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RELATIONSHIPS AMONG AVOIDANT PERSONALITY DISORDER, SOCIAL ANXIETY DISORDER, AND NORMATIVE PERSONALITY TRAITS: A TWIN STUDY

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Avoidant personality disorder (AvPD) and social anxiety disorder (SAD) share risk factors to a substantial degree, and both are characterized by the experience of anxiety in social situations. The authors investigated whether these disorders are differentially related to the Big Five personality traits. They also examined the underlying genetic and environmental influences on these associations. A population-based sample of 1,761 female twins was interviewed at baseline, and 1,471 of these were re-interviewed 10 years later. Associations between AvPD, SAD, and personality traits were investigated with multivariate biometric analyses. The authors found that AvPD and SAD are differentially related to several personality traits at the phenotypic, genetic, and environmental level. The genetic and environmental liability to AvPD could be fully accounted for by the genetic and environmental factors influencing SAD and personality. The findings may increase current etiological understanding of these disorders and inform future classification and treatment efforts.

Avoidant personality disorder (AvPD) and social anxiety disorder (SAD) are both characterized by fear and avoidance of social situations (Bogels et al., 2010). Several authors have argued that AvPD and SAD lie on a continuum of severity, with AvPD representing a severe form of SAD (cf. Stein & Stein, 2008). Others contend that AvPD may differ meaningfully from SAD, and that more research on potential disorder-specific features of AvPD is neces-

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sary to improve our understanding of the AvPD–SAD relationship (Bogels et al., 2010; Eikenaes, Hummelen, Abrahamsen, Andrea, & Wilberg, 2013; Marques et al., 2012). Persons with AvPD and SAD may differ in their psychological makeup, such as on normative personality traits. Identifying such differences and their underlying causes could lead to increased knowledge about the motivations, cognition, and affects of these disorders, as well as improved classification and treatment.

One way to assess whether AvPD and SAD are separable constructs is to study the factor structure of their diagnostic criteria. We recently performed factor analyses in a population-based sample of young adult women and identified an underlying two-factor structure where the diagnostic criteria for each diagnosis appeared to reflect distinct, although correlated, constructs (Torvik et al., 2016). The results are in line with a previous study of a clinical sample (Huppert, Strunk, Ledley, Davidson, & Foa, 2008).

Another way to investigate the extent of common and disorder-specific features is to study the structure of the underlying genetic and environmental risk factors influencing AvPD and SAD. This can be done by using multivariate twin models, where the extent of genetic and environmental overlap between the disorders can be estimated (Rijsdijk & Sham, 2002). Our research group has published the only two twin studies on the relationship between AvPD and SAD. In the first study, the correlation between genetic factors influencing AvPD and SAD did not differ significantly from unity, while environmental risk factors were specific to each disorder (Reichborn-Kjennerud, Czajkowski, Torgersen, et al., 2007). In a follow-up study, utilizing the increased statistical power associated with a new wave of assessment, we identified substantially overlapping, but partially distinct, genetic risk factors (Torvik et al., 2016). In addition, a partial overlap in environmental risk factors for the two disorders was identified. Taken together, findings from previous factor analytic and biometric studies are at odds with an explanatory model which holds that AvPD and SAD differ only in degree of severity and not in kind. Instead, current empirical findings suggest that AvPD and SAD are correlated but separable constructs, with differences in both the genetic and environmental risk factors for the two disorders. There is a need for further studies to identify the sources of these differences.

One possibility is that AvPD and SAD are differentially related to normative personality traits, and that they share genetic and environmental influences with such traits to an unequal extent. A considerable body of research has demonstrated substantial covariance between personality traits and various psychiatric disorders, with distinguishable patterns of associations across different types of psychopathology (Kotov, Gamez, Schmidt, & Watson, 2010; Samuel & Widiger, 2008). While analyses of observed phenotypes will allow us to test whether individuals with AvPD and SAD differ with respect to personality traits, twin analyses can provide additional information on the degree to which observed differences are due to genetic and/or environmental overlap (Rijsdijk & Sham, 2002).

Disorder-specific phenotypic associations with personality traits for AvPD and SAD have not been investigated in previous population-based studies, and no twin study addressing the association has been published.

However, the phenotypic relationship with personality traits has been assessed separately for AvPD and SAD in several studies. In these investigations, personality has most commonly been assessed according to the Five Factor Model of Personality (FFM), which comprises the following traits: neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness (cf. John, Naumann, & Soto, 2008). In a meta-analysis (Samuel & Widiger, 2008), significant associations were identified between AvPD and all FFM traits, with a high positive correlation with neuroticism, a high negative correlation with extraversion, and low to moderate negative associations with openness to experience, agreeableness, and conscientiousness. A similar pattern has been found between SAD and neuroticism and extraversion, while results on the remaining three traits have been mixed (Bienvenu et al., 2001; Kaplan, Levinson, Rodebaugh, Menatti, & Weeks, 2015; Kotov et al., 2010). In a twin study, Bienvenu, Hettema, Neale, Prescott, and Kendler, (2007) examined the relationship between SAD, neuroticism, and extraversion further. The results indicated that genetic factors underlying neuroticism and extraversion account entirely for the genetic vulnerability to SAD. No twin studies on AvPD and FFM traits have been published.

The lack of previous comparative studies prevents us from having strong, a priori expectations about differences between AvPD and SAD in their relationship to various FFM traits. As such, the majority of our analyses in the current study are of an exploratory nature. However, an older, theoretically oriented literature lends useful clues. According to Millon's (1969) original descriptions, AvPD involves a broad pattern of avoidant behavior that also includes nonsocial situations. Millon's assertion is supported by findings from more recent studies (Taylor, Laposa, & Alden, 2004). Avoidance of situations characterized by novelty, risk, or any form of strong emotions is not well captured by the SAD construct and may be a distinguishable feature of AvPD (Alden, Laposa, Taylor, & Ryder, 2002; Arntz, 1999). The FFM trait openness to experience involves a general tendency to seek new experiences and activities (cf. John et al., 2008). Hence, we expected to observe lower levels of openness to experience in AvPD compared to SAD, and we predicted that AvPD shares genetic and environmental influences with this FFM trait to a greater extent than SAD.

