Longitudinal Predictors of Alcohol-Related Harms During the Transition to Adulthood

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While a range of factors have been found to increase the likelihood of alcohol-related harms among young people, little is known about their relative importance. This article aimed to identify the risks for alcohol-related harms at an age when alcohol use and problems tend to peak in Australia (19–20 years). A wide range of concurrent and antecedent factors from multiple domains were examined using path analysis, including individual characteristics, family environment, and externalising and internalising problems. The sample comprised of 941 individuals from the Australian Temperament Project, a large longitudinal community-based study. The path model controlled for current risky drinking and revealed a number of variables that were significant longitudinal predictors of alcohol-related harms within each of the domains, including adolescent antisocial behaviour and drinking behaviour, low agreeableness, impulsivity, and paternal drinking levels. The potential for developmental prevention approaches to reduce alcohol-related harms by targeting externalising behaviour problems, interpersonal influences, and individual characteristics is discussed.

Key words: alcohol-related harms; longitudinal analysis; risk factors; risky drinking; transition to adulthood.

The transition to adulthood, generally defined as 18 to 25 years, is a period of extensive role exploration without clear normative expectations for many young people in Western industrialised countries (Arnett, 2011). A relatively high incidence of risk behaviours and mental health problems are observed during this time (Smart & Sanson, 2005). In particular, the extent to which young people report experiencing harms arising from alcohol use, such as risky sexual activity and violence (O’Malley, 2004), is a major public health concern in Australia and other Western countries. Both risky alcohol use and alcohol-related harms reach a lifetime peak during the early twenties (Muthén & Muthén, 2000). Data from the Australian National Drug Strategy Household Survey suggests that approximately half of 20–29 year-olds drink at levels which place them at risk for short-term harms (e.g., injury), and nearly a fifth drink at levels that place them at risk of lifetime or long-term harm, such as impaired liver function (Australian Institute of Health and Welfare, 2011).

Alcohol consumption is by definition a prerequisite for alcohol-related harms, and a moderate relationship between alcohol consumption and associated harms has been consistently observed (Selin, 2005; Stice, Barrera, & Chassin, 1998). However, harms do not always result from consumption—that is, some young people drink but report few negative consequences (Schulenberg & Maggs, 2002). Given that alcohol intake and resulting harms do not have a direct correspondence, there may be other factors that contribute to a young person’s likelihood of experiencing alcohol-related harms (Stice et al., 1998). Identifying such factors has important implications for prevention and intervention efforts. Reducing the risk of alcohol-related harms is particularly relevant to countries such as Australia, which take a harm minimisation approach and focus on preventing the adverse consequences associated with...
alcohol consumption rather than eradicating drinking itself (McMorris, Hemphill, Toumbourou, Catalano, & Patton, 2007).

Risk Factors for Alcohol-Related Harms

Previous research has suggested a number of child and adolescent factors that may accentuate young peoples’ risk for alcohol-related harms in the context of drinking, including individual characteristics, aspects of the family environment, and externalising and internalising behaviour problems. The following section provides an overview of studies that have examined risk factors for alcohol behaviours over the transition to adulthood. Given the paucity of research examining precursors of alcohol-related harms, we also canvas studies examining antecedents of other alcohol behaviours, including alcohol consumption.

Individual characteristics

Gender is a factor that has been consistently associated with differences in levels of alcohol use and resulting harms. Specifically, males tend to drink more and experience higher rates of alcohol use disorders, although gender differences are reducing as gender roles change in contemporary society (Zucker, 2008).

A growing body of literature suggests that early temperament factors (typically seen as the “core” of aspects of developing personality, including affective, activational, and attentional dimensions; Sanson, Hemphill, & Smart, 2002) are associated with vulnerability to alcohol problems. For example, Caspi et al. (1997) found that temperament at age 3 was associated with alcohol dependence at age 21: undercontrolled children, described as irritable, impulsive and non-persistent in their activities, and confident (adventure seeking) children, who were easy to engage and displayed an active interest in an unfamiliar situation, were generally at greater risk of later alcohol dependence and health problems than children classified as inhibited, reserved, or well-adjusted. There is also mounting evidence for the relevance of later personality style (Conrod, Castellanos-Ryan, & Mackie, 2011), with higher neuroticism/negative emotionality and lower agreeableness and conscientiousness implicated in more negative alcohol outcomes (Ruiz, Pincus, & Dickinson, 2003).

Behavioural and emotional self-regulation deficiencies have also been implicated in the development of alcohol-related problems (Simons, Carey, & Gaher, 2004). In particular, traits related to impulsivity, disinhibition, or behavioural undercontrol are strongly associated with alcohol misuse, both in the general population (Sher, Grekin, & Williams, 2005) and in college students (MacKillop, Mattson, Anderson MacKillop, Castelda, & Donovick, 2007). This risk is particularly salient in the context of poorer family management (Hill et al., 2010).

