)
DZF	0.063(−0.082, 0.206)	0.132(−0.009, 0.268)	0.072(−0.093, 0.233)
OS	0.145(0.030, 0.255)	0.124(0.015, 0.231)	0.027(−0.116, 0.170)
Father–son*	0.148(0.071, 0.224)	0.111(0.039, 0.181)	0.164(0.079, 0.245)
Father–daughter	0.175(0.108, 0.240)	0.114(0.047, 0.178)	0.172(0.101, 0.240)
Mother–son	0.152(0.079, 0.223)	0.084(0.016, 0.151)	0.103(0.018, 0.185)
Mother–daughter	0.150(0.085, 0.213)	0.159(0.098, 0.219)	0.146(0.074, 0.146)
Spouses	0.058(−0.008, 0.126)	0.078(0.078, 0.141)	0.055(−0.020, 0.129)

*Parent offspring and spouse correlations constrained to be equal across zygosity groups.
Confidence intervals are shown between brackets.

result is consistent with previous studies and it can be explained by dominance genetic effects and/or competitive sibling interaction. These possibilities are tested in the genetic analysis. Furthermore, the parent–offspring correlations show a very consistent picture with correlations around 0.150 that point to the presence of additive genetic effects. The low spouse correlations indicate that there is not assortative mating for trait anger. The large difference between MZ and DZ cross time correlations imply that the stability of trait anger across years is probably due to genetic factors, with dominance genetic effect among them. However, it must be noted that both MZ and DZ correlations across time are lower than those within time; whereas the parent–offspring correlations are very similar within and across time. This shows that the cross time correlations of the twin pairs are also affected by the lower stability of the trait during adolescence.

Means, standard deviations and stability coefficients for the parental and offspring samples are summarized in Table II.

Genetic Analyses

The path diagram in Figure 1 represents the general genetic model that is being tested.

The diagram represents the model for an opposite sex twin pair and their parents where the first born twin is a male and the second born twin is a female. This is an ADE model where the variance of anger is assumed to be explained by additive genetic factors, dominance genetic factors and environmental factors not shared by the members of the same family. At first, different parameters are estimated for males and females. The latent factors placed above the phenotypes from 1991 are those sources of variance common to 1991 and 1993 or, in other words, the stable sources of variance. The latent factors placed above the phenotypes from 1993 represent the sources of variance specific to 1993 that were not present in 1991. The phenotype in 1991 is explained by the influence of the common factor, and the phenotype in 1993 is due to the sum of the influences of the common factor and the specific 'novelty' factor. i.e. the variance of anger for T191 is decomposed as follows $S^2_{T191} = a^2_{11} + d^2_{11} + e^2_{11}$, while the variance of anger for T193 is partitioned as $S^2_{T193} = a^2_{21} + a^2_{22} + d^2_{21} + d^2_{22} + e^2_{21} + e^2_{22}$.

Table II. Descriptives. Mean and Standard Deviations within Time in 1991 and 1993 for Anger and Age

	Twins		Parents	
	Mean	SD	Mean	SD
Anger				
1991	16.87	5.19	16.49	4.67
1993	17.08	5.20	16.19	4.41
Cross time correlation	0.573		0.671	
Age				
1991	17.69	2.24	46.68	5.49
1993	17.76	3.06	46.98	5.42

The cross time correlation or stability coefficient is also shown of anger.

It is assumed that the amount of variance explained by each component is proportional in the parental and offspring generations. The parameter γ is placed in the model to account for the difference of variance between them observed in the saturated model. Resemblance between parents and offspring is explained by the additive genetic variance that they share. Each parent shares with each twin 50% of the additive genetic variance. Dominance genetic effects are those due to the interaction or combination of alleles at a particular locus. Offspring receive only one allele from each parent, not a combination of two alleles (Plomin *et al.*, 2001). For that reason dominance is not transmitted from parents and offspring and thus, there is not a D path from parents to offspring.

Given that the DZ correlations are less than twice the MZ correlations, and that most of them are not significantly different from zero, the shared environment is not included in the model.

DZ twins resemble each other because they share 50% of there genetic variance, inherited from their parents. They also share 25% of the dominant genetic variance. MZ twins share the totality of both the additive and the dominant genetic variances.

