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Hannah Friedman March 25, 2019

The Genetic and Environmental Etiology of Social Phobia in Youth and the Etiological Overlap Between Shyness, Extraversion, and Social Phobia

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Abstract

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This study examined the genetic and environmental etiology of social phobia and the etiological overlap between social phobia, shyness, and extraversion in youth. We used a representative sample of twins (N = 874 twin pairs) ages 4 to 19 whose parents rated them on measures of psychopathology, temperament, and personality. Results of the univariate behavior genetic analyses suggested significant additive genetic and nonshared environmental influences on social phobia. Bivariate genetic analyses indicated a moderate positive genetic and nonshared environmental correlation between shyness and social phobia. There was also a small negative genetic and environmental correlation between social phobia and extraversion and shyness and extraversion. Together, these results suggest that social phobia has genetic and unique environmental causes and may be etiologically related to shyness and extraversion.

Keywords: Social phobia, shyness, extraversion, behavior genetics

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Tables and Figures

The Genetic and Environmental Etiology of Social Phobia in Youth and the Etiological Overlap Between Shyness, Extraversion, and Social Phobia

Social phobia (SP) is a common anxiety disorder characterized by a persistent fear of perceived or actual scrutiny from others, physical symptoms of anxiety or panic attacks while in threatening social settings, and an avoidance of social or performance situations (Kessler & Merikangas, 2004). Individuals with social phobia also often present with a fear of acting in a manner that is embarrassing or that could result in rejection (American Psychiatric Association, 2013). The genesis of social phobia is unclear, but studies suggest that several factors, including biological, developmental, and social mechanisms, play a role in its etiology (Hudson & Rapee, 2000). Behavior genetic studies may be particularly useful in studying the etiology of social phobia, as they can quantify the magnitude of genetic and environmental influences that contribute to the development of the condition. Several of these studies (Hallett, Ronald, Rijsdijk, & Eley, 2009; Kendler, Neale, Kessler, Heath, & Eaves, 1992) suggest that anxiety disorders in both children and adults are moderately heritable and influenced by nonshared environmental factors. Few studies, however, have specifically examined the etiology and heritability of social phobia, and even fewer have examined this in both children and adolescents. Because social phobia is an early onset condition and may be a risk factor for more serious psychopathology (Chavira & Stein, 2005), it is particularly important to examine its genetic and environmental causes in children and adolescents. In addition, examining the possible etiological overlap between social phobia, shyness, and extraversion in this population may help determine the extent to which genetic vulnerability to social phobia is indexed by basic personality traits.

Structure of Social Phobia

Though social phobia is currently represented in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013) as a discrete disorder, studies employing taxometric methods have supported a dimensional model for this condition as well as other forms of psychopathology (Kollman, Brown, Liverant, & Hoffman, 2006). Ruscio and Watson (2010) examined the latent structure of social phobia and found that social phobia is a dimensional construct that includes a range of mild to severe presentations of social anxiety. Research suggests that facets of social phobia, including the "performance only" specifier, which refers to fear that is restricted to speaking or performing in public, and fear of negative evaluation are also dimensional constructs that demonstrate continuous relationships with social fears (Boyers et al., 2017; Ruscio & Watson, 2010).

Examination of the underlying structure of social phobia symptoms has revealed similar structures across assessment instruments and types of samples (Cox, Clara, Sareen, & Stein, 2008). Factor analysis of social phobia symptoms in a treatment-seeking clinical sample identified factors related to social interactional anxiety, such as fears of being observed by others and public speaking anxiety (Saffren, Turk, & Heimberg, 1998). Similarly, factor analysis of the Social Phobia Scale (SPS) and Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1989) in clinical and control samples identified distinct dimensions of social phobia, including a fear of social interactions and a fear of scrutiny, that fall into two distinguishable social phobia areas. Additional studies using the SIAS and SPS have supported these unique dimensions and have found that social interaction fears are more strongly correlated with low positive affect, while performance fears are more robustly related to high physiological arousal. Similarly, examination of the factor structure of social phobia in nationally representative samples has

identified three dimensions including fear of public performance, fear of being observed, and social interaction fears (Iza et al., 2014).

In children, a three-factor solution has emerged that includes fear of negative evaluation from peers, social avoidance and distress in new situations, and generalized social avoidance and distress (LaGreca & Stone, 1993). Indeed, children with social phobia commonly present with one or more social fears. These fears may lead to impaired peer and interpersonal relations, as children with the condition tend to speak less frequently, are less likely to start interactions, and often avoid anxiety-provoking social situations (Chavira & Stein, 2005). Children with social phobia also often display impaired social skills. In one study, Spence and colleagues (Spence, Donovan, Brechman-Toussaint, Strauss, & Milton, 1999) examined whether children with social phobia demonstrated social skills deficits and negative self-evaluations using observations in school and laboratory settings. Their findings indicated that children with social phobia were rated as significantly less socially competent by peers and adults and tended to view their own social performance more negatively.

Because children with social phobia typically avoid public speaking tasks, giving presentations, and communicating with authority figures, they may also be at risk for impaired academic functioning (Van Ameringen, Mancini, & Farvolden, 2003). Social phobia predicts chronic school refusal in young children and underachievement and dropout rates in older children and adolescents (Kessler, Foster, & Saunders, 1995). Epidemiological studies in children indicate that social phobia co-occurs frequently with other psychiatric disorders, including major depression, generalized anxiety disorder, specific phobias, and substance abuse (Chartier, Walker, & Stein, 2003), such that 66% of children with social phobia suffered from at least one other disorder (Acaturk, Graff, Straten, Have, & Cuijpers, 2008).

Cognitive-behavioral therapy and pharmacotherapies are considered first-line interventions for social phobia; however, over 80% of children with social phobia do not receive treatment (Burstein et al., 2011). This lack of treatment may contribute to an increased likelihood of later occupational or social impairment and instances of other psychiatric conditions in adulthood (Van Ameringen et al., 2003). Considering the negative implications of untreated social phobia in children, it is particularly important to examine its etiological factors, as this effort may help to increase our understanding of social phobia.

