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A prospective longitudinal model predicting early adult alcohol problems:

Evidence for a robust externalizing pathway

Alexis C. Edwards, PhD[1], Charles O. Gardner, PhD, Matthew Hickman, PhD, and

Kenneth S. Kendler MD

Virginia Institute for Psychiatric and Behavioral Genetics, Department of Psychiatry,

Virginia Commonwealth University, Richmond, VA

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[1]Corresponding author:

Department of Psychiatry

VCU PO Box 980126

Richmond, VA 23298-0126

Ph: 1-804-828-8591; Fax: 1-804-828-1471

aedwards5@vcu.edu

Abstract

Background

Risk factors for alcohol problems (AP) include biological and environmental factors that are relevant across development. The pathways through which these factors are related, and how they lead to AP, are optimally considered in the context of a comprehensive developmental model.

Method

Using data from a prospectively assessed, population-based UK cohort, we constructed a structural equation model that integrated risk factors reflecting individual, family, and peer/community-level constructs across childhood, adolescence, and young adulthood. These variables were used to predict AP at age 20.

Results

The final model explained over 30% of the variance in liability to age 20 AP. Most prominent in the model was an externalizing pathway to AP, with conduct problems, sensation seeking, AP at age 17.5, and illicit substance use acting as robust predictors. In conjunction with these individual-level risk factors, familial AP, peer relationships, and low parental monitoring also predicted AP. Internalizing problems were less consistently associated with AP. Some risk factors previously identified were not associated with AP in the context of this comprehensive model.

Conclusions

The etiology of young adult AP is complex, influenced by risk factors that manifest across development. The most prominent pathway to AP is via externalizing and related behaviors. These findings underscore the importance of jointly assessing both biologically influenced and environmental risk factors for AP in a developmental context.

Key Words: ALSPAC, alcohol problems, development, externalizing pathway

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Introduction

        Alcohol use and problems are complex phenotypes that are influenced by both biological (e.g., genetic) and environmental factors (Goldman *et al.*, 2005, Kendler *et al.*, 2011b). Risk factors for the development of alcohol problems (AP) include exposures experienced during childhood, adolescence and adulthood (Henkel, 2011, Prescott *et al.*, 1997, Sher *et al.*, 2005); furthermore, factors at the level of the individual, the family, and the peer/community environment are all relevant to risk. These factors are likely interrelated over development. Delineating the complex cascades of risk over time is critical to improving our understanding of the etiology of AP, as well as to refine programs aimed at education, prevention and early intervention.

        Family history – a reflection for both genetic risk and familial-environmental risk – has been robustly associated with AP (Cloninger *et al.*, 1981, Kendler *et al.*, 2011b), as have childhood physical and/or sexual abuse and neglect (Fergusson *et al.*, 2013, Kendler *et al.*, 2000). The role of early socioeconomic status (SES) is inconsistent (Hanson and Chen, 2007), potentially because the relationship might vary across different alcohol use outcomes (Kendler *et al.*, 2014). Environmental risk factors for adolescent alcohol use include peer group deviance (Hoffmann and Bahr, 2014) and low parental monitoring (Dick *et al.*, 2007).

        Individual-level risk factors for AP have often been conceptualized as reflecting two major pathways: externalizing and internalizing (Babor *et al.*, 1992, Cloninger *et al.*, 1981, Del Boca and Hesselbrock, 1996, Windle and Scheidt, 2004). For example, Windle and Scheidt (2004) describe a "negative affect" subtype and a "chronic/antisocial personality" subtype, which are distinguished in part by their presentation of higher levels of anxiety and depression (the former subtype) versus higher levels of alcohol consumption and impairment, along with symptoms of adult antisocial behavior (the latter). The use of other substances, generally considered a manifestation of

externalizing tendencies, has often been associated with alcohol problems (Babor *et al.*, 1992, Blanco *et al.*, 2013). This is likely due at least in part to genetic and/or environmental risk factors common to alcohol and other drug use (Kendler *et al.*, 2011a, Wetherill *et al.*, 2014). Personality and temperament are also indicators of risk (Sher *et al.*, 2005): neuroticism (Prescott *et al.*, 1997), impulsivity (McGue *et al.*, 1997), and extraversion (Kilbey *et al.*, 1998, Prescott *et al.*, 1997) have all been associated with AP.

