Heritability of Type-D Personality

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Objective: To quantify the influence of genes and environment on individual differences in type-D status, and the type-D subcomponents negative affectivity and social inhibition. Type-D personality independently predicts poor prognosis in patients with cardiovascular disease. However, no previous study has determined the heritability of type-D personality. **Methods:** This study determined type-D personality by applying the "combination of scales" method to survey data collected by the Netherlands Twin Register in 3331 healthy, young adult twins. Using structural equation modeling (SEM), the relative contributions of additive genetic, nonadditive genetic, and nonshared environmental factors to the variance in type-D and its subcomponents were determined. **Results:** SEM indicated that type-D personality was substantially heritable (52%). The subcomponents negative affectivity and social inhibition were equally heritable, with broad heritability estimates of 46% and 50%. Although negative affectivity was determined by additive genetic effects and nonshared environment, individual differences in social inhibition were predominantly determined by nonadditive genetic effects and nonshared environment. **Conclusions:** This study provides strong evidence that genes are important in determining individual differences in type-D personality and the type-D subcomponents negative affectivity and social inhibition. **Key words:** type-D personality, twins, heritability, negative affectivity, social inhibition.

A = additive genetic variance component; **ABQ** = Amsterdam Biographical Questionnaire; **C** = shared environmental variance component; **D** = nonadditive (dominance) genetic variance component; **DZ** = dizygotic; **E** = nonshared environmental variance component; **MZ** = monozygotic; **NA** = negative affectivity; **SI** = social inhibition; **STAI** = Spielberger Trait Anxiety Inventory; **TWND** = Twin type-D scale; **YASR** = Young Adult Self-Report.

INTRODUCTION

The distressed (type-D) personality has been associated with adverse health outcomes in coronary artery disease (1–3), heart failure (4–6), and peripheral arterial disease (7), independent of other biomedical and psychological risk factors. Defined as the tendency to experience negative emotions and to inhibit the expression of these emotions in a social context, type-D personality represents a synergy of the common traits negative affectivity (NA) and social inhibition (SI) (8,9). NA is a broad personality trait that is defined by the tendency to experience negative emotional states across time and situations, and comprises on one hand dysphoria, and on the other hand, feelings of tension and worry (10). Although type-D individuals experience a wide range of negative emotions, type-D personality comprises more than negative emotions alone. Due to the inclusion of the SI component, representing the stable tendency to inhibit the expression of emotions, thoughts, and behaviors in social interaction, the type-D construct also indicates that a combination of SI and NA is essential when investigating the effects on clinical outcome (9,11). Only those individuals that score high on both subcomponents are at increased risk. Type-D personality is fairly prevalent (between 13% and 32.5%) among the general population in multiple Western-European countries (7,8,12,13). The prevalence of type-D personality is higher (between 26% and 53%) in cardiovascular patients (4,7,8,12,14).

Twin studies have consistently shown that heritability estimates for neuroticism range between 31% and 59% (15–23), and that this genetic influence is stable across the adult life span (19). The heritability of SI, or related traits, is not as well established. Two twin studies in young children (3–4 year olds) have shown that individual differences in behavioral inhibition, a close correlate of SI, are determined by genetic influences. Eley and colleagues (24) used a shyness/inhibition construct in 4 year olds and showed that the heritability for this behavioral style was 76% in boys and 66% in girls. Derks and colleagues (25) reported heritabilities for withdrawn behavior, and estimated genetic influences to be around 55% to 58% for boys and around 40% to 50% for girls. In adults, only one study reported on the heritability of a behavioral inhibition-like construct (26), namely, the fear of negative evaluation. Results showed that 48% of the individual differences were explained by genes. No study so far has addressed the heritability of type-D personality or simultaneously examined its two subcomponents, NA and SI, in a genetic analysis.

In the present study, the relative influence of genes and environment on individual differences in type-D status, and the type-D subcomponents, NA and SI, are quantified, using data from a large population-based sample of healthy, adolescent, and young-adult twins. A bivariate analysis is used to determine the extent to which genetic influences on NA and SI overlap. Because the two type-D subcomponents are independent of each other (9), we expect they only share a limited amount of genetic variance.