Etiology of Social Phobia

Behavior genetic analyses in adults have suggested that social phobia symptoms are moderately heritable (Hettema, Prescott, Myers, Neale, & Kendler, 2005; Mosing et al., 2009). These analyses rely on twin designs, which examine the genetic and environmental influences of a given condition or trait. The classical twin design partitions the variance in a given trait into that due to additive genetic factors (A), nonadditive genetic factors (D), shared environmental factors (C), and non-shared environmental factors (E). These constructs are derived from differences in the correlations between monozygotic (MZ; identical) twins, who share, on average, 100% of their segregating genes and dizygotic (DZ; fraternal) twins, who share 50% of their genes. The twin model assumes that MZ and DZ twins experience identical trait-relevant environments (Kendler, Neale, Kessler, Heath, & Eaves, 1993) and thus, greater correlations between MZ than DZ twins imply genetic influences and similar twin correlations imply environmental influences (Mosing et al., 2009).

Etiology of social phobia in adults. Several twin studies have examined the genetic and environmental influences of social phobia and other anxiety disorders in adults. These studies have consistently found support for the role of additive genetic and non-shared environmental

influences in social phobia, but have found little evidence of nonadditive genetic or shared environmental influences (Hettema et al., 2005; Scaini, Belotti, & Ogliari, 2014). Studies examining the etiology of anxiety disorders, including social phobia, specific phobias, and agoraphobia, have found evidence of appreciable additive genetic influences ranging from .30 (social phobia) to .39 (agoraphobia) and nonshared environmental influences ranging from .61 (agoraphobia) to .80 (social phobia; Kendler et al., 1992; Kendler, Myers, Prescott, & Neale, 2001).

Because phobias and anxiety disorders often co-occur, twin studies frequently examine the extent to which common genetic and environmental factors influence multiple anxiety disorders. In one such study, Mosing et al. (2009) examined the familial aggregation and co-morbidity of major depression, panic disorder, agoraphobia, and social phobia in adult twins. Social phobia was moderately heritable ($h^2 = .39$) and displayed significant genetic correlations with major depression ($r = .76$), agoraphobia ($r = .53$), and panic disorder ($r = .60$). In an earlier twin study, Hettema et al. (2005) examined the shared etiological influences on six anxiety disorders (generalized anxiety disorder, panic disorder, agoraphobia, animal phobia, situational phobia, and social phobia) in adult twins and found that the genetic covariance across these conditions was best explained by two common additive genetic factors, suggesting a shared genetic diathesis for these conditions.

Etiology of social phobia in children. In children, as in adults, there is evidence that genetic and environmental factors play a role in the etiology of social phobia. These studies have found moderate heritability estimates ($h^2 = .54$) for social anxiety in children and largely support the role of additive genetic and non-shared environmental influences. Behavior genetic analyses on social phobia have found estimates of additive genetic influences ranging from .28 to .61 and

non-shared environmental influences ranging from .25 to .32 (Hallett et al., 2009; Ogliari et al., 2006).

Few twin studies in children have supported the role of shared environmental influences in social phobia and other anxiety disorders. A study examining the etiology of anxiety-related behaviors, including social anxiety, negative cognitions, and fear, found evidence of significant additive genetic and nonshared environmental factors, with estimates of additive genetic influences ranging from .46 (negative affect) to .61 (social phobia) and non-shared environmental influences from .24 (fear) to .40 (negative cognitions). Shared environmental influences, however, accounted for only 7 percent of the variance in social phobia and 9 percent in negative cognitions (Hallett et al., 2009). Another study on child and adolescent twins assessed the genetic and environmental influences on panic/somatic anxiety, social phobia, generalized anxiety disorder, and separation anxiety disorder. The best-fitting model for all of the disorders included additive genetic and non-shared environmental influences, and there was no evidence of shared environmental influences in these disorders (Ogliari et al., 2006).

Studies examining the extent to which common genetic and environmental factors influence multiple anxiety disorders, such as social phobia, in children have yielded similar results to the previously discussed adult studies (Waszcuk, Zavos, Gregory, & Eley, 2014). In one such study in adolescent twins, Nelson et al. (2000) examined the etiology of social phobia, co-morbid psychiatric conditions, and the risk of suicidal ideation and attempts. A common additive genetic component explained a significant proportion of the variance in social phobia, major depressive disorder, and alcohol dependence (Nelson et al., 2000). Ogliari et al. (2010) also found that genetic influences explained a significant (58-99%) amount of the covariation between

symptoms of social phobia, generalized anxiety disorder, separation anxiety, and panic disorder in a sample of child and adolescent twins.

Molecular genetic studies. Though not the main focus of this paper, molecular genetic studies may provide relevant information in the study of the etiology of social phobia; much of this literature, however, has been inconsistent in its findings. In one study, Rowe et al. (1998) examined the relation of a polymorphism in the dopamine transporter gene (*DAT1*) to symptoms of six internalizing disorders (generalized anxiety disorder, major depressive disorder, panic disorder, separation anxiety, specific phobia, and social phobia). This study found a positive association between the *DAT1 10*-repeat allele and all six of the disorders. Another study found no association of social phobia with the serotonin transporter protein (*5HTT*) gene or the $5HT_{2A}$ receptor ($5HT_{2A}R$) gene (Stein, Chartier, Kozak, King, & Kennedy, 1998). In contrast, a later study examined candidate gene associations with withdrawn behavior, which is a risk factor for social phobia, and found that two genes (*HTR2A* and *ADRA2A*) were associated with withdrawn behaviors (Rubin et al., 2013).

Genome-wide association studies (GWAS) have also been used to examine genetic variants associated with social phobia and other anxiety disorders. In contrast to candidate gene studies, genome-wide association studies examine the association between millions of variants across the genome with the phenotype of interest without a priori hypotheses about which variants will be implicated. One GWAS found that genetic variants within the *PDE4B* gene were significantly associated with anxiety and stress-related disorders at the genome-wide level (Meier et al., 2018). Another GWAS examined common genetic effects across generalized anxiety, panic, agoraphobia, specific phobia, and social phobia and identified two SNPs, rs1709393 on the *LOCI152225* locus and rs1067327 on the *CAMKMT* gene, which were

associated with shared risk across the disorders (Otowa et al., 2016). Finally, a recent GWAS specifically examining genetic risk variants for social anxiety also identified two SNPs, rs708012 on Chromosome 6 and rs78924501 on Chromosome 1, which suggest a genetic basis for the disorder (Stein et al., 2017).

