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Title: The effects of social anxiety on alcohol and cigarette use across adolescence: Results from a longitudinal twin study in Finland

Year: 2016

Version: Accepted version (Final draft)

Please cite the original version:

Savage, J. E., Kaprio, J., Korhonen, T., Pulkkinen, L., Rose, R. J., Verhulst, B., & Dick, D. M. (2016). The effects of social anxiety on alcohol and cigarette use across adolescence: Results from a longitudinal twin study in Finland. Psychology of Addictive Behaviors, 30(4), 462-474. https://doi.org/10.1037/adb0000183

HHS Public Access

Author manuscript

Psychol Addict Behav. Author manuscript; available in PMC 2017 June 01.

Published in final edited form as:

Psychol Addict Behav. 2016 June ; 30(4): 462–474. doi:10.1037/adb0000183.

The Effects of Social Anxiety on Alcohol and Cigarette Use across Adolescence: Results from a Longitudinal Twin Study in Finland

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Abstract

Conflicting reports exist on the direction of the relationship between social anxiety (SA) and alcohol/cigarette use (AU/CU) and alcohol/nicotine dependence (AD/ND), with both positive and negative associations reported. A prospective, longitudinal sample of Finnish twins (n=1906) was used to test potential explanations for these discrepancies. Specifically, this study used peer, parent, and teacher ratings of SA, and a clinical interview screening item for Social Anxiety Disorder (SAD-Sc) to examine associations between SA and AU/CU and AD/ND from early adolescence into young adulthood. Peer-rated SA was negatively associated with AU, CU, and AD from age 14 through age 22, implying a protective effect (β=−0.01 to −.03). Teacher- and parent-rated SA associations were in the same directions but weaker or non-significant, indicating that aspects of SA that are recognizable by peers may be most relevant to AU/CU. Self-reported SAD-Sc was also negatively associated with AU, but positively associated with AD symptoms in young adulthood (β=0.38). Our findings partially support the existence of different associations between SA and AU versus AD, but only in the context of SAD-Sc rather than trait SA. Neither trait SA nor SAD-Sc significantly predicted ND symptoms, although SAD-Sc was associated with both cigarette abstinence and daily smoking. These findings suggest that adolescent SA is modestly associated with lower AU/CU, although there may be some individuals with more severe SA who

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The authors have no conflicts of interest to report.

develop alcohol problems later in life. There was little evidence of a common underlying liability contributing to both SA and alcohol/cigarette use.

Keywords

social anxiety; alcohol; nicotine; adolescents; twins

Alcohol and cigarette use in adolescence are associated with a number of negative health and interpersonal outcomes (Kypri et al., 2009; Windle et al., 2008) and predict the development of alcohol and nicotine use disorders in adulthood, particularly when initiation occurs at an early age (Doubeni, Reed, & Difranza, 2010; Grant & Dawson, 1997; Rose, Winter, Viken, & Kaprio, 2014). Given the relevance of peer influences on substance use in adolescence (Windle et al., 2008), a potentially important factor impacting adolescent alcohol and cigarette use is social anxiety (SA), which is an intense fear of being negatively judged by others accompanied by psychological and physiological symptoms and, typically, an avoidance of social situations (see Morris, Stewart, & Ham, 2005 for a review). SA is common among adolescents, with 46% of 12-year-olds and 55% of 17-year-olds reporting a fear of social situations (Kashani & Orvaschel, 1990). Researchers typically characterize SA as a dimensional, temperament-like trait or, in its more severe manifestation, a clinical diagnosis of Social Anxiety Disorder (SAD). Although much research has examined the association between SA/SAD and substance use, the nature of this relationship remains unclear. Below, we briefly review the research on the relationship between SA/SAD and alcohol and cigarette use/problems. We use the following abbreviations: AU - alcohol use (frequency/quantity), CU - cigarette use (frequency/quantity), AUDs/AD – alcohol use disorders/alcohol dependence, and ND – nicotine dependence.

Social anxiety and alcohol use

Adults

Among adults, large epidemiological surveys and clinical samples have consistently found elevated rates of comorbidity between SAD and AUDs (Buckner, Timpano, Zvolensky, Sachs-Ericsson, & Schmidt, 2008; Grant et al., 2005; Schneier et al., 2010; Thomas, Thevos, & Randall, 2009). However, one longitudinal study found that individuals with subclinical SAD (social fears without avoidance), but not those meeting diagnostic criteria, had more frequent heavy/binge drinking and higher rates of AUDs (Crum & Pratt, 2001).

Young adults

A meta-analysis of 44 studies (Schry & White, 2013) concluded that self-reported trait SA is negatively associated with AU but positively associated with alcohol problems in college students. There has been mixed evidence as to whether young adults with higher trait SA choose to drink more during laboratory social stress tasks (see Battista, Stewart, and Ham [2010], for a review). As for SAD, one prospective study of young adults found that, for women, SAD predicted development of an AUD three years later (Buckner & Turner, 2009).

Adolescents

Adolescent studies have had mixed findings. Trait SA and AU have been positively associated in two studies (Ohannessian, 2014; Zehe, Colder, Read, Wieczorek, & Lengua, 2013), but not a third (Blumenthal, Leen-Feldner, Frala, Badour, & Ham 2010). Further, Tomlinson, Cummins, & Brown (2013) showed that both very low and very high levels of trait SA were positively associated with drinking initiation, and Pardee, Colder, & Bowker, (2014) determined that SA was protective against AU in early adolescence but predicted higher AU at older ages. Studies examining SAD have also been inconsistent: SAD had no association with AU in one (Wu et al., 2010; Fröjd, Ranta, Kaltiala-Heino, & Marttunen, 2011), prospectively predicted lower adult AU in another (Fröjd et al., 2011), and prospectively predicted higher likelihood of onset of adult regular use and hazardous use in another (Zimmerman et al., 2003). Two studies have shown a positive prospective association between adolescent SAD and adult AD (Buckner, Schmidt, et al., 2008; Zimmerman et al., 2003).