In the current study, we examine the effects of a wide variety of environmental, familial, and individual-level factors on risk of early adult AP in a comprehensive longitudinal model. We utilize a large, prospectively assessed cohort from the UK where dense phenotypic information is available. Our design offers critical advantages over previous studies. While prior research has examined different aspects of the model described in the current study, few previous studies have had the opportunity to include such a wide range of potential risk/protective factors collected on a single sample. In many cases, we are also able to examine multiple measures of constructs potentially implicated in the etiology of AP, thereby determining if effects are time-specific. Another important advantage of our design is that participants are prospectively assessed, eliminating the possible risk of recall bias. Finally, the ALSPAC sample is community-based, improving the likelihood of generalizability of findings, and the size of the study enables us to detect modest effect sizes, which is especially important in the context of a comprehensive model.

Methods

Sample

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a cohort-based sample recruited in southwest England. ALSPAC recruited 14,541 pregnant women resident in Avon, UK with expected dates of delivery 1st April 1991 to 31st

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December 1992. 14,541 is the initial number of pregnancies for which the mother enrolled in the ALSPAC study and had either returned at least one questionnaire or attended a "Children in Focus" clinic by 19/07/99. Of these initial pregnancies, there was a total of 14,676 fetuses, resulting in 14,062 live births and 13,988 children who were alive at 1 year of age. Subsequent phases of enrolment increased the sample size over time. The phases of enrolment are described in more detail in the cohort profile papers (Boyd *et al.*, 2013, Fraser *et al.*, 2013). For the current analyses, full or partial data were available for 9720 participants. The study website contains details of all the data that is available through a fully searchable data dictionary (http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/). Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees.

Measures

Other than sex, values for each predictor variable were sum scores derived from a series of individual items (see below and Supplemental Material). Where the focal individual provided non-missing responses for at least half of the items for a particular variable, a pro-rated score was calculated (exceptions noted in Supplemental Material). If necessary, variables were transformed to reduce skewness and were converted to z-scores so that all were on a similar metric.

Outcome variables. Although age 20 AP was our major outcome variable for our model, we also included age 17.5 AP in our model. This decision was based on the strong association between AP across ages: we reasoned that risk factors relevant to age 17.5 AP could potentially provide critical context in the multivariate model with age 20 AP as the final outcome. In addition, this approach allowed us to test whether some risk factors were also important in late adolescence/early adulthood or if age 20 AP would only be predicted by age 17.5 AP. Thus, in univariate analyses with AP at age

17.5 *or* age 20 as the outcome variable, we examined the effects of predictors described below.

The major outcome variable for these initial analyses was age 20 AP, which was derived from 20 self-reported items regarding problematic alcohol use. These items and the approach to deriving scores have been previously described (Salvatore *et al.*, 2014). Briefly, these items are from the Alcohol Use Disorders Identification Test (AUDIT), items aimed at assessing DSM-IV Alcohol Dependence criteria, and additional items reflecting negative consequences due to drinking. Factor analysis in Mplus Version 6.11 (Muthén and Muthén, 1998-2011) indicated that a single-factor model provided an adequate fit to the data. Scores were square-root transformed and converted to z-scores for use as outcome variables in the current analyses.

Predictive variables. A broad range of potentially predictive factors, many of which were assessed at multiple ages, was tested for their association with age 17.5 or age 20 AP in univariate models; these are listed here and in Table 1. Those variables included in the final model are also described in additional detail in the Supplementary Material. Early life/exogenous factors included maternal and paternal alcohol consumption and AP; parental SES; and parental physical or emotional cruelty.