The goal of the current investigation was to address the relationship between AvPD, SAD, and normative personality traits at the phenotypic, genetic, and environmental levels. We had the following specific aims: first, to estimate the associations between FFM traits and AvPD and SAD; second, to test whether AvPD and SAD are differentially related to FFM traits; third, to estimate the degree to which genetic and environmental influences are shared between FFM traits and the two disorders; fourth, to test whether the degree of genetic and environmental overlap with FFM traits differs between AvPD and SAD; and fifth, to estimate to what extent differences in FFM traits explain differences between AvPD and SAD at the level of genetic and environmental risk factors.

METHOD

STUDY SAMPLE

The twins were identified through information from the Medical Birth Registry of Norway, established January 1, 1967, which receives mandatory notification of all live births and stillbirths of at least 16 weeks' gestation. The Norwegian Institute of Public Health Twin Panel (NIPHTP) is based on all Norwegian twins born between 1967 and 1979. During this period, 15,370 twins were born in Norway.

In 1998, 12,698 twins (6,349 twin pairs) were invited to a questionnaire study, to which 8,045 persons (63%) responded (3,334 pairs and 1,377 single responders). Data from the current report stem from an interview-based study based on this twin sample. The "Axis I and Axis II psychiatric disorders in Norwegian Twins" (AI/AII study) was conducted between 1999 and 2004 (Wave 1), with a second wave of data collection between 2010 and 2011 (Wave 2). In Wave 1, all complete pairs from the 1998 questionnaire study in which both twins had agreed to further contact (3,153 pairs) were invited, as well as 68 pairs unintentionally drawn directly from the NIPHTP. Altogether, 2,801 twins (1,776 females) were assessed at personal interview for *DSM-IV* Axis I and Axis II disorders. For practical reasons, 231 interviews (8.3%) were conducted by telephone. The response rate was 44%.

Wave 2 was performed using the same instruments. However, to maximize the participation rate, all the interviews were conducted by telephone. In addition, a self-report questionnaire tapping normative personality traits was administered to the participants. Of the 2,801 participants in Wave 1, 17 had withdrawn their consent to participate in further research, 14 had unknown addresses, and 12 had died, leaving 2,758 eligible individuals who were invited to participate in the follow up study. After two written reminders and a final telephone contact to nonresponders, 2,284 twins (1,482 females) were interviewed in Wave 2, resulting in a response rate of 82.8%.

Among females, 187 reported having lifetime AvPD, SAD, or both disorders at least once. At Wave 1, 1,761 females had valid data for AVPD and SAD; 445 monozygotic (MZ) pairs, 256 dizygotic (DZ) pairs, and 359 single twins (of which 341 in dizygotic opposite-sex [DZO] pairs). Partially complete data were available for 15 women. At Wave 2, 1,471 females had complete data for personality traits, AVPD, and SAD; 354 MZ, 174 DZ, and 415 single twins (of which 285 were in DZO pairs). Partially complete data were available for 11 women. Only 45 males in our sample met the diagnostic criteria for AvPD and/or SAD. Even considering dimensional representations of the disorders, the prevalence was too low to permit biometric modeling. Consequently, only female twins were included in our study. The mean age of the women was 28.6 years (*SD* = 4.3, range 19–36) at Wave 1 and 37.8 (*SD* = 3.8, range 30–44) at Wave 2. The mean number of years of education was 14.8 (*SD* = 2.5, range 9–26) at Wave 1 and 15.6 (*SD* = 2.9, range 9–30) at Wave 2. A total of 527 women (29.7%) were married and 627 (35.4%) were living in cohabitation at Wave 1, while 764 women (51.6%) were married and 382 (25.8%) were living in cohabitation at Wave 2.

In Appendix Table A1, we present the occurrence of full and subthreshold AvPD and SAD for both women and men at Wave 1 and Wave 2. There were no notable differences in occurrence in the different twin groups. Appendix Table A2 provides information on rates of six *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM-IV*; American Psychiatric Association, 1994) Axis I disorders among women with AvPD and SAD. The stability and change of AvPD and SAD among women across the two waves of assessment have been presented in a previous report (Torvik et al., 2016)

Twin zygosity was determined by a combination of questionnaire items and genotyping. The misclassification rate was estimated to be less than 1.0% (Harris, Magnus, & Tambs, 2006), which is unlikely to substantially bias results (Neale, 2003).

ETHICS

After a complete description of the study, written informed consent was given by all participants, and the study protocol was approved by the Regional Committee for Medical and Health Research Ethics and the Norwegian Data Inspectorate.

MEASURES

Avoidant Personality Disorder. A Norwegian version of the Structured Interview for *DSM-IV* Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1995; see also Helgeland, Kjeldsberg, & Torgersen, 2005) was used to assess *DSM-IV* AvPD. The *DSM-IV* criterion associated with each question was rated as 0 = "not present," 1 = "subthreshold," 2 = "present," and 3 = "strongly present." The SIDP-IV uses the "5-year rule," meaning that behaviors, cognitions, and feelings that predominated for most of the past 5 years are considered to be representative of an individual's personality.

We used a dimensional modeling approach, constructing ordinal count variables based on the number of endorsed diagnostic criteria. Dimensional representations are often considered a better conceptualization of PDs than categorical clinical diagnoses (Widiger & Samuel, 2005). To optimize statistical power and produce maximally stable results, we summed the number of criteria with a score of 1 or higher; that is, each AvPD criterion scored ≥ 1 increased the count variable by 1. This variable was meant to reflect AvPD traits that do not necessarily constitute a clinical diagnosis. Multiple threshold tests previously performed by our research group indicate that the use of individual subthreshold AvPD criteria reflect varying levels of severity on a single continuum of liability (Reichborn-Kjennerud, Czajkowski, Neale, et al., 2007).

High counts were infrequent. To avoid empty cells in the twin analyses, we truncated the AvPD variable by collapsing the upper categories for the summed score. The resulting variable included six ordered categories (0–5). Our research group has previously performed multiple threshold tests which indicate that the number of positive criteria for AvPD represent different de-

grees of severity of the disorder (Reichborn-Kjennerud, Czajkowski, Neale, et al., 2007).