Social anxiety and cigarette use

In adults, trait SA has been linked to greater CU, ND, and difficulty quitting smoking (Buckner, Farris, Schmidt, & Zvolensky, 2014; Lopes et al., 2002). SAD has similarly been positively associated with current/lifetime smoking status, ND, and lack of success in smoking cessation (Cougle, Zvolensky, Fitch, Sachs-Ericsson, 2010; Goodwin, Zvolenksy, Keyes, & Hasin, 2012; Lasser et al., 2000), although another study found that SAD had no association with ND but did predict cravings and higher avoidance-related motivation to smoke (Kimbrel, Morissette, Gulliver, Langdon, & Zvolensky, 2014). For young adults, one study found that trait SA positively predicted coping-related smoking behaviors in social situations (Watson, VanderVeen, Cohen, DeMaree, & Morell, 2012). In adolescents, one study found that trait SA was associated with lower actual likelihood of smoking but a higher urge to smoke in social situations (Henry, Jamner, & Whalen, 2012), while another found that SA differentially predicted higher/lower CU under conditions of higher/lower peer approval of smoking (Zehe et al., 2013). Adolescent SAD has been associated with heavier CU (for boys; Wu et al., 2010) and with ND, but not with initiation or regular use (Sonntag, Wittchen, Höfler, Kessler, & Stein, 2000).

Rationale and aims of the current study

The previous section identified numerous positive and negative SA-substance use associations reported in the literature. Positive findings are consistent with prominent theories suggesting that socially anxious individuals use alcohol and nicotine to self-medicate their anxiety symptoms, reduce tension, and facilitate social interactions (see Bacon and Ham [2010], Battista et al. [2010], Buckner et al. [2013], and Morris et al. [2005] for reviews). Negative relationships may be due to those with SA avoiding social situations that would provide them with access to and/or encourage the use of alcohol and cigarettes (e.g. parties), which are particularly important for adolescents who have not reached the legal age to purchase them (Fergusson & Horwood, 1990; Mayeux, Sandstrom, & Cillessen, 2008). In the present study, our goal was to investigate several potential factors that might

explain the discrepancies in the reported findings on the SA-substance use relationship, testing whether differential associations may be attributable to effects of age/developmental phase, or different aspects of how the substance use outcome and/or SA construct is measured across studies. We build upon the existing literature by using a longitudinal epidemiological sample of Finnish twins to elucidate the nature of the association between SA and alcohol and cigarette use from early adolescence to young adulthood. Our specific aims were fivefold:

1. To replicate the findings of Pardee et al. (2014), and to extend these to nicotine use, to determine whether the direction of association between SA and substance use changes between adolescence and young adulthood. We hypothesized that SA would be negatively related to alcohol and cigarette use in adolescence when peer social interactions are particularly influential to substance use, but positively related to substance use in adulthood when alcoholic beverages and cigarettes can be easily and independently acquired.

2. To bridge across the many studies in which either consumption or problem measures (but not both) were assessed, in order to determine whether conflicting directions of association with SA may be due to the differences in type of substance use outcome. Theory suggests that socially anxious individuals may not have higher levels of consumption, but may develop psychological dependence through coping-motivated substance use (Bacon & Ham, 2010). Here we investigate cross-sectional and longitudinal associations of SA with measures of both consumption and problems, hypothesizing that SA would be negatively associated with frequency of use, but positively associated with AD/ND symptoms.

3. To unify findings from studies utilizing assessments of SAD diagnoses, which have typically shown positive associations with substance dependence, and those using dimensional assessments of trait SA, which have been less consistent. Bacon & Ham's (2010) Avoidance-Coping model and findings from Crum and Pratt (2001) suggest that there may be distinct associations with substance use between SA measures that explicitly include avoidance criteria (e.g. clinical SAD) and dimensional measures of trait SA/shyness, because some socially anxious individuals may use alcohol or nicotine to cope with their anxiety in social situations while others avoid social situations where exposure to substance use is most prevalent. The current study assesses dimensional measures of trait SA and a clinical screening item of SAD symptoms (including an avoidance criterion). A positive association between SAD and substance dependence is well established, but we hypothesize that experience of SAD symptoms will be associated with lower frequency of use and trait SA with higher frequency of use in adulthood.

4. To compare peer, parent, and teacher ratings of early adolescent SA in predicting trajectories of substance use. Virtually all studies in this area have used self-report scales or interviews to assess SA/SAD, with a few studies of youth including parent reports (e.g. Henry et al., 2012; Wu et al., 2010). Self-

reports and parent reports, however, are plagued by problems such as social desirability bias (Kashani & Orvaschel, 1990), and informants from different social domains may each perceive facets of SA that differently relate to substance use. Based on previous work (Clemans et al., 2014), we hypothesized that peer ratings of SA would more strongly predict substance use than parent or teacher ratings due to peers' uniquely relevant position to appraise social behaviors.

5. To estimate the magnitude of genetic and environmental effects on trajectories of adolescent/young adult substance use and conduct an exploratory investigation of the potential for etiological overlap between SA and substance use. Twin studies have indicated that substance use in early adolescence is almost entirely attributable to environmental factors shared by siblings (peers, rearing environment), with stronger impacts of genes in adulthood (Dick et al., 2007; Kendler, Schmitt, Aggen, & Prescott, 2008). SA effects on substance use may thus become more prominent in adulthood when legal/social barriers to access are lessened and genetic predispositions play a larger role. The extent to which a shared genetic or environmental liability may underlie the SA-substance use relationship is unclear. As reviewed by Buckner et al. (2013), a few studies find elevated familial risk across SAD and AUDs; however, a large twin study found that SAD and AUDs were influenced by distinct genetic factors (Kendler, Prescott, Myers, & Neale, 2003). Here we extend the previous research by modeling the genetic and environmental underpinnings of developmental trajectories of substance use, and provide a novel exploration of the potential shared etiology between SA and AU, CU, and ND.

Methods

Sample

Participants in this study were from the *FinnTwin12* sample (Kaprio, 2006; Kaprio, Pulkkinen, & Rose, 2002; Pulkkinen et al., 1999), a prospective longitudinal study of five sequential cohorts of Finnish twins beginning at age 12 and continuing, at present, into their mid-20s. Importantly, as the legal age for purchasing alcohol and cigarettes in Finland is age 18, this longitudinal study covers the transitional period in which participants gain independent legal access to these substances. In Finland, all individuals are assigned a personal identification number at birth that is linked to their parental data at the Population Register Centre. From this registry, twins born from 1983 to 1987 were identified as individuals born on the same day to the same mother and were invited to participate in the *FinnTwin12* study, permitting an unbiased sampling strategy that included all twins born in Finland during that time. The study was approved by the Ethics Committee of the Hospital District of Helsinki and Uusimaa and the IRB of Indiana University, Bloomington. A subset of these individuals (~40%) was selected for more intensive study. Most were selected at random, with about 30% being chosen on the basis of high parental alcoholism scores to enrich the sub-sample with "at-risk" individuals (Rose, Dick, Viken, Pulkkinen, & Kaprio, 2004). These individuals received peer, parent, and teacher assessments at age 12, clinical interviews at ages 14 and 22, and self-report questionnaires at ages 12, 14, 17, and 22, as

described below. Parental permission was given to collect in-school peer and teacher ratings for 93% of these respondents at age 12, and 99% of school principals gave permission to conduct these assessments in school. A total of 25,106 pupils in 503 classes participated in the peer assessments.

