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Gender and direction of effect of alcohol problems and internalising symptoms in a longitudinal sample of college students

Lina E. Homman, MSc¹, Alexis C. Edwards, PhD², Seung Bin Cho, PhD^{2,3}, Danielle M. Dick, PhD³, and Kenneth S. Kendler, M.D.²

¹Institute of Clinical Sciences, Block B. Queens University Belfast. Royal Victoria Hospital. Belfast, BT12 6BA, Northern Ireland, UK

²Virginia Institute for Psychiatric and Behavioral Genetics, Department of Psychiatry, Virginia Commonwealth University. PO Box 980126, Richmond, VA, USA, 23298-0126

³Departments of Psychology and African American Studies, Virginia Commonwealth University, PO Box 842509, Richmond, VA 23284-2509

Abstract

Background—Alcohol problems and internalising symptoms are consistently found to be associated but how they relate to each other is unclear.

Objective—The present study aimed to address limitations in the literature of comorbidity of alcohol problems and internalising symptoms by investigating the direction of effect between the phenotypes and possible gender differences in college students.

Method—We utilised data from a large longitudinal study of college students from the United States (N=2607). Three waves of questionnaire-based data were collected over the first two years of college (in 2011–2013). Cross-lagged models were applied to examine the possible direction of effect of internalising symptoms and alcohol problems. Possible effects of gender were investigated using multigroup modelling.

Results—There were significant correlations between alcohol problems and internalising symptoms. A direction of effect was found between alcohol problems and internalising symptoms but differed between genders. A unidirectional relationship varying with age was identified for males where alcohol problems initially predicted internalising symptoms followed by internalising symptoms predicting alcohol problems. For females, a unidirectional relationship existed wherein alcohol problems predicted internalising symptoms.

Conclusions/Importance—We conclude that the relationship between alcohol problems and internalising symptoms is complex and differ between genders. In males, both phenotypes are

Correspondence should be addressed to: Lina E. Homman., Institute of Clinical Sciences, Block B. Room 02.032. Queens University Belfast. Royal, Victoria Hospital. Belfast, BT12 6BA, Northern Ireland, UK., Telephone: 02890635363, Fax: 02890 235900, L.Homman@qub.ac.uk.

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predictive of each other, while in females the relationship is driven by alcohol problems. Importantly, our study examines a population-based sample, revealing that the observed relationships between alcohol problems and internalising symptoms are not limited to individuals with clinically diagnosed mental health or substance use problems.

Keywords

Alcohol; internalising symptoms; depression; anxiety; longitudinal; college sample; cross-lagged modelling

Introduction

Prevalence of internalising symptoms (IS) and alcohol use problems (AP) (symptoms of alcohol abuse and dependence) increase notably in adolescence, in particular as individuals enter college (Costello et al., 2003; O'Malley Johnston., 2002; Wechsler et al., 2000). IS is a term describing conditions where an individual focuses problems inwards and include symptoms of social withdrawal, anxiety and depression (Achenbach & McConaughy, 1992). The two phenotypes often co-occur and the presence of either generally results in a 2–4x increased odds of having the other, as compared to individuals without either (Costello et al., 2003; Lewinsohn et al., 1998; Trautman et al., 2015). AP and IS each presents a health problem on its own, but when the two co-occur, it can result in increased severity of symptoms, increased risk of suicide, less adaptive functioning and less effective treatment (Jackson et al., 2005; Rao, 2006; Subramaniam et al., 2007). To avoid the adverse effects of comorbid AP and IS and to improve prevention and treatment efforts, it is important to understand how AP and IS are associated.

The relationship between the two phenotypes is complex and the degree to which IS is a consequence of AP versus vice versa is unclear. The majority of previous research focuses on AP's relationship to depression or anxiety, both of which are consistently correlated with AP (Wolitzky-Taylor et al., 2012). Evidence supports a higher order classification of depression and anxiety as one internalising factor (Krueger, 1999; Mineka et al., 1998; Watson, 2005). This is also supported by research in genetics, suggesting that the genetics of generalised anxiety and depression are highly correlated (Kendler et al., 2003). When an internalising factor is accounted for, a specific internalising disorder on its own has not been shown to contribute any additional information on the relationship between AP and IS (Kushner et al., 2012). Moreover, it is often more practical to study IS as a single dimension in population-based studies due to the low prevalence of anxiety and depressive disorders in the general population (Goodman et al., 2010).

