Nine-Year Stability of Type D Personality: Contributions of Genes and Environment

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Objective: To assess longitudinal changes in genetic and environmental influences on Type D personality and its subcomponents negative affectivity (NA) and social inhibition (SI) over a 9-year period. Most personality constructs have good retest reliability over long periods, with stability attributed to genes, and changes to environmental factors. Type D personality is stable across an 18-month period and is influenced by genetic factors. However, there is no knowledge on long-term stability, and the contributions of genes and environment to that stability. **Methods:** Type D personality was determined from survey data collected in 1991 (n = 3235; mean age = 17.3 years), 1997 (n = 3133; mean age = 25.3 years), and 2000 (n = 4456; mean age = 29.6 years) in a population sample of healthy twins. Multivariate structural equation modeling was employed. **Results:** Type D heritability ranged from 50% in 1997 to 34% in 2000, with the same genetic factor affecting Type D at all time points. Heritability of SI ranged from 49% (1991) to 42% (2000), with the same genetic factor influencing SI at all times. Heritability estimates for NA ranged from 45% (1991) to 40% (2000), with one genetic factor influencing NA at all times, and one genetic factor influencing NA at the second and third occasions. Different environmental factors acted on Type D, NA, and SI at each of the three measurement occasions. **Conclusion:** Type D personality and both subcomponents are stable over time, which is largely due to genetic factors. Different unique environmental factors influence the Type D components at different occasions. **Key words:** Type D personality, heritability, environmental influences, standard error of the mean.

A = additive genetic variance; **C** = common or shared environmental variance; **D** = dominance genetic variance; **DZ** = dizygotic; **E** = unique or nonshared environmental variance; **GWAS** = genome-wide association study; **MZ** = monozygotic; **NA** = negative affectivity; **SI** = social inhibition; **SNP** = single nucleotide polymorphism.

INTRODUCTION

Personality traits are developmentally dynamic constructs that exhibit both change and stability throughout the life span (1). In general, good retest stability over lengthy time spans in adulthood is observed for personality constructs, such as neuroticism and extraversion (2,3). Type D personality, which refers to the combined effect of negative affectivity (NA) (tendency to experience negative emotions) and social inhibition (SI) (tendency to inhibit self-expression in social interaction), has shown good short-term temporal stability (up to 18-month retest period) in adult cardiac patients (4,5). As genetic factors may contribute to the change as well as the continuity in a trait, part of the stability in Type D personality might be caused by genetic factors that are expressed continuously over time. Bratko and Butkovic (6) demonstrated that the 4-year stability of the Eysenck personality traits—extraversion, neuroticism, and psychoticism—was determined mainly by genetic factors, whereas change over that period was mainly due to environmental factors. Even over much longer time periods, neuroticism seems to be influenced by a stable set of genes, as a large study (7) with a follow-up of 22 years revealed.

In a study by Kupper et al. (8), we showed that individual differences in Type D classification and the scores on its continuous subscales were, for a large part, determined by genetic influences. However, it is not known whether these genetic and environmental components influencing individual differences in Type D personality and its subcomponents remain stable over longer periods of time. Therefore, the aim of the current investigation was to assess longitudinal changes in the genetic and environmental influences on Type D personality caseness and scores on the subcomponents NA and SI over a 9-year period.

METHODS

Study Population

All participants were registered with the Netherlands Twin Register (9). They were part of the adolescent and adult twin family cohort that was recruited through city councils or volunteer participation from 1991 onward and took part in a longitudinal survey study on health, life-style, and personality. Details on the recruitment procedure and sample characteristics of this cohort are described elsewhere (9,10). This study focuses on the data collections in 1991, 1997, and 2000, when information relevant to Type D personality and its subcomponents was obtained. Thirty participants were excluded due to too many missing values resulting in missing composite Type D personality scores. Twin pairs aged <13 years (n = 112 in total) at any of the measurement occasions were excluded from genetic modeling as well as participants aged >75 years (n = 10), who were excluded from the 2000 data set.