METHODS

Participants

All participants were registered with the Netherlands Twin Register (27). They were part of the adolescent and adult twin family cohort that was recruited through city councils and in 1991 was sent a survey containing personality and psychopathology inventories, and questions about health, demographic background, and lifestyle. Completed questionnaires were obtained for 3384 twin individuals from 1695 families.

For 317 same-sex twin pairs, zygosity determination was based on DNA polymorphisms, using 11 highly polymorphic genetic markers; for the remaining twin pairs, zygosity was based on the answers to questions on the

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likeness of the twins and whether family members and others can distinguish between the twins. Twenty-seven families (53 individuals) did not provide DNA or questionnaire-based information on zygosity, and were excluded from further analysis. The correspondence between DNA and questionnaire-based zygosity was 97% (28).

The final sample consisted of 552 monozygotic males (MZM), 487 dizygotic males (DZM), 766 monozygotic females (MZF), 565 dizygotic females (DZF), and 961 dizygotic twin individuals of opposite sex (DOS). The mean age was 17.2 ± 2.26 (standard deviation, SD) years (range = 12–24). The Ethics Committee of the Vrije Universiteit University Hospital approved the study protocol.

Assessment of Type-D Personality

We assessed type-D personality in the twins by applying the "combination of scales" method (11,14,29) to the survey data. The 1991 survey included three scales: the Amsterdam Biographical Questionnaire (ABQ) (30), the Spielberger Trait Anxiety Inventory (STAI) (31), and the Young Adult Self-Report (YASR) (32), which had items similar to the items from the original type-D questionnaire (DS14) (8) in content and wording. After the combination of scales method, 20 items were selected and used to determine type-D status (Table 1).

The constructed scale (TWND) was validated by administering these items together with the DS14 to a large group of first year students from

TABLE 1. Structural Validity and Internal Consistency of TWND and DS14

Item	Source	Principal Components Analysis		
		Factor I	Factor II	Internal Consistency[c]
TWND[a]				
1[d] Makes new friends easily	YASR	0.13	0.67	0.56
3 Prefers limiting social contacts	ABQ	0.31	0.37	0.34
6[d] Easy to make new acquaintances	ABQ	0.10	0.63	0.49
8 Difficulties to unbend at a cheerful party	ABQ	0.13	0.47	0.39
10 Is a closed kind of person	YASR	0.14	0.58	0.48
11 Tend to behave inconspicuous	ABQ	0.09	0.72	0.61
14[d] Is a talkative kind of person	ABQ	0.03	0.81	0.70
16 Is often shy	YASR	0.10	0.60	0.49
18[d] Quick and certain in way of acting	ABQ	0.39	0.45	0.40
19[d] Talks too much	YASR	−0.14	0.62	0.44
				$\alpha = 0.81$
2 Worries too much	STAI	0.62	0.14	0.54
4 Sometimes feels miserable without reason	ABQ	0.58	0.12	0.49
5 Often moody	ABQ	0.55	0.09	0.47
7 Feeling that difficulties are piling up	STAI	0.71	0.07	0.61
9 Mood often goes up and down	STAI	0.72	0.20	0.66
12 Tense when thinking of recent worries	STAI	0.70	0.00	0.58
13 Feelings of failure	STAI	0.72	0.01	0.61
15 Nervous and tense kind of person	ABQ	0.62	0.23	0.55
17 Bothered by unimportant thoughts	STAI	0.72	0.07	0.62
20 Things in life don't go as they should	ABQ	0.47	0.05	0.38
				$\alpha = 0.85$
DS14[b]				
1[d] Makes contact easily when meeting people		0.83	0.07	0.74
3[d] Often talks to strangers		0.66	−0.04	0.53
6 Feels inhibited in social interactions		0.75	0.34	0.71
8 Difficulties to start a conversation		0.81	0.07	0.73
10 Is a closed kind of person		0.78	0.10	0.69
11 Rather keeps other people at a distance		0.73	0.10	0.65
14 Doesn't find the right things to talk about		0.75	0.25	0.69
				$\alpha = 0.88$
2 Makes a fuss about unimportant things		−0.08	0.62	0.49
4 Often feels unhappy		0.21	0.75	0.66
5 Is often irritated		0.02	0.70	0.57
7 Takes a gloomy view of things		0.28	0.76	0.69
9 Often in a bad mood		0.23	0.72	0.63
12 Often worries about something		0.23	0.71	0.60
13 Is down in the dumps		0.19	0.83	0.76
				$\alpha = 0.86$

EV = Eigenvalue; TWND = Twin type-D scale; ABQ = Amsterdam Biographical Questionnaire; STAI = Spielberger Trait Anxiety Inventory; YASR = Young Adult Self Report.