Personality, Temperament, and Social Phobia

In order to better understand the development of psychopathology, researchers have also examined the extent to which major temperaments or personality traits are related to, or serve as risk factors for, phobias and other psychiatric conditions. Much of this research has examined the relationship between shyness and social phobia and extraversion and social phobia, both of which will be discussed in turn. Many of the defining symptoms of social phobia, such as social skills deficits and a fear of negative evaluations, are also present in individuals who are shy (Chavira, Stein, & Malcarne, 2002). Unlike social phobia, which is a psychiatric disorder, shyness is primarily conceptualized as a temperament trait that is moderately stable from early childhood through adolescence (Coplan, Arbeau, & Armer, 2008). While the onset of social phobia is usually in childhood or adolescence, shyness tends to appear much earlier in life, often in infancy (Turner, Beidel, & Townsley, 1990). The prevalence rates of shyness are also higher than that of social phobia. Burstein, Ameli-Grillon, and Merikangas (2011) found that almost 50% of U.S. adolescents considered themselves shy at some point during the last 12 months, while only 8.6% of the sampled adolescents met diagnostic criteria for social phobia. Shyness and social phobia also differ in their natural course and impact, as shyness tends to be less chronic and impairing than social phobia (Turner et al., 1990). Although the two conditions have several differences, studies have indicated that they are highly correlated and that shyness in

childhood may predict internalizing disorders, such as social phobia, later in life (Heiser, Turner, Beidel, & Roberson-Nay, 2009).

Genetic etiology of shyness in children. There is a considerable amount of evidence that supports the role of genetic and non-shared environmental factors in the etiology of shyness and related traits in children and adolescents. Emde et al. (1992) measured shyness, emotion, and cognition in 14-month-old twins and found a high heritability rate ($h^2 = .49$) and non-significant common environmental influences for shyness. Another study examined the genetic and phenotypic structure of five anxiety-related behaviors, including shyness/inhibition, in pre-school twins. A measure of shyness/inhibition was included to assess temperamental contributions to the anxiety-related behaviors. This study found significant non-shared environmental ($e^2 = .24$) and additive genetic influences ($a^2 = .76$; Eley et al., 2003) and noted that while shyness/inhibition displayed a moderate level of genetic overlap with the other anxiety scales, it also had some degree of unique genetic influences. This finding may explain why only some children who are shy later develop anxiety disorders while others do not. Evidence for genetic influences on shyness have also been found in older children and adolescents, with heritability estimates ranging from .49 to .67 (Saudino et al., 1995).

Extraversion. Major personality traits in adults have also been studied in relation to the development of psychopathology. Most of the extant research in this area has focused on the positive association between neuroticism and anxiety disorders (Kotov, Gamez, Schmidt, & Watson, 2010). Recently, however, scholars have started to assess the etiology of and extent to which extraversion may be associated with anxiety disorders, such as social phobia. Extraversion is defined as an individual's tendency to be sociable, energetic, and experience positive emotions (e.g., happiness). Extraversion is also associated with several correlates of social behavior such

as meeting new people and being involved with social organizations (Bienvenu, Hettema, Neale, Prescott, and Kendler). Accordingly, research suggests that social phobia (Mulder & Aken, 2014) and shyness (Afshan, Askari, & Manickam, 2015), which both involve distress and avoidance of social settings, are negatively correlated with extraversion (Watts, Poore, Lilienfeld, & Waldman, under review).

Like other personality traits, extraversion appears to be influenced by genetic and non-shared environmental factors. Studies examining the etiology of extraversion in adults have found evidence of moderate heritability estimates ranging from .41 to .53. The remaining variance has largely been attributed to non-shared environmental influences (Jang, Livesley, & Vemon, 1996; Pederson, Plomin, McClearn, Friberg, & Sarason, 1988).

Studies of traits related to extraversion, such as positive emotionality, have also found significant genetic influences. One of these studies on adult twins reared apart used the Multidimensional Personality Questionnaire to assess the differential heritability of personality variables. Tellegen et al. (1988) found that the individual variability in positive emotionality, an extraversion correlate, was influenced by significant additive genetic ($a^2 =$. 40), shared environmental ($c^2 =$. 22), and non-shared environmental influences ($e^2 =$. 38). These results largely support other studies that have found appreciable additive genetic and non-shared environmental influences for extraversion in adults (Floderus-Myrhed, Pederson, & Rasmuson, 1980; Rose, Koskenvuo, Kaprio, Sarna, & Langinvainio, 1988).

Although somewhat less is known about the genetic etiology of extraversion in children and adolescents, the existing literature suggests that extraversion is influenced by additive genetic, non-additive genetic, and non-shared environmental factors. Little support exists for the role of shared environmental influences in extraversion in children. Heritability estimates for

extraversion and its correlates in infants and toddlers range from .35 to .47, which generally mirror the moderate heritability rates of extraversion in adults (DiLalla & Jones, 2000).

Studies examining the etiology of extraversion and its correlates (e.g., surgency and sociability) in children have found evidence of significant additive genetic and non-shared environmental influences, with estimates ranging from .20 to .47 and .48 to .59, respectively (Buss & Plomin, 1984; DiLalla & Jones, 2000; Gillespie, Evans, Wright, & Martin, 2004; Watts, Poore, Lilienfeld, & Waldman, under review). Some studies, however, have supported the role of non-additive genetic factors, which accounted for .31 to .33 of the variance in an adolescent twin sample aged 12-18 (Rettew, Rebollo-Mesa, Hudziak, Willemsen, & Boomsma, 2008).

Associations Between Social Phobia, Shyness, and Extraversion

Research on different personality traits (e.g., neuroticism) and anxiety disorders in adults has suggested that genetic influences account, at least in part, for some of the association between these different constructs (Eley & Plomin, 1997). A small body of research further indicates that there are significant relations between extraversion and social phobia in adults. In one study, Bienvenu et al. (2007) examined the extent to which extraversion and neuroticism indexed the genetic and environmental risk factors for agoraphobia, animal phobia, and social phobia in adult twins and found that genetic influences on extraversion and neuroticism fully accounted for the genetic liability in agoraphobia and social phobia. This study also noted that the greatest risk for agoraphobia and social phobia was comprised of genetic liability to both low extraversion and high neuroticism levels.

Another study assessed possible developmental factors in the etiology of social phobia in adults and found support for the relation between low extraversion and social phobia (Stemberger et al., 1995). Stemberger et al. (1995) also observed that 72% of adults with social

phobia in this study reported a history of childhood shyness. It is not clear from these findings, however, if low extraversion and shyness served as precursors or vulnerability factors for social phobia. Additional studies have found negative phenotypic correlations between social phobia and extraversion and its correlates in adults (Kotov et al., 2010; Watson, Clark, & Carey, 1988), college undergraduates (Afshan et al., 2015), and children (Mulder & Aken, 2014). There are no studies that we know of that examine the etiological overlap between social phobia, shyness, and low extraversion.