In 40.1% of the same-sex twin pairs, zygosity determination was based on deoxyribonucleic acid polymorphisms, whereas for the remaining twin pairs, zygosity was based on the answers to questions on the likeness of the twins and whether family members and others can distinguish between the twins. The correspondence between deoxyribonucleic acid and questionnaire-based zygosity is 97% (11). Ninety-four twin individuals (n = 75 families) had too many missing data and were excluded from further analysis. Finally, completed questionnaires with valid Type D information were present for 3,235 twin individuals (n = 1,602 pairs) in 1991, for 3,133 twin individuals (n = 1,301 pairs) in 1997, and for 4,456 twin individuals (n = 1621 pairs) in 2000. Figure 1 depicts a flowchart of the three measurement occasions, indicating per occasion exclusions, newly recruited twins, the number of twins in follow-up, and the participation rate. Numbers represent the sample for which

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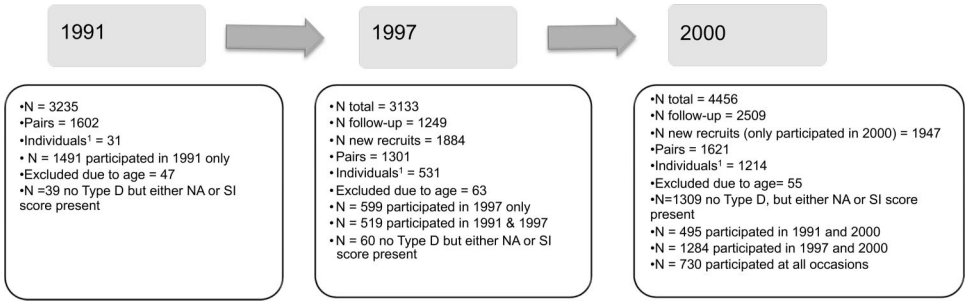

Figure 1. Inclusion flow chart. Exclusions due to age overlap, as subjects may have been excluded from multiple data sets. Numbers presented on participation rate represent the sample for which a Type D classification could be made. N follow-up is the total amount of participants in follow-up (which may be larger because participants may have either a negative affectivity (*NA*) or social inhibition (*SI*) score and no Type D classification). [1]Individuals = individuals from complete and from incomplete pairs.

a Type D classification could be made and may be higher for the subcomponents of Type D personality, as specified in the figure.

The Ethics Committee of the VU University Medical Center approved the study protocol.

Assessing Type D Personality

Type D personality was assessed by the 20-item Type D scale that was extensively described and validated in previous research (8,12) on the heritability of Type D personality. All items were scored on a 3-point scale from 0 (false) to 2 (true), with sum scores ranging from 0 to 20. Because one of the items of the Type D assessment was not included in the 2000 survey ("Do you feel that few things in your life go as they should?"), we applied the last observation carried forward procedure (13) and imputed the score on this item from the earlier 1997 survey. This procedure underestimates the variability and is, therefore, considered a conservative procedure. Because of missing data for several NA component items in the 2000 survey, NA total scores could not be calculated for part of the total sample, leading to a difference in N for NA and SI at the third measurement occasion (Fig. 1, box 2000). At least seven of the ten items had to be present to calculate the subcomponent scores. In case of ≤3 missing values, items that were missing were replaced with the mean. Whenever a score on one of the subcomponents was missing, combined with a low (< median) score on the other component, this person was automatically classified as non-Type D, as both subcomponents should be above the predefined cut-off level. Type D caseness was determined by a median split on both subscales (NA and SI) for each measurement occasion (median split NA = 5 [1991] and 3 [1997 and 2000]; median split SI = 7 [1991] and 8 [1997 and 2000]).

Statistics

Test Retest Reliability

To examine the individual change over time, we first calculated intraclass correlations at population level, using reliability analysis in SPSS 17.0 (SPSS Inc., Chicago, Illinois) for NA, SI, and Type D personality classification. As data from twin relatives are not independent, we performed the reliability analysis twice, comprising half of the monozygotic (MZ) and dizygotic (DZ) twin pairs in each analysis.

Genetic Modeling

To answer the question to which extent genes, shared environment, and nonshared environment contribute to the variance of Type D personality and the subcomponents NA and SI at each measurement occasion, and the stability of these constructs over time, quantitative genetic variance decomposition models were fit to the observed raw data (including data from complete and incomplete twin pairs), using the structural equation modeling program Mx (Mx: Statistical Modeling, Department of Psychiatry, Virginia Commonwealth University, Richmond, Virginia). First, multivariate unconstrained models were fit to test the assumptions of the twin model, i.e., equal means (thresholds in case of Type D personality between zygosities, equal variances between zygosities and sexes, and equal covariances between the sexes). For Type D personality, heritability was assessed using a liability-threshold model, which assumes a latent, normally distributed liability to being affected that is manifest as a categorical phenotype (14). The underlying distribution was modeled to have one threshold, which allows for two categories: affected (Type D) and unaffected (non-Type D). The final, most parsimonious, unconstrained model provided the twin correlations for each measurement occasion. With this final model, multivariate variance decomposition was initiated. Because of multiple testing, a $p < .01$ was considered significant. The methodology of behavioral genetic twin studies and how to interpret the results has been reviewed by McCaffery and colleagues (15).