The phenotypes of the twins are connected through reciprocal paths. Those paths and their corresponding parameters represent the direct phenotypic effects that the twins have on each other, and thus the sibling interaction effects. It is assumed that the amount of influence that they exert on each other is equal, but different interaction parameters are estimated for same sex male twins (i_1), same sex female twins (i_2), and opposite sex twins (i_3). Further details about the derivation of the expected variances and covariances and the effects of the presence of sibling interaction on the model expectations can be found in Neale and Cardon (1992) and Eaves (1976).

Two constraints are imposed in the model: (1) Sibling interaction effects are constrained to be equal in 1991 and 1993; and (2) the total amount of variance explained by each component A, D, and E is also constrained to be equal in 1991 and 1993 (i.e. $a_{11}^2 = a_{21}^2 + a_{22}^2$). This constraint ensures that the estimates are stable and replicable effects. The 750 families that participated two times provide the information relative to stability. Those families that participated once in 1991 or in 1993 provide information relative to replication across samples.

Table III shows the results of the model fitting sequence. The general model shows an excellent fit according to the negative AIC and the RMSEA below 0.05. Departing from the general model, model 2 is fitted to test for sex differences in the variance components of anger, constraining the male's and female's parameters to be equal (i.e. $a_{m11} = a_{f11}; a_{m21} = a_{f21}; a_{m22} = a_{f22}$ for additive genetic effects, and likewise for the dominance and non-shared environmental factors). There was a significant decrease of fit as a consequence of the constraint and thus, it cannot be assumed that the same proportion of variance is explained by each component in males and females.

Models 3 and 4 test for sex differences in the sibling interaction parameter, first equating it for same sex male and female pairs, and subsequently equating them to the opposite sex pairs. Both models fit the data as well as the general model, so it can be assumed that there are not significant differences in the amount of sibling interaction effects across sexes.

Models 5–8 are intended to test for the significance of certain parameters. In model 5, all dominance genetic effects for males are fixed to zero (i.e. $d_{m11} = d_{m21} = d_{m22} = 0$), and model 6 does the same with the female's dominance components. Table III shows the χ^2 change produced in models 5 and 6 with respect to the general model. Model 5 suffers a significant decrease of fit, while model 6 can be considered as good as the general model. Thus, dominance genetic effects are necessary to explain the variance of anger for males, but not for females.

Under the label of model 7, departing from model 4, where a single sibling interaction parameter was estimated for all pairs, the sibling interaction effects are fixed to zero ($i_1 = i_2 = i_3 = 0$). The change of fit compared to model 4 is not significant ($p > 0.01$) indicating that the constraint can be held and thus, the sibling interaction effects are zero in the population.

Model 8 constrains the scalar 'γ' to one. Such a model implies the assumption that the parental and

Table III. Model Fitting Results

MODEL	−2LL	DF	CT[a]	χ^2	df	*p*	χ^2, DF (vs. SAT)[b]	AIC	RMSEA
1/General model	78,938.80	13,485					363.81, 200	−36.18	0.018 (0.014, 0.021)
Tests of sex differences: parameters are constrained to be equal across males and females within zygosity groups									
2/1 + ADE♂ = ADE♀	78,966.61	13,494	1	27.809	9	0.001			
3/1 + i♂♂ = i♀♀	78,942.62	13,486	1	3.817	1	0.051			
4/1 + i♂♂ = i♀♀ = i♂♀	78,945.22	13,487	3	2.6	1	0.106			
Significance tests: parameters are constrained to be zero (the scalar is constrained to equal 1)									
5/1 + D♂ = 0	78,974.66	13,488	1	35.85	3	0.000			
6/1 + D♀ = 0	78,943.46	13,488	1	4.65	3	0.199			
7/4 + i = 0	78,950.39	13,488	4	5.17	1	0.023			
8/1 + scalar = 1	79,116.57	13,486	1	177.72	1	0.000			
Specific effects in 1993: Novelty effects only present in 1993 are constrained to be zero									
9/1 + d_{22} = 0 ♂ and ♀	78,957.62	13,487	1	18.81	2	0.000			
10/9 + a_{22} = 0 ♂ and ♀	78,970.50	13,489	1	31.70	4	0.000			
Combination of Constraints: parameter constraints which did not produce a deterioration of the fit are put together progressively to obtain a final model									
1/General model	78,938.80	13,485					363.81,200	−36.18	0.018 (0.014, 0.021)
2/1 + i♂♂ = i♀♀	78,942.62	13,486	1	3.816	1	0.051	367.63,201	−34.37	0.018(0.014, 0.021)
3/2 + i♂♂ = i♀♀ = i♂♀	78,946.22	13,487	2	3.597	1	0.057	371.23,202	−32.77	0.018 (0.014, 0.021)
4/3 + i = 0	78,950.39	13488	3	4.170	1	0.041	375.40,203	−30.60	0.018 (0.014, 0.021)
5/ 4 + D♀ = 0	78,955.62	13491	4	5.23	3	0.156	380.63,206	−31.37	0.018 (0.014, 0.021)

[a]CT = compared to model #.