Peer/family/social environmental factors tested were stressful life events, parental monitoring, bullying, and peer group deviance. Individual-level predictive variables included scores on the Strengths and Difficulties Questionnaire (total and/or subscales; Goodman et al., 2000); symptoms of attention-deficit hyperactivity disorder; symptoms of conduct disorder and antisocial behavior; religious interest; symptoms of major depression; personality constructs from the International Personality Item Pool (Ehrhart *et al.*, 2008), which included openness, agreeableness, conscientiousness, neuroticism, and extraversion; a modified version of Arnett's Inventory of Sensation

Seeking (Arnett, 1994); scores on the Self-Rating of the Effects of Alcohol (SRE) questionnaire (Schuckit *et al.*, 1997); and illicit substance use.

Predictive variables were based on self-reports, maternal reports, and maternal partner reports; in some cases, composite variables were constructed based on multiple reporters. Variables were prospectively assessed; see Table 1 for average age at assessment. Variables that were associated with the outcome (at $p<0.05$) were retained for inclusion in the first iteration of the multivariable structural equation model.

Statistical Modeling

Predictive variables from the initial univariate analyses retained for inclusion in the first iteration of the multivariable structural equation model if they were associated with the outcome (at $p<0.05$). In the multivariate model, variables were arranged as follows. Early life/exogenous variables were ordered first (earliest) in the model. All other variables were placed in the model in order of their age of measurement. After the variable order was established, we constructed a saturated model where each upstream variable had paths to all downstream variables. We then sequentially pruned paths from the model as follows. First, starting from the top down, we removed all variables with p-values greater than 0.5 (for the null hypothesis that the "true" value of the path was zero). We then performed a second pass removing all with p-values greater than 0.2. Similar iterations were performed of p-values of 0.1, 0.05, and 0.01.

We next switched to using effect sizes (based on standardized path coefficients) for determining which paths to set to 0. On first pass, we removed all paths with effect sizes less than 0.05. This was repeated using 0.06, and finally 0.071 (which corresponds to 0.5% of the variance) as minimum standardized path coefficients. Thus all paths that remain in the final model have p-values less than 0.01 and contributed to at least 0.5% of the variance in the dependent variable. By using this approach we were

able to obtain a final model that was simplified as much as possible without seriously degrading model fit or loss of explanatory power for the outcome variables of interest.

To deal with problems of missing data, models were fit with maximum likelihood. Thus we are operating under the assumption that missing values are missing at random. Under this assumption, we assume that missing values come from the same covariance matrix as the observed values so that missing values are "predictable" (with a measurable level of uncertainty) from the observed variables for the proband.

Results

Model fitting

Model fitting resulted in a final model with acceptable fit (CFI=0.968, TFI=0.948) that explained 30.7% of the total variance of age 20 AP, and 30.6% of the total variance of age 17.5 AP. The model fitting process resulted in the exclusion of many potential predictors, including parental cruelty, childhood physical abuse, scores on the Self-Rating of the Effects of Alcohol scale, and all but one measure of internalizing problems. Variables retained in the final model are described below and listed in Table 1, which also lists variables that did not meet inclusion criteria for the final model. Table 2 provides total and indirect effect sizes between each predictor and AP at age 17.5 and age 20. We focus on the association between risk factors and age 20 AP below, as that was the primary outcome of interest.

Early life predictors

Four exogenous or early life predictors (sex, parental SES, paternal alcohol problems, and maternal alcohol problems) remained in the final model (Figure 1). Of these, paternal alcohol problems and parental SES had direct effects on AP at age 20, which accounted for 62% and 98% of their total effect sizes, respectively (Table 2). Men were more likely to have AP than women, as were participants with higher parental SES.

Other variables measured prior to age 13 that remained in the final model were conduct problems, positive peer relationships, low parental monitoring, and peer deviance. Positive peer relationships were associated with greater AP; higher levels of conduct problems and peer deviance, and lower levels of parental monitoring predicted higher levels of AP. The effect size of this early measure of conduct problems, an individual-level factor, was lower than those of the familial/peer environment measures.