Covariates

Because the age range of our sample was rather large (14–75 years), and sex differences may exist for Type D personality subcomponents, we tested for the significance of the effects of age and sex on the means (NA and SI) and thresholds (Type D classification) in the unconstrained models. If significant, they were taken into account in the variance decomposition analyses, using linear regression modeling (16).

Variance Decomposition

For all variables, variance was decomposed from raw data (including both complete and incomplete pairs to provide a more accurate variance estimation) into either latent factors A, C, and E or latent factors A, D, and E, dependent on whether the twin correlations suggested the presence of nonadditive genetic variance (D) referring to variance due to the interaction effect of two alleles that define the genotype at one locus; D is likely when the DZ correlation is less than half the size of the MZ correlation (17).

In longitudinal genetic analyses, one may choose from several models, depending on data requirements and research questions. The current study employed a Cholesky model (Fig. 2); as in this model, factors are constrained to affect later, but not earlier, time points. A Cholesky model is the most general way in which the variance-covariance structure of the longitudinal data can be decomposed into its genetic and environmental parts. Its model is suitable to address research questions concerning the magnitude of genetic and/or environmental influence at each occasion, and the extent to which genetic and environmental influences overlap across time. Data should be present on more than one measurement occasion to be included in the covariance calculations. Data from subjects that only participated once could only be used for the variance calculations.

Significance of individual path coefficients was tested by constraining them to zero and comparing the nested models by likelihood ratio tests. Specifically, it was tested whether an AE model was preferred over an ADE or ACE model. Subsequently, it was tested whether individual differences in Type D caseness, NA and SI, were influenced by a common set of genes or that, at one or more measurement occasions, a significant amount of specific genetic variance was present. To this end, we tested the presence of a third genetic factor by constraining genetic paths a33 (and d33) to zero. Then, a22 (and d22) were constrained to zero to establish whether there were time-specific genetic factors for the second measurement occasion. We additionally tested the significance of the effects of genetic factors on later time points

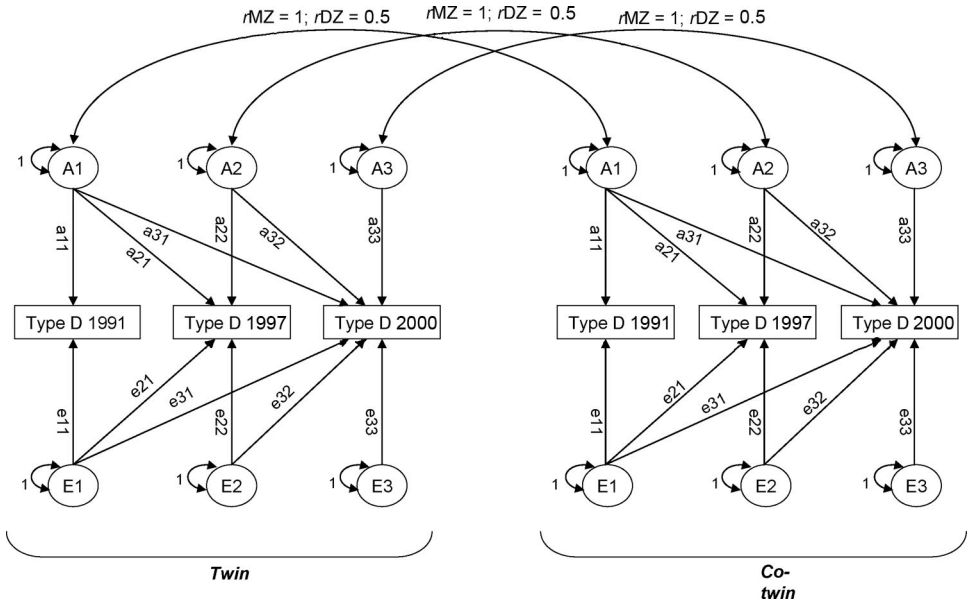

Figure 2. Trivariate Cholesky decomposition of Type D personality over time. This trivariate path diagram only includes components A and E. Components C and D have been left out for clarity reasons. For C, the decomposition of relations is similar to A, with the exception that both *r*MZ and *r*DZ are 1. For D, the decomposition of relations is similar to A, with the exception that *r*DZ = 0.25. *Single-headed arrows* represent causal paths; *double-headed arrows* represent correlational paths. *A* = additive genetic variance; C = common or shared environmental variance; D = dominance genetic variance; *E* = unique, nonshared, environmental variance.