The present study utilized this intensive subset of the *FinnTwin12* sample (n = 1906). The sample was 48.8% female, with 36.0% monozygotic (MZ) twins, 32.0% same-sex dizygotic (DZ) twins, 30.3% opposite-sex DZ twins, and 1.7% twins of uncertain zygosity. Participants were mailed self-report questionnaires in four waves: at age 11–12 (hereafter referred to as age 12), within 2 months of their 14th birthday, within 3 months of reaching age 17.5, and between ages 20–26 (average age of 22 with 96% between ages 21 and 23, hereafter referred to as 22). Eighty-seven percent of twins and their parents who were contacted responded to the Wave 1 study at age 12, and 90%, 92%, and 73% of the target sample participated at age 14, age 17, and age 22, respectively (Kaprio 2006; Kaprio 2013). Participants who dropped out were more likely to be male and had higher peer-rated SA and behavioral problems, although they did not significantly differ on alcohol or cigarette use measures at ages 12 or 14.

Measures

Social anxiety—At age 12, peer assessments were conducted in participants' classrooms using the Multidimensional Peer Nomination Inventory (MPNI), which has been described in detail elsewhere (Pulkkinen et al., 1999). The present study used the MPNI's Social Anxiety subscale, which consisted of two items: "Which of your classmates are shy with other kids?" and "Which of your classmates are frightened or nervous about new things or new situations?" and had an internal consistency reliability of α = 0.68/0.81 for girls/boys (Pulkkinen et al., 1999). The nomination process required students to choose, from a printed list of all classmates' names, up to three male and three female classmates that best fit the description or behavior portrayed by each item. Each individual's score on an item was calculated as the percentage of total votes that he or she received out of the total votes possible (range of 0–100); scale items were then averaged. Parents and teachers were administered the same items, but these appeared in the form of statements rather than nominations, and they rated each behavior on a four point scale (from 0 = *does not apply* to 3 = *applies in a pronounced way*). Internal consistency reliability was α = 0.69/0.74 for teacher reports of girls/boys and α = 0.46/0.42 for parent reports of girls/boys (Pulkkinen et al., 1999). Self-reports were not collected with this measure.

Substance use frequency—At age 14, participants were asked how often they drank alcohol (four response options from "never/I don't drink alcohol" to "once a week or more"), whether they had ever tried smoking, and how often they smoked cigarettes (five response options from "I have tried smoking but I don't smoke" to "I smoke at least once each day", with an additional option that did not directly measure frequency, "I am trying to or have quit smoking"). The same alcohol and cigarette frequency questions were asked at ages 17 and 22, but included expanded response categories (see Table 1 for full categories). For drinking frequency at ages 17 and 22, the response options of "a couple of times a week" and "daily" were combined due to low frequency of daily drinking (< 2%). For

smoking frequency, having never tried smoking was combined with the response option of "I have tried smoking but don't smoke" (both having a frequency of zero), while the response option "I am trying to or have quit smoking" was coded as missing, as the actual frequency of smoking could not be determined from this response option (endorsed by 10% or less of the respondents at each age). The ordinal smoking frequency variable was thus coded as 0 = non-smoker, 1 = less than once a week, 2 = once a week or more, 3 = 1–9 cigarettes per day, 4 = 10–19 cigarettes per day, and 5 = 20 or more cigarettes per day.

Clinical interviews—At age 14 and age 22, participants were administered the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999), a clinical interview that assessed lifetime DSM-IV AD symptoms among those who had initiated alcohol use. Lifetime ND symptoms were assessed at age 22 among ever regular smokers, using the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991). Additionally, at age 22, the SSAGA provided a dichotomous lifetime screening measure of DSM-IV SAD symptoms ("SAD-Sc"), assessed as the following question: "Some people have an excessive amount of fear about doing certain things in front of people, such as speaking or eating in a restaurant. In such situations, their fear is severe enough to cause enough anxiety, embarrassment or nervousness that they avoid them. Are there situations that ever caused you any of these feelings?" These structured psychiatric interviews were conducted by trained interviewers in person during a clinic visit or via telephone.

Data Analysis

Differential effects of age—First, we conducted latent growth curve (LGC) analyses to model trajectories of alcohol and cigarette use frequency across time, and to estimate the effects that peer-, parent-, and teacher-rated trait SA (as measured prospectively by the MPNI at age 12) had on these trajectories. These models test for dynamic effects of SA on substance use across different phases of adolescence/young adulthood. These latent growth curve models included measures of drinking or smoking frequency across three waves of data collection, at ages 14, 17, and 22. In these models, latent growth factors (intercept, slope, quadratic) are assumed to underlie the function of change in substance use over time (Preacher, Wichman, MacCallum, & Briggs, 2008). Because there were three measured time points, we could estimate a model with either two latent growth factors (intercept and slope), two latent growth factors plus an autoregressive path that encompasses the direct effect of substance use at one assessment on substance use at the subsequent assessment, or three latent growth factors (intercept, slope, and quadratic effects). If included, the autoregressive path can be estimated two ways: (a) a consistent autoregressive effect across time; or (b) two distinct coefficients representing the effect of age 14 use on age 17 use and the separate effect of age 17 use on age 22 use, allowing for unequal effects between early and late adolescence. For drinking and smoking frequency, we tested both of these autoregressive models plus the two- and three-factor non-autoregressive models, and chose the best-fitting model based on the lowest Akaike's Information Criteria (AIC; Akaike, 1987) and Bayesian Information Criteria (BIC; Schwartz, 1978) and significance of the autoregressive parameters.

For all models, the effects of SA and sex were regressed on the latent factor means. Individuals who had initiated alcohol or cigarette use outside of the home environment by age 12 (n = 408) were excluded from the analysis to eliminate potential influences of substance use on individuals' age 12 SA levels. These analyses were carried out in the OpenMx package version 2.3.1 (Boker et al., 2015) for R statistical software version 3.1 (R Core Team, 2015), using full information maximum likelihood estimation. Interaction effects between sex and SA were tested by including a multiplicative Sex SA term in the regression model, and testing the change in model fit (chi-square test of the -2*loglikelihood) after constraining the interaction term to zero.