Problematic levels of both alcohol use and IS have been reported in college students (American College Health Association, 2012; O'Malley & Johnston, 2002). Both phenotypes increase notably in adolescence and continue to do so in early adulthood (Costello et al., 2003; O'Malley Johnston., 2002; Wechsler et al., 2000). An additional increase in alcohol use is frequently observed in college students; often considered to be due to the absence of parental supervision and easier access due to other peers of legal age and other drinking peers (Carter et al., 2010; O'Malley & Johnston, 2002). In addition, alcohol

use, AP and IS are hypothesised to increase as a consequence of the significant life changes associated with starting college, making this transitional time a vulnerable period (Keyes et al., 2012; Lewinsohn et al., 1998). Such changes include freedom from parental supervision, new responsibilities such as financial responsibilities, and several possibly stressful factors such as academic stress, establishing and maintaining relationships, developing an identity, and transitional stress from living with parents to living with other students (Upcraft & Gardner, 1998). Stressful factors are often associated with an increase in problems such as AP and IS (Lewinsohn et al., 1998), suggesting college is a time period which may increase the likelihood of developing AP and/or IS in some individuals.

Several studies have attempted to elucidate the relationship between AP and IS, but results are inconsistent. Within phenotype continuity has been generally identified as well as an association between the two phenotypes (Angold et al., 1999; Marmorstein, 2010). Evidence suggests that a shared liability (genetic and/or environmental) likely contribute to their co-occurrence (Kendler et al., 2003). It has been hypothesised that there is a direction of effect between the phenotypes: one phenotype might precede and/or trigger the other (Fergusson et al., 2009; Stice et al., 1998; Wolitzky-Taylor et al., 2012), or the relationship may be of a reciprocal nature (Marmorstein, 2010; Needham, 2007). However, the literature is not consistent in regards to direction of effect. There is evidence supporting that IS (O'Neil et al., 2011; Stice et al., 1998), depression (Wolitzky-Taylor et al., 2012), and anxiety (Buckner & Schmidt, 2009) predicts/precedes AP. On the contrary, there is evidence that AP predicts/precedes IS (Trim et al., 2007), depression (Fergusson et al., 2009) and anxiety (Schuckit, 1996). Reviews of direction of effect between the phenotypes similarly lack consistency. Reviews have shown that AP predicts depression (Boden & Fergusson, 2011) and anxiety (Allan et al., 1995), while other indicates that IS predicts AP (O'Neil et al., 2011).

It is important to understand whether one phenotype predicts the other in order to improve treatment and prevention. However, majority of these studies may be biased due to either recall bias of using retrospective information, or due to have looked at wide age ranges at each measuring point, possibly resulting in issues as the relationship of the phenotypes has been shown to change with age (O'Neil et al., 2011). Additionally, some studies address alcohol problems in conjunction with other substances rather than only alcohol. For example, Marmorstein and colleagues (2010) investigated direction of effect between depression and substance dependence in adolescence. Furthermore, both phenotypes peak in late adolescence/early adulthood, in particular in college, and their presence at this age is strongly predictive of its presence later on in life. Adolescence is therefore an important period of time to understand how the phenotypes are related. Moreover, in order to investigate possible direction of effect between the phenotype, longitudinal studies are necessary. However, longitudinal studies addressing the relationship of AP and IS (rather than depression or anxiety separately) in a sample of college students are highly spare and those which do exist are inconsistent in their findings.