In mid-adolescence (ages 13-16.5), only individual-level variables were predictive of AP in the final model. The personality constructs of extraversion and low conscientiousness, sensation seeking, and symptoms of major depression and conduct disorder were all positively associated with AP.

In late adolescence (age 17.5 and older), both individual-level and environmental variables were predictive of age 20 AP. Peer group deviance and stressful life events were positively associated with the outcome, with the former having a reasonably large effect size. Age 17.5 AP and illicit substance use also predicted age 20 AP. As expected, the former was by far the strongest predictor in the model: it had the highest effect size of any predictor in the model, and only 6% of the effect was indirect. Finally, two individual-level variables assessed at age 18, sensation seeking and illicit substance use, were associated with age 20 AP.

As shown in Table 2, nearly all variables' effects were mediated to some extent. Indeed, only 5 (of 19) predictors in the final model had a direct effect on age 20 AP: higher parental SES, paternal alcohol problems, age 17.5 AP, and age 18 sensation seeking and illicit substance use (only direct effects were possible for the last two). Primary mediators included conduct problems, peer group deviance, and the predictors most temporally proximal to age 20 AP.

Discussion

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This report describes a comprehensive developmental model delineating the complex pathways leading to early adult alcohol problems. Our use of a large, longitudinal population-based cohort, densely assessed across 20 years for a broad range of risk factors, represents a substantial advance over previous studies that have been limited by sample size, scope, and/or reliance on retrospective reporting. Our results provide important insight to the contextual effects of environmental and individual risk factors for AP. The final model accounted for over 30% of the variance in age 20 AP, a figure comparable to several previous studies of older adults (Dubow *et al.*, 2008, Kendler *et al.*, 2011b, Pitkanen *et al.*, 2008), and higher than a model of adult alcohol consumption in a British sample (Maggs *et al.*, 2008). Below, we detail our most noteworthy findings.

First, we found strong evidence of a developmental externalizing pathway to AP (Figure 2), beginning in early adolescence with conduct difficulties at age 11y8m and peer deviance at 12.5. This continues into the mid- to late-teen years as exemplified by low conscientiousness (age 13.5), sensation seeking (ages 13.5 and 18), subsequent conduct problems (age 15.5), and substance use (age 17.5 and 18). In addition, conduct problems and sensation seeking are prominent mediators of many other predictors. These results are consistent with prior reports of associations between externalizing problems and alcohol use/AP (Fergusson *et al.*, 2007, Hesselbrock and Hesselbrock, 2006, Pardini *et al.*, 2007, Whelan *et al.*, 2014, Zucker, 2008), including studies that have used the current sample (Heron *et al.*, 2013, Kendler *et al.*, 2013). These individual-level externalizing problems mediate, and are mediated by, low parental monitoring and peer group deviance, similar to previous reports (Hussong, 2002, Nash *et al.*, 2005, Patterson *et al.*, 1989, Steinberg *et al.*, 1994). Critically, we provide evidence that this externalizing pathway is robust to the inclusion of a wide variety of other risk factors, thereby demonstrating its unique predictive ability.

Second, we observe a strong, and almost entirely direct, relationship between AP at age 17.5 and age 20. Thus, understanding risk for late adolescent AP is quite informative for predicting risk for age 20 AP. However, it is not sufficient: not only do factors assessed at age 18 (sensation seeking and illicit substance use) have substantial effects on age 20 AP, but some earlier risk factors' primary mediation paths do not principally involve age 17.5 AP. This demonstrates that, despite the fact that adolescent AP is considered a robust risk factor for problems in adulthood (McCambridge *et al.*, 2011), the developmental pathways leading to AP extend beyond late adolescence. This finding has potential implications for prevention efforts, in that it reveals that preventing problems at 17.5 does not entirely mitigate risk for later problems.