Finally, previous alcohol use in adolescence has also been found to predict experiences of alcohol-related harms. Drawing on longitudinal person-centred methodologies, Toumbourou et al. (2004) found that trajectories of alcohol use over adolescence (17–19 years) predicted experiences of alcohol-related harms in young adulthood (21 years), even when controlling for concurrent alcohol consumption. For example, males and females who had stable high-alcohol use trajectories in adolescence were over two and four times more likely to experience alcohol-related harms at 21 years, respectively, compared to stable non- or infrequent drinkers.

Family environment

Previous research has consistently identified family history of alcoholism and higher parental drinking as risk factors for heavier alcohol use and alcohol-related problems over the lifespan (Walden, Iacono, & McGue, 2007). While the findings of the moderate heritability (40–60%) of alcohol use disorders (Goldman, Oroszi, & Ducci, 2005) suggest that genetics may partially account for these relationships, the role of the family environment, particularly socialisation processes within the family, has also been shown to be important (Jacob et al., 2003).

Specifically, lack of parent–child connection, parental support, and parental control predict alcohol-related problems in American college students (Fischer, Forthum, Pidcock, & Dowd, 2007). In addition, parental monitoring of young peoples’ behaviour is associated with lower alcohol use and fewer negative consequences among college students (Wood, Mitchell, Read, & Brand, 2004). Lower family socioeconomic status (SES) may pose an additional risk for alcohol problems. This may be due to the greater environmental challenges and poorer health behaviours associated with lower SES status (McLaughlin et al., in press).

Externalising and internalising behaviours

There is considerable evidence of an externalising developmental pathway to alcohol-related problems and disorders (Zucker, 2006). Prospective longitudinal studies indicate that childhood and adolescent antisocial behaviours (such as aggression, property destruction, theft) and disorders (conduct and oppositional defiant disorders) are associated with later alcohol abuse and dependence (Harford & Murthen, 2000; Pardini, Raskin White, & Stouthamer-Loeb, 2007). Those who continue to display antisocial behaviour into adulthood are also at higher risk for developing alcohol use disorders (McGue & Iacono, 2005).

The possibility of an internalising pathway to alcohol-related harms has received less attention but is gaining increasing empirical support (Hussong, Jones, Stein, Baucom, & Boeding, 2011). A number of studies report that depressed mood in childhood and adolescence is linked to an earlier onset of alcohol use, alcohol-related problems in early adolescence and alcohol dependence in young adulthood (Crum et al., 2008; Pardini, Lochman, & Wells, 2004). Cross-sectional studies further suggest that depression and alcohol use disorders frequently co-occur; for example, the Australian National Survey of Mental Health and Well Being found that Australian adults with an alcohol disorder were four times more likely to have an affective disorder (Burns & Teesson, 2002). Similarly, university students who identify themselves as problem drinkers also report more depressive symptoms than other students (Eshbaugh, 2008). Findings are less clear regarding connections between anxiety, alcohol consumption, and alcohol-related problems. Some studies suggest that individuals with high levels of anxiety in adolescence show a lower risk of problem drinking in adulthood (Pardini et al., 2004). However, other studies have found elevated levels of anxiety among hazardous or problem drinkers compared to lower-level or non-problem drinkers (e.g., Vanheusden et al., 2008), and the Australian National Survey of Mental Health and Well Being reported that adults with an
alcohol disorder were three times more likely to have an anxiety disorder (Burns & Teesson, 2002).

**Limitations of Previous Research**

There are a number of gaps in existing knowledge about predictors of alcohol-related harms during the transition to adulthood. First, a significant limitation of the available literature is that most studies have examined developmental antecedents of alcohol use or alcohol use disorders, rather than of alcohol-related harms. Since alcohol intake is only a moderate predictor of consequent harms, the factors that increase risk for alcohol-related harms (as distinct from use) remain unknown. Likewise, studies that examine alcohol use disorders provide limited insight into this issue as the majority of young people who experience harms arising from alcohol consume alcohol at sub-clinical levels. In addition, although some studies have brought together ecological models of the development of alcohol use or alcohol disorders (e.g., Kramer et al., 2008), few have done this for alcohol-related harms, and thereby examined whether the identified developmental antecedents to alcohol-related harms remain significant when the broader developmental context and individual’s competencies and drinking behaviours are taken into account. This is important to ascertain in order to identify which factors, or combination of factors, pose the greatest risk for alcohol-related harms. Furthermore, only a small number of studies have been able to prospectively examine developmental precursors from early in life, with most numbers using only short-term longitudinal data (Englund, Egeland, Oliva, & Collins, 2008). Samples have also been limited in generalisability, with the majority limited to college students (thus failing to represent a significant proportion of young people) and US samples (which may have limited relevance for countries such as Australia that have distinct harm minimisation policy approaches to alcohol; Beyers, Toumbourou, Catalano, Arthur, & Hawkins, 2004).