Interrater reliability at Wave 1 was assessed based on scoring by two raters of 70 audiotaped interviews. Intraclass and polychoric correlations for the number of endorsed AvPD criteria at the subthreshold level were 0.96 and 0.97, respectively. At Wave 2, two interviewers rescored 95 audiotaped interviews. Intraclass and polychoric correlations for the number of endorsed criteria at subthreshold level were 0.84 and 0.92, respectively.

Social Anxiety Disorder. Lifetime *DSM-IV* SAD diagnosis was assessed using the Norwegian version of the computerized Composite International Diagnostic Interview (CIDI; Wittchen & Pfister, 1997; see also Landheim, Bakken, & Vaglum, 2003). This is a structured diagnostic interview developed by the World Health Organization (Wittchen, 1994; Wittchen, Lachner, Wunderlich, & Pfister, 1998). Individuals who feared at least one social situation and who were worried about showing anxiety symptoms, but who did not otherwise satisfy diagnostic criteria, were considered to have subthreshold SAD. This definition has been used in previous studies (Torvik et al., 2016). We used a dimensional modeling approach, constructing ordinal count variables for SAD at both waves of assessment (No SAD = 0, subthreshold SAD = 1, and SAD = 2).

As a result of skipping patterns, it was not possible to rescore audiotaped interviews. However, the CIDI has shown good test–retest and interrater reliability in other studies (Wittchen, 1994; Wittchen et al., 1998).

Normative Personality Traits. Personality traits were assessed with the Big Five Inventory (BFI; John & Srivastava, 1999), a 44-item self-report inventory measuring the five basic personality traits specified by the FFM: neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness. Each of the 44 items in the BFI is rated on a 5-point Likert scale from 1 (*disagree a lot*) to 5 (*agree a lot*). Scores on each of the five personality traits are calculated as the sum of scores on the respective items. All five variables were continuous and approximately normally distributed.

Means, standard deviations, and the Cronbach's alpha obtained for the BFI subscales, as well as correlations between the subscales, are presented in Appendix Table A3.

Validity. The validity of our measures of AvPD and SAD have been described previously (Torvik et al., 2016). The BFI has satisfactory reliability and validity, widespread international use, and a translated version previously used successfully in Norway (DeYoung, 2006; Engvik & Follesdal, 2005).

Procedure. Interviewers at Wave 1 were mainly senior clinical psychology graduate students and experienced psychiatric nurses. Interviewers at Wave 2 included senior clinical psychology graduate students, experienced psychiatric nurses, and experienced clinical psychologists who were interviewers also at Wave 1. For the SIDP-IV, interviewers were trained by one psychiatrist and two psychologists with extensive previous experience with

the instrument. For the CIDI, interviewers received a standardized training program administered by teachers certified by WHO. The interviewers were supervised closely during the data collection period. At both waves of data collection, the SIDP-IV interview was conducted after the CIDI interviews, which helped interviewers to distinguish long-standing behaviors from temporary states resulting from Axis I disorders. Different interviewers assessed each twin in a pair.

STATISTICAL ANALYSES

Definition of the AvPD and SAD Variables. Using structural equation modeling, we have previously shown that a model of separate, although highly correlated, risk factors accounts for AvPD and SAD (Torvik et al., 2016). In this model, one factor influenced AvPD at Wave 1 and Wave 2 and was constrained to influence AvPD equally strongly at both time points, whereas the other factor was constrained to influence SAD equally strongly at Wave 1 and Wave 2. These latent, time-invariant variables were used in the current study. The advantages of performing analyses based on these latent constructs are that they are free from the random measurement errors that bias the estimates from each point of data collection, and that they represent stable features of both AvPD and SAD.

Phenotypic Analyses. First, we estimated polyserial correlations between AvPD and each FFM trait and between SAD and each FFM trait. Next, we addressed whether AvPD and SAD are differentially related to personality traits. We performed a chi-square difference test to assess whether a model where the correlations between AvPD and all personality traits and SAD and all personality traits were allowed to take different values provided a significantly better fit to the data than an alternative model where the correlations with all FFM traits were fixed to be equal for AvPD and SAD. The same test was subsequently performed for each of the five FFM traits separately. Statistical dependency between cotwins and within individuals at the two time points was accounted for by using a sandwich estimator (i.e., the complex sample option) for the robust weighted least squares (WLSMV) in Mplus 7.31 (Muthén & Muthén, 2012).

Biometric Analyses. Next, we fitted multivariate twin models to investigate the structure of the underlying genetic and environmental influences on the observed covariance between AvPD, SAD, and FFM traits. In the classical biometric twin model, individual differences in liability are assumed to arise from three latent factors: additive genetic (A), that is, genetic effects that combine additively; common or shared environment (C), which includes all environmental effects that contribute to their similarity; and individual-specific or unique environment (E), which includes all environmental effects that make the twins different plus random measurement error (Jinks & Fulker, 1970). Because MZ twins share all their genes and DZ twins share, on average, 50% of their segregating genetic material, additive genetic effects contribute twice as much to the resemblance in MZ compared to DZ twins. By