Differential effects of the SA rater—To compare the predictive power of parent, teacher, and peer ratings of SA, the best fitting LGC model of alcohol/cigarette use was run with the peer-, parent-, and teacher-rated measures of SA regressed on the latent factor means. Rater differences in the magnitude and significance of the effect of SA on the latent growth factor means were then compared by constraining the parameters to equality and testing the change in model fit with a chi-square test in the difference of the −2*loglikelihood.

Differential effects of substance use outcomes—We used linear regression to assess the relationships of SA with AD symptoms at ages 14 and 22 and with ND symptoms at age 22, including main effects of sex and a multiplicative Sex SA interaction term. Comparison of these models with the latent growth curve models demonstrates whether the SA-substance use relationship differs when substance dependence, rather than frequency, is the measured outcome.

Differential effects of SA measures—We used chi-square tests and linear regression to investigate the association between SAD-Sc and substance use frequency and dependence symptoms. As SAD-Sc was assessed only at age 22, we looked at its associations with concurrent alcohol and cigarette use frequency and concurrent AD and ND symptoms. These analyses were conducted in SPSS 21, using ordinary least squares linear regression for continuous outcomes (AD/ND symptom counts) and chi-square tests for ordinal outcomes (frequency). We employed SPSS's Complex Sampling procedure to account for correlated observations within twin pairs and obtain corrected standard errors. Main effects of sex and a multiplicative Sex SAD-Sc interaction term were included in the regression models.

Genetic and environmental effects—Because this was a twin sample, the variance in the latent growth factors in the LGC models, as well as the residual variance in the measured variables, could be decomposed into contributions from additive genetic effects (A), common environmental effects (C), and unique environmental effects (E). This is possible in a twin sample because monozygotic (MZ) twins have the same genomic sequence, while dizygotic (DZ) twins share, on average, half of their segregating genetic variation, but both types of twins share all of their common environment (defined as factors which contribute to the similarity between twins in a pair). Unique environmental factors are exposures and experiences that make twins less similar. The contribution of each of these

sources of variance can be estimated by comparing the relative similarity of DZ twins to that of MZ twins. Using the standard principles and assumptions of biometrical modeling of twins (Neale & Cardon, 1992), we estimated the amount of variance in the latent and residual factors accounted for by each of these sources.

We also leveraged this genetically informative sample to identify potential mechanisms underlying the covariance between SA and substance use. To do so, we examined cross-twin cross-trait correlations between SA/SAD-Sc and substance use frequency/problems in MZ versus DZ pairs. These are the correlations between one twin's SA and their co-twin's substance use. As described above, higher correlations in MZ twins relative to DZ twins indicate that the cross-trait covariance is driven by shared genetic influences.

Results

Sample descriptives

Table 1 shows the frequencies of alcohol and cigarette use across the sample ages. Substance use frequencies all increased with age. The average age 12 peer-rated social anxiety score was 11.50 (SD = 14.00; range: 0–100; 15% rated as "0"), and males had lower mean SA than females (males: M = 10.04, SD = 12.79; females: M = 13.02, SD = 15.03; $t(1826.9) = -4.66$, $p < .001$, Cohen's d = .21). The average age 12 parent-rated and teacher-rated SA scores were, respectively, 0.81 and 0.86 (range: 0–3; 16% and 24% rated as "0"), with males again having lower scores than females (parent: $t(1773) = -2.98$, $p = .003$, d = .15; teacher: $t(1886) = -3.29$, $p = .001$, d = .13). At age 22, the average number of lifetime AD symptoms was 2.08 (SD = 2.82), the average number of ND symptoms during heaviest lifetime smoking period among ever regular smokers was 2.53 (SD = 2.13), and 18.9% of the sample endorsed having ever experienced symptoms of SAD on the lifetime SAD-Sc screening measure.

Latent growth curve models

For the LGC models of both alcohol and cigarette use frequency, the two latent factor model with no autoregressive paths was the best-fitting model for the data based on fit criteria (results available upon request from the first author). Results from the specific hypotheses tested by this model are presented in the following sections.

Differential effects of SA raters

Correlations for SA ratings between informants were previously found to be 0.24 to 0.32 (Pulkkinen et al., 1999). The upper panel of Table 2 presents correlations between rater reports of SA and substance use frequency measures across waves, which were negative for all measures and were strongest with peer-rated SA. For the LGC models, full results are presented in the next section, but we first compared the effects of parent-, peer-, and teacher-rated SA on the latent growth factors to determine which model to present. Peer-rated SA scores were transformed to be on the same 0–3 scale as parent/teacher ratings. Regression coefficients for each rater from the LGC model are presented in the lower panel of Table 2. SA ratings had a negative association with alcohol and cigarette use trajectories (intercept and slope latent growth factors) across all informants. However, these associations were

statistically significant only for peer-rated SA (both latent growth factors) and teacher-rated SA (slope only). Parameter estimates for peer-rated SA were larger than those of parents and teachers, and these could not be constrained to equality without a significant decrement in model fit ($p < .05$) for both latent growth factors in the alcohol model and for the intercept in the cigarette use model. We therefore interpret the rest of our results using the peer ratings of SA.

Differential effects of age

Results from the LGC models using peer-rated SA are presented in Figures 1 and 2. There was an overall increase in substance use with age in this sample, as indicated by the positive mean value of the latent growth slope. Frequency of drinking began at a higher level and had a greater increase across time than smoking frequency in this sample, likely due to the high proportion of individuals at each assessment who did not smoke regularly. With both drinking and smoking frequency, peer-rated SA had a small but significant negative effect on the latent means of both the intercept and slope. The negative effects on the intercept indicate that individuals with higher levels of social anxiety used alcohol and cigarettes less frequently at age 14. Contrary to our hypothesis, there was not a positive effect of SA on the slopes; instead, the negative effects indicated that higher SA was also associated with a slower rate of increase in AU and CU into late adolescence/young adulthood.

Differential effects of substance use outcome

The latent growth curve analyses indicated a consistent negative association between trait SA and substance use frequency in adolescence and into young adulthood. We next tested the direction of association between trait SA and AD/ND symptoms. Age 12 peer-rated SA was also negatively associated with AD at age 14 ($\beta = -.004$, $p < .001$) and again at age 22 ($\beta = -.01$, $p = .001$). Age 12 peer-rated SA was negatively but not significantly associated with ND at age 22 ($\beta = -.007$, $p = .383$). Parent- and teacher-rated SA also had negative directions of association (age 14 AD: $\beta = -.02/-.02$; age 22 AD: $\beta = -.02/-.07$; ND: $\beta = -.15/-.12$, respectively) but these were not significant (all p's > .2).