The Current Study

Although the literature reviewed thus far suggests the presence of genetic and environmental influences in the etiology of social phobia in youth, there are not any studies focusing solely on the genetic etiology of social phobia across a wide age range of children and adolescents. It is, therefore, the primary purpose of this study to use a twin study design to investigate the genetic and environmental factors in the etiology of social phobia in a large child and adolescent twin sample. The extant literature also suggests that social phobia may be positively related to shyness and negatively associated with extraversion in adults; however, as far as we know, no research exists that examines the possible etiological overlap between social phobia, shyness, and extraversion in youth. To address this gap in the literature, a secondary aim of this study is to examine the extent to which social phobia, shyness, and extraversion are influenced by common etiological mechanisms.

Several hypotheses were generated. First, it was hypothesized that there would be moderate genetic and nonshared environmental influences on social phobia in youth with little evidence of shared environmental influences. Drawing from the adult literature (e.g., Beinvenu et al., 2007), it was also hypothesized that extraversion would be negatively associated with social phobia and

shyness and that shyness would be positively associated with social phobia. Additionally, it was predicted that the genetic factors that influence extraversion and shyness would also significantly affect social phobia in children. This study contributes to a better understanding of the role of temperament and personality in the development and etiology of social phobia in children. The findings of this study serve to quantify the genetic and environmental influences of social phobia in youth and clarify the etiological overlap between social phobia, low extraversion, and shyness.

Method

Participants

The present sample consisted of 2,498 individuals (51% female) aged 4 to 16 (M_{age} = 8.5 years, *SD* = 2.9). The sample was drawn from the Georgia Twin Registry, which is a population-based twin registry comprised of 4- to 18-year old twins and their siblings living in Georgia at the time of original data collection. Parents in the registry provided ratings on a series of personality and psychopathology measures. Mothers typically completed the questionnaires (53%), and the remaining questionnaires were completed either by fathers (1%) or mothers and fathers together (46%). Completed questionnaire data was available for 405 (46%) MZ pairs and 469 (54%) same- and opposite-sex DZ pairs. Twenty-nine percent (N = 253) of the sample was comprised of siblings of twins, which were included in phenotypic analyses. Twin zygosity was determined through a 9-item parental report questionnaire regarding the twins' physical similarity that has previously been validated against DNA polymorphisms (Bonnelykke, Hauge, Holm, Kristoffersen, & Gurtler, 1989). The ethnic background of participants was 82% Caucasian, 11% African American, 1% Hispanic, and 6% other/mixed ethnicity.

Measures

Emory Combined Rating Scale. The twins' primary caregivers completed the Emory Combined Rating Scale (ECRS; Waldman et al., 1998), which assesses symptoms of common DSM-IV (American Psychiatric Association, 1994) childhood psychiatric disorders, including attention-deficit hyperactivity disorder, oppositional defiant disorder, separation anxiety disorder, generalized anxiety disorder, major depressive disorder, agoraphobia, panic disorder, specific phobia, obsessive-compulsive disorder, and social phobia. Items are rated on a 5-point likert scale from 0 (*not at all representative of the child*) to 4 (*very much representative of the child)*. The 10-item social phobia scale included statements such as child "worries about what to say or how to act when around new people" or "gets tongue-tied or doesn't talk when around new people." Internal consistency (Cronbach's alpha = .76) of the social phobia scale was high. Scores on the social phobia scale were averaged to produce a total score.

Emotionality, Activity, and Sociability Temperament Survey (Buss & Plomin, 1984). The Emotionality, Activity, and Sociability Temperament Survey (EAS) is a 20-item parent report measure that assesses the dimensions of emotionality, activity, sociability, and shyness. Items are rated on a 5-point likert scale from 0 (*does not describe the child at all*) to 4 (*describes the child very well*). The shyness scale included items such as "s/he tends to be shy" or "s/he takes a long time to warm up to strangers." Scores on the shyness scale were averaged to produce a total score. The EAS has satisfactory psychometric properties in infants and children (Abulizi, Pryor, Michel, Melchior, & Van der Waerden, 2017) and high internal consistency (Cronbach's alpha = 0.85 for all scales; Cronbach's alpha = 0.79 for shyness scale).

Big Five Personality Questionnaire (BFPQ; Lanthier, 1993). The Big Five Personality Questionnaire is comprised of 60 personality adjectives that reflect the personality domains of the five-factor model. It was created by combining adjectives from well-established five-factor

model inventories (Goldberg, 1990; McCrae & Costa, 1997) and comprises descriptors from Big 5 inventories such as the Big Five Inventory (John, Donahue, & Kentle, 1991) and the NEO Personality Inventory-Revised (Costa & McCrae, 1992). The five domains included Openness to Experience, Conscientiousness, Extraversion, Agreeableness, and Neuroticism. Each dimension had 12 items, which were rated on a 5-point likert scale from 0 (*does not describe the child at all*) to 4 (*describes the child very well*). The extraversion scale (Cronbach's alpha = 0.77) contained adjectives such as "energetic," "takes charge," and "enthusiastic." Scores on the extraversion scale were averaged to produce a total score.

Data Analyses

We conducted all analyses using Mplus version 8 (Muthén & Muthén, 1998-2017) using full-information maximum likelihood estimation (FIML) to handle any missing data, which produces less biased parameter estimates compared with listwise and pairwise deletion in the presence of missing data (Enders & Bandalos, 2001). In the phenotypic analyses, we used the cluster option to account for the nonindependence of siblings nested within families.

Zero-order correlations. Phenotypic zero-order correlations between social phobia, shyness, and extraversion were estimated. Cross-twin and cross-trait correlations were also estimated separately for MZ and DZ twins to infer the common genetic and environmental influences underlying phenotypic relations among the variables. Genetic influences were suggested if the cross-twin and cross-trait correlations were greater for MZ than DZ twins.

Model-fitting analyses. Biometrical modeling approaches (Neale & Cardon, 1992) were used to decompose the phenotypic variance in social phobia, shyness, and extraversion and estimate the contribution of latent factors corresponding to additive genetic (A), nonadditive genetic (D), shared environmental (C), and non-shared environmental (E) influences. We used a

set of goodness-of-fit indices, including the χ^2 test statistic, root mean square error of approximation (RMSEA), Comparative Fit Index (CFI), Tucker-Lewis index (TLI), Bayesian Information Criteria (BIC), and Standardized Root Mean Square Residual (SRMR), to evaluate and select the best fitting model.