**The Current Study**

The Australian Temperament Project (ATP), a large-scale longitudinal study that has followed individuals and their families from infancy to adulthood, provides a valuable opportunity to examine a broad range of influences across different developmental stages and domains of functioning on experiences of alcohol-related harms during the transition to adulthood. The current study aimed to identify unique predictors of alcohol-related harms among individuals aged 19 to 20 years from three domains: (a) individual characteristics (gender, temperament, personality, impulsivity, previous experiences of alcohol intoxication); (b) family environment factors (maternal and paternal drinking, maternal monitoring, family socioeconomic status); and (c) externalising and internalising problems (depression, anxiety, antisocial behaviour). The specific aims of the study were (a) to determine whether the factors within each of these domains showed a unique predictive association with alcohol-related harms when the effects of current risky drinking and other variables were taken into account, and (b) to identify the most prominent predictors associated with alcohol-related harms from these domains.

As shown in Figure 1, we predicted that all of these factors would impact on alcohol-related harms, beyond the effect of their association with risky drinking. Frequency of risky drinking alcohol use was included in the model as a covariate given its immediate causal relationship with harm outcomes. Given that previous research has suggested that antecedents proximal in time explain the greatest variance in alcohol outcomes and that effects of earlier relationships often occur through their association with intermediate functioning (Zucker, 2008), we hypothesised that the influence of childhood experiences would occur through their impact on adolescent experiences in these domains. In developing this model, we were mindful of the likely relationships between predictor variables and included these in the model, including the relationships between inflexibility and neuroticism, depression, and anxiety (Betts, Gullone, & Allen, 2009; Windle, 1989), depression and neuroticism (Roelofs, Huibers, Peeters, & Arrnitz, 2008), depression and anxiety (Hale, Raajmakers, Muris, van Hoof, & Meeus, 2009), antisocial behaviour and alcohol use (Zucker, 2006), and between maternal and paternal drinking (Keller, Cummings, Davies, & Mitchell, 2008). This hypothesised model is summarised in Figure 1.

**Method**

**Participants**

Participants were members of the Australian Temperament Project (ATP). The ATP is a longitudinal study that has collected 15 waves of data predominantly by mail surveys on domains including temperament, personality, parenting practices, health, social skills, peer relationships, sociodemographic indices, and internalising and externalising problems such as depressive symptoms and substance use (for more details, see Prior, Sanson, Smart, & Oberklaid, 2000). The original ATP cohort comprised 2,443 4–8 months old infants and their families who were recruited through the maternal and child health centres in 1983. They were selected to be representative of the Australian state of Victoria. The University of Melbourne and Australian Institute of Family Studies Human Research Ethics Committee provide ethical approval for the project.

At 19 to 20 years, approximately two thirds were still involved in the study. Attrition has been slightly higher in families experiencing socio-economic disadvantage or with parents born outside Australia. Importantly however, the retained and non-retained subsamples show no substantial differences in scores on any of the infancy characteristics measured at the study’s commencement (O’Connor et al., 2011). The study thus continues to be representative of the diverse range of young people’s attributes. In addition, although attrition is unlikely to be a significant influence on the results, socio-economic status has been controlled for in the present analyses.

Information relating to alcohol consumption and alcohol-related harms was collected from age 13–14 years (in 1996) and approximately every 2 years thereafter, giving four waves of data collection to age 19–20 years. As alcohol consumption is a necessary precondition for harmful alcohol use consequences, the present study focuses on data from current drinkers (those 19–20-year olds who reported consuming an alcoholic drink on...
one or more days within the last month) and who also answered questions on alcohol-related harms. In order to investigate the effects of past alcohol consumption on current alcohol-related harms, only those who had provided data relating to alcohol for at least two of the three previous data waves were included. The present study thus involved a subset of 941 young adults (419 males). The small number of participants (n = 70) who did not respond to questions on alcohol use for at least two of the three previous waves of data collection and were removed from the analysis were not significantly different from the retained sample on infant characteristics (F(5, 654) = 0.64, p = 0.67, or concurrent drinking behaviours (F(2, 987) = 1.00, p = 0.37).