[a] Factor I: EV = 5.48; Factor II: EV = 2.86.

[b] Factor I: EV = 5.45; Factor II: EV = 2.74.

[c] Corrected item-total correlations.

[d] Reversed item.

Tilburg University (n = 293, 21.5% male). Each item from the TWND was rated on a 3-point Likert scale from 0 (false) to 2 (true); each item from the DS14 was rated on a 5-point Likert scale from 0 (false) to 4 (true). Subjects scoring high on both NA and SI, as determined by either median split (TWND) or a cut-off of 10 (DS14), were classified as type-D. To determine whether the TWND scale would validly capture the characteristics of type-D personality, factor analysis and reliability analysis (SPSS 14.0, Chicago, Illinois) were used to examine the internal-structural validity of the TWND scale. Cross-tabulation (SPSS 14.0) enabled direct comparison between type-D status based on the TWND and based on the DS14.

Genetic Modeling

Genetic and environmental influences on variation in type-D personality, TWND-based NA and SI scores were analyzed in a classical twin design (which assumes that on average DZ twins share half as many genes in common by descent as MZ twins and that they experience the same shared environmental influences (equal environment assumption)) using the structural equation modeling (SEM) package Mx (Mx: Statistical Modeling) (33).

Type-D Personality

Heritability of type-D personality was assessed using a liability-threshold model, which assumes a latent, normally distributed liability to being affected, that is manifest as a categorical phenotype (34). For type-D personality, the underlying distribution was modeled to have one threshold, which allows for two categories: affected (type-D) and unaffected (nontype-D). Sources of variation in type-D liability considered in the modeling were additive genetic factors (A), nonadditive genetic factors (D), shared environmental influences (C), and nonshared (unique) environmental factors (E). In an analysis that only includes twins, C and D are confounded, and SEM cannot discriminate between the two. Consequently, variance was decomposed 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 is likely when the MZ correlation >2 * DZ correlation (35)). Nested submodels (AE, CE, E) were compared with either the full ACE or ADE by constraining path coefficients to zero, to get at the most parsimonious and best fitting model. Sex differences were tested by allowing the magnitude of the genetic and environmental effects to be different for males and females and by allowing the correlation between the genetic factors for the opposite-sex twins to be less than the theoretical 0.50. For all models, different thresholds were estimated for males and females, allowing for differences in the prevalence of type-D personality between males and females. The fit and parsimony of the various models were judged using likelihood ratio tests.

NA and SI Subcomponents

The genetic modeling analyses were carried out in several steps. First, in a bivariate saturated model, assumptions of the twin model were tested, such as equal means between zygosities, equal variances between zygosities and sexes, and equal covariances between the sexes. In addition, the effects of the variables age and gender on the mean were assessed. Because of multiple testing, a $p < .01$ was considered significant. The final outcome defined the most parsimonious unconstrained model, which provided the twin correlations and the cross-twin-cross-trait correlations. With this final model variance decomposition was initiated.

Similar to the liability threshold model above, variance was decomposed 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 is likely when the MZ correlation >2 * DZ correlation (35)). 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 NA and SI were influenced by a common set of genes, or that a significant amount of specific genetic variance was present. Similar tests were performed for the environmental component. The AIC (36) was used to judge the relative fit of nested and un-nested models.

RESULTS

Validity of the TWND Measure of Type-D Personality

Results from the factor analysis on the student dataset confirmed the two-factor structure underlying type-D personality for both questionnaires (TWND and DS14). Scree plots showed that succeeding factors were of much less importance (Eigenvalue ≤ 1) and explained a minor proportion of the variance. For both questionnaires, the factor loadings for the individual items are reported in Table 1. Cronbach's α and item-total correlations indicated a high level of internal consistency within these two factors for both questionnaires. When assessed with the TWND, the subcomponents shared 12% of the variance ($r = .35$; $p < .001$); the subcomponents shared 10% of the variance ($r = .31$; $p < .001$) when assessed with the DS14.