(i.e., a21 [and d21] and a32 [and d32]). Similar tests were performed for the environmental component, except for the occasion-specific environmental influences (e22 and e33), as these include measurement error. In each final model, genetic and environmental correlations were extracted from the standardized variance covariance matrices. In addition, phenotypic tracking correlations were calculated for each final model, following this formula (example given for the relationship between NA assessments in 1991 and 1997):

$$r_{TR} = (\sqrt{h^2_{1991}} \times r_g \times \sqrt{h^2_{1997}}) + (\sqrt{e^2_{1991}} \times r_e \times \sqrt{e^2_{1997}})$$

RESULTS

Sample Characteristics

Table 1 contains the average age of the sample, the prevalence for Type D personality, and descriptives for NA and SI for each of the measurement occasions. Results show that NA levels decreased slightly, whereas SI levels increased slightly over time. Type D prevalence ranged between 25.9% and 27.9% in men, and between 35.2% and 38.4% in women. The increase in standard deviation of the average age may be explained by the inclusion of additional older-aged twins in 1997 and 2000.

TABLE 1. Sample Characteristics of the Three Subsequent Time Points

	1991	1997	2000
Men			
Age	17.2 (2.4)	24.3 (9.4)	28.6 (10.6)
Type D (%)	27.8	25.9	26.9
NA	4.7 (3.9)	3.2 (3.7)	3.4 (3.9)
SI	7.5 (4.3)	8.2 (3.9)	8.3 (3.9)
Women			
Age	17.2 (2.4)	25.8 (10.5)	30.4 (11.7)
Type D (%)	35.3	38.4	37.2
NA	5.8 (4.2)	4.8 (4.3)	4.9 (4.5)
SI	7.9 (4.8)	8.7 (4.1)	8.6 (4.1)

NA = negative affectivity; SI = social inhibition; E = unique or nonshared environmental variance.
Numbers represent mean (standard deviation), unless otherwise specified.

Nine-Year Retest Stability

The reliability analysis showed that, for NA, the intraclass correlations over three measurement occasions were 0.72 (95% confidence interval [CI], 0.66–0.77) and 0.78 (95% CI, 0.73–0.81) for each half of the twin population. SI also showed high retest reliability, as demonstrated by intraclass correlations of 0.82 (95% CI, 0.78–0.85) and 0.83 (95% CI, 0.80–0.86). For the dichotomous classification of Type D personality, the 9-year retest stability was less, with intraclass correlations of 0.58 (95% CI, 0.50–0.65) and 0.62 (95% CI, 0.54–0.68) for each half of the twin population. There were no sex differences in retest stability for all measures. All correlations were significant at the $p < .001$ level.

Type D Personality

Assumptions of the Twin Model

Multivariate analyses of the data at the three measurement occasions showed that prevalence of Type D personality did not differ for MZ and DZ twins and dizygotic twins of opposite sex ($p > .01$). At all three measurement occasions, thresholds showed significant sex differences. Type D prevalence was always higher in women compared with men ($p < .01$). Furthermore, there were no significant sex differences in the MZ and DZ twin correlations ($p > .05$). Prevalence of Type D personality at all time points and accompanying twin correlations are presented in Tables 1 and 2, respectively. Based on the difference in MZ and DZ twin correlations

TABLE 2. Twin Correlations for Type D Personality and Subcomponents Negative Affectivity and Social Inhibition for Each Measurement Occasion

	Type D Personality (Polychoric)		Negative Affectivity		Social Inhibition	
	MZ	DZ	MZ	DZ	MZ	DZ
1991	0.55	0.20	0.47	0.22	0.50	0.08
1997	0.49	0.21	0.43	0.22	0.49	0.12
2000	0.39	0.11	0.41	0.21	0.42	0.12

MZ = monozygotic; DZ = dizygotic.