Measures

Alcohol-related harms

The level of alcohol-related harms was assessed at 19–20 years by ten items adapted from the Victoria Adolescent Health Survey (Hibbert, Caust, Patton, Rosier, & Bowes, 1996). Participants were asked to indicate on a 3-point Likert scale (0 = never, 1 = once or twice, 2 = more often) the number of times in the last year their alcohol use had caused them to (a) get so drunk they were sick or passed out, (b) have trouble at home, work or school the next day, (c) get injured or have an accident, (d) become violent and get into a fight, (e) have sex with someone which they later regretted, (f) get into trouble with the police, (g) be unable to remember what happened the night before, (h) be asked to leave a party, pub or club because they were drunk, (i) feel that they were unable to stop drinking once they’d started, and (j) feel irritable or depressed when alcohol wasn’t available. Answers to the questions were combined to create an alcohol-related harm score with a possible range from 0–20 (M = 2.47, SD = 2.75), with higher scores indicating a higher level of harms (α = 0.78).

Alcohol consumption

At 13–14 and 17–18 years, alcohol consumption was measured according to self-reported frequency of intoxication in the last month (how many days in the last month they had enough alcohol to feel its effects; range 0–30 days). Following the Australian national health guidelines for risk of short-term harms that were in operation at the time of the study (NHMRC, 2001),
frequency of risky drinking was assessed at age 19–20 as the number of days in the last month that participants reported drinking seven or more standard drinks if male, or five or more standard drinks if female.

**Demographic covariates**

Gender was coded as 1 = male and 2 = female. Parental socio-economic status (SES) was assessed when participants were 13–14 years by parental report, as a composite of both parents’ educational and occupational levels. Educational level was measured by a reversed version of Brotherton, Kotelac, and Hammond’s (1979) 8-point scale from “primary” to “postgraduate” level. Occupational level was measured by Broom, Jones, and Zubrzycki’s (1974) 8-point scale, from “housewife/student/unemployed” to “upper professional.”

**Predictors**

The predictors included in the model are summarised in Table 1. Further details are available upon request. Variables are grouped under three domains: individual characteristics, family environment, and demographic covariates.

**Data analysis**

The path analysis was performed using AMOS 19 software (Arbuckle, 2010). Model estimations were based on a covariance matrix and used maximum likelihood estimates, which work well with large sample sizes (Tabachnick & Fidell, 2001). The model was identified, and the estimation process converged. All parameter estimates were within the range of permissible values. The model diagram shows standardised parameter estimates, $\beta$, which represent the effect of a given predictor variable on the dependant variable after accounting for the remaining relationships in the model.

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**Table 1** Description of Measures Assessing Risk and Protective Variables

<table>
<thead>
<tr>
<th>Variable, age assessed and informant</th>
<th>Measure derivation, example items and response categories and reliability (where relevant)</th>
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<tbody>
<tr>
<td><strong>Individual characteristics</strong></td>
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<tr>
<td>Early temperament at 3–4 years (P): Inflexibility, persistence, approach</td>
<td>Adapted from the Childhood Temperament Questionnaire (Thomas &amp; Chess, 1977). Inflexibility was measured by nine items (e.g., “If my child is upset, it is hard to comfort him/her”, $\alpha = 0.79$), persistence was measured by seven items (e.g., “When a toy or game is difficult, my child quickly turns to another activity”, $\alpha = 0.83$), and approach was measured by seven items (e.g., “When unknown adults visit our home, my child is immediately friendly and approaches them”, $\alpha = 0.84$). Responses were measured on a 6-point scale from “almost never” to “almost always.”</td>
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<td>Personality at 17–18 years (S): Extraversion, agreeableness, conscientiousness, neuroticism</td>
<td>Adapted from Goldberg’s (1992) five-factor personality items, which consist of nine-step bipolar rating scales that range from (1) very (Trait A) through (5) neutral (Trait A and Trait B) to (9) very (Trait B). Extraversion was measured by five items (e.g., “Unenergetic-energetic”, $\alpha = 0.74$). Agreeableness was measured by five items (e.g., “Rude-polite”, $\alpha = 0.70$). Conscientiousness was measured by five items (e.g., “Reliable-unreliable”, $\alpha = 0.76$). Neuroticism was measured by five items (e.g., “Nervous-at ease”, $\alpha = 0.70$)</td>
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<td>Impulsivity at 15–16 years (S)</td>
<td>ATP devised scale—five items (e.g., “I avoid activities where I might get injured”, $\alpha = 0.67$), rated on a 6-point Likert scale from “never” to “always.”</td>
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**Family environment**

Parental drinking when participants were 17–18 years (P): Maternal, paternal

Parental report was used where possible to assess predictors to avoid bias arising from single source data. In choosing which time points to use measures from, we were constrained by the data available; for example, impulsivity was not measured at 17–18 years and hence the measure at 15–16 years was used instead. Temperament was measured in early childhood rather than early adolescence as early temperament provides the foundation for later personality development.