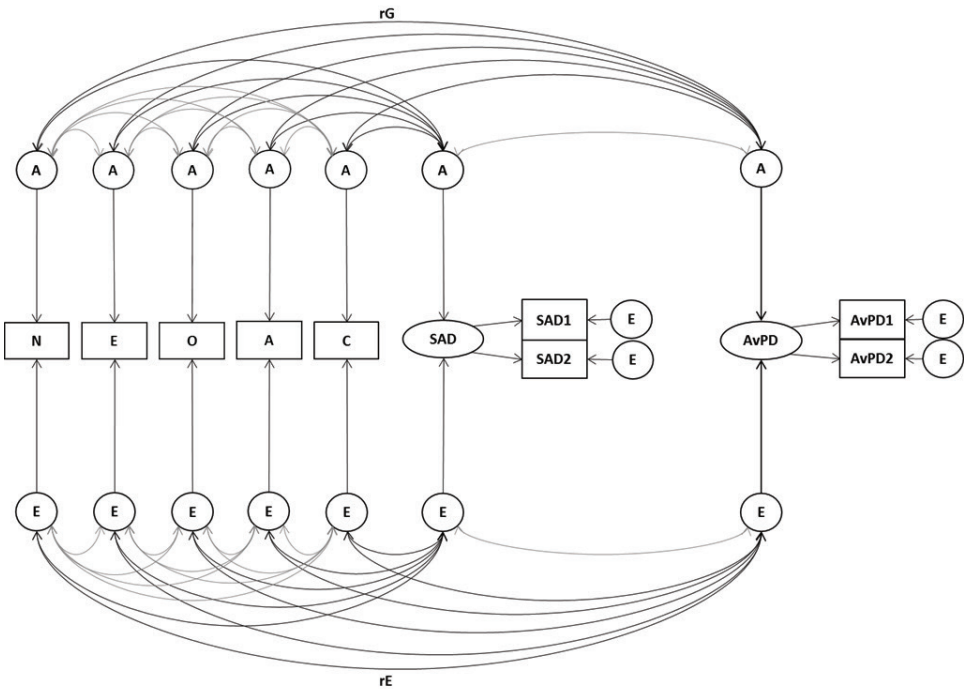

FIGURE 1. The path diagram illustrates the correlated factor model. By convention, the squares represent observed variables, while circles are used for the latent variables. The double-headed curved arrows represent the genetic and environmental correlations between each of the five personality traits and SAD and AvPD. These are the paths that were selectively constrained to be equal in the nested submodels. The single-headed arrows represent regression coefficients. N = Neuroticism; E = Extraversion; O = Openness to experience; A = Agreeableness; C = Conscientiousness; A = Latent genetic factor; E = Latent environmental factor; rG = genetic correlation; rE = environmental correlation.

definition, MZ and DZ twins share all of their C factors and none of their E factors.

In univariate twin analyses, data on cotwin similarity for a single phenotype are used to partition the observed variance into A , C, and E components (Rijsdijk & Sham, 2002). In multivariate analyses, the twin model can be extended to explore the contributions of genetic and environmental factors to the observed covariance between two or more phenotypes (Martin & Eaves, 1977).

In the present study, the latent, time-invariant risk factors for AvPD and SAD were analyzed together with all five personality traits, yielding a measurement model with nine observed variables (AvPD and SAD measured at two time points, and five personality traits measured at Wave 2). A graphical presentation of this model is given in Figure 1. We have previously shown that AvPD and SAD are best accounted for by an AE model; that is, there were no contributions from C factors to the covariance between the disor-

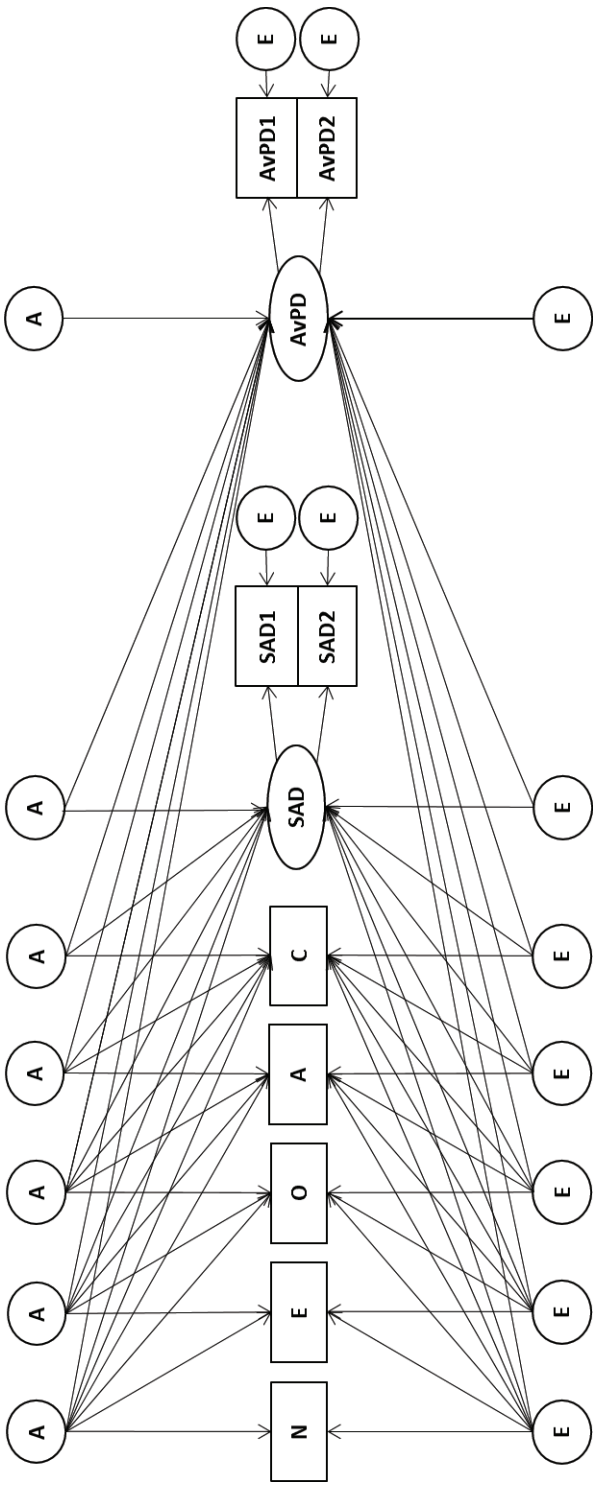

FIGURE 2. The path diagram illustrates the Cholesky decomposition approach. By convention, the squares represent observed variables, while circles are used for the latent variables. The single-headed arrows represent regression coefficients. The latent "A" and "E" variables that the latent AvPD variable is regressed upon represent genetic and environmental effects that are unique to AvPD. N = Neuroticism; E = Extraversion; O = Openness to experience; A = Agreeableness; C = Conscientiousness; A = Latent genetic factor; E = Latent environmental factor.