Third, we demonstrate that effects of sex and the early life influences of parental SES and paternal alcohol problems are persistent, impacting early adult AP via both direct and indirect paths. We conducted *post hoc* multi-group analyses to further investigate the potential effect of sex. In those analyses, the sexes were modeled separately and we tested whether parameter estimates differed significantly across sex; results indicated that they did not. Thus although males are more likely to develop AP, our results suggest that pathways to AP are consistent across the sexes. A previous study using this sample reported a complex relationship between parental SES and alcohol outcomes at ages 16 and 18 (Kendler *et al.*, 2014); the current results, in which the direction and magnitude of effect differs for age 18 and age 20 AP, further confirms that the association is nuanced. Higher parental SES could confer easier access to alcohol, though we are unable to address this hypothesis using the current data. Maternal alcohol problems are a much less robust risk factor than paternal problems. Findings from previous studies have been inconsistent with respect to the impact of paternal versus maternal drinking problems (e.g., (Bohman *et al.*, 1981, Cloninger *et al.*, 1981). In less extensive models examining the relationship between parental and

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offspring alcohol problems in the current sample, parent-specific effects were less discrepant (Kendler *et al.*, 2013), raising the possibility that the true effects of parental alcohol problems must be considered through the lens of comprehensive developmental models such as that described here. These results warrant follow-up.

Fourth, despite previous reports of an "internalizing subtype" of alcohol problems (Del Boca and Hesselbrock, 1996, Hesselbrock and Hesselbrock, 2006), we found only modest evidence of such a pathway. Despite the examination of multiple measures of internalizing symptoms, across various ages, only age 16y6m symptoms of major depression was retained in the final model. Previous studies of the ALSPAC sample have demonstrated a positive relationship between internalizing problems and alcohol misuse (Edwards *et al.*, 2014), but those effects apparently diminish in the context of externalizing-related predictors. Studies in other samples have also demonstrated more extensive positive relationships between various manifestations of internalizing problems and alcohol problems (Kendler *et al.*, 2011b, Mezquita *et al.*, 2014, Prescott *et al.*, 1997).

Additional risk factors were peripheral to the central externalizing pathway to AP. Positive peer relationships, assessed at age 11.75, could confer risk by increasing exposure or access to alcohol, given that alcohol use during this period frequently occurs within the context of peer groups (Hussong, 2000, 2002). Similarly, the positive association between extraversion and AP could be related to peer interactions, as has been demonstrated in previous research (Knyazev, 2004). We also note that stressful life events, which were assessed at age 17.5, were predictive of AP. The events included in this scale range from a death in the family to academic problems to problems with parents; future analyses might examine whether specific events are riskier or whether the *accumulation* of stressors is more important, regardless of the nature of those events.

Finally, we note that a number of previously implicated AP risk factors were not supported in the current model. Childhood abuse and neglect were insufficiently predictive of AP to warrant inclusion in the final model, contrary to previous studies' reports of their relationship with AP (Herrenkohl *et al.*, 2013, La Flair *et al.*, 2013, Mezquita *et al.*, 2014, Potthast *et al.*, 2014). We also found no support for a unique relationship between scores on the SRE scale and AP, despite evidence that SRE scores are related to earlier drinking measures and peer drinking in this sample (Schuckit *et al.*, 2008a, Schuckit *et al.*, 2008b). Furthermore, in many cases only one assessment of a given risk factor was included in the final model – e.g., conduct disorder at 15.5 was included, but not at age 8.5, 12.5, or 13.9 (see Table 1). Similarly, antisocial behavior at ages 18 and 20 was not included in the final model. These exclusions are possibly due to the close relationships among these variables and those that *were* included in the final model (e.g., conduct problems, low conscientiousness, sensation seeking, illicit drug use), largely rendering the excluded variables redundant.