**Externalising and internalising problems**

Depression at 13–14 years (S) Short Mood and Feelings Questionnaire (Angold et al., 1995), 12 items (e.g., “I don’t enjoy anything at all”, $\alpha = 0.80$) rated on a 3-point scale from “rarely or never” to “very often.”

Anxiety at 13–14 years (S) Adapted from the Revised Behavior Problem Checklist (Quay & Peterson, 1987)—five items rated on a 3-point Likert scale from “rarely/inever” to “very often,” e.g., “I get anxious and scared,” $\alpha = 0.71$.

Antisocial behaviour at 13–14 years (S) Short form of the Moffitt and Silva (1988) Self Report of Delinquency Scale—eight items (e.g., “In the last 12 months, have you . . . stolen something $\alpha = 0.74$ Responses were 1 = “not at all,” 2 = “once,” and 3 = “more than once.”

**Note.** S = self-report, P = parent report. For continuous measures, higher scores indicate more of the attribute.
A number of fit statistics were examined. We used chi-square as an indicator of fit problems. Significant values suggest that the sample correlation matrix and the model correlated matrix are significantly different. However, chi-square is known to be affected by large sample size (Schermelleh-Engel, Moosbrugger, & Muller, 2003). We also examined the Adjusted Goodness of Fit Index (AGFI), which is the GFI adjusted for the degrees of freedom. AGFI values greater than 0.80 are taken to reflect acceptable fit (Schermelleh-Engel et al., 2003). The Root Mean Square Error of Approximation (RMSEA) is based on the non-centrality parameter, and takes particular account of the error of approximation, with values below 0.08 taken to indicate a good fit in this study (Brown & Cudeck, 1993). The normed fit index (NFI) compares the $\chi^2$ of the model to the $\chi^2$ value of the independence model, with values greater than 0.95 indicating a good fit (Tabachnick & Fidell, 2001). The chi-square difference test is a popular means of comparing model fit that was used to examine whether changes in chi-square were statistically significant (Byrne, 2001).

Results

Descriptive statistics and intercorrelations between variables are included in Table 2. Levels of missing data averaged 5%. Missing data was estimated using the expectation-maximization algorithm (Enders, 2006).

Full Model

The hypothesised model (see Figure 1) was estimated and found to be a poor fit for the data ($\chi^2 = 1713.85$, d.f. = 155, $p < 0.001$; RMSEA = 0.11; AGFI = 0.77; NFI = 0.47). Hence, we performed model trimming, removing non-significant paths one at a time starting from the smallest loading path. Following this procedure, maternal drinking, the personality dimensions of conscientiousness, extraversion, and neuroticism at mid/late adolescence, and gender and SES were removed from the model. The path from the frequency of intoxication at 13 to 14 years to the frequency of intoxication at 17 to 18 years and the covariances hypothesised between predictors (with the exception of the covariance between antisocial behaviour and frequency of intoxication at 13–14 years) were also removed. This significantly improved model fit ($\Delta \chi^2 = 1140, p < 0.001$), although fit was still judged to be relatively poor ($\chi^2 = 573.21$, d.f. = 78, $p < 0.001$; RMSEA = 0.08; AGFI = 0.88; NFI = 0.66).

Next, we performed model building. First, as most of the mid/late adolescence intermediate functioning dimensions were removed from the model, direct paths from each of the childhood/early adolescent dimensions to both risky drinking and alcohol-related harms were tested, and significant paths were retained. This process resulted in the addition of two paths (a) a direct path from frequency of intoxication at 13 to 14 years to risky drinking at 19 to 20 years, and (b) a direct path from antisocial behaviour at 13 to 14 years to alcohol-related harms at 19–20 years. It also caused the removal of five childhood/early adolescent dimensions from the model (i.e., inflexibility, persistence, approach–sociability, depression, and anxious–fearful), which did not have significant loadings to either of the later alcohol measures.

Second, the modification indices were examined and indicated a number of changes to improve model fit that were judged to be conceptually coherent. Specifically, three paths were added (see discussion for conceptual explanation) from antisocial behaviour at 13 to 14 years to maternal monitoring at 17 to 18 years, agreement at 17 to 18 years, and impulsivity at 15 to 16 years. Following these modifications, the model was substantially improved ($\Delta \chi^2 = 479, p < 0.001$) and was judged a good fit for the data ($\chi^2 = 94.19$, d.f. = 19, $p < 0.001$; RMSEA = 0.07; AGFI = 0.95; NFI = 0.91). The final model is presented in Figure 2 and reveals that greater alcohol-related harms in the transition to adulthood were related to higher antisocial behaviour at 13–14 years, impulsivity at 15–16 years, and higher frequency of intoxication, paternal drinking, and lower agreeableness at 17–18 years. The model also demonstrates that these effects were significant after controlling for concurrent risky drinking. Greater alcohol-related harms during the transition to adulthood were also related to risky drinking at the same age. Overall, these factors accounted for 23% of the variance in alcohol-related harms.