Differential effects of social anxiety measure

Finally, we investigated whether SAD-Sc had a different relationship with substance use frequency and problems than dimensional/trait SA. At age 22, individuals who endorsed lifetime SAD-Sc drank less frequently (or not at all) than those who had never experienced SAD symptoms ($\chi^2[7.9] = 18.01$, $p = .025$, Cramer's $V = .125$). They were also less likely to have tried smoking in the first place, but more likely to smoke daily if they had tried smoking ($\chi^2[6.9] = 15.14$, $p = .036$, Cramer's $V = .114$). In addition, endorsement of SAD-Sc at age 22 was positively associated with concurrent AD symptoms ($\beta = 0.38$, $p = .001$). This effect was somewhat stronger among current smokers ($\beta = 0.62$, $p = .004$) than non-smokers ($\beta = 0.14$, $p = .199$), but the interaction term was only marginally significant ($\beta = -0.38$, $p = .086$). However, SAD-Sc was not significantly related to ND symptoms ($\beta = .08$, $p = .735$). Despite their opposite associations with AD, trait SA and SAD-Sc were correlated – peer-rated SA at age 12 positively predicted endorsement of SAD-Sc at age 22 (odds ratio: 1.02, $p < .001$, 95% confidence interval: 1.01–1.03) – though the magnitude of this association was relatively modest.

Sex effects

For both alcohol and cigarette use LGC models, sex had a significant positive effect on the intercept and negative effect on the slope. Females thus began with higher frequency of use than males at age 14, but increased at a slower rate than males as they aged. We also added sex by SA interaction terms to all of the LGC and linear regression models to test for sex differences in the relationship between SA and substance use. All interaction terms were non-significant, (p's > .3), with two exceptions. First, peer-rated SA and sex had a significant interaction in predicting age 22 AD symptoms (β = 0.18, p = .001), meaning that the negative relationship between SA and AD was stronger for boys than girls. Follow-up analyses were conducted to compare the difference in simple slopes and regions of significance for the effect of SA between sexes (Preacher, Curran, & Bauer, 2006). These analyses indicated that the simple slope of association between SA and AD symptoms was negative for boys (estimate: −0.020, 95% CI: −0.027, −0.013) but did not differ from zero for girls (estimate: −0.002, 95% CI: −0.010, 0.006). Therefore, SA was protective against young adult alcohol problems in boys but not girls.

Second, in the smoking frequency LGC models, sex had a modest but significant interaction with peer-rated SA in predicting the intercept (β = −0.05, p = .04) and slope (β = 0.02, p = .04) of the trajectory of smoking frequency across adolescence. The association between SA and early adolescent smoking frequency at age 14 (intercept) was more strongly negative for girls than for boys, with the estimate for girls as −0.051 (95% CI: −0.080, −0.022) and for boys as −0.004 (95% CI: −0.034, 0.023). However, for the change in smoking frequency across time (LGC slope), the negative effect of SA on the slope was attenuated for girls relative to boys. Follow-up analyses indicated that the association between SA and the latent growth slope did not differ from zero for girls (estimate: −0.004, 95% CI: −0.020, 0.012), but was significantly negative for boys (estimate: −0.021, 95% CI: −0.030, −0.013). These results indicate that higher SA was protective against early adolescent smoking for girls but not boys; however, higher SA was protective against the increase in smoking behavior across adolescence for boys.

Genetic and environmental contributions

The proportions of variance and covariance in AU/CU attributable to A, C, and E across ages, as implied by the parameters estimated in the LGC models (Figures 1 and 2), are displayed in Table 3. There were significant genetic influences on individuals' initial drinking frequency (intercept) and their level of change across time (slope), as well as significant age-specific genetic and unique environmental effects at ages 17 and 22. The sources of variance contributing to the latent growth factors and residual measures of smoking frequency could not be as well disentangled; however, common environmental factors did significantly influence smoking trajectories, and the overall effects of A, C, and E on smoking frequency differed significantly from zero (as shown in Table 3). Across substances, the contribution of A and E tended to increase with age while the overall contribution of C decreased, with the largest proportion of effects from C factors contributing to initial drinking and smoking frequency at age 14.

Cross-twin cross-trait correlations between SA/SAD-Sc and substance use are presented in Table 4. For both MZ and DZ twins, almost all of the correlations were non-significant. We do not see a consistent pattern of larger magnitudes in MZ versus DZ twins across the multiple measures of SA/SAD and substance use/dependence that would be indicative of a shared genetic or environmental etiology between the two constructs. However, significant correlations in MZ (but not DZ) twins between SA and smoking frequency (ages 17 and 22) and drinking frequency (age 17), and for SAD-Sc and drinking frequency (age 14) suggest the possibility of a genetic overlap in the etiology of these constructs that may warrant further investigation.

Discussion

Summary of findings

In a longitudinal sample of Finnish twins, the present study examined possible factors contributing to conflicting reports of the direction of association between social anxiety and substance use by testing whether differential associations may be attributable to age, rater reports of SA, SA measure, or substance use outcome. We found that peer ratings of SA were more strongly related to substance use outcomes than parent or teacher ratings, and that peer-rated social anxiety traits at age 12 had a small, but statistically significant, negative effect on the trajectories of alcohol and cigarette use frequency from age 14 to 22. Although the coefficients were small (−0.01 to −0.03), the range of peer-rated SA was 0–100, so they can still indicate large differences in substance use frequency between individuals at the extreme ends of the social anxiety distribution. The direction of association between SA and substance use frequency did not change across time. Age 12 peer-rated SA also negatively predicted AD symptoms at both ages 14 and 22, but was not significantly related to ND. Self-reported endorsement of a SAD screening item in young adulthood was likewise negatively associated with substance use frequency, but, in contrast, were positively associated with AD symptoms, and did not have a significant relationship with ND symptoms. Additionally, we found that shared environmental factors contributed to initial AU/CU while genetic and unique environmental effects had greater impacts later in adolescence/young adulthood. Twin correlations did not offer robust evidence of a shared genetic or environmental liability between SA and substance use.

Relationship between social anxiety and substance use

Our findings provide some support for the hypothesis that distinct aspects or severity levels of SA may contribute to different substance use patterns, as SAD-Sc and peer-rated trait SA showed opposite directions of association with AD symptoms. These two facets of SA are correlated but potentially substantively distinct, and may reflect differences in severity of SA or differential avoidance versus coping pathways (e.g. Bacon & Ham, 2010; Crum & Pratt, 2011). Alternatively, there may be other moderating factors determining whether socially anxious individuals avoid substance use or become dependent; for example, Santesso, Schmidt, & Fox (2004) proposed that high SA in combination with high sociability leads to substance use/dependence to facilitate desired social interactions.