TABLE 1. Polyserial Correlations Between Personality Traits and AvPD and SAD

	AvPD	SAD
Neuroticism	.54**	.54**
Extraversion	–.75**[a]	–.49**
Openness	–.22**[a]	.05
Agreeableness	–.26**[a]	–.18**
Conscientiousness	–.34**	–.36**

**$p < .01$. [a]Different polyserial correlation with personality trait across AvPD and SAD, $p < .05$.

ders. Correlations between the genetic factors influencing AvPD, SAD, and the personality traits were estimated, as were correlations between the corresponding environmental factors. Next, we specified a submodel in which the genetic and environmental correlations with all five personality traits were constrained to be of equal magnitude for AvPD and SAD, and compared the fit to models without these constraints. Next, a similar analysis was performed for each personality trait separately.

We proceeded to perform Cholesky decomposition analyses, which are algebraically equivalent to the "correlated factors" solution described above, but quantify the relationships between the phenotypes in a different manner (Loehlin, 1996). In multivariate Cholesky analyses, the shared and specific components of genetic and environmental variance can be estimated sequentially for the phenotypes in the model. We used this approach to assess the extent to which the five normative personality traits and SAD together accounted for genetic and environmental variance in AvPD, and to estimate the extent to which there were residual influences unique to AvPD. A graphical illustration of the Cholesky decomposition analysis is presented in Figure 2.

In our analyses, the fit of constrained, nested submodels was compared with the full model by assessing the difference in –2 times log likelihood (Δ-2LL), which is asymptotically χ^2 distributed. If the difference in χ^2 is nonsignificant, a simpler, more restricted model is preferred. In addition, we used the Akaike information criterion (AIC), a parsimony corrected measure of model fit (Akaike, 1987). Models with low AIC value are preferred. The models were fitted to raw data using full information maximum likelihood (FIML) for categorical data as the estimation procedure in OpenMx 2.0 (Neale et al., 2016) for R 3.1.2 (R Core Team, 2014). The FIML method utilizes all data, from both complete and incomplete twin pairs, and provides better estimates in structural equation models than traditional missing data methods (Enders & Bandalos, 2001). Because the AvPD and SAD variables were ordinal, we used a liability-threshold model, assuming that ordered categories are imprecise indicators of unobserved, normally distributed liabilities (Falconer, 1965).

RESULTS

PHENOTYPIC ANALYSES

Results from the analyses of phenotypic associations between AvPD, SAD, and FFM traits are presented in Table 1 (polyserial correlations) and Table

TABLE 2.Fit Indices for Models of Phenotypic Relationships Between Personality Traits, AvPD, and SAD

Model	$\Delta\chi^2$	Δ *df*	*p* Value
All personality traits equal	97.22	5	.00
Neuroticism equal	0.01	1	.93
Extraversion equal	47.63	1	.00
Openness equal	55.73	1	.00
Agreeableness equal	4.44	1	.04

$\Delta\chi^2$ = chi-square value, Δ *df* = difference in degrees of freedom.

2 (model fit indices). As shown in Table 1, a positive association was identified between AvPD and neuroticism. AvPD was negatively associated with extraversion, openness to experience, agreeableness, and conscientiousness. We found a substantial, positive association between SAD and neuroticism; a negative association with extraversion, agreeableness, and conscientiousness; and no statistically significant correlation with openness to experience.

In a chi-squared difference test, the polyserial correlations between the two disorders and all FFM traits could not be constrained to equality (Table 2). Studying each trait separately, we found that AvPD and SAD were differentially related to extraversion, openness to experience, and agreeableness. For each of these traits, there were stronger negative correlations with AvPD.

BIOMETRIC ANALYSES

In Appendix Table A4, we present polychoric correlations within MZ and DZ twin pairs, as well as heritability estimates for all FFM traits and for AvPD and SAD at both waves of assessment.

The genetic correlations between AvPD, SAD, and personality traits are presented in Table 3. For AvPD, we found a positive genetic correlation with neuroticism, a strong negative correlation with extraversion, and somewhat weaker negative genetic correlations with openness to experience, agreeableness, and conscientiousness. Genetic influences on SAD were highly positively correlated with genetic influences on neuroticism and substantially negatively correlated with extraversion. More modest genetic correlations were identified for openness to experience, agreeableness, and conscientiousness.

As shown in Table 4, the measurement model where the genetic correlations between AvPD and FFM traits and SAD and FFM traits were constrained to be of equal value had a significantly worse fit to the data than a model where the correlations were estimated freely. Furthermore, fixing correlations to be equal for one personality trait one at a time, we found that the genetic correlations differed for AvPD and SAD on neuroticism, extraversion, and openness to experience. A stronger negative correlation with extraversion and openness to experience was found for AvPD, while a stronger positive correlation was identified between SAD and neuroticism.

Estimates of environmental correlations are presented in Table 3. A positive environmental correlation was found between AvPD and neuroticism, while negative associations were identified for the remaining four personality traits. A similar pattern of environmental correlations was found for SAD.

TABLE 3. Genetic and Environmental Correlations Between Personality Traits and AvPD and SAD

	Genetic Correlations		Environmental Correlations	
	AvPD	**SAD**	**AvPD**	**SAD**
Neuroticism	.56*[a]	. 81*	.53*[b]	.22*
Extraversion	–.79*[a]	–.60*	–.74*[b]	–.33*
Openness	–.18*[a]	.11	–.28*[b]	–.06
Agreeableness	–.42*	–.29*	–.16*	–.09
Conscientiousness	–.50*	–.30*	–.15*	–.35*

*$p < .05$. [a]Different genetic correlation with personality trait across AvPD and SAD, $p < .05$. [b]Different environmental correlation with personality trait across AvPD and SAD, $p < .05$.

Model fit indices for the tests of environmental correlations are shown in Table 4. A measurement model where all environmental correlations between AvPD, SAD, and FFM traits were constrained to be equal did not fit well. Neuroticism, extraversion, and openness to experience were differentially related to the two disorders. Again, stronger negative correlations with extraversion and openness to experience were identified for AvPD. The positive environmental correlation for neuroticism was comparably higher with AvPD than with SAD.

In Appendix Table A5, we provide all the estimated genetic and environmental correlations across all primary variables in the measurement model.