Limitations

The findings reported here should be considered in light of several limitations. First, the ALSPAC sample is relatively young and not yet through the risk period for the development of alcohol problems. Some individuals will develop such problems later in life via pathways distinct from those observed here. Indeed, the internalizing subtype of AP probably has a later onset (Del Boca and Hesselbrock, 1996), potentially explaining why support for an internalizing pathway to AP was limited in this young sample. Accordingly, later waves of data collection may reveal other diverse pathways of risk. In addition, drinking behaviors could differ between US and UK populations, particularly given the earlier legal drinking age in the UK; thus these results warrant replication in a US sample.

In some cases, our modeling approach was limited by the availability of variables at different ages. For example, personality characteristics are evident prior to the available measure at age 13.5, and might actually influence conduct difficulties at age 11.7. Furthermore, our model trimming approach involved excluding measures based on their predictive power, rather than on the age at assessment. Thus, although symptoms of major depression were assessed at age 12.5, they did not strongly predict risk until later in adolescence, so only the later measure was retained. We do not intend to imply that the risk pathway cannot begin prior to the appearance of each factor in the final model. Despite these idiosyncrasies of model fitting, a shift in ordering of these factors in the model would be unlikely to dramatically impact the substantive findings of the study, e.g., an externalizing pathway to AP would likely remain evident.

Our AP outcome is not a diagnostic measure. However, age 20 AP is strongly correlated with age 20 alcohol dependence symptom count (r=0.65; p<0.0001) and with an age 20 alcohol dependence diagnosis (r=0.46, p<0.001), suggesting that our measure of AP is a useful indicator of clinical risk. Attrition in the ALSPAC sample is of some concern, though we have made an effort to address this by using ML in our modeling. Finally, different approaches to modeling are possible and could lead to varying conclusions. For example, we tested related predictors (e.g., conduct problems/antisocial behavior) independently rather than as trajectories, in part due to inconsistencies in assessment items over time. In addition, our structural equation model assumes that the predictor variables act additively and linearly in their impact on AP, which in some cases may be unrealistic.

Conclusions

In summary, using a prospective sample densely assessed from birth through age 20, our comprehensive developmental model indicates that factors at the level of the family environment, social environment, and individual combine to influence risk to early

adult AP. Early life factors, such as parental alcohol problems and SES, have long-lasting effects on AP. In early adolescence, an externalizing pathway to AP is initiated, which involves conduct problems, association with deviant peers, sensation seeking, and illicit substance use. The lack of evidence for several previously implicated risk factors merits additional study in the current sample as well as replication in other samples using comparable methods.

The multifactorial nature of these predictors, and in particular the predictive utility of extrinsic factors such as parental SES and peer group deviance, indicates that reductive biological models will fail to sufficiently explain developmental risk for AP. Rather, consistent with findings from twin and family studies indicating that approximately half the variance in AP is due to genetic factors (Verhulst *et al.*, 2014), the model described here provides support for the importance of both biologically influenced, individual-level factors (e.g., personality constructs, psychopathology) *and* environmental factors, which impact risk through complex and intertwined relationships. To further understand the etiology of AP, both domains must be considered jointly.

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Table 1. Variables tested for association with age 17.5 or age 20 AP.

Construct	Included in final model	Not included in final model	
Exogenous/early childhood	Sex	Maternal alcohol consumption (during/after pregnancy)	
	Maternal alcohol problems	Partner[1] alcohol consumption	
	Partner alcohol problems	Physical/emotional cruelty toward child	
	Parental SES		
Individual Level	SDQ at 11.7y – conduct problems subscale	SDQ at 3.9y – total score	Religious interest at 12.75y
	SDQ at 11.7y – peer problems subscale	SDQ at 6.75y – total score	MD symptoms at 12.5y
	CD at 15.5y	SDQ at 8.1y – total score	MD symptoms at 13.5y
	MD symptoms at 16.5y	SDQ at 9.6y – total score	MD symptoms at 17.5y
	Extraversion at 13.5y	SDQ at 11.7y – total score (some subscales used)	Mood at 18y
	Conscientiousness at 13.5y	ADHD at 7.6y	Neuroticism at 13.5y