Discussion

This study aimed to identify longitudinal predictors from multiple domains—including individual characteristics, family environment factors, and behaviour problems—that increase vulnerability for alcohol-related harms among early adult alcohol users. The analysis sought to separate predictors of harm from those predicting risky drinking. The results reveal a number of individual and family environment factors over childhood and adolescence that predict alcohol-related harms in young adulthood. These effects were observed beyond their impact on alcohol use; that is, their impact was not attributable to these individuals simply drinking more but suggests that these risks impact on how the young person responds to alcohol. The final path model included predictors from across domains but emphasised more proximal measurement points and critical early adolescent transitions. Another recurring theme was the likely role of a broad impulsive or disinhibitory trait as an underlying causal mechanism for these effects.

The results revealed that young people engaging in antisocial behaviours in early adolescence might be particularly at risk of experiencing alcohol-related harms in young adulthood. Similarly, individuals displaying lower levels of agreeableness (who have been found by previous studies to have a tendency towards irritable, hostile, and suspicious behaviour e.g., Costa, McCrae, & Dembro, 1989; Miller & Lynam, 2001) were also more vulnerable to harms. This concords with the previous research suggesting that harmful patterns of alcohol use may be more prominent among individuals with antagonistic, disagreeable traits or who display antisocial behaviour in adolescence, and supports the proposal of an externalising pathway towards later alcohol involvement and problems (Zucker, 2006). Antisocial behaviour is characterised by the inability or unwillingness to inhibit behavioural impulses, even when negative consequences are likely to result, and this underlying impulsivity or disinhibition is likely to contribute to externalising pathways towards problem alcohol outcomes (Zucker, 2008). The findings also suggest the importance of adolescent drinking...
Table 2: Intercorrelations and Descriptive Statistics for Variables Used in the Analyses