Prevalence of type-D was 18.5% when measured with the original DS14, and 26.7% when measured with the proxy questionnaire (TWND). An acceptable association present between the two methods of type-D assessment ($\chi^2 = 68.385$; $p < .001$) also demonstrated by a κ of 0.46 ($p < .001$). The correlation between NA subscales of the DS14 and the TWND was high ($r = .75$; $p < .001$) as well as the correlation between the SI subscales of both questionnaires ($r = .77$; $p < .001$). The above results denote sufficient validity of the 20-item TWND scale as a measure of type-D personality. We therefore used the TWND scale to determine type-D personality in the twin database.

Heritability of Type-D Personality

Prevalence of type-D personality did not differ between MZ, DZ and DOS twins ($p > .07$). Type-D prevalence was higher for women (30.9%) compared with men (22.8%) ($p < .001$), but no sex differences were found in the covariances. There was no significant influence of age. The resulting most parsimonious saturated model indicated that the monozygotic twin correlation was 0.55 (95% confidence interval (CI) = 0.42–0.65), whereas the dizygotic twin correlation was 0.22 (95% CI = 0.10–0.33). Based on these twin correlations, a model including additive (A) and nonadditive (D) genetic effects in concert with nonshared environmental factors (E) was chosen to commence variance decomposition modeling.

The ADE model could be reduced to an AE model, with different prevalences of type-D personality in males and females without a significant loss of fit (Table 2). The heritability estimate for type-D personality in this final, most parsimonious model was 52% (95% CI = 41–62).

Heritability of NA and SI

On average, the MZ twins scored 4.98 ± 3.95 (SD) and the DZ twins 5.52 ± 4.12 on the NA subcomponent (maximum score = 20). For SI, averages of 7.80 ± 4.62 for MZ twins and 7.59 ± 4.53 for DZ twins were observed (maximum score = 20). A significant zygosity effect was found for the mean NA score, which was slightly higher in DZ twins compared with MZ twins. Males and females did not differ in their mean SI score, but females had significantly higher NA scores than

TABLE 2. Model Fitting Results for Type-D Personality

Number	Model	$\Delta\chi^2$	Δdf	Vs.	p
1	ADE[a]	—	—		
2	ADE[b]	19.721	1	1	<.001
3	$ADE_{males} = ADE_{females}$	0.624	3	1	.89
4	AE	0.653	1	3	.42
5	E	73.318	1	4	<.001

[a] Full model, sex differences in prevalence of type-D.
[b] No sex differences in prevalence of type-D.
$\Delta\chi^2$ = difference in χ^2 between nested and reference model; Δdf = amount of degrees of freedom that are gained in the nested model; Vs. = reference model.
The final model (4) includes an additive genetic and nonshared environmental factor in the presence of sex differences in the prevalence of type-D.

TABLE 3. Twin Correlations From the Constrained Saturated Model

	MZ	DZ
Negative affectivity	0.46 (0.40–0.52)[a]	0.22 (0.16–0.28)
Social inhibition	0.50 (0.45–0.56)	0.09 (0.02–0.15)
Cross-twin-cross-trait correlations	0.16 (0.11–0.21)	0.08 (0.03–0.12)

MZ = monozygotic twins; DZ = dizygotic twins (both same-sex and opposite sex); cross-twin-cross-trait correlations = the correlation between the negative affectivity score for one twin of a pair and the social inhibition score for the other twin of a pair.
[a] The 95% confidence intervals are given between the parentheses. All correlations are significant at $p < .05$ level.

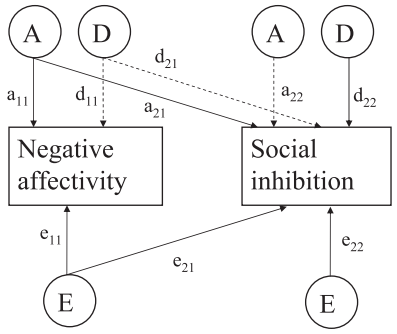

Figure 1. Variance decomposition model for bivariate analysis. Representation of a triangular (Cholesky) variance decomposition into additive genetic (A), nonadditive genetic (D) and nonshared environmental influences (E). a_{11} through a_{22} = path coefficients for the additive genetic variance component; d_{11} through d_{22} = path coefficients for the nonadditive genetic variance component; e_{11} through e_{22} = path coefficients for the nonshared environmental variance component; dashed line = nonsignificant path, dropped from the final model.