(Table 2), it was decided to start out with an additive genetic, dominance genetic, unique or nonshared environmental variance (ADE) decomposition, including sex effects on the thresholds.

Variance Decomposition

The results of the multivariate structural equation modeling are presented in Table 3. As demonstrated in Table 3, an AE model fit the data best as compared with an ADE or E (very poor fit, data not shown) model. Next, we determined whether a single genetic factor influenced Type D personality over time. The most parsimonious solution with unstandardized path coefficients (standardized coefficients in brackets) is visualized in Figure 3*a*. One common genetic factor (A1) affected individual differences in Type D personality at all measurement occasions over the 9-year time period, with the

TABLE 3. Model Fitting Results for Type D Personality and Subcomponents Negative Affectivity and Social Inhibition

Model #	Content of the Model	Fit of the Model (−2LL)	# Estimated Parameters	Fit Comparison			
				$\Delta\ \chi^2$	$\Delta\ df$	Versus	*p*
			A. Type D Personality				
1	ADE	12581.09	24				
2	AE	12584.41	18	3.32	12	1	.99
			Genetic Path Coefficients				
3a	AE - a32 a33	12591.72	16	7.29	2	2	.03
3b	Model 3a–a22	12591.86	15	0.13	1	3a	.72
			Environmental Path Coefficients				
4a	Model 3b–e31&e32	12657.11	14	73.32		3b	<.001
4b	**Model 3b–e21**	**12593.29**	**13**	**1.43**		**3b**	**.23**
			B. Negative Affectivity				
1	ADE	59240.81	26				
2	AE	59240.81	20	0	6	1	1.00
			Genetic Path Coefficients				
3a	**AE–a33**	**59240.94**	**19**	**0.14**	**1**	**2**	**.71**
3b	Model 3a–a32	59256.48	18	15.54	1	3a	<.001
3c	Model 3a–a22	59255.64	18	14.70	1	3a	<.001
			Environmental Path Coefficients				
4a	Model 3a–e31&e32	59514.91	18	273.97	2	3a	<.001
4b	Model 3a–e21	59252.15	18	11.35	1	3a	<.001
			C. Social Inhibition				
1	ADE	57919.22	21				
2	AE	57951.59	15	32.37	6	1	<.001
			Genetic Path Coefficients				
3a	ADE–a33, d33, a32, d32	57921.62	17	2.40	4	1	.80
3b	**Model 3a–a22&d22**	**57919.99**	**15**	**0.68**	**2**	**1**	**.68**
			Environmental Path Coefficients				
4a	Model 3b–e31&e32	58205.95	13	285.96	2	3b	<.001
4b	Model 3b–e21	57950.66	14	30.67	1	3b	<.001

−2LL = −2 log likelihood; $\Delta\ \chi^2$ = difference in −2LL between two compared models; $\Delta\ df$ = difference in *degrees of freedom* between two compared models; *p* indicates whether the more restricted model fits the data better compared to an earlier model, with significance indicating a worse fit; *A* = additive genetic variance; *D* = dominance genetic variance; *E* = unique or nonshared environmental variance.

Column "Versus" indicates the number of model to which the more restricted model is compared. Because of multiple testing, a $p < .01$ is considered a significant worsening of fit.

Bold faced line indicates best-fitting model.