FFM traits could fully account for the difference in etiology between AvPD and SAD. After adjusting for all FFM traits and SAD using Cholesky decomposition, we estimated the factor loadings of AvPD-specific genetic and environmental effects to be 0.02 (95% CI [0.00, 0.13]) and 0.23 (95% CI [0.00, 0.40]), respectively. Constraining both of these paths to zero did not result in a significant deterioration of model fit.

DISCUSSION

Our main finding is that AvPD and SAD are differentially related to several normative personality traits. This holds true at the level of observed phenotypes, but also when the underlying structure of genetic and environmental influences is explored. A second important finding from our investigation is that etiological factors underlying FFM traits could fully account for the differences in genetic and environmental influences on AvPD and SAD.

Consistent with prior research, we found a positive relationship with neuroticism and a negative association with extraversion for both AvPD and SAD in the phenotypic analyses (Kotov et al., 2010; Samuel & Widiger, 2008). The results for the remaining three FFM traits are also in line with previous findings.

The analyses of observed associations between AvPD, SAD, and personality traits showed lower openness to experience in individuals with AvPD than in individuals with SAD, a finding compatible with our a priori hypothesis of differences between the two disorders with respect to their association with this trait. We speculate that the lower openness to experience among individuals with AvPD is related to a disorder-specific feature of nonsocial

TABLE 4. Fit Indices for Models of the Genetic and Environmental Relationship Between Personality Traits, AvPD, and SAD

	Genetic Relationships				Environmental Relationships			
Model	**Δ –LL**	**Δ *df***	**Δ AIC**	***p* Value**	**Δ –LL**	**Δ *df***	**Δ AIC**	***p* Value**
All personality traits equal	47.08	5	37.08	.00	38.17	5	28.17	.00
Neuroticism equal	7.14	1	5.14	.01	7.39	1	5.39	.01
Extraversion equal	4.77	1	2.77	.03	12.16	1	10.16	.00
Openness equal	9.71	1	7.71	.00	4.13	1	2.13	.04
Agreeableness equal	1.00	1	–1.01	.32	.33	1	–1.67	.56
Conscientiousness equal	4.00	1	2.00	.05	3.48	1	1.48	.06

Note. Base model: No constraints on correlations, Akaike's information criterion (AIC) value = 3400.80, *df* = 15007. Δ -LL = difference in log likelihood, Δ *df* = difference in degrees of freedom, Δ AIC = difference in Akaike's information criterion.

avoidance, which has been thoroughly described in an older, less empirically based literature (cf. Millon, 1969).

We also identified more substantial negative correlations between AvPD and extraversion and agreeableness. Our findings differ from the results in a previous study of a patient sample, where comparably lower levels of conscientiousness and higher levels of neuroticism among individuals with AvPD were identified (Hummelen, Wilberg, Pedersen, & Karterud, 2007). Population-based studies are better suited than clinical studies when the extent of co-occurrence between different behavioral phenotypes is assessed (Berkson, 1946). The treatment-seeking part of a general population is more likely to suffer from several concurrent psychiatric disorders. Patients may also have a different personality structure than individuals with psychiatric disorders who do not receive treatment. It is possible that the discrepancy of findings between our study and the clinical study can be explained by the use of different types of study samples.

In the first twin study with simultaneous assessment of AvPD and SAD in relation to normative personality traits, we show that genetic risk for these disorders overlaps to a substantial degree with genetic factors that contribute to individual differences in FFM personality. We find especially high estimates for the traits of neuroticism and extraversion. Substantial genetic overlap between SAD and these two traits has been demonstrated in previous studies (Bienvenu et al., 2007; Hettema, Neale, Myers, Prescott, & Kendler, 2006). In the current study, we demonstrate a similar pattern of correlations for AvPD.

An important finding in our study is that the disorders differ in the degree to which they share genetic and environmental influences with neuroticism, extraversion, and openness to experience. With these results, we extend the findings from our previously published study of shared and specific risk factors for AvPD and SAD (Torvik et al., 2016) and show that these FFM traits are a source of disorder-specific genetic and environmental influences. Indeed, the results imply that personality traits serve an important role in accounting for differences between AvPD and SAD. While Torvik et al. identified genetic and environmental risk factors for AvPD that were not shared with SAD, the current results indicate that FFM traits and SAD together may

account for all the genetic and environmental liability to AvPD. Because we studied the time-invariant features of SAD and AvPD, our results apply only to genetic and environmental effects that were stable across the two time points. Time-specific effects also influence individual variation in AvPD and SAD, and the overlap between such effects was not assessed in our study.

A notable finding from the biometric analyses is that while the phenotypic correlation with neuroticism appears to be equal for both disorders, the degree of genetic and environmental overlap with this trait seems to be disorder-specific. We did not expect a priori to find a more substantial environmental overlap between AvPD and neuroticism and a larger overlap of genetic factors between SAD and this personality trait. Cautious interpretation is warranted until this finding is replicated and, ideally, relevant specific genetic variants and environmental factors are identified.

Our study may contribute to an improved understanding of the AvPD–SAD relationship. The identification of differential genetic and environmental associations with personality traits advances current knowledge about the etiology of these disorders. Furthermore, our findings could provide hypotheses about results from gene-finding studies and studies with measures of specific environmental risks (e.g., stressful life events). It has been robustly shown in previous studies that AvPD shares underlying risk factors with psychotic disorders, common symptom disorders, and other personality disorders (Fogelson et al., 2007; Kendler et al., 2011; Roysamb et al., 2011). Nevertheless, the current results support a relatively parsimonious explanatory model where all genetic variants and environments that affect stable aspects of AvPD can be identified among those that contribute to individual variability in SAD and normative personality traits.

The interpretation of our results in the light of the theoretical literature on the relationship between personality traits and psychopathology is not straightforward. Among several explanatory models, our findings are in accordance with a common liabilities model, where personality traits are associated with AvPD and SAD due to shared underlying etiological factors (cf. Widiger & Smith, 2008). However, direct unidirectional or bidirectional causal relationships between personality traits and the disorders may also exist. Also, the relationship between the traits and AvPD and SAD may be pathoplastic; that is, the disorders and personality traits affect the presentation of one another. In such a model, AvPD and SAD could be conceptualized as one disorder that is expressed and recognized in two different forms depending on an individual's personality traits. Although a comprehensive discussion of nosological implications of our findings is beyond our scope here, we contend that the increased knowledge about shared and distinct features of AvPD and SAD can inform future efforts to classify these disorders.