There are several mechanisms which may account for the comorbidity of two phenotypes (Neale & Kendler, 1995). Two are of particular interests in regards to the association between AP and IS in college students. Each mechanism proposes that one phenotype may increase the risk of the other and thereby function as a risk factor on its own. Firstly, alcohol

use increases significantly among students in college and thereby the risk of AP (O'Malley & Johnston, 2002; Yu & Shacket, 2001). AP are hypothesised to directly cause IS due to the bio-psycho-social consequences of acute and chronic alcohol abuse and dependence (George et al., 1990; Rao, 2006). For example, a consequence of alcohol abuse is decreased levels of serotonin, resulting in lowered mood (Rao, 2006). AP may also have detrimental effects on family, academic work, legal issues, health and social life which in turn may result in IS (Foster et al., 1999). Secondly, IS may increase the likelihood of developing AP via the self-medication hypothesis (Khantzian, 1997). Small doses of alcohol act as a stimulant and a depressant, resulting in a perceived reduction in symptoms of IS (Carrigan & Randall, 2003). Alcohol use as a method of coping may lead to a lack of developing efficient coping mechanisms to deal with stressors and may exacerbate the problem (Marmorstein et al., 2010). Moreover, all of the above mechanisms may also work simultaneously or trigger each other, resulting in a bi-directional relationship. For example, self-medication of IS with alcohol use may promote continued alcohol use and misuse which via its psychopharmacological effects (such as a decrease of serotonin), could increase the symptoms of IS.

Gender differences

Gender differences in the prevalence of AP and IS, as well as in the relationship between the phenotypes, have been consistently shown. Females generally exhibit a higher prevalence of IS throughout adolescence and into young adulthood (Costello et al., 2003; Poulin et al., 2005; Windle & Windle, 2006) while males consistently report a higher prevalence of AP compared to females (Kivimäki et al., 2014). The literature is conflicting in regards to whether directional pathways of AP and IS differ between genders. A large proportion of studies provide evidence that the associations between AP and IS are stronger in females than in males (Clark et al., 1997; Costello et al., 2003; Dawson et al., 2010), some indicate that the association is stronger in males than females (Marmorstein & Iacano, 2011) while others find no difference by gender (Fergusson et al., 2009; Brook et al., 1998; Marmorstein, 2009). Moreover, it is hypothesised that IS is a stronger predictor of AP in females and normally precedes AP while in males AP is a stronger predictor of IS and normally precedes IS (Deykin et al., 1992; Moscato et al., 1997; O'Neil et al., 2011). However, the results from studies examining the relationship between AP and IS are currently inconclusive.

Present study

Using a longitudinally-assessed sample of college students, the present study aims to i) establish whether an association exists between AP and IS, ii) whether the direction of effect between the phenotypes is present and iii) investigate gender differences within the presentation of AP and IS. We hypothesise that an association between phenotypes is present in the form of a bidirectional relationship. We base this hypothesis on two observations. Firstly, both phenotypes share risk factors, and additional environmental risk factors for both phenotypes increase as individuals enter college, increasing the risk for both phenotypes. Secondly, as both phenotypes are likely to increase at this time, they are also likely to trigger each other according to the two main mechanisms accounting for the association between the two phenotypes; suggesting a bi-directional relationship. Based on previous literature, we also hypothesise that females report a stronger between phenotype association. The

present study adds to the literature by examining a longitudinal sample of college students, addressing the topic longitudinally rather than cross-sectionally in a sample of increased vulnerability to both phenotypes.

Method

Procedure and sample

This study used data from the longitudinal study Spit for Science (S4S) (Dick et al., 2014). This is an ongoing study of college students at a large, urban, public university in the Mid-Atlantic region of the United States. Students 18 years or older were invited to take part as they started their first fall semester of college. Students who did not participate were offered a second chance of participating the following spring. S4S data from the first cohort is included in this analysis as this is the only cohort with a sufficient number of data collection points at the time of writing. Study data were collected and managed using Research Electronic Data Capture (REDCap) electronic data capture tools hosted at Virginia Commonwealth University (Harris et al., 2009).