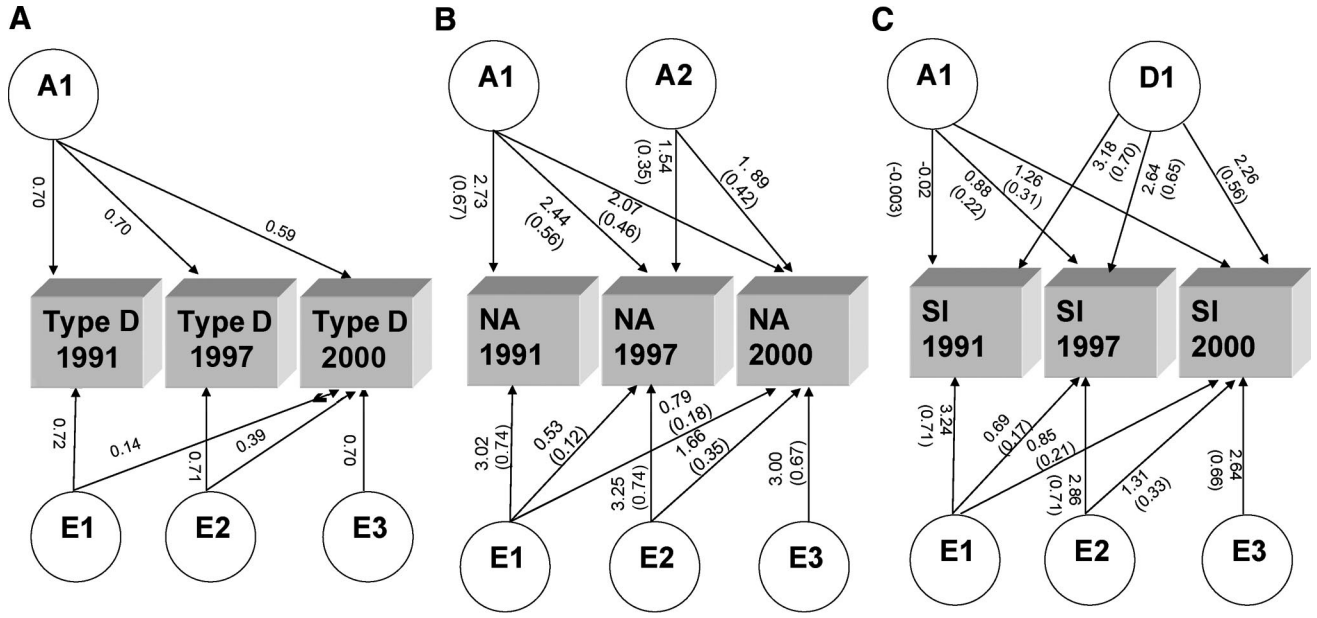

Figure 3. Final models for Type D personality, negative affectivity and social inhibition. *A* = additive genetic factor; *D* = nonadditive genetic factor; *E* = unique, nonshared, environmental factor. Numbers in *b* and *c* represent the unstandardized path coefficients with the standardized path coefficients in brackets. As the unstandardized and standardized path coefficients are the same in the Type D threshold analysis (standard deviation in this analysis is 1), only one number is printed (Fig. 3*a*).

TABLE 4. Estimates (95% Confidence Interval) of Genetic and Environmental Variance Components

	A (%)	D (%)	E (%)
Type D personality			
1991	49 (42–57)	—	51 (43–58)
1997	50 (41–55)	—	50 (45–59)
2000	34 (26–41)	—	66 (59–74)
Negative affectivity			
1991	45 (39–50)	—	55 (50–61)
1997	43 (38–48)	—	57 (52–62)
2000	40 (39–44)	—	60 (56–61)
Social inhibition			
1991	0 (0–13)	49 (40–54)	51 (46–57)
1997	5 (5–20)	43 (23–52)	53 (47–58)
2000	10 (0–27)	32 (14–45)	58 (54–64)

A = additive genetic effects; *D* = dominance genetic effects; *E* = unique (nonshared).

heritability estimate for Type D personality being 50% in 1991, 47% in 1997, and 36% in 2000 (Table 4). Heritability of Type D personality decreased significantly from the 1991 to the 2000 measurement occasions, but the confidence intervals overlapped for the 1991 and 1997 measurement occasions and for the 1997 and 2000 measurement occasions. Because one shared genetic factor influenced Type D classification at all time points, the genetic correlation was 1.

For the unique environmental component, we could remove the influence of the first environmental factor (E1) on individual differences in Type D personality in 1997 without reducing the fit of the model (model 4b). Overall, the environmental variance decomposition for Type D personality showed that the unique environmental factors affecting Type D personality were different for each time point, and of differing magnitude (Table 4 and Fig. 3*a*), accounting for 50% to 64% of the variance. The environmental correlation between 1991 and 1997 could be removed and, thus, is 0; the environmental correlation between 1997 and 2000 was 0.49, whereas the correlation between 1991 and 2000 was 0.17. The phenotypic tracking correlation indicating the covariation in Type D at different measurement occasions was moderate to large, between 0.49 and 0.69, with the genetic contribution to this tracking correlation (59% to 100%) being much higher than the environmental contribution (0% to 41%).