TABLE 4. Genetic Model Fitting Results for Negative Affectivity and Social Inhibition

Number	Model	$\Delta\chi^2$	Δdf	Vs.	p
1	Full ADE_{NA}/ Full ADE_{SI}				
2	AE_{NA}–ADE_{SI}	0.196	2	1	.91
3	AE_{NA}–AE_{SI}	22.589	1	2	<.001
4	2, without shared $E_{NA\text{-}SI}$	24.722	1	2	<.001
5	2, without shared $A_{NA\text{-}SI}$	43.491	1	2	<.001
6	2, without specific A_{SI}	0	1	2	1.000

NA = negative affectivity; SI = social inhibition; full model = no constraints; $\Delta\chi^2$ = difference in χ^2 between nested and reference model; Δdf = amount of degrees of freedom that are gained in the nested model; Vs. = reference model.
The final model (6) excludes the following paths in Figure 1: d11, d21, and a22.

males (5.8 versus 4.7). Age was not a significant covariate for either NA or SI. NA variances and both NA and SI covariances did not differ for males and females. SI variances were significantly smaller for males compared with females. This difference was addressed by performing a scalar correction for the SI subcomponent (male values for SI were multiplied by 1.105), before calculating the twin correlations and the variance decomposition analysis. In the most parsimonious saturated model, the twin correlations and cross-twin-cross-trait correlations for NA and SI were estimated (Table 3).

To estimate the genetic and environmental contributions to the variance of NA and SI, and to determine to which extent individual differences in NA and SI derive from a common genetic or environmental factor, NA and SI were examined in a bivariate variance decomposition analysis, for which the ADE model is presented in Figure 1. The model fitting results for the several successive hypotheses that were tested are presented in Table 4. Because significant sex differences were found for the mean NA score, the effect of sex on the mean NA scores was included in the analyses. The initial ADE model was reduced to an AE model for NA without a significant reduction in statistical fit (Model 2). For SI, an ADE model represented the data best (Models 2 and 3). It was further tested whether genetic and/or environmental variance was shared between the two traits. To this end, it was tested whether paths a_{21} and e_{21} were significantly different from zero (Models 4 and 5), which they were. Results further showed that all additive genetic variance of SI was shared with NA (Model 6). The phenotypic correlation between NA and SI in this dataset was 0.27 ($p < .001$). The genetic correlation, reflecting the amount of overlap between gene sets influencing both traits, was 1.00 for additive genes and 0.00 for nonadditive genes. The contribution of these shared additive genes to the phenotypic correlation was 60% (95% CI = 45–74). The nonshared environmental component was best represented by its original triangular variance decomposition: no nonshared environmental path coefficient could be set to zero without a significant loss of statistical fit (Model 4). The environmental correlation, reflecting the amount of overlap in environmental elements influencing both traits, was 0.20 (95% CI = 0.13–0.27). The contribution of these nonshared environmental components to the phenotypic correlation was 40% (0.26–0.55). In the best fitting, final model (Model 6), 46% of the variance in negative affectivity was

TABLE 5. Heritability Estimates for Negative Affectivity and Social Inhibition

	A	D	E
Negative affectivity	0.46 (0.40–0.52)[a]	—	0.54 (0.48–0.60)
Social inhibition	0.06 (0.03–0.09)	0.44 (0.38–0.49)	0.50 (0.45–0.56)

A = additive genetic component; D = nonadditive genetic component; E = nonshared environmental component.
[a] The 95% confidence intervals are given between the parentheses.

explained by additive genetic influences, whereas 50% of the individual differences in social inhibition was explained by genetic factors (44% of which was attributed to nonadditive genetic effects). The remaining variance in both SI and NA was accounted for by nonshared environmental influences. A summary of these estimates is given in Table 5, and the nonsignificant paths are depicted as dashed lines in Figure 1.