The initial wave of data collection occurred in the fall of 2011 when the students were incoming freshmen, the second in the spring of 2012 and the third in the spring of 2013. A total of 2715 participants took part in the study out of which 2607 participants were included in the present analyses. Participants were excluded due to not answering any of the alcohol problems (N=16) items, internalising symptoms items (N=57), or items about ethnicity (N=58). In the initial wave, 2056 took part, 2221 took part in the second wave (N=659 participants joined the study in the second wave) and 1349 in the final wave. Only 36.5% of the sample took part in all three waves while 71.71% of the sample took part in two of the waves. Participants with complete and incomplete data across the waves did not differ significantly on IS or AP. Being female predicted an increase in the odds of participating in all waves compared to males (OR=1.52, 95% CI=1.29-1.80, $p<.001$). The sample consisted of 1035 males and 1572 females. The full sample consisted of 15.29% Asians, 19.54% African Americans, 5.93% Latin Americans, 52.07% Whites, 6.87% of more than one race and less than 1% American Indians and Native Hawaiians. The mean age in wave 1 was 18.49 years ($SD=.61$), in wave 2 19.05 years ($SD=.71$) and in wave 3 19.96 years ($SD=.39$). Informed consent was given by all participants for participation in the study.

Measures

Internalising symptoms—Eight items from the SCL-90 (Derogatis et al., 1973) measuring IS were used in all 3 waves. Of the 8 items included, 4 were related to depression and 4 to anxiety. Items related to depression were; feeling blue, loss of interest, hopeless about future, and worrying too much. Anxiety items concerned being scared for no reason, fearful, spells of panic and nervousness. Participants were asked to report symptoms from the last 30 days. All the items had a likert response scale of 5 options ranging from “not at all” to “extremely”. The items of the SCL-90 were hypothesised to represent an underlying IS scale. Exploratory factor analysis (EFA) and confirmatory factor analysis (CFA) were performed to test this hypothesis. EFA was conducted on a random subset of the sample and was verified with CFA on the remaining sample. Moreover, internal reliability was good for

wave 1 ($\alpha = .87$), wave 2 ($\alpha = .87$) and wave 3 ($\alpha = .89$). Participants missing more than half (4) of the items were excluded (wave 1: 3.31%; wave 2: 4.05%; wave 3: 3.78%). For participants for whom 4 or more items were present, the items were summed. For participants with missing data on 1-4 items, the missing items were replaced with the mean of the non-missing items.

Alcohol problems—AP were measured with 9 items adapted from the Semi-Structured Assessment of the Genetics of Alcoholism (Bucholz et al., 1994). These items address craving (cravings in situations where drinking was not possible) as well as symptoms of alcohol dependence (AD) in accordance with DSM-IV (tolerance, wanting to stop/cut down on drinking, intending not to drink/getting drunk but drinking/getting drunk anyway, given up activities due to drinking, no time for other activities due to drinking, continued drinking even though it was causing problems, withdrawal symptoms). We concluded that we were assessing AP rather than AD as we only considered symptoms rather than making a diagnosis. Items were scored on a three-point ordinal response scale: “never”, “1–2 times” “3 or more times”. Participants missing data on more than 4 of the items were excluded (wave 1: 0.96%; wave 2: 1.27%; wave 3: 1.04%). A sum score of AP was created for symptoms occurring “3 or more times” for participants with 5 or more non-missing values. Scores were pro-rated for those participants who had responded to all 5-8 items. Participants who had responded that they had: never drank alcohol, never been drunk, never drank regularly or never had more than 3 drinks, were not administered the 9 items, and were considered to not have any AP (coded as 0) (16.06 %). The analysis was also performed assigning these participants as having missing AP data, results did not differ significantly (not reported). Internal reliability was fair; wave 1: $\alpha = .64$, wave 2: $\alpha = .65$, wave 3: $\alpha = .69$.

Statistical analysis

Within phenotype predictability and direction of effect between phenotypes were tested using cross-lagged models. A primary model consisted of regression paths where one phenotype predicted the other phenotype at the immediate subsequent time point (cross-lagged paths). One phenotypes' initial and immediately following waves were allowed to predict consecutive waves (auto-regressive paths). A correlation between AP and IS in wave 1 was estimated. This concluded our primary model. Factor analyses was not incorporated as sum scores were satisfactory; alcohol items have well established psychometric properties with high homogenous factor loadings, and EFA for IS indicated that sum scores were an acceptable method.

Two variants of the primary model were tested for the best possible fit of the data. First, a within phenotype path from time point 1 to time point 3 was added to the models since strong correlations were identified between them. Second, residual correlations between AP and IS at time points 2 and 3 were added to the model. These residual correlations represent the shared, or common, effects of excluded predictors. Without residual correlations the model assumes the cross-sectional relationship between the two variables to be explained by the relationship between preceding variables; this is a strong assumption and may lead to overestimated cross-lagged relationships. To test whether variants of the primary model resulted in a better fit of the data the different models were estimated and compared.