[b]Chi-squared and degrees of freedom of the model compared to the saturated model. Indicates the goodness of fit of the model.

the offspring generations have the same variance. The large decrease in the goodness of fit indicates that the difference in variability between generations is not negligible, and must be included in the model.

Models 9 and 10 are intended to find out whether there are novel genetic effects active in the second survey in 1993 that were not present during the first survey in 1991 ($d_{22}=0$ and $a_{22}=0$). The results show that neither dominance nor additive genetic effects specific to 1993 can be dropped from the model.

Finally, a series of models are fitted accumulating the previous results with the purpose of finding the most parsimonious explanation of the data. Each of these models is compared to the saturated model to obtain general indices of goodness of fit, as well as to the immediately previous model. Table IV shows the parameter estimates of these five models.

Model 1 is the general model. Models 2 and 3 constrain the sibling interaction effects across sexes. In model 4 the sibling interaction effects are removed. In model 5 dominance genetic effects are removed for the female sample. All five models show a satisfactory fit to the data with negative AIC and an RMSEA lower than 0.05. According to the χ^2 comparisons with the immediately previous models, none of the progressive constraints produce a significant decrease of fit ($p>0.01$). Choosing among the 5 models following strict statistical criteria would lead to the selection of the general model as the best explanation of the data, as it shows the lowest AIC. But the differences between the models are so slight that the RMSEA does not even change from one to another. It can also be observed in Table IV how the parameter estimates for the variance components and the partitioning of the cross time correlation barely change from one model to another (they all fall within the 95% confidence interval of the last model). Clearly the most controversial part of the model fitting sequence in terms of goodness of fit and parameter estimates is the sibling interaction, in line with previous literature (e.g. Sims *et al.*, 1991). It is clear from models 2 and 3 that the effects can be equated across sexes. The

Table IV. Parameter Estimates for Models 1–5, Where Relevant Constraints are Progressively Accumulated

Model		1	2	3	4	5
Variance decomposition males	A91	0.26	0.25	0.25	0.24	0.23(0.145, 0.324)
	A93	0.26	0.25	0.25	0.24	0.23(0.145, 0.324)
	D91	0.34	0.31	0.29	0.25	0.26(0.126, 0.377)
	D93	0.34	0.31	0.29	0.25	0.26(0.126, 0.377)
	E91	0.39	0.44	0.46	0.51	0.51(0.429, 0.613)
	E93	0.39	0.44	0.46	0.51	0.51(0.429, 0.613)
Variance decomposition females	A91	0.29	0.30	0.30	0.29	0.34(0.279, 0.406)
	A93	0.29	0.30	0.30	0.29	0.34(0.279, 0.406)
	D91	0.11	0.15	0.13	0.09	–
	D93	0.11	0.15	0.13	0.09	–
	E91	0.59	0.54	0.57	0.62	0.66(0.594, 0.720)
	E93	0.59	0.54	0.57	0.62	0.66(0.594, 0.720)
Cross time correlation decomposition	A♂	0.40	0.38	0.39	0.37	0.35(0.225, 0.499)
	A♀	0.40	0.41	0.40	0.40	0.47(0.363, 0.499)
	D♂	0.31	0.29	0.26	0.24	0.26(0.032, 0.463)
	D♀	0.14	0.18	0.16	0.12	–
	E♂	0.29	0.33	0.35	0.39	0.39(0.227, 0.566)
	E♀	0.45	0.41	0.43	0.48	0.53(0.422, 0.636)
Genetic and environmental correlations across time	A♂	0.97	0.98	0.99	0.98	0.98
	A♀	0.87	0.86	0.87	0.88	0.88
	D♂	0.58	0.59	0.59	0.62	0.65
	D♀	0.82	0.79	0.80	0.89	–
	E♂	0.47	0.47	0.48	0.48	0.48
	E♀	0.49	0.48	0.49	0.50	0.51
Sibling interaction	Males	−0.08	−0.05	−0.03	–	–
	Females	−0.02	−0.05	−0.03	–	–
	Opp sex	−0.00	−0.01	−0.03	–	–
		0.85	0.85	0.85	0.85	0.85