Behavior genetic analyses. Univariate analyses were conducted to estimate the genetic and environmental influences underlying social phobia, shyness, and extroversion. In behavior genetic models, additive genetic influences are correlated 1.0 among MZ twins who share, on average, 100% of their genes. Additive genetic influences among DZ twins who share 50% of their genes are correlated 0.5. Nonadditive genetic influences are correlated 1.0 for MZ twins and 0.25 for DZ twins because DZ twins receive the same genes from both parents 25% of the time. Shared environmental influences are correlated 1.0 for both MZ and DZ twins because they comprise environmental influences that are common to twins. Nonshared environmental influences are uncorrelated across twins because they constitute unique environmental influences to each twin.

We fit all possible combinations of univariate behavior genetic models, including (1) ACE models, which estimated the proportion of variance in social phobia symptoms attributable to additive genetic (A), shared environmental (C), and nonshared environmental (E) influences; and (2) ADE models, which estimated nonadditive genetic influences (D) instead of shared environmental influences (C). We fit a series of reduced models, including (3) AE models, which included additive genetic and nonshared environmental influences, and (4) CE models that included shared and nonshared environmental influences. These models were also tested with sibling interaction parameters to account for possible rater contrast effects.

Bivariate analyses. Biometrical model-fitting analyses were also used to estimate the extent to which the etiological influences on social phobia, shyness, and extraversion were correlated. We tested two models in our bivariate analyses, based on the results of the univariate models. First, we fit bivariate AE models for social phobia and shyness, social phobia and extraversion, and shyness and extraversion. We then fit an AE model with rater contrast effects to each of these combinations, given evidence from the univariate model results that indicated sibling competition or cooperation effects or rater effects.

Results

Phenotypic analyses.

Descriptive statistics and phenotypic correlations between constructs of interest are displayed in Table 1. There was a significant positive correlation between social phobia and shyness ($r = 0.43$) and negative correlations between social phobia and extraversion ($r = -0.35$) and shyness and extraversion ($r = -0.61$).

Univariate analyses.

We fit univariate behavior genetic models of social phobia, shyness, and extraversion in which each of the variables was decomposed into latent genetic and environmental influences. Results of the univariate analyses are displayed in Table 3, with the best fitting model for each construct shown in bold. The best-fitting model for social phobia yielded evidence of additive genetic ($a^2 = 0.45$) and nonshared environmental ($e^2 = 0.55$) influences. The parameter estimates for shyness indicated appreciable additive genetic ($a^2 = 0.78$) and nonshared environmental influences ($e^2 = 0.27$) with rater contrast effects ($s = -0.20$; $p = 0.00$). A similar pattern emerged for extraversion. The best-fitting model for extraversion yielded moderate additive genetic ($a^2 =$

0.62) and nonshared environmental influences ($e^2 = 0.38$), as well as a significant rater contrast effect (s = -0.20; p = 0.00).

Bivariate analyses.

We tested a series of bivariate behavior genetic models to account for the covariance of genetic and environmental influences among social phobia, shyness, and extraversion. Results of the bivariate models are displayed in Table 4, with the best-fitting model shown in bold. A model containing additive genetic ($r_a = 0.51$) and nonshared environmental influences ($r_e = 0.39$) with rater contrast effects emerged as the best-fitting model for social phobia and shyness (see Figure 1). Consistent with the results from univariate analyses, rater contrast effects were only found for shyness (s = -0.16; p = 0.00). A model containing additive genetic ($r_a = -0.37$) and nonshared environmental ($r_e = -0.31$) influences with rater contrast effects emerged as the best-fitting model for the covariation between extraversion and social phobia (see Figure 2). Consistent with univariate results, rater contrast effects were only observed for extraversion (s = -0.17; p = 0.00). A model with additive genetic ($r_a = -0.30$) and nonshared environmental ($r_e = -0.37$) influences with rater contrast effects was the best-fitting model for the covariation between shyness and extraversion (see Figure 3). Rater contrast effects were present for both shyness and extraversion s = -0.17; p = 0.00).

Discussion

The current study assessed the genetic and environmental etiology of social phobia and the etiological overlap between shyness, extraversion, and social phobia in a large child and adolescent twin sample. Three hypotheses were examined: 1) extraversion would be negatively associated with social phobia and shyness, whereas shyness would be positively associated with social phobia, 2) there would be moderate genetic and nonshared environmental influences on

social phobia in youth with little evidence of shared environmental influences, and 3) the genetic and environmental factors that influence extraversion and shyness would also significantly affect liability to social phobia in children. The current paper adds to the literature by highlighting the significant role of additive genetic and nonshared environmental influences in the etiology of social phobia in children and adolescents. Importantly, research on the genetic and environmental factors influencing social phobia in youth may contribute to a better understanding of the condition. This study also illustrated the etiological overlap between social phobia, shyness, and extraversion in youth, a finding that adds to the adult literature on the etiological overlap between social phobia and low extraversion (Bienvenu et al., 2007).

Interpretation of Findings

Consistent with previous literature (Bienvenu et al., 2007; Heiser et al., 2009) and our hypotheses, we observed moderate negative phenotypic associations between shyness and extraversion and social phobia and extraversion; conversely, we found a moderate positive association between shyness and social phobia.

In line with many previous studies (Hallett et al., 2009; Ogliari et al., 2006), our univariate social phobia behavior genetic analyses yielded evidence of appreciable additive genetic and nonshared environmental influences with no evidence of nonadditive genetic or shared environmental influences. Nevertheless, the absence of shared environmental influences contrasts with some studies of child anxiety symptoms and disorders that have found significant shared environmental influences in separation anxiety (Topolski et al., 1996), fear, and general distress (Eley et al., 2003). The contrasting finding within the current study might be due to the informant type used with each sample. For example, Thapar and McGuffin (1995) assessed child anxiety symptoms and found evidence of additive genetic and nonshared environmental

influences in parent ratings; however, child reports indicated the presence of shared enviromental influences. The present study only used parent report, which may account for the lack of shared environmental influences. The age and sex of the participants in a specific sample may also explain the absence or presence of shared environmental influences. Past literature has suggested that shared environmental influences are more prevalent in early childhood psychopathology, whereas additive genetic and nonshared environmental influences increase in late childhood and adolescence (Garcia et al., 2013). Indeed, Eley et al. (2003) noted the significant role of shared enviromental influences in anxiety-related behaviors in pre-school children. The current study age range was 4 to 16 with a mean of 8.5. Including older children in our sample may explain the lack of shared environmental influences.