Initially, a model including all paths (the paths of the primary model and the variant models) was estimated. Paths of the variant models were then constrained in succession and compared to the model estimating all paths. Paths were retained if constraining them resulted in a significantly worse fit to the data. The best model was selected by highest CFI and lowest RMSEA, SRMR, BIC and χ^2 . A good model fit was suggested by values of: CFI > .95, RMSEA < .06, SRMR < .08, and lowest possible BIC value (Hu & Bentler, 1999). A model estimating all paths is presented in Figure 1.

To assess for gender differences, multigroup analysis testing was performed in order to assess for differences of parameters between males and females. An initial model (to which all other models were compared) allowed all parameters (according to the best model fit of the data for the full sample) to be estimated separately by gender. A second model fixed all parameters to be equal between genders to test whether an overall difference between genders could be observed. If an overall difference was observed, further stepwise analysis was conducted constraining one set of parameters equal across genders while freeing all other parameters in order to test which exact parameter(s) differed between genders. The parameters were fixed in the following order; 1) correlation (and residual correlations if applicable); 2) cross-lagged paths; and 3) autoregressive paths (within phenotype paths). Models were compared by comparing the χ^2 difference to the initial model. Data was prepared and descriptive results obtained using STATA 12 and cross-lagged models fit in Mplus 7.11 (Muthén & Muthén, 2013). All analyses in Mplus were performed using Full Information Maximum Likelihood (FIML) to take account of missing values.

Results

EFA and CFA of SCL-90 items

EFA supported a one-factor solution according to eigenvalues and scree plot. The factor loadings were high at each wave (range: .70–.81 (wave 1); .73–.82 (wave 2); .75–.86 (wave 3)). CFA confirmed a 1-factor model, with a good fit as indicated by high CFI (CFI=.95) and a low RMSEA (RMSEA=.054).

Descriptive statistics

Means and standard deviations of the distribution of AP and IS within the sample are presented in Table 1. Mean AP increased throughout the study for both males and females. Mean IS increased between wave 1 and 2 and decreased between waves 2 and 3. Females consistently reported higher levels of IS than males and males reported higher levels of AP than females in all waves.

Correlations of AP and IS across waves are presented in Table 2. Within phenotype correlations indicated that IS were significantly correlated across waves. Likewise, AP were significantly correlated across waves. Results indicated significant cross-sectional correlations between AP and IS within each time point. Longitudinal between phenotype correlations indicated that; i) AP at wave 1 were correlated with IS at wave 2 but not at wave 3, ii) IS at wave 1 was correlated with AP at wave 2 and iii) IS at wave 2 was correlated with

AP at wave 3. Results were consistent across genders with one exception; ii) was significant for males but not females.

Cross-lagged models

A cross-lagged model was estimated for AP and IS from wave 1 to wave 3; standardized results are presented in Table 3. Constraining wave 1 to predict wave 3 within AP or IS decreased the fit significantly, as did constraining the residual correlations between AP and IS in wave 2 and 3. A model estimating all paths was retained as it provided the best model fit, and a good model fit according to RMSEA, SRMR, CFI and χ^2 .

The final path estimates for the cross-lagged model are presented in Figure 1. The significant autoregressive paths indicated within phenotype continuity. AP and IS at wave 1 were significantly correlated. Residual correlations between AP and IS at wave 2 and 3 were significant. AP in wave 1 significantly and positively predicted IS at wave 2, but AP at wave 2 did not significantly predict IS at wave 3. IS at wave 1 did not significantly predict AP at wave 2 but IS at wave 2 significantly and positively predicted AP at wave 3. Constraining non-significant paths to zero did not change the fit of the model (χ^2 difference=1.35, df=2), which suggests that the non-significant paths did not have an impact on the model fit. A model estimating all paths was therefore used.