DISCUSSION

The findings emerging from our present study in 3331 twins showed that the presence of type-D personality is substantially heritable (52%). In addition, genetic factors were shown to influence both type-D subcomponents. The heritability for NA was 46%, due to additive genetic factors. For SI, heritability was 50%, mostly due to nonadditive or dominance genetic effects.

For NA, our present findings are comparable with those reported in multiple previous studies (15–23) on related personality traits such as neuroticism, and emphasize the difference between negative affect (referring to mood state), which was found not to be heritable (37) and NA, a stable predisposition to experience negative emotions that shows a substantial genetic contribution. This study is the first to report heritability estimates for type-D personality and SI. Our estimate of 50% broad heritability for SI lies close to previous heritability reports on behavioral inhibition in children (24,25) and fear of negative evaluation in adults (26). Contrary to our findings, these previous studies reported only additive genetic effects, whereas we also found nonadditive (dominance) genetic effects for SI. This discrepancy might be partly due to the age of the subjects, but it may also reflect a true difference in the nature of the constructs (fear/behavioral inhibition versus social inhibition) assessed.

The two type-D subcomponents, NA and SI, were only moderately correlated ($r = .27$). The bivariate genetic modeling results showed that the shared (additive) genetic component between NA and SI, although significant, was only modest (6%). The fact that only a limited amount of genetic variance was shared between NA and SI strengthens the notion that type-D personality is a composite of two independent traits, working additively to yield the type-D personality.

Type-D personality has proven to be a strong, independent predictor of cardiovascular morbidity (38), and all-cause and cardiac mortality (11,14) in cardiac patients. This robust relationship is most likely mediated by physiological mechanisms affecting the cardiovascular system. A plethora of studies already found most standard cardiovascular risk factors to be substantially influenced by genes (38–45). However, only a very limited amount of studies have actually tested whether these genetic influences are (partly) shared with genetic influences on psychological characteristics, and these few publications showed mixed results (46–48). One study estimated the genetic and environmental contributions to the covariation of depressive symptoms and individual components of the metabolic syndrome and found that the associations between these individual components and depressive symptoms were attributable to environmental factors (46), whereas another study showed significant genetic correlations for depressive symptoms with hypertension and heart disease (48). A very recent study in a large community sample ($n = 6148$) from Sardinia found no evidence for shared genetic determinants between neuroticism and cardiovascular risk factors, such as blood pressure and intima-media thickness (47). Future research on type-D personality may want to determine whether type-D personality and biomedical risk factors (partly) fall back on the same genetic and/or environmental factors.

Although the implications of this study's findings are promising, they should be interpreted prudently, in the light of several limitations of the study design. In designing a proxy scale to assess type-D, we tested the eligible items in a newly composed list (TWND) together with the original instrument. It is of note that the presentation of the items in the twin survey differed from the presentation in the validation sample; in the twin survey, the items were incorporated in their original questionnaires. Another limitation is that the average age of the patient populations in which type-D is an independent predictor of cardiovascular morbidity and mortality is much higher than the average age of the twin population the present analyses were based on. Although age was not a significant confounder in the present age range, it may be that over time different genes come into play, and that the heritability estimates in this young population do not represent the same genes that at later age cause individual differences in type-D personality. A necessary next step is to determine whether the heritability of type-D personality is stable over the course of time. A second limitation is that in a classic twin design, the presence of additive genetic, nonadditive genetic effects and shared environmental effects are confounded and cannot be estimated simultaneously. Disentangling the contributions of shared environmental and dominance genetic effects requires additional data from, for example, twins reared apart, half-siblings or nonbiological siblings reared together (49). Based on the difference between MZ and DZ twin correlations, we decided to model dominance genetic effects. Therefore, in the

present sample, common environmental factors did not show a main effect on type-D status or subcomponent scores. This does, however, not exclude interaction effects to take place between shared environmental factors and genetic factors. These were not included in the present study.

In conclusion, this study provides strong evidence that genes are important in determining individual differences in type-D personality and its subcomponents. Future elucidation of the genetic and environmental factors influencing type-D personality and cardiovascular risk factors may help explain differences in cardiovascular prognosis in type-D and non-type-D patients.

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