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<td>7. Approach (3–4 years)</td>
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<tr>
<td>8. Externally (17–18 years)</td>
<td>0.02**</td>
<td>0.04</td>
<td>0.00</td>
<td>0.05</td>
<td>0.15**</td>
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<tr>
<td>9. Externalizing problems (17–18 years)</td>
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<tr>
<td>10. Conscientiousness (17–18 years)</td>
<td>0.18**</td>
<td>0.12**</td>
<td>0.07</td>
<td>0.11**</td>
<td>0.01</td>
<td>0.05</td>
<td>0.17**</td>
<td>0.06</td>
<td>0.11**</td>
<td>0.01</td>
<td>0.32**</td>
<td>0.54**</td>
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<tr>
<td>11. Neuroticism (17–18 years)</td>
<td>0.02</td>
<td>0.12**</td>
<td>0.01</td>
<td>0.05</td>
<td>0.07**</td>
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<tr>
<td>12. Impulsivity (15–16 years)</td>
<td>0.21**</td>
<td>0.23**</td>
<td>0.05</td>
<td>0.19**</td>
<td>0.05</td>
<td>0.15**</td>
<td>0.03</td>
<td>0.16**</td>
<td>0.20**</td>
<td>0.17**</td>
<td>0.19**</td>
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<tr>
<td>13. Maternal drinking (17–18 years)</td>
<td>0.10**</td>
<td>0.11**</td>
<td>0.04</td>
<td>0.17**</td>
<td>0.00</td>
<td>0.03</td>
<td>0.01</td>
<td>0.03</td>
<td>0.04</td>
<td>0.06</td>
<td>0.02</td>
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<tr>
<td>14. Paternal drinking (17–18 years)</td>
<td>0.13**</td>
<td>0.10**</td>
<td>0.03</td>
<td>0.11**</td>
<td>0.04</td>
<td>0.03</td>
<td>0.08**</td>
<td>0.00</td>
<td>0.00</td>
<td>0.02</td>
<td>0.03</td>
<td>0.01</td>
<td>0.33**</td>
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<tr>
<td>15. Maternal monitoring (17–18 years)</td>
<td>0.16**</td>
<td>0.15**</td>
<td>0.14**</td>
<td>0.17**</td>
<td>0.04</td>
<td>0.06</td>
<td>0.06</td>
<td>0.04</td>
<td>0.12**</td>
<td>0.08**</td>
<td>0.00</td>
<td>0.11**</td>
<td>0.01</td>
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<tr>
<td>16. Depression (13–14 years)</td>
<td>0.07**</td>
<td>0.06</td>
<td>0.13**</td>
<td>0.04</td>
<td>0.14**</td>
<td>0.08**</td>
<td>0.03</td>
<td>0.19**</td>
<td>0.18**</td>
<td>0.23**</td>
<td>0.39**</td>
<td>0.04</td>
<td>0.00</td>
<td>0.05</td>
<td>0.10**</td>
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<tr>
<td>17. Anxious-fearful (13–14 years)</td>
<td>—</td>
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<tr>
<td>18. Antisocial behaviour (13–14 years)</td>
<td>0.26**</td>
<td>0.20**</td>
<td>0.48**</td>
<td>0.28**</td>
<td>0.11**</td>
<td>0.08**</td>
<td>0.05</td>
<td>0.02</td>
<td>0.17**</td>
<td>0.11**</td>
<td>0.00</td>
<td>0.18**</td>
<td>0.07**</td>
<td>0.05</td>
<td>0.28**</td>
<td>0.26**</td>
<td>0.05</td>
<td>0.05</td>
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<tr>
<td>19. SES (13–14 years)</td>
<td>—</td>
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<tr>
<td>20. Gender</td>
<td>0.11**</td>
<td>0.10**</td>
<td>0.01</td>
<td>0.13**</td>
<td>0.01</td>
<td>0.18**</td>
<td>0.00</td>
<td>0.06</td>
<td>0.20**</td>
<td>0.18**</td>
<td>0.11**</td>
<td>0.28**</td>
<td>0.04</td>
<td>0.03</td>
<td>0.13**</td>
<td>0.13**</td>
<td>0.22**</td>
<td>0.14**</td>
<td>0.02</td>
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<tr>
<td>Mean</td>
<td>2.47</td>
<td>4.39</td>
<td>2.98</td>
<td>3.12</td>
<td>2.99</td>
<td>3.13</td>
<td>6.14</td>
<td>6.75</td>
<td>6.88</td>
<td>4.28</td>
<td>3.30</td>
<td>2.83</td>
<td>3.25</td>
<td>3.16</td>
<td>0.37</td>
<td>0.67</td>
<td>0.70</td>
<td>0.70</td>
<td>3.91</td>
<td>55.5%</td>
</tr>
<tr>
<td>SD</td>
<td>2.75</td>
<td>4.09</td>
<td>1.10</td>
<td>3.41</td>
<td>0.79</td>
<td>1.05</td>
<td>0.80</td>
<td>1.10</td>
<td>1.02</td>
<td>1.15</td>
<td>1.17</td>
<td>0.74</td>
<td>0.89</td>
<td>0.85</td>
<td>0.70</td>
<td>0.29</td>
<td>0.41</td>
<td>1.32</td>
<td>1.42</td>
<td></td>
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<tr>
<td>Min</td>
<td>0</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>1.22</td>
<td>1.00</td>
<td>1.00</td>
<td>1.60</td>
<td>1.20</td>
<td>2.40</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.71</td>
<td>1.00</td>
<td></td>
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</table>
| Max                                   | 20    | 30.00 | 15.00 | 3.00  | 6.00  | 5.86  | 6.00  | 9.00  | 9.00  | 9.00  | 5.43  | 4.00  | 4.03  | 1.58  | 2.00  | 8.00  | 7.67  | *p < 0.05; **p < 0.01.
patterns in establishing pathways towards risky alcohol use and alcohol-related harms in young adulthood. Frequency of intoxication over adolescence was significantly associated with later alcohol use, consistent with a large body of research, primarily based on university students, showing that drinking in secondary school is strongly predictive of hazardous or risky drinking in the first year out of school (Read, Wood, & Capone, 2005). Moreover, individuals with established patterns of drinking by late adolescence may be at particular risk of alcohol-related harms during the transition to adulthood, supporting the findings of previous studies that associate the development of alcohol-related harms during the transition to adulthood with the frequency of drinking at earlier ages and the age of alcohol initiation (e.g., Fergusson, Lynskey, & Horwood, 1994; Toumbourou et al., 2004).

Paternal drinking behaviours were also predictive of alcohol-related harms in early adulthood. Previous research has consistently identified a family history of alcoholism and higher parental drinking as risk factors for heavier alcohol use and alcohol-related problems over the lifespan (Walden et al., 2007). Apart from genetic contributions, the family environment shaped by a parent with an alcohol problem may also contribute to the formation of impulsive, disinhibited traits through inconsistent parenting practices, and parents’ modeling of problematic substance use (King et al., 2009).