Gender differences

Results of multigroup analyses of a cross-lagged model (including all paths as suggested by model estimation for the full sample) of AP and IS are presented in Table 4. Multigroup analyses between genders indicated a significant difference in χ^2 between a model in which parameters were freely estimated across genders and one in which estimates were fixed to be the same across genders. Further analyses investigating which variables differed between genders indicated that differences by genders were only significant with respect to autoregressive paths. However, when investigating the models for each gender separately, other gender differences were also observed.

All paths and their significance by gender are displayed in Figure 1. All autoregressive paths were significant for both genders and indicated higher coefficient for females than males. The correlation between AP and IS did not differ significantly between genders at wave 1 and residual correlations did not differ at wave 2. A residual correlation in wave 3 were not significant for males but was for females. AP at wave 1 significantly predicted IS at wave 2 for both genders, however, IS at wave 2 significantly predicted AP at wave 3 for males but not for females. No other cross-lagged paths were significant.

Discussion

Using a large longitudinally assessed college sample, this study examined: a) longitudinal relationships in terms of direction of effect and b) gender differences in the relationship between AP and IS. Correlations between AP and IS indicated a significant relationship cross-sectionally and to some extent longitudinally. Cross-sectional correlations were consistent across both genders while longitudinal correlations were less pronounced among females. A direction of effect between AP and IS was observed; i) initial level of AP was

positively predictive of IS at the following time point ii) IS at the second wave was positively predictive of AP at the third wave. One main difference was observed between genders: IS in wave 2 was predictive of AP in wave 3 for males but not for females. Additionally, stability over time was observed within each phenotype as indicated by significant auto-regressive paths, where predictive power was stronger among females. As expected, AP increased throughout the study. IS initially increased followed by a decrease. It is possible that the initial increase of IS may be attributed to the stress of starting college, such as academic stress and dealing with changes. These factors of stress may decrease as individuals settle into college, thereby decreasing IS.

We observed a positive unidirectional relationship changing with wave among the full sample, as well as within males. AP predicting IS is supported by previous studies as well as IS predicting AP (Marmorstein, 2010; Hartka et al., 1991). IS may occur due to bio-psychosocial consequences of AP and adverse social effects causing stress and IS. IS may in turn be self-medicated with alcohol, therefore increasing the risk of continued AP. However, to our knowledge, the unidirectional relationship changing with age in the present study has not previously been observed in a college sample. If AP were consistently predictive of IS or IS was consistently predictive of AP in each wave, it would suggest that stable factors at each wave were determinants of AP and IS. Yet, only AP in wave 1 was predictive of IS (AP in wave 2 was not predictive of IS), suggesting that factors determining direction of effect changed with time. It is from the present study not clear what these factors may be. However, if direction of effect of the two phenotypes is dependent on changeable factors, it supports the inconsistencies found in the previous literature. We urge for future studies to replicate the present findings and examine what these factors may be.

For females, AP predicted IS but IS did not predict AP. With some exceptions, (Gilman & David, 2001), the present results are not consistent with the majority of literature which suggests that IS predicts or precedes AP in females. According to the present study, AP should be considered a risk factor for the development of IS in females but not vice versa. It is possible that that our results differ from previous research findings due to the use of a broader construct of IS (rather than just depression or anxiety). When depression and anxiety are investigated separately with regards to their relationship to AP, depression is consistently found to predict AP in females while anxiety is sometimes associated with decreased levels of AP; due to the potential for increased anxiety levels caused by chronic alcohol use, or alternatively, anxiety related to situations in which alcohol is consumed (Krank et al., 2011). It is also plausible that IS does in fact predict AP earlier on in adolescence, however this is not captured in the present study due to the limited age range.

Two possible explanations could be considered as to why the relationship of AP and IS differs between genders. Firstly, it has been hypothesised that symptoms may be more toxic in the specific gender in which they occur less frequently, referred to as “the gender paradox” (Keenan et al., 1999). According to this theory, symptoms may be more difficult to acknowledge and process, resulting in the problem becoming more detrimental in the gender in which it is less common. The hypothesis suggests that AP are more detrimental in females as AP are less common in females. The theory further suggests that as IS is less common in males than in females and more difficult to process in males, IS in males may be more likely

to be self-medicated with alcohol than in females. Secondly, a possible explanation could lie within the observed consistent correlation of externalising disorders to both internalising disorders and alcohol problems (Costello et al., 2003). Externalising disorders are generally more common in males than females (Siminoff et al., 1997). It is possible that males suffering from internalising disorders respond by exhibiting symptoms of externalising behaviours such as AP, and AP is on the continuum of externalising disorders (Krueger et al., 2005). Females may simply not self-medicate via this route, however, why males and females differ in the present study is not clear. Further studies are needed to clarify why the present results differ somewhat from the majority of previous findings.