Our results also suggested that there might be unique associations between SA and different facets of substance use (e.g. frequency versus problems), supporting the idea that SA is negatively associated with frequency but positively associated with problems or dependence, as others have previously found (Morris et al., 2005; Schry et al., 2013). However, this was true only for individuals endorsing SAD-Sc, who were more likely to abstain from drinking and smoking but had higher levels of AD; peer-rated trait SA was negatively associated with both AU/CU frequency and AD. Surprisingly, neither SA nor SAD-Sc was related to ND despite several previous studies finding that effect (Buckner et al., 2014; Goodwin et al., 2012; Sonntag et al., 2000). Given the mixed findings of effects in the literature, it may be that the SA-substance use association is simply not very robust and fluctuates with sample and methodological characteristics.

Finally, our results for peer-rated SA did not reveal a reversal in the direction of association between SA and substance use frequency or problems from early adolescence to young adulthood. As we did not have clinical assessments of SAD in early adolescence, it is undetermined whether or not that is also true for SAD. These findings indicate a protective effect of SA against substance use and problems from adolescence through young adulthood, but longer-term follow-ups are necessary to determine whether that persists beyond young adulthood. A previous study demonstrated that teacher-rated anxiety at age 14 had a continued negative association with drinking frequency (but not alcohol problems) at ages 27 and 42 among females (Pitkänen, Kokko, Lyyra & Pulkkinen, 2008). However, because a positive association between SAD-Sc and AD symptoms was already evident at age 22 in our study, it is also possible that the persistent negative association we found between peer-rated SA and substance use/problems in young adulthood was driven by a distinct relationship with trait SA versus SAD as previously theorized.

Rater comparisons

In addition, we found that aggregate peer ratings of SA in early adolescence were stronger predictors of substance use than either parent or teacher ratings of SA at the same age. Consistent with previous research (Martin-Storey et al., 2011), these results indicate that peers are particularly well suited to provide assessments of behavioral and emotional constructs in children and adolescents, especially those relevant to interpersonal social functioning. It is interesting to note that parental SA ratings were not significantly associated with substance use, perhaps indicating a systematic bias in how parents' evaluate their children's emotional functioning – or that they are not exposed to the aspects of their children's social behavior that is important to adolescent substance use (e.g. peer interactions). Research using the same sample and assessment as the present study (Pulkkinen et al., 1999) has found that peer reports are more reliable than either parent or teacher reports of a child's behavioral and emotional traits, and other studies have found that young children can accurately identify constructs such as SA and other internalizing traits in their same-age peers, even more so than adults (Miers, Blöte, & Westenberg, 2010). Clemans and colleagues (2014) similarly found that peer ratings of behavior were better predictors of associated outcomes than parent or teacher ratings. This suggests that, when possible, peer reports should be collected in research involving younger age groups in order to provide more accurate and useful indicators than might be obtained from other sources.

Genetic and environmental contributions to substance use

Consistent with previous studies (Kendler et al., 2008; Dick et al., 2007), the twin models showed an increasing contribution of genetic and unique environmental factors and a decreasing contribution of shared environmental factors to substance use with older age. The shared environment maintained a relatively high importance to smoking frequency (~30% of the variance) even in young adulthood. Genetic factors had a significant impact on both the intercept and slope of drinking frequency, indicating that this trajectory is genetically influenced, with additional strong time-specific influences of genes and unique environmental factors at ages 17 and 22. The covariance between substance use at ages 14, 17, and 22 was primarily driven by common environmental factors, indicating that intra-individual similarity in substance use between early adolescence and later in life is largely due to impacts of the familial environment on initial levels of substance use. The small cross-twin cross-trait correlations indicated that genetic and shared environmental factors are largely not overlapping between SA and substance use, although further investigation is needed. Specifically, significant correlations within MZ pairs between SA and drinking/smoking frequency is suggestive of a shared genetic etiology, although inconsistency across ages and measures (SA versus SAD-Sc and substance frequency versus dependence) highlights the need for caution in such an inference. A thorough investigation of this using formal bivariate biometric modeling is beyond the scope of this investigation and likely underpowered in our sample, but would be a worthwhile direction for future studies.

Limitations

There are several limitations that influence how this study's findings should be interpreted. Self-report measures of substance use, particularly by underage participants, may not be entirely accurate, though there are few practical alternatives to this method. The two items used to create the SA measure in the peer nominations, and the single SAD screening item in the clinical interview may not be fully representative of the true construct of social anxiety. Low internal consistency for some of the SA measures (e.g. α = .42–.46 for parent-rated SA) indicate this may not be the best construction of SA, although the reliability of peer-rated SA was higher (α = .68–.81). Further research with more thorough assessments of SA would provide greater support for the conclusions of this study. However, our study is strengthened by using measurements of SA from aggregated reports of classroom peers who are uniquely situated to evaluate social behaviors in their peers, which should decrease any individual source of bias and improve the reliability and validity of the measure. While this sample of twins was ascertained through unbiased measures and is representative of the population of Finland, caution should be taken in generalizing these findings. Twins did not differ from their classroom peers with respect to mean social anxiety (Pulkkinen et al, 1999), which shows that twins represent the Finnish adolescent population very well. As with virtually all longitudinal studies, sample attrition may reduce the reliability of the results, as we noted there were some significant differences between the characteristics of those who did and did not respond to all waves of the study. Our use of a full information maximum likelihood estimator in the latent growth curves model greatly reduces the potential biases due to sample attrition. In addition, excluding individuals with early (pre-age 12) initiation of substance use ensured that SA ratings were not influenced by substance use itself, but may obscure the relationship between SA and early adolescent substance use. We note that

including these individuals in the analyses led to virtually identical estimates of the association between SA and substance use. We also interpreted the differences in direction of association between trait SA and SAD-Sc to mean that these are distinct constructs with different relationships to substance use, but these were also assessed by different methods (informant versus self-report), which might account for differences. The fact that peer-rated SA predicted SAD-Sc provides some convergent validity, but this possibility should be considered. It may also be that self-perceptions of SA are more relevant to substance use than other indicators.

Despite these limitations, the present study provided a detailed examination of the relationship between SA and substance use, and the numerous factors that complicate this relationship. The longitudinal design of this study allows for stronger inferences to be drawn about the lifelong relationship between SA and substance use, as well as the developmental course of substance use, than cross-sectional studies that examine only a single time point. This study's findings, as always, should be considered in light of its limitations, but the measures from multiple raters, prospective longitudinal design, and multiple methods of examining the SA-substance use relationship should encourage confidence in the findings.