Our current findings may be relevant for clinical practice because the identification of distinct features of AvPD could inform the design of prevention and treatment programs. Overall, relatively few data are available on the comparative effects of treatments for AvPD and SAD, but previous findings indicate that AvPD is associated with a larger symptom burden, more relapses, and a slower rate of functional improvement during and after treatment with cognitive-behavioral therapy (Feske, Perry, Chambless, Ren-

neberg, & Goldstein, 1996; Oosterbaan, van Balkom, Spinhoven, de Meij, & van Dyck, 2002). Normative personality traits have been described as possible moderators of treatment responsivity to psychotherapy (Sanderson & Clarkin, 2002). We speculate that the distinct link to low openness to experience in AvPD and lower levels of agreeableness and extraversion in persons with AvPD compared to persons with SAD can have a negative impact on the establishment of therapeutic rapport, the motivation to question existing cognitive biases, and patients' tendency to drop out of treatment programs. Knowledge about group-level personality differences between AvPD and SAD can be useful for clinicians in their planning and provision of care to individual patients. There is a need for future clinical trials testing treatment programs developed specifically to attend to distinct features of AvPD.

Our study represents one step toward the identification of the disorder-specific features of AvPD. In addition to further investigations of the relationship between AvPD, SAD, and personality traits, there is a need to address other potential sources of differences in future twin studies. Findings from previous studies of phenotypic associations indicate that AvPD is more strongly related to depression than SAD is (Huppert et al., 2008). Results by Marques et al. (2012) indicate that emotional guardedness in close interpersonal relationships is a distinguishable feature of AvPD. Eikenaes and colleagues (Eikenaes, Egeland, Hummelen, & Wilberg, 2015; Eikenaes et al., 2013; Eikenaes, Pedersen, & Wilberg, 2016) have found differences in attachment styles and personality dysfunction across AvPD and SAD in a Norwegian patient sample, as well as an increased level of self-reported experience of childhood neglect among AvPD patients. A significantly elevated prevalence of AvPD in first-degree relatives of patients with schizophrenia has also been identified, and AvPD may be genetically related to the schizophrenia spectrum disorders (Fogelson et al., 2010). Such features of AvPD and SAD should be further explored in future population-based twin studies.

LIMITATIONS

The strengths of the present study include the use of structured diagnostic interviews, longitudinal follow-up, and a population-based twin sample, but some limitations must be mentioned.

First, substantial attrition was observed in this sample from the National Medical Birth Registry through two previous questionnaire studies to the first wave of interviews. However, previous detailed analyses have shown that cooperation was strongly predicted by female sex, monozygosity, and higher educational status, but not by symptoms of psychiatric disorders and substance abuse (Tambs et al., 2009). Further attrition from Wave 1 to Wave 2 was low, with a participation rate of 82.2% at Wave 2. Attrition in the MZ female group was somewhat lower than in the DZ female group, but it is unlikely that this has biased the current results. Participation in Wave 2 was predicted by high education ($p < .001$ adjusted for sex and age), female sex ($p = .003$), and monozygosity ($p = .001$). Nonparticipants in Wave 2 had, on average, 0.82 more subthreshold personality disorder criteria than participants ($p < .001$). Of the 10 PDs assessed at Wave 1, criteria were significantly

higher in nonparticipants in Wave 2 only for antisocial personality disorder (0.09 criteria difference, $p < .001$) and narcissistic personality disorder (0.09 criteria difference, $p = .002$). Neither the total number of Axis I disorders nor any specific disorder was significantly higher in the nonparticipation group.

Second, only females were included in our analyses, and our findings cannot necessarily be extrapolated to the male population. Third, we studied young Norwegian adults, and the results may not apply to other age groups or individuals from a different ethnic background than the participants. Fourth, due to a limited number of SAD cases, we could not distinguish between generalized and performance-only SAD. It is possible that the relationship with both AvPD and personality traits differs between these types of SAD. Fifth, FFM traits were measured only at Wave 2. Thus, random measurement error in these variables was not accounted for, and studies of the longitudinal relationship between FFM traits and AvPD and SAD could not be performed. Sixth, findings from a recent multigenerational family study indicate assortative mating for AvPD and SAD (Isomura et al., 2015). Absence of assortative mating is an underlying assumption of biometric twin models; if assortative mating is present, it may introduce bias to the estimated genetic and environmental correlations (Rijsdijk & Sham, 2002). Seventh, the relatively low number of participants with either AvPD or SAD in our sample indicates a risk of Type II error. The results from the Cholesky analyses must be interpreted with caution because we may not have had sufficient statistical power to identify small true genetic and environmental influences unique to AvPD. Despite the absence of significant disorder-specific etiological effects on AvPD in our analysis, we cannot strongly infer that such effects are not present. Eighth, the sequencing of variables in the Cholesky decomposition ideally reflects the assumed temporal ordering of the variables. We made no such assumption in our current analyses. Our primary goal of the variance decomposition was not temporality, but rather to test the presence of any residual genetic or environmental variance in AvPD while accounting for all genetic and environmental influences on personality traits and SAD. The presence of residuals or specific effects on the final variable is not biased by the absence of temporality assumptions.

CONCLUSION

AvPD and SAD are differentially related to several normative personality traits at the phenotypic, genetic, and environmental levels. Our current results indicate that normative personality traits may account for the previously identified differences in underlying genetic and environmental risks for the two disorders.