Although, the present findings in relating to gender differences should be treated with caution. The findings for the full sample support a pathway from IS to AP, and while multigroup analysis does not suggest that pathways differ by gender, analyses by gender does suggests cross-lagged differences on one account (IS in wave 2 predicts AP in wave 3 in males only). This discrepancy is most likely due to that only one of the four cross-lagged paths is actually different between the genders, resulting in that no difference is significantly observable overall.

Strengths and limitations

This study had a number of strengths and limitations. Three main strengths of the study are notable. The study i) employed a large population based sample which was ii) homogenous in terms of age, setting and circumstances providing a base from which some generalisations can be drawn and iii) approaches the topic in a more detailed manner than some previous studies in terms of analysis and in regards to phenotypes examined.

Mechanisms, potential shared risk factors, and possible mediators underlying the association between AP and IS were not explored in this study; nor were potential mechanisms for underlying gender differences. Accounting for shared risk factors (if we knew exactly what they were) would be of interest but not doing so does not undermine statements about direction of effect as these paths would be still be present as shared risk factors would increase the risk of both phenotypes. To understand how AP and IS interact with each other and why they are associated, other factors must be investigated such as environmental and genetic influences, personality, age and other disorders such as externalising disorders.

The study was restricted to a limited number of measurement points. Although the study aimed to address a specific age range, it would be advantageous to have measures from an earlier age through adolescence and into early adulthood in order to better understand the possible effects of age. In addition, the study had large assessment intervals which limits the ability to detect the proximal factors. The results should therefore be interpreted with caution and future studies should aim to have smaller assessment intervals.

A major limitation were missing data: participation rates dropped in the final wave of the study. Prior analyses indicated that participants who did not take part in the final wave of the study did not differ significantly on measures of interest. Nevertheless, this does not necessarily mean there are no differences in longitudinal pathways. To address this issue a post-hoc sensitivity analyses was performed. The results supported our initial results. A

model fit estimating all paths was good (RMSEA=.019; CFI=1.00; SRMR=.008). A cross-lagged model indicated significant autoregressive paths and correlations. Significant cross-lagged paths were observed from AP wave 1 to IS wave 2 (.11, $p<.001$), for both males (.09, $p<.05$) and females (.13, $p<.001$), and from IS wave 2 to AP wave 3 (.07, $p<.01$), for males only (.14, $p<.01$). The results of the sensitivity analyses indicate that the shifting sample does not have a significant impact on interpretation of the results. Or in other words, the results suggest that the measures of interest and their relationship may not be related to whether participants took part or not in the final wave of the study.

Concluding remarks

The findings from this large population-based sample provide additional support for a positive relationship between AP and IS in college students. We observed relationships of direction between the two phenotypes, but the relationship differed depending on gender. The results suggest that the increase of alcohol use during college is a risk factor for the development of both AP and IS and that the observed relationships are not limited to individuals with clinically diagnosed mental health or substance use problems. Additional research is warranted to replicate the current findings, as well as to investigate whether the observed processes between AP and IS continue into adulthood.

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Glossary

Alcohol problems

Symptoms of alcohol dependence and alcohol abuse as defined by DSM and ICD

Internalising symptoms

A construct describing a phenomena where an individual turns problems inwards (as opposed to outwards (externalising symptoms)) and consists of symptoms such as social withdrawal, anxiety and depression

Cross-lagged models

A statistical method of investigating direction of effect of events over time

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Fig. 1.