Conclusion

The present study provided evidence that the conflicting direction of association between SA and alcohol use may be due to different aspects or levels of severity of SA, with some socially anxious individuals avoiding heavy substance use, and others with clinically significant distress/impairment (SAD symptoms) being more likely to develop dependence, given initiation of use. An understanding of what factors differentiate these individuals could aid in identifying those most in need of prevention and/or treatment programs. In addition, we found evidence that both trait SA and SAD are related to lower frequency of alcohol and cigarette use across both adolescence and young adulthood. Further research is needed to extend this investigation beyond young adulthood, and to determine what aspects of social anxiety differ between SAD and trait SA/shyness to determine why one appears to increase risk for alcohol problems while the other does not. This underlying etiology may have clinical implications for treating the root cause of alcohol problems among socially anxious individuals; for example it may be that coping motives (Ham et al., 2007), inexperience with drinking due to avoidance of social situations (Clerkin & Barnett, 2012), or specific types of fears (Morris et al., 2005) may be driving hazardous use. Though SA was not associated with nicotine dependence in this sample, previous reports of its relationship to craving, coping-related smoking, and lower quitting success suggest that certain aspects of SA may be important to address in prevention and treatment of nicotine use among socially anxious individuals. Although genetic influences on alcohol and cigarette use increased from adolescence to young adulthood, the lack of concurrent increases in the association between SA and substance use, as well as the lack of correlation for these constructs across twins, suggests that environmental factors are the primary cause for their association. Once identified, these factors could present modifiable targets for prevention and treatment programs.

Acknowledgments

Funding for this study was provided by grants UL1TR000058 from the National Institutes of Health's National Center for Advancing Translational Science and T32MH020030 from the National Institute of Mental Health (JES), U01DA024413, R25DA026119, and R37DA018673 from the National Institute on Drug Abuse (BV), K01AA018755, AA015416, and AA015416-S1 (DMD) and AA12502, AA00145, and AA09203 (RJR) from the National Institute on Alcohol Abuse and Alcoholism, grants 100499, 205585, 141054, 118555, 265240, 263278, and 264146 from the Academy of Finland (JK), and the Academy of Finland Centre of Excellence Programme (LP and JK). The funding sources had no further role in the study design, data analysis, writing of the report, or decision to submit this manuscript for publication.

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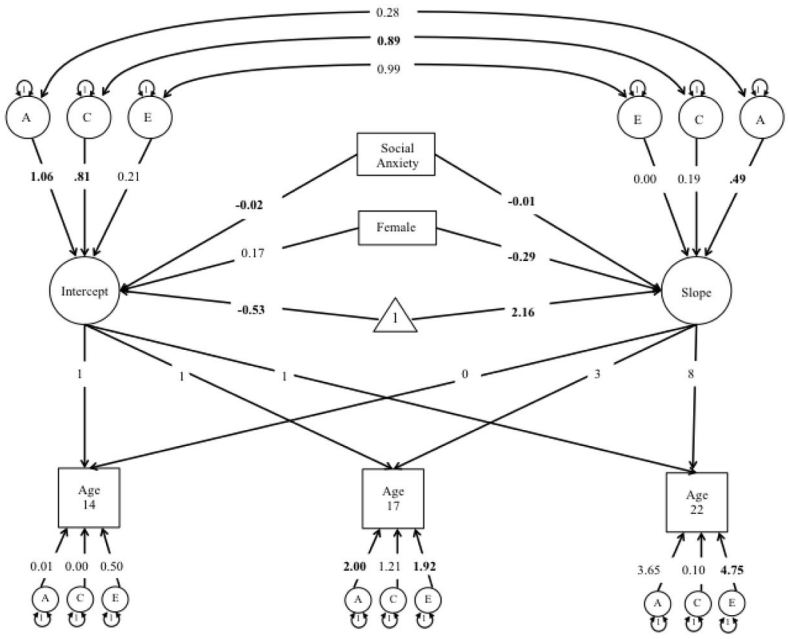

Figure 1.

Path diagram of a biometric latent growth curve model representing the effect of peer-rated social anxiety (SA) at age 12 on drinking frequency across adolescence and young adulthood. Parameter estimates are unstandardized. Bolded values significantly differ from zero, $p < .05$.

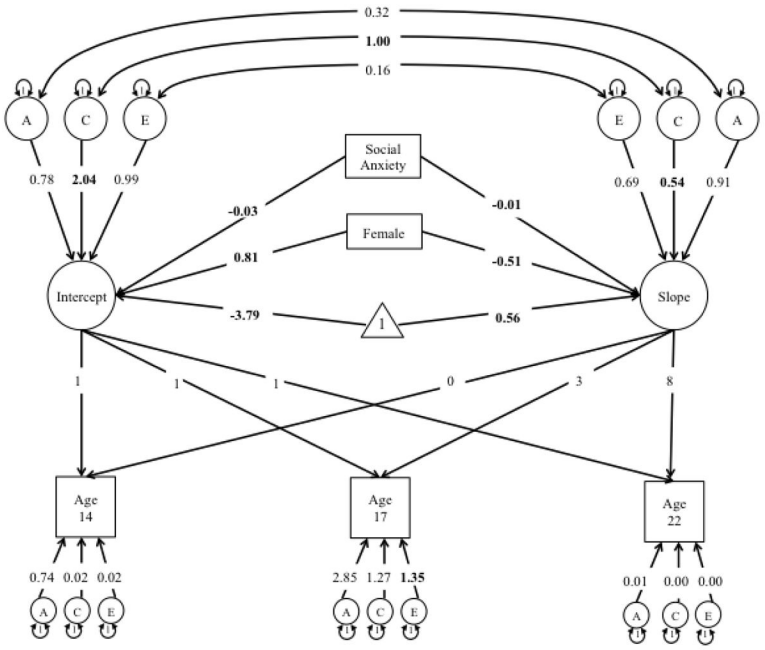

Figure 2.

Path diagram of a biometric latent growth curve model representing the effect of peer-rated social anxiety (SA) at age 12 on cigarette smoking frequency across adolescence and young adulthood. Parameter estimates are unstandardized. Bolded values significantly differ from zero, $p < .05$.