To our knowledge, this is the first study in youth to demonstrate that social phobia, temperament traits, and personality are influenced by some common etiological factors. Our findings suggest that social phobia and shyness have a positive genetic and environmental correlation, indicating that the etiological factors that influence the development of social phobia also influence the development of trait shyness. Conversely, there was a negative genetic and nonshared environmental association between social phobia and extraversion, which is consistent with previous findings in adults (Bienvenu et al., 2007). We also observed negative genetic and nonshared environmental correlations between shyness and extraversion. These findings suggest that the greatest genetic risk for social phobia involves genetic liability to both high shyness and low extraversion. However, the negative correlation between the etiological influences on shyness and extraversion was small, indicating that, although these phenotypes have a moderate phenotypic correlation, they display some distinct etiology.

The finding of significant nonshared environmental correlations between social phobia, shyness, and extraversion is also notable. This result suggests that the presence of specific environmental factors that are not shared between twins, such as an individual's experiences and responses to certain life events, may be related to the development of social phobia or levels of extraversion or shyness. This is in line with evidence that suggests that negative childhood social outcomes or aversive social events (e.g., criticism, teasing, or exclusion by others) increase the risk of developing both social phobia and higher levels of shyness (Chartier et al., 2001; Stemberger et al., 1995), whereas positive reactivity to daily experiences and familial relationships may contribute to increased extraversion levels (Horwitz, Luong, & Charles, 2008).

A discussion of rater contrast effects in the univariate and bivariate analyses is warranted. Rater contrast effects were not present for social phobia; however, they emerged for shyness and extraversion in both analyses. Contrast effects may be due to a parental rater bias in which a parent contrasts twins when rating behaviors or symptoms. They may also be due to a sibling interaction effect, which involves twins developing different behaviors as a reaction to each other's behaviors (Eley et al., 2003). Our results support past studies that have found sibling interaction or rater contrast effects for temperament (Eley et al., 2003), and the EAS scales specifically (Neale & Stevenson, 1989). Rater contrast effects are common in temperament studies, as parents may overemphasize small temperament differences between their two twins. The lack of sibling contrast effects for social phobia is interesting considering the anxiety these individuals experience during social interactions. If one member of a twin pair is more avoidant and has a co-twin who often speaks or interacts for him, it is possible that the twin who interacts more for himself and his sibling would gain increased social experience. Theoretically, this may

result in sibling interaction effects; however, this hypothesis was not supported in the current study

Implications

The results of the current study contribute to the literature on the etiology of social phobia in youth. To our knowledge, this is also the first study to demonstrate a genetic overlap between social phobia and temperament or personality traits. These results have several implications. First, the parameter estimates from the tested models may help inform more genetic and environmentally robust social phobia, shyness, and extraversion phenotypes in children and adolescents. Second, the finding that social phobia and shyness display moderate additive genetic correlations implies that the observed relationship between these two constructs is not simply due to their overlapping symptom and presentation profile. This result suggests that a common set of genes may pleiotropically influence one's vulnerability to social phobia and shyness and supports the search for genetic markers that influence both social anxiety and intermediate temperament phenotypes related to social anxiety (e.g., shyness, introversion, or behavioral inhibition). Smoller et al. (2008) noted the association between *RGS2* and childhood behavioral inhibition, shyness, and risk for social anxiety, suggesting that these phenotypes are influenced to some extent by the same genes.

The negative genetic and environmental correlations between social phobia and extraversion and extraversion and shyness also suggest the importance of low extraversion (introversion) and high shyness as personality risk factors for social phobia and support the search for genes that influence these variables. A study by Stein, Schork, and Gelernter (2004) found that *ADRB1*, a polymorphism of the β_1-adrenergic receptor gene, was associated with low extraversion and high shyness. Additional molecular genetics studies are needed to better understand the specific

genetic mechanisms underlying social phobia and possible targets for novel therapeutic treatments.

Additionally, the present study results may also have implications for the distinction between personality, temperament, and psychopathology. The findings of common genetic origins between shyness and social phobia and unique pathways between shyness, extraversion, and social phobia refute the null hypothesis of personality-psychopathology research, which suggests that the observed co-occurrence between personality traits and psychopathology is explained by criterion overlap instead of common factors (Jang, Wolf, & Larstone, 2006). The current study instead lends support to the common cause or spectrum hypothesis that posits that the association between personality and psychopathology is due to shared genetic or environmental causes (Andersen & Bienvenu, 2011). Under the spectrum hypothesis, it is possible that shyness and low extraversion act as common expressions of a shared underlying diathesis for anxiety. This diathesis may be expressed either as abnormal personality or temperament characteristics or, in the setting of extreme stress, as clinically diagnosed social phobia. Conversely, it may also be that social phobia represents dimensional extremes of personality or temperament. Additional twin studies are needed to examine the unclear distinction and influence of shared genetic factors on the expression of personality and psychopathology.

Limitations

The results of this study should be interpreted in light of several limitations. First, there are limitations inherent to the twin design. Our results are based on several assumptions including the absence of assortative mating and the equal environment assumption. The equal environment assumption suggests that identical and fraternal twins experience the same enviromental influences related to the behavioral phenotypes being studied (Hettema et al.,

2006). If this assumption is violated, the greater similarity between identical twins may be due to their increased environmental similarity instead of genetic factors, which would bias obtained results. Second, there were low rates of psychopathology in the community sample that was used. Our results, therefore, may not fully generalize to child and adolescent populations who have been clinically diagnosed with social phobia or other forms of psychopathology.

An additional limitation is that the data on social phobia, shyness, and extraversion characteristics relied solely on parental report. Previous studies have noted differences between parent- and self-reports of child internalizing and psychopathology symptoms (Grills & Ollendick, 2002; Weems, Feaster, Horigian, & Robbins, 2011). Evidence also suggests that using both parent and child or adolescent self-reports (Bögels & van Mellick, 2004; Hope et al., 1999) contributes unique information beyond that obtained from parent-report alone. Future twin studies might utilize multi-informant methodology to more clearly investigate the etiology of social phobia and temperament or personality characteristics. Finally, this study relied solely on cross-sectional data of social phobia, shyness, and extraversion. Temperament characteristics are early emerging traits that are relatively stable through middle childhood (Neppl et al., 2010). Given their early onset, they may be likely to develop before psychiatric disorders. Future twin studies should seek to assess the longitudinal, as well as concurrent, etiology of the associations between temperament, personality, and psychiatric conditions.