Cross-lagged model of AP and IS over three time points (total (female/male))

Arrows from one phenotype to preceding phenotype represents regression paths where one phenotype predicts the other at immediately subsequent time point (cross-lagged paths) as well as one phenotype' initial and immediately preceding waves to predict consecutive waves (auto-regressive paths). The arrow between AP and IS at w1 represents a correlation. The arrow between AP and IS at w2 and w3 represents residual correlations. Values printed along arrows are regression coefficients for the full sample, females and males. Non-significant paths are presented as grey arrows and non-significant coefficients are presented in grey. Significant paths are significant at minimum $p < .05$. IS=Internalising Symptoms; AP=Alcohol Problems; w=wave.

Table 1

Descriptive statistics for alcohol problems (AP) and internalising symptoms (IS), by gender and wave

	Full sample	Female	Male
	Mean (SD)	Mean (SD)	Mean (SD)
AP ^a wave 1	.47 (1.02)	.47 (1.03)	.49 (1.01)
AP wave 2	.49 (1.00)	.48 (1.00)	.51 (1.01)
AP wave 3	.68 (1.20)	.62 (1.15)	.79 (1.29)
IS ^b wave 1	14.69 (5.98)	15.44 (6.10)	13.50 (6.10)
IS wave 2	16.56 (6.21)	17.38 (6.18)	15.24 (6.00)
IS wave 3	15.65 (6.38)	16.41 (6.55)	14.27 (5.81)

^a Sum score of 9 AP items adapted from the Semi-Structured Assessment of the Genetics of Alcoholism (range 0–9).

^b Sum score of 8 IS items from the SCL-90 (range 8–40)

Table 2
Correlations of internalising symptoms (IS) and alcohol problems (AP) by gender

	IS			AP		
	Wave 1	Wave 2	Wave 3	Wave 1	Wave 2	Wave 3
<i>Full sample</i>						
IS	Wave 1	–				
	Wave 2	0.59 ***	–			
	Wave 3	0.51 ***	0.51 ***	–		
AP	Wave 1	0.13 ***	–0.02	–		
	Wave 2	0.06 *	0.16 ***	0.04 ***	–	
	Wave 3	0.05	0.15 ***	0.12 ***	0.39 ***	0.52 ***
<i>Female</i>						
IS	Wave 1	–				
	Wave 2	0.58 ***	–			
	Wave 3	0.51 ***	0.50 ***	–		
AP	Wave 1	0.12 ***	0.12 ***	0.02	–	
	Wave 2	0.03	0.14 ***	0.04 ***	0.54 ***	–
	Wave 3	0.04	0.14 ***	0.12 ***	0.43 ***	0.58 ***
<i>Male</i>						
IS	Wave 1	–				
	Wave 2	0.61 ***	–			
	Wave 3	0.51 ***	0.50 ***	–		
AP	Wave 1	0.16 ***	0.19 ***	0.09	–	
	Wave 2	0.11 **	0.20 ***	0.04 ***	0.42 ***	–
	Wave 3	0.10	0.21 ***	0.20 ***	0.33 ***	0.43 ***

*** p<.001,

** p<.001,

* p<.05

Table 3

Cross-lagged analysis model fit of alcohol problems and internalising symptoms

	Parameters	RMSEA	χ^2 (df)	CFI	SRMR	BIC
All paths estimated	25	0.014	2.97 (2)	1.00	0.007	49524.34
No path from Wave 1 to Wave 3	23	0.100	109.96*** (4)	0.95	0.040	49615.57
Residual correlations constrained	23	0.053	34.08*** (4)	0.99	0.021	49539.70

p<.001

Table 4

Multigroup analysis of cross-lagged analysis by gender

	Parameters	RMSEA	χ^2	CFI	SRMR	BIC	χ^2 difference
All parameters free	50	0.021	6.35 (4)	1.00	0.010	49543.03	
All parameters fixed	37	0.024	29.96* (17)	0.99	0.032	49464.23	23.61* (13)
Correlations fixed	47	0.010	7.87 (7)	1.00	0.013	49520.91	1.52 (3)
Cross path fixed	46	0.019	11.95 (8)	1.00	0.020	49517.12	5.60 (4)
Autoregressive paths fixed	44	0.032	23.30** (10)	0.99	0.027	49512.72	16.95** (6)

p<.001,**
p<.001,*
p<.05