Negative Affectivity

Assumptions of the Twin Model

The multivariate assumption tests in the saturated models for NA showed that, for all measurement occasions, the means of NA were not different in MZ twins compared with DZ twins ($p > .01$). Sex was a significant covariate of NA at all measurement occasions, with men scoring lower than women ($p < .001$). Both sex and age were included as covariates in the subsequent variance decomposition. Importantly, there were no significant differences in variances between zygosities ($p > .001$). There were differences in variance between the sexes for the second and third measurement occasions ($p < .001$), which disappeared after scalar correction (essentially multiplying male data at these two measurement occasions so that male and female variances become equal; $p > .05$). Covariances were found to be equal for males and females. The resulting twin correlations are displayed in Table 2.

Variance Decomposition

Based on the difference in MZ and DZ twin correlations, variance decomposition was initiated with an ADE model.

Multivariate variance decomposition showed that an AE model fits the data better compared with an ADE or E (very poor fit, data not shown) model (Table 3, model fitting results; Table 4, summary of heritability estimates). Age showed a small negative linear association with mean NA scores ($r_{1997} = -.02$; $r_{2000} = -.02$). An additional research question was to determine whether there was a single genetic factor that influenced NA over time or that new genetic components came into play as time progressed. The most parsimonious solution for NA is visualized in Figure 3*b*, displaying unstandardized (standardized) path coefficients. One common genetic factor (A1) affected individual differences in NA at all measurement occasions over the 9-year time period. An additional genetic component (A2) was present that affected individual differences at both the second and third measurements of NA. Overall, heritability was stable, between 40 and 45%, with overlapping confidence intervals. Total variance decreased from 18.4 to 16.0 over the 9-year time period. The genetic correlations between the three measurement occasions for NA were 0.94 (1991–1997), 0.82 (1997–2000), and 0.97 (1991–2000), respectively.

The environmental variance decomposition for NA had to remain in full Cholesky decomposition, as omitting paths from the model led to worse fitting models (Table 3, model fitting results). The final model showed that the environmental variance in NA ranged between 55% and 60% (Fig. 3*b* and Table 4) and that less than half of this variance was shared between time points, visualized in moderate environmental correlations ($r_{e1991–1997} = .16$; $r_{e1997–2000} = .48$; $r_{e1991–2000} = .23$). The phenotypic tracking correlation indicating the covariation in NA at different time points was moderate to large, between 0.45 and 0.70, with the genetic contribution (60% to 80%) to the phenotypic tracking correlation being substantially larger than the environmental contribution (20% to 40%).

Social Inhibition

Assumptions of the Twin Model

The assumptions tests showed no differences in SI means between zygosities and sexes ($p > .05$), nor were there differences in variance ($p > .05$). There was no significant regression effect of age on mean SI levels ($p > .05$). As covariances also were equal for males and females ($p > .05$), age and sex differences were not included in the final variance decomposition. The resulting twin correlations are displayed in Table 2. Based on the difference in MZ and DZ twin correlations, it was decided to start out model fitting with an ADE variance decomposition.

Variance Decomposition

Multivariate variance decomposition, including all measurement occasions, showed that both A and D had to be retained in the model for SI (Table 3, model fitting results), as both AE and E models provided a worse fit to the data (E model not shown). Results showed that one common additive genetic factor (A1) and one common dominance genetic factor (D1) affected individual differences in SI at all measurement occasions over the 9-year time period. No additional genetic factors came into play over time. Overall, the final model showed that broad heritability (A + D) was stable, with estimates between 42% and 49%, with overlapping confidence intervals (Table 4). Total variance decreased from 20.6 to 16.1 over the 9-year time period. Needless to say, because only one genetic additive component and one genetic dominance component influenced SI at all time points, the genetic correlation was 1.

For the unique environmental component, the final model showed that the original Cholesky decomposition resulted in the best fit with the unique environmental components accounting for 51% to 58% of the variance. The environmental correlations between the three measurement occasions of SI were moderate ($r_{e1991–1997} = .24$; $r_{e1997–2000} = .28$; $r_{e1991–2000} = .48$), indicating that less than half of the environmental variance was shared between time points. The phenotypic tracking correlations were rather large, between 0.58 and 0.66, with the genetic contribution (60% to 79%) being substantially larger than the environmental contribution (21% to 40%). The most parsimonious solution for SI, including unstandardized (standardized) path coefficients, is illustrated in Figure 3*c*.