Future Directions

Given that little research exists on the causes and etiological correlates of social phobia in youth, future research should continue to use behavior genetic techniques to explore genetic and environmental risk factors in this population. Specifically, future longitudinal studies may examine shifts in genetic and environmental risk factors of social phobia, shyness, and low

extraversion throughout development, which may provide information on whether there are new genetic influences that emerge overtime or if different sources of shared or nonshared enviromental factors increase one's liability to anxiety in specific age groups.

Additionally, future studies may consider examining sex differences in the etiology of social phobia, shyness, and extraversion. Higher rates of social phobia have been reported in girls (Essau, Conradt, & Petermann, 1999), and the frequency and intensity of the disorder in girls often increases with age (Asher, Aderka, & Asnaani, 2017). Evidence also suggests that genetic influences on anxiety and inhibited temperaments tend to be greater for girls than boys (Feignon, Waldman, Levy, & Hay, 2001). Few studies have found sex differences in the heritability of extraversion (Eaves, Heath, Neale, Hewitt, & Martin, 1998). Future research with a larger sample size may examine whether genetic and environmental risk factors for social phobia, shyness, and extraversion vary between boys and girls. Lastly, studies may seek to cross-validate and extend the current study findings by using multiple measures (e.g., behavioral observation, self-report, or multi-informant report) of social phobia, shyness, and extraversion.

Conclusion

This study examined the etiology of social phobia and its etiological overlap with shyness and extraversion in a large child and adolescent twin sample. The results emphasize the role of additive genetic and nonshared environmental influences in the etiology of social phobia in youth. They also indicate that the greatest genetic risk for social phobia involves genetic liability to both high shyness and low extraversion. Findings of additive genetic influences across children of different ages suggest that social phobia has a strong genetic basis. The results of this paper call for additional molecular genetics studies that may identify genes or alleles that confer

risk for social phobia. Future studies on normative temperaments or personality traits related to social phobia are also needed to better understand the genetic basis of this condition.

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Table 1. *Descriptive statistics and phenotypic correlations between social phobia, shyness, and extraversion.*

	α	M (SD)	SP	S	E
Social Phobia (SP)	0.76	1.06 (0.45)			
Shyness (S)	0.85	1.37 (0.86)	0.43**		
Extraversion (E)	0.77	2.44 (0.56)	-0.35**	-0.61**	

Note. ** $p<.01$

Table 2. *MZ and DZ Correlations.*

	Twin 2		
Twin 1	1	2	3
MZ twins			
1. Social Phobia (SP)	**.45**		
2. Shyness (S)	**.25**	**.57**	
3. Extraversion (E)	-.05	**-.35**	**.44**
DZ twins			
1. Social Phobia (SP)	**.21**		
2. Shyness (S)	.03	-.04	
3. Extraversion (E)	**-.41**	.04	**-.19**

Note. MZ = monozygotic; DZ = dizygotic. Bolded is $p < .01$.

Table 3. *Summary of fit statistics and parameter estimates for univariate behavior genetic models.*

	Model Fit Statistics							Parameter Estimates			
Model	**X^2 (df)**	**CFI**	**TLI**	**BIC**	**RMSEA (90% CI)**		**SRMR**	**A^2**	**C^2/D^2**	**E^2**	**s**
Social Phobia											
ACE	10 (5)	0.70	0.88	1993	0.05	(0.00, 0.09)	0.13	0.45	0.00	0.55	-
ADE	13 (5)	0.53	0.81	1991	0.06	(0.02, 0.10)	0.13	0.25	0.23	0.52	-
CE	WOULD NOT CONVERGE										
AE	**12 (6)**	**0.64**	**0.88**	**1986**	**0.05**	**(0.00, 0.09)**	**0.13**	**0.45**	**-**	**0.55**	**-**
ACE+S	WOULD NOT CONVERGE										
ADE+S	14 (5)	0.50	0.80	1988	0.06	(0.02, 0.10)	0.12	0.61	0.00	0.42	-0.08
CE+S	17 (4)	0.37	0.79	2009	0.06	(0.10, 0.19)	0.14	-	0.29	0.71	-
AE+S	14 (5)	0.49	0.80	1989	0.06	(0.02, 0.10)	0.12	0.61	-	0.42	-0.08
Shyness											
ACE	40 (5)	0.62	0.85	4102	0.13	(0.09, 0.17)	0.10	0.48	0.00	0.52	-
ADE	15 (5)	0.89	0.96	4072	0.07	(0.03, 0.11)	0.06	0.00	0.55	0.45	-
CE	105 (6)	0.00	0.64	4152	0.20	(0.17, 0.23)	0.14	-	0.24	0.76	-
AE	48 (6)	0.54	0.85	4096	0.13	(0.10, 0.17)	0.10	0.48	-	0.52	-
ACE+S	8 (4)	0.96	0.98	4070	0.05	(0.00, 0.10)	0.06	0.78	0.00	0.26	-0.20
ADE+S	4 (4)	1.00	1.00	4066	0.00	(0.00, 0.07)	0.04	0.00	0.67	0.33	-0.10
CE+S	87 (5)	0.00	0.64	4158	0.20	(0.16, 0.24)	0.14	-	0.07	0.89	0.09
AE+S	**10 (5)**	**0.95**	**0.98**	**4064**	**0.05**	**(0.00, 0.09)**	**0.06**	**0.78**	**-**	**0.27**	**-0.20**
Extraversion											
ACE	25 (5)	0.36	0.74	1548	0.13	(0.08, 0.18)	0.14	0.30	0.00	0.70	-
ADE	17 (5)	0.61	0.84	1538	0.10	(0.05, 0.15)	0.12	0.00	0.36	0.64	-
CE	44 (6)	0.00	0.60	1556	0.16	(0.12, 0.21)	0.16	-	0.15	0.85	-
AE	30 (6)	0.23	0.74	1542	0.13	(0.09, 0.18)	0.14	0.30	-	0.70	-
ACE+S	14 (4)	0.68	0.84	1541	0.10	(0.05, 0.16)	0.12	0.63	0.00	0.39	-0.20
ADE+S	10 (4)	0.81	0.90	1536	0.08	(0.02, 0.14)	0.10	0.00	0.53	0.47	-0.10
CE+S	37 (5)	0.00	0.60	1563	0.16	(0.12, 0.21)	0.16	-	0.07	0.92	0.04
AE+S	**18 (5)**	**0.60**	**0.84**	**1535**	**0.10**	**(0.05, 0.15)**	**0.12**	**0.62**	**-**	**0.38**	**-0.20**