APPENDIX

TABLE A1. Prevalences of AvPD and SAD

	Women		Men	
	Wave 1 *n* (%)	Wave 2 *n* (%)	Wave 1 *n* (%)	Wave 2 *n* (%)
TB.AvPD diagnosis				
Not present	1,724 (97.5)	1,449 (97.9)	1,006 (98.6)	795 (99.1)
Present	45 (2.5)	31 (2.1)	14 (1.4)	7 (0.9)
Subtreshold AvPD criteria				
0	969 (54.8)	981 (66.3)	565 (55.4)	595 (74.2)
1	348 (19.7)	238 (16.1)	225 (22.1)	102 (12.7)
2	215 (12.2)	116 (7.8)	95 (9.3)	55 (6.9)
3	115 (6.5)	54 (3.7)	70 (6.9)	24 (3.0)
4	53 (3.0)	50 (3.4)	34 (3.3)	15 (1.9)
5 or more	70 (4.0)	41 (2.8)	31 (3.0)	11 (1.4)
SAD				
No SAD	1,372 (77.4)	1,187 (80.1)	854 (83.8)	713 (88.9)
Subthreshold	312 (17.6)	196 (13.2)	142 (13.9)	76 (9.5)
SAD	89 (5.0)	98 (6.6)	23 (2.3)	13 (1.6)
Total *n*	1,776 (100.0)	1,482 (100.0)	1,024 (100.0)	802 (100.0)

AvPD = Avoidant personality disorder; SAD = Social anxiety disorder.

TABLE A2. Rates of *DSM-IV* Axis I Disorders in Women With AvPD and SAD

	AvPD				SAD			
	Wave 1		Wave 2		Wave 1		Wave 2	
DSM-IV disorder	*n* (%)	*r* (*SE*)	*n* (%)	*r* (*SE*)	*n* (%)	*r* (*SE*)	*n* (%)	*r* (*SE*)
Major depressive disorder	21 (47.7)	0.40 (0.07)	16 (51.6)	0.34 (0.09)	39 (43.8)	0.42 (0.06)	49 (50.0)	0.41 (0.06)
Generalized anxiety disorder	6 (13.6)	0.39 (0.11)	7 (22.6)	0.47 (0.10)	14 (15.7)	0.51 (0.07)	21 (21.4)	0.59 (0.07)
Panic disorder	8 (18.2)	0.39 (0.10)	9 (29.0)	0.50 (0.09)	23 (25.8)	0.59 (0.06)	26 (26.5)	0.61 (0.06)
Agoraphobia	14 (31.8)	0.49 (0.08)	12 (38.7)	0.57 (0.08)	30 (33.7)	0.59 (0.06)	36 (36.7)	0.70 (0.05)
Specific phobias	18 (40.9)	0.21 (0.08)	18 (58.1)	0.41 (0.08)	45 (50.6)	0.35 (0.06)	47 (48.0)	0.39 (0.06)
Alcohol use disorder	6 (13.6)	0.22 (0.11)	1 (3.2)	–0.02 (0.17)	17 (19.1)	0.37 (0.06)	12 (12.2)	0.36 (0.09)

AvPD = Avoidant personality disorder; SAD = Social anxiety disorder; *r* = Polychoric correlation; *SE* = standard error.

TABLE A3. Response Characteristics Among Women by Subscales of the Big Five Inventory

Subscale	Means (*SD*)	α	N	E	O	A	C
N	2.58 (0.69)	0.85	1.00	—	—	—	—
E	3.51 (0.65)	0.85	–0.45 (0.02)	1.00	—	—	—
O	3.30 (0.56)	0.80	–0.02 (0.03)	0.28 (0.02)	1.00	—	—
A	3.99 (0.42)	0.71	–0.39 (0.02)	0.27 (0.02)	0.06 (0.03)	1.00	—
C	3.94 (0.46)	0.72	–0.44 (0.02)	0.32 (0.02)	–0.02 (0.03)	0.40 (0.02)	1.00

SD = Standard deviation; α = Cronbach's alpha; N = Neuroticism; E = Extraversion; O = Openness to experience; A = Agreeableness; C = Conscientiousness.

TABLE A4. Within-Pair Correlations and Heritability Estimates Among Women

	MZ correlations [95% CI]	DZ correlations [95% CI]	Heritability [95% CI]
N	0.45 [0.36, 0.53]	0.16 [0.01, 0.30]	0.45 [0.36, 0.55]
E	0.53 [0.45, 0.60]	0.30 [0.15, 0.43]	0.51 [0.42, 0.60]
O	0.50 [0.42, 0.58]	0.14 [–0.01, 0.28]	0.48 [0.39, 0.57]
A	0.27 [0.17, 0.36]	0.09 [–0.06, 0.24]	0.26 [0.16, 0.36]
C	0.43 [0.34, 0.51]	0.21 [0.06, 0.35]	0.43 [0.34, 0.52]
AvPD1	0.38 [0.28, 0.48]	0.30 [0.16, 0.43]	0.39 [0.29, 0.48]
AvPD2	0.35 [0.22, 0.48]	0.32 [0.13, 0.51]	0.38 [0.25, 0.49]
SAD1	0.46 [0.33, 0.59]	0.29 [0.09, 0.49]	0.47 [0.34, 0.59]
SAD2	0.52 [0.38, 0.66]	0.34 [0.10, 0.59]	0.53 [0.39, 0.65]

MZ = Monozygotic twin pairs; DZ = Dizygotic twin pairs; N = Neuroticism; E = Extraversion; O = Openness to experience; A = Agreeableness; C = Conscientiousness; AvPD1 = Avoidant personality disorder at Wave 1; AvPD2 = Avoidant personality disorder at Wave 2; SAD1 = Social anxiety disorder at Wave 1; SAD2 = Social anxiety disorder at Wave 2.

TABLE A5. Genetic and Environmental Correlations Between AvPD, SAD, and Normative Personality Traits

	N	E	O	A	C	AvPD	SAD
N	1.00	–0.41	–0.04	–0.36	–0.31	0.53	0.22
E	–0.44	1.00	0.27	0.30	0.30	–0.74	–0.33
O	–0.01	0.27	1.00	0.11	0.06	–0.28	–0.06
A	–0.34	0.19	–0.08	1.00	0.35	–0.16	–0.09
C	–0.51	0.33	–0.13	0.51	1.00	–0.15	–0.36
AvPD	0.56	–0.78	–0.17	–0.42	–0.50	1.00	0.82
SAD	0.81	–0.60	0.11	–0.30	–0.30	0.62	1.00

Note. Genetic correlations below the diagonal. Environmental correlations above the diagonal. N = Neuroticism; E = Extraversion; O = Openness to experience; A = Agreeableness; C = Conscientiousness; AvPD = Avoidant personality disorder; SAD = Social anxiety disorder.

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