Table 1

Frequency of alcohol and cigarette use across adolescence and young adulthood

Measure	Age 14 N (%)	Age 17 N (%)	Age 22 N (%)
Drinking Frequency			
Never/don't drink	1062 (62.3)	135 (10.2)	55 (4.5)
Once a year or less	--	56 (4.2)	11 (0.9)
2–4 times a year [a]	370 (21.7)	119 (9.0)	56 (4.6)
About once every 2 months	--	134 (10.1)	61 (5.0)
About once a month [a]	220 (12.9)	175 (13.2)	110 (9.0)
A couple of times a month	--	367 (27.7)	289 (23.6)
Once a week [a]	53 (3.1)	237 (17.9)	350 (28.6)
A couple of times a week	--	102 (7.7)	278 (22.7)
Daily	--	1 (0.1)	15 (1.2)
Smoking Frequency			
Never tried	957 (56.4)	366 (27.2)	168 (13.7)
Tried but don't smoke	504 (29.7)	361 (27.4)	446 (36.4)
Trying to/have quit [b]	78 (4.6)	80 (6.1)	113 (9.2)
Less than once a week	42 (2.5)	56 (4.2)	73 (6.0)
Once a week or more but not daily	45 (2.7)	76 (5.8)	63 (5.1)
1–9 cigarettes daily [a]	72 (4.2)	239 (18.1)	151 (12.3)
10–19 cigarettes daily	--	127 (9.6)	169 (13.8)
20+ cigarettes daily	--	14 (1.1)	41 (3.3)
Total N	*1705*	*1326*	*1225*

Note: Dashes indicate response options that were not presented at age 14.

[a] Response option differed slightly at age 14, see text for details;

[b] Response option was excluded from analyses.

Table 2

Comparison of the association with substance use between peer-, parent-, and teacher-rated social anxiety at age 12. Top panel: correlations with substance use at each measurement occasion; bottom panel: regression coefficients predicting latent growth factors (Figs. 1–2) underlying alcohol and cigarette use trajectories from age 14–22.

Outcome	Peer	Parent	Teacher
Drinking Frequency			
Age 14	−0.18***	−0.05	−0.14**
Age 17	−0.12**	−0.01	−0.01
Age 22	−0.10*	−0.11*	−0.05
Smoking Frequency			
Age 14	−0.21*	−0.04	−0.01
Age 17	−0.13*	−0.09	−0.02
Age 22	−0.17**	−0.13*	−0.05
Drinking Frequency			
Intercept	−0.44*	−0.06	−0.10
Slope	−0.24*	−0.06	−0.09*
Smoking Frequency			
Intercept	−1.06*	−0.17	−0.26
Slope	−0.35*	−0.07	−0.14*

Note: Polyserial correlation coefficients are reported for the ordinal frequency measures. Correlations were conducted with one twin from each pair and estimates were similar when replicated in their co-twins.

* $p < .05$,

** $p < .01$,

*** $p < .001$

Table 3

Proportions of variance and covariance in alcohol and cigarette use frequency attributable to additive genetic effects (A), common environmental effects (C), and unique environmental effects (E) across adolescence and young adulthood. Diagonal elements represent the variance in alcohol/cigarette use frequency at each age while off-diagonal elements represent the covariance between frequency measures across ages.

Drinking Frequency			
Age	**Age 14**	**Age 17**	**Age 22**
Proportions due to Additive Genetic (A) factors			
Age 14	0.54 (0.31–0.76)		
Age 17	0.29 (−0.06–0.65)	0.41 (0.21–0.60)	
Age 22	−0.02 (−0.63–0.60)	0.40 (0.04–0.75)	0.42 (0.17–0.6)
Proportions due to Common Environmental (C) factors			
Age 14	0.32 (0.11–0.53)		
Age 17	0.66 (0.33–0.95)	0.34 (0.17–0.50)	
Age 22	0.94 (0.38–1.00)	0.55 (0.23–0.85)	0.23 (0.11–0.43)
Proportions due to Unique Environmental (E) factors			
Age 14	0.14 (0.09–0.21)		
Age 17	0.05 (−0.04–0.18)	0.25 (0.20–0.33)	
Age 22	0.08 (−0.10–0.25)	0.05 (0.00–0.18)	0.35 (0.27–0.45)
Smoking Frequency			
Age	Age 14	Age 17	Age 22
Proportions due to Additive Genetic (A) factors			
Age 14	0.18 (0.14–0.55)		
Age 17	0.13 (−0.04–0.52)	0.44 (0.44–0.55)	
Age 22	0.14 (−0.02–0.66)	0.40 (0.12–0.66)	0.46 (0.16–0.69)
Proportions due to Common Environmental (C) factors			
Age 14	0.66 (0.28–0.86)		
Age 17	0.74 (0.61–0.94)	0.37 (0.19–0.42)	
Age 22	0.75 (0.53–0.94)	0.38 (0.14–0.52)	0.30 (0.14–0.51)
Proportions due to Unique Environmental (E) factors			
Age 14	0.16 (0.14–0.16)		
Age 17	0.13 (−0.04–0.14)	0.19 (0.14–0.20)	
Age 22	0.11 (0.09–0.36)	0.22 (0.15–0.23)	0.25 (0.22–0.26)

Note: Variance component estimates are derived from the latent growth curve models (Figs. 1–2). Ninety-five percent confidence intervals are shown in parentheses.

Table 4

Cross-twin cross-trait correlations between social anxiety and substance use measures

	Peer-rated SA		SAD Symptoms	
	rMZ	**rDZ**	**rMZ**	**rDZ**
Drinking Frequency				
Age 14	−0.04	−0.10	0.35*	0.09
Age 17	−0.25*	−0.07	0.10	0.05
Age 22	−0.07	−0.12	0.16	0.11
Smoking Frequency				
Age 14	−0.07	−0.07	0.03	0.03
Age 17	−0.27*	−0.03	0.01	0.04
Age 22	−0.24*	−0.06	0.11	0.09
AD Symptoms				
Age 14	−0.10	−0.03	−0.04	−0.10
Age 22	−0.05	−0.04	−0.04	−0.13
ND Symptoms (Age 22)	0.04	0.03	−0.08	0.03

Note: Pearson correlation coefficients are reported for pairs of continuous measures (peer-rated SA & AD/ND symptoms), polyserial correlation coefficients for continuous-ordinal pairs (peer-rated SA & frequency; SAD & symptoms), and polychoric correlation coefficients for categorical-ordinal pairs (SAD & frequency). SA = trait social anxiety, SAD = social anxiety disorder symptom endorsement, rMZ = correlation for monozygotic twin pairs; rDZ = correlation for dizygotic twin pairs, AD = alcohol dependence, ND = nicotine dependence.

* $p < .05$