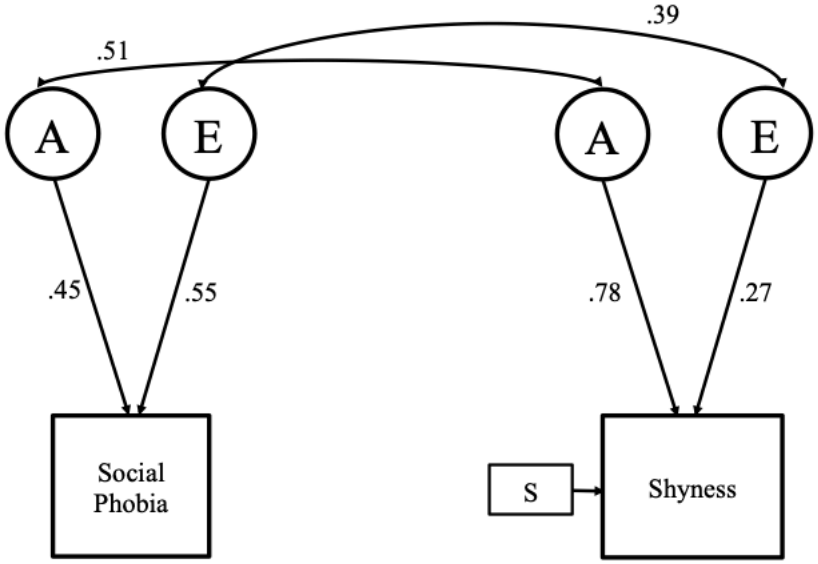
Note. Best-fitting model is bolded.

Table 4. *Summary of fit statistics and parameter estimates for bivariate behavior genetic models.*

	Model Fit Statistics						Parameter Estimates			
Model	**X^2 (df)**	**CFI**	**TLI**	**BIC**	**RMSEA (90% CI)**	**SRMR**	**r_a**	**$r_{c/d}$**	**r_e**	**s**
Social Phobia and Shyness										
AE	80 (18)	0.86	0.91	6238	0.09	(0.07, 0.11)	0.11	0.50	-	0.40	-
AE + S	**32 (17)**	**0.94**	**0.96**	**5653**	**0.05**	**(0.02, 0.07)**	**0.10**	**0.51**	**-**	**0.39**	**-0.16**
Social Phobia and Extraversion										
AE	49 (20)	0.76	0.86	3412	0.06	(0.04, 0.08)	0.12	-0.38	-	-0.32	-
AE+S	**38 (19)**	**0.83**	**0.89**	**3365**	**0.05**	**(0.03, 0.07)**	**0.11**	**-0.37**	**-**	**-0.31**	**-0.17**
Shyness and Extraversion										
AE	45 (18)	0.78	0.86	3424	0.06	(0.04, 0.08)	0.12	-0.38	-	-0.32	-
AE+S	**41 (16)**	**0.80**	**0.85**	**3422**	**0.06**	**(0.04 0.08)**	**0.11**	**-0.36**	**-**	**-0.30**	**-0.17**

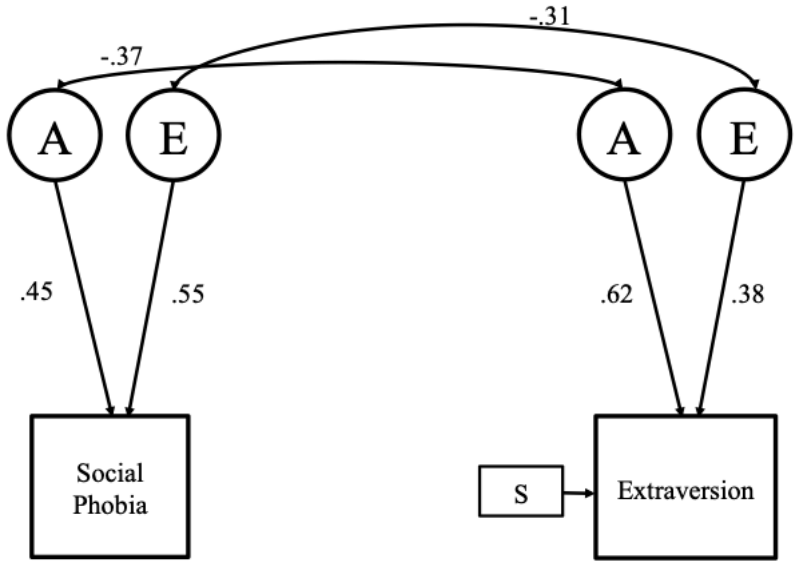
Note. Best-fitting model is bolded. r_a = additive genetic correlation; $r_{c/d}$ = shared environmental/nonadditive genetic correlation; r_e = nonshared environmental correlation.

Figure 1.



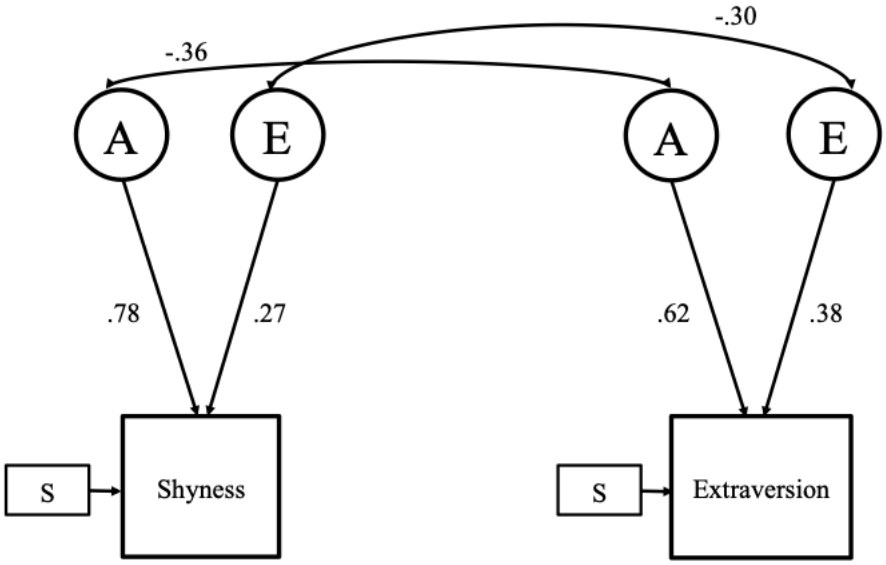
Best-fitting bivariate model for social phobia and shyness.

Figure 2.



Best-fitting bivariate model for social phobia and extraversion.

Figure 3.



Best-fitting bivariate model for shyness and extraversion.