Model 5 is the final selected model and includes confidence intervals between brackets.

overall estimate of the sibling interaction effects in model 3 is −0.03. Following a strict statistical argument based on the p value ($p > 0.01$), sibling interaction effects should be removed from the model. Furthermore, previous analysis showed that, one by one the interaction effects are estimated as: $i♂♂91 = -0.03$ ($p = 0.332$), $i♂♂93 = -0.12$ ($p < 0.001$), $i♀♀91 = -0.04$ ($p = 0.155$), $i♀♀93 = 0.01$ ($p = 0.649$), $i♂♀91 = 0.02$ ($p = 0.301$), $i♂♀93 = 0.03$ ($p = 0.226$). Only the effect for males in 1993 is significantly different from zero. That might be a spurious estimate due to the low MZM first born 1993 variance. When the effects are combined across the 1991 and 1993 samples, the effect does not differ significantly from zero. There is no theoretical reason to support an interaction effect that is observed exclusively in 1993, but not in 1991, especially given that the mean age of both samples is the same. The effect is neither stable (in those pairs who participated two times) nor replicable (in those pairs who participated once).

Model 5 is selected as the best and most parsimonious explanation of the data. The broad heritability of anger is 0.49 for males and 0.34 for females. About half of the genetic variance in males is due to dominance interaction, whereas no dominant effects are found in the female population. The decomposition of the cross time correlations shows that stability across time is due, 61% to genetic effects, and 39% to non-shared environmental effects for males, while for females genetic effects explain 47% and non-shared environmental effects explain 53% of the stability. The genetic correlation shows that additive effects on anger are very stable, as 88% for females, and 98% for males of the genes that explain the variance in 1991 are still effective in 1993. The dominance genetic correlation is a bit lower (0.65 for males), as well as the environmental correlation (0.48 for males, 0.51 for females), which implies that although a large part of the dominance genetic and environmental effects present in 1991 are also present in 1993, a great deal of new dominance genetic effects and environmental sources of variance become relevant 2 years later.

DISCUSSION

The present study has explored the genetic and environmental sources of variance on anger through a powerful design. It includes a large sample of families of MZ and DZ twins and their parents, and the repeated measurement of the trait, leading to a clarification of previous contradictory results. A review of previous studies revealed that the finding of low DZ correlations compared to the MZ correlations has been a common factor in the field. The explanations provided for this phenomenon vary from study to study. Loehlin (1986) proposed three possible mechanisms to explain such pattern of results. Non-additive genetic effects and competitive sibling interaction are two of the candidate explanations that have been considered and tested in this article. The large sample size and the availability of parental data make this study a unique opportunity to detect and distinguish between those two mechanisms.

The results show that the sources of variance on anger differ across sexes. For males 23% of the variance is due to additive genetic effects and 26% to dominance genetic effects. For females 34% of the variance is due to additive genetic effects, and the remaining is explained by non-shared environmental influences. There was no consistent evidence to confirm the presence of competitive sibling interaction effects. The estimation of the broad heritability lies within the range of previous studies. Variations in the precise estimates might be due to the failure of most studies to consider sex differences and/or and dominance genetic effects (i.e. Carmelli *et al*., 1988; Koskenvuo *et al*., 1981; Meininger *et al*., 1988; Pedersen *et al*., 1989; Rahe *et al*., 1978; Sims *et al*., 1991; Tambs *et al*., 1992). The main problem of previous studies is the lack of power. Detecting and distinguishing between dominance and sibling interaction effects, while testing for sex differences, requires large samples and, preferably different kinds of relatives (Rietveld *et al*., 2003).

In the present study genetic variance explains 15% more variance of anger in males than in females. The study of sex differences in the causes of personality traits that increase the risk to CHD might be relevant and informative, as the risk to suffer coronary problems is larger in males than in females (Dolan *et al*., 1992). A lower heritability in females is consistent with previous studies on genetic markers where serotonin gene polymorphisms have been found to be related with anger on males, but not in females (Manuck *et al*., 1999). These results could imply different prevention and therapeutic programs for males and females, with the former more focused on biological genetic related sources of variance, and the former more directed to environmental factors e.g. life style.