The current findings also revealed that some risk factors for alcohol-related harms and risky alcohol consumption are distinct, suggesting the importance of examining both the overlapping and distinct etiological pathways to these outcomes. For example, the personality factor of agreeableness uniquely predicted alcohol-related harms—consistent with previous research suggesting the relevance of altruistic, trusting, and straightforward personality styles (Ruiz et al., 2003)—but had no direct association with risky drinking. In contrast, low parental monitoring of behaviour during late adolescence shared a direct association with risky drinking (consistent with, e.g., Wood et al., 2004) but no direct relationship with alcohol-related harms. The moderate path loading (0.31) from risky drinking to harms also reiterates the lack of a 1:1 correspondence between use and harmful outcomes. Together, these findings suggest the importance of disentangling predictors of use and harms in future research, and for interventions to focus on both reducing risky alcohol consumption and harms.

Also noteworthy is the finding that certain factors that have been associated with harms in previous research did not significantly predict alcohol-related harms in the current multivariate model, either directly or indirectly through risky drinking. In particular, there was no evidence for an internalising pathway towards alcohol-related harms. Furthermore, there were no effects of early temperament once other factors were taken into account, perhaps because the effects of temperament dimensions were subsumed by later personality characteristics. This reiterates the importance of examining predictors within multivariate models, in order to avoid the possibility of overestimating effects that can be explained by other factors.

**Strengths and Limitations**

There are several important strengths to the current project. Variables were measured prospectively, reducing the biases often associated with retrospective recall and allowing longitudinal associations to be detected. The study also included a larger range of factors than most previous studies. Use of a representative community sample of 19–20-year olds and examination of alcohol-related harms, rather than alcohol abuse or dependence also allowed inferences to be drawn about
the important predictors of subclinical alcohol problems in the broader young adult population, rather than the smaller group of young people with diagnosable alcohol use disorders.

A number of limitations to the current study also warrant consideration. To ensure that our sample comprised current drinkers, we included only those participants who reported that they had consumed alcohol in the last 30 days. We also included only those participants that had completed two of the three adolescent surveys on alcohol use, resulting in a subsample comprising 81% of responding participants at age 19–20 years. It is possible that this criterion for inclusion may have resulted in the selection of a sample that drank more heavily than the general population, and these results should be generalised with caution. Furthermore, as the study was directed at identifying predictors of alcohol-related harm among current alcohol users, these findings do not reflect the experiences of abstainers who may encounter harms as a result of other people’s drinking.

We were also limited by the data available and were not able to measure concurrent factors associated with alcohol use, such as alcohol control skills or settings-based interventions (Brennan, Moore, Byrne, & Murphy, 2011). We were not able to explain all of the variance in alcohol-related harms, suggesting that these competencies and environmental contingencies might also impact on both alcohol use and experiences of alcohol-related harms. Additionally, a number of the measures included in the study were ATP devised, and although they have good reliability, validity work is ongoing.

Implications and Future Directions

In regards to intervention, the findings suggest that as well as interventions to directly reduce risky alcohol use and harms through managing environmental contingencies, harm reduction could also encompass a focus on early pathways to reduce early adolescent risk factors. In particular, these interventions might focus on addressing factors during the transitions into and out of secondary schooling that appear to be impacting on post-schooling alcohol behaviours. One such risk factor contributing to harm was alcohol use at different ages, suggesting support for the policy target of reducing alcohol use over earlier developmental periods. Given the apparent complexity of the aetiology of alcohol-related harms and the multiple domains from which risk factors were identified, prevention and intervention efforts that address multiple aspects of young people’s lives in an individualised manner are likely to be most effective. Indeed, evidence in favour of targeted prevention and early intervention programmes rather than universal programmes is growing (Conrod, Stewart, Comeau, & Maclean, 2006; Foxcroft & Tsertsvadze, 2012). For example, long-term effects have been demonstrated for “PreVenture,” a school-based, personality-targeted intervention designed to reduce alcohol use and problem drinking in adolescents who show elevated risk for alcohol-related problems and other emotional and behavioural difficulties based on their personality profiles (Conrod et al., 2011).

These results reveal the relative contributions of a broad spectrum of factors in predicting alcohol-related harms, and thus provide an excellent starting point for a more hypothesis-driven exploration of the pathways that lead to alcohol-related harms in future research. Future research may well broaden explorations of antecedents of alcohol-related harms to incorporate biopsychosocial models of development including high-risk genes (Windle, 2010).

Conclusions

The current findings confirm and extend previous studies in identifying a broad range of risk factors for alcohol-related harm among young adult alcohol users. Above and beyond the effects of drinking history, adolescent antisocial behaviour, low agreeableness, impulsivity, and paternal drinking levels were all implicated in the experience of alcohol-related harms. Interventions to reduce alcohol-related harms could be extended from a focus on alcohol use to consider targeting broader developmental domains including externalising behaviour problems, interpersonal influences, and individual characteristics.

Acknowledgements

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Smart, D., & Sanson, A. (2005). What is life like for young Australians today, and how well are they faring? Family Matters, 70, 46–53.