	Sensation seeking at 13.5y	CD at 8.5y	Openness at 13.5y
	Sensation seeking at 18y	CD at 12.5y	Agreeableness at 13.5y
	Illicit substance use at 17.5y	CD at 13.9y	SRE at 12.5y
	Illicit substance use at 18y	Antisocial behavior at 18y	SRE at 13.5y
		Antisocial behavior at 20y	SRE at 17.5y
		Religious interest at 9.6y	
Peer/family/social environment	PGD at 12.5y	PGD at 10.5y	Bullying at 8.5y
	PGD at 17.5y	PM at 11.6y	Bullying at 10.5y
	PM at 12.5y	PM at 13.5y	Bullying at 12.5y
	SLE at 17.5y	PM at 15.5y	Bullying at 17.5y
		SLE at 8.6y	

[1]"Partner" refers to the focal child's mother's partner. At the first ALSPAC assessment, the partner was the child's father in >99% of cases.

SES – socioeconomic status; SDQ – Strengths and Difficulties Questionnaire; CD – conduct disorder; MD – major depression; SRE – Self-Reported Effects of Alcohol; PGD – peer group deviance; PM – parental monitoring; SLE – stressful life events

Table 2. Standardized total and indirect effects on age 17.5 and age 20 AP. Where appropriate, the most pronounced mediation paths between predictors and age 20 AP are described.

Predictor Variable	Age 17.5		Age 20		Primary Mediation Paths for Age 20 AP
	Total Std. Effect	Indirect Std. Effect	Total Std. Effect	Indirect Std. Effect	
Sex	0.046	0.046	0.082	0.082	SS 13y6mo, SS 18y
Parental SES	-0.024	-0.024	0.138	-0.003	-
Paternal Alcohol Problems	0.099	0	0.115	0.044	AP 17y6mo
Maternal Alcohol Problems	0.022	0.022	0.013	0.013	-
Conduct Problems 11y8m	0.045	0.045	0.031	0.031	-

Good Peer Relationships 11y8mo	0.101	0.014	0.047	0.047	AP 17y6mo
Low Parental Monitoring 12y8mo	0.13	0.13	0.090	0.090	SS 13y6mo, SS 18y
Peer Group Deviance 12y8mo	0.161	0.161	0.098	0.098	CD - AP 17y6mo PGD 17y6mo - AP 17y6mo
Extraversion 13y6mo	0.128	0.044	0.078	0.078	AP 17y6mo
Low Conscientiousness 13y6mo	0.097	0.097	0.065	0.065	SS 13y6mo, SS 18y
Sensation Seeking 13y6mo	0.102	0.102	0.130	0.130	SS 18y
Conduct Disorder 15y6mo	0.286	0.15	0.146	0.146	AP 17y6mo, PGD17y6mo - AP 17y6mo
Major Depression Symptoms 16y6mo	0.066	0.066	0.035	0.035	PGD17y6mo - AP 17y6mo

Peer Group Deviance 17y6mo	0.341	0.12	0.187	0.187	AP 17y6mo, ISU 17y6mo - AP 17y6mo
Stressful Life Events 17y6mo	0.108	0.025	0.055	0.055	AP 17y6mo
Illicit Substance Use 17y6mo	0.254	n/a	0.185	0.185	AP 17y6mo, ISU 18y
Alcohol Problems 17y6mo	n/a	n/a	0.442	0.027	SS 18y
Sensation Seeking 18y	n/a	n/a	0.161	0.009	ISU 18y
Illicit Substance Use 18y	n/a	n/a	0.096	-	n/a

Std=standardized; y=year; mo=month; SS=sensation seeking; AP=alcohol problems; CD=conduct disorder; PGD=peer group deviance; ISU=illicit substance use

Figure Captions

Figure 1.

Final full model with standardized path estimates. Variables and their corresponding paths are color-coded by approximate developmental time frame.

Figure 2.

Variables falling under the rubric of "externalizing" are highlighted (with other variables in grey), along with the corresponding path estimates, to illustrate the externalizing pathway from early adolescence to age 20 AP.