DISCUSSION

Type D personality is a risk factor for poor prognosis and death in cardiovascular disease (18–20). However, the reason why Type D patients are more vulnerable is still elusive and may reflect biological (21–23) but also behavioral factors (24). The purpose of the current study was to determine the etiology of stability and change in Type D personality and its subcomponents over a time period of 9 years. The results point toward two primary conclusions. First, the stability of NA and SI, as well as Type D personality caseness, seems to be primarily a function of enduring genetic influences. Second, change in NA may be due to both new genetic influences and nonshared environmental factors, whereas for Type D classification and SI only nonshared environmental factors seem responsible for change. These results are consistent with previous literature on personality constructs. Individual differences in neuroticism have shown to be influenced by a stable set of genes over very large periods of time (>20 years) (7). A study by Bratko and Butkovic (6) demonstrated that the 4-year stability of the Eysenck personality traits—extraversion, neuroticism, and psychoticism—during the transition period from adolescence into young adulthood was determined mainly by genetic factors, whereas change over that period was mainly due to environmental factors.

A longitudinally stable set of genes explained between 42% and 49% of the variance in SI in the current population, displaying both additive and dominance genetic effects, which was also reported in our previous cross-sectional behavioral-genetic analysis of Type D personality (8). SI is a construct that is related to social interaction anxiety and fear of negative evaluation. Previous family and twin heritability reports on these constructs have shown that (social) anxiety (25–27) and

fear of negative evaluation (28) are heritable to a similar degree and that this influence is stable over time (26). None of these studies, however, reported on dominance genetic factors.

Very large-scale genome-wide association studies (GWAS) may help disentangle the very complex biological basis of emotional functioning. So far, one GWAS was performed for Type D personality and two GWAS were performed for neuroticism. The GWAS on Type D personality showed that the most significant single nucleotide polymorphism (SNP) associations occurred in or near genetic regions important for immune function and neuronal plasticity. Additional associations were found with SNPs that also were reported to have associations with, among others, autism, hypertension, diabetes, and inflammation (29). The two GWAS on neuroticism may provide information on potential candidate genes for Type D personality. Both GWAS on neuroticism suggested the involvement of a different gene, as one study (30) showed an association of one SNP within the PDE4D gene, involved in cyclic adenosine monophosphate degradation, thereby affecting cell signaling. The other study (31) found an association with MAMDC1, coding for a neuronal adhesion molecule involved in regulating neuronal migration and axonal guidance. This latter finding would fit the theory well that neuroticism predisposes to mood disorders, as other neuronal adhesion molecules have been previously associated with chronic stress and mood disorders (32).

The current findings should be interpreted with appropriate caution, as there are some limitations to this study. First, because the Type D scale (DS14) was not available for these twin data sets, an extensively validated 20-item proxy was used to assess Type D personality and its subcomponents, and a median split was used to classify persons as having a Type D personality (just like in earlier versions of the Type D scale, DS16) (12). Notably, the median was lower for the 1991 data as compared with the data from 1997 and 2000. Second, for SI, large confidence intervals for A and D were observed, and the additive genetic component was estimated to be very small. Computer simulation by Eaves (33) showed that although the estimate of broad heritability (A + D) may be stable, large fluctuations in estimates of A and D may occur. Future studies that include information on many different genetic relationships (e.g., twins, siblings, half-siblings, parent-offspring) are needed to reliably separate additive genetic influences from nonadditive genetic influences. A further limitation is that the 1991 sample was drawn from a homogeneous sample of young adult twins, whereas the 1997 and 2000 samples had a broader age range and included middle and older-aged twins. Although the age of the twins did not affect the mean score of SI, it should be noted that the emergence of a second genetic factor influencing individual differences in NA at the second and third measurement occasions might be a reflection of this inclusion modification, as well as the difference in the median of NA and SI in 1991, on the one hand, and in 1997 and 2000, on the other hand. In addition, the current sample had a high attrition rate, as well as a steady inflow of newly recruited twins. The samples at the three time points, therefore, differ from each other to a certain extent with respect to the presence of participants. Attrition was unrelated to Type D personality classification; it is, therefore, unclear how this could have affected the results.

In conclusion, genetic factors contributed to stability in individual differences in Type D personality over time, whereas different environmental factors affected Type D personality over time. The latter indicates that behavioral intervention would be feasible and useful in Type D patients. Future studies are encouraged to examine the genetic covariation between Type D personality and general physiology and to search for candidate genes for Type D personality in GWAS.

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