Although the present study has helped to clarify some issues, there are still some characteristics of the data that need to be explained. Observing the summary correlations under the light of the model fitting results can raise some questions. The presence of dominance genetic variance in males is mostly expressed in larger MZ correlations in males than in females. That increases the MZ/DZ ratio and suggests dominance effects. But there is still the fact that most DZ correlations are not significantly different from zero, and that parent–offspring correlations are in the order of the DZ correlations, pointing to the need of other explanations for the large MZ correlations or low DZ correlations besides dominance genetic effects. The present study has raised serious doubts over the competitive sibling interaction hypothesis. Reed *et al*. (1991) studied the effects of placentation on a set of Type A personality measures. The authors found greater similarity in monochorionic pairs than in dichorionic pairs, so that correlations in MZ dichorionic pairs were similar to the DZ correlations, less than half the MZ monochorionic correlations. Reed *et al*. interpret this result as a special violation of the EEA. Future studies could differentiate between monochorionic and dichorionic MZ pairs, and take that into account into the genetic models. In that vein, Loehlin (1986), modelled a MZ specific latent factor that improved significantly the fit of the genetic model. Loehlin interpreted the factor as either configurational genetic effects and/or shared environments specific to MZ twins. In a study where data on chorionicity are available, Loehlin's MZ factor could be included for the monochorionic MZ pairs, but not for the dichorionic.

The main limitation of the present study to generalize the results might be the age of the twins. During adolescence and young adulthood personality traits are still quite unstable and more sensitive to changes in the non-shared environment (Caspi and Roberts, 2001; Reiss *et al*., 2000). Such instability could be responsible for the unclear pattern of variances. During adulthood, between 30 and 50 years of age personality stabilizes and the effects of genes gain importance. However, the inclusion of parental data in the sample has helped to reach a strong conclusion regarding the effect of additive genetic effects, as the parent–offspring correlations showed a very consistent picture. Furthermore, it can be observed in Table A.1 of the Appendix A, that among the complete pairs, there are 2549 twin pairs from 12 to 24 years old, and 2108 parental couples from 33 up to 73 years old. Thus, the adult generation comprises close to half of the sample, and the variance components can be assumed to be equal between the two generations, after a difference in variance is accounted for by a scalar. Future longitudinal studies that cover adolescence and adulthood will advance the understanding of dominance genetic effects and their stability thorough the life span, and distinguish between developmental and generational changes in the variance architecture. With that purpose, we are planning to collect data on anger within the 7th survey of the NTR.

The current tendency to move from the molar idea of TABP to a more elementary level will also help to disentangle the sources of variance and mechanisms that increase the risk of suffering CHD. The present study has shown that the use of large samples and family designs facilitates the reliable detection of important factors like sex differences or dominance genetic effects, and rule out negligible factors like sibling interaction, otherwise detected by weaker designs. To explore the nature of the TABP, several studies like this that consider different molecular characteristics from personality variables to biological endophenotypes or environmental factors—i.e. anger, hostility, blood pressure, serotonine levels, or work overload—will lead to the possibility to select a multivariate phenotype that comprises the *toxic* configuration prone to suffer CHD and its genetic and environmental determinants.

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APPENDIX

Table A.1. Descriptive Statistics for Age Distribution by Survey Participation

		1991					1993				
		N	Mean	SD	Min	Max	*N*	Mean	SD	Min	Max
Twins	91 and 93	913	17.37	2.24	13.08	22.46	913	19.63	2.27	15.00	24.82
	91	697	18.07	2.21	12.60	24.55					
	93						939	15.97	2.58	12.00	24.62
Father	91 and 93	768	47.41	5.30	35.81	71.14	768	49.49	5.32	37.91	73.76
	91	529	48.07	5.9	35.37	38.46					
	93						811	46.31	4.82	35.91	72.58
Mother	91 and 93	768	45.28	4.75	34.08	60.41	768	47.57	4.78	36.46	63.16
	91	529	45.80	5.53	33.79	63.65					
	93						811	44.16	4.49	34.17	61.73

Data from complete twin pairs and parents who participated in 1991, 1993 or both are summarized in detail here. Those families that have random missing values (115 twin pairs, and 556 parents) are not reported in this table but were used in the analyses.

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