Risk Factors for Adolescent Drinking

An Introduction

Larsen, H.; Wiers, R.W.

DOI
10.1111/acer.13029
Publication date
2016
Document Version
Final published version
Published in
Alcoholism - Clinical and Experimental Research

Vol. 40, No. 4
April 2016

Risk Factors for Adolescent Drinking: An Introduction

Helle Larsen and Reinout W. Wiers

ADOLESCENTS ARE PRONE to involvement in a variety of risk-taking behaviors (Steinberg, 2008), with alcohol use and misuse being one of the most popular forms of risk taking. During the past few decades, a variety of constructs have been associated with adolescent alcohol use and misuse. Many of the recent findings are covered in this Virtual Issue (http://onlinelibrary.wiley.com/journal/10.1111/(ISSN)1530-0277/homepage/virtual_issue.htm). The 7 papers in the Virtual Issue cover a broad range of risk factors from individual characteristics, such as impulsivity and genetics, to environmental factors, such as peers and drinking contexts. Numerous studies have indicated that environmental, genetic, and developmental factors all influence behavioral outcomes, often in a complex interplay (e.g., Belsky et al., 2009; van der Zwaluw and Engels, 2009). Specific contextual factors may facilitate, attenuate, or exacerbate individual risk factors for adolescent alcohol use. As a case in point, it should be noted that the extent to which alcohol use is considered a risk-taking behavior is likely to depend on the cultural context. In cultures where adolescent alcohol use is illegal and strongly discouraged (like the United States), it can be a marker of deviance, while in Mediterranean cultures, where the norm is to introduce adolescents to drinking in the context of a family meal, this is not the case (e.g., Rolando et al., 2012). Note that in both kinds of cultures, heavy drinking during adolescence would typically be considered deviant. Kuntsche and colleagues (2013) recently concluded, based on data from 38 European and North American countries, that early drunkenness, but not early drinking, is a strong risk factor for later problems. Likewise, in peer groups, an increase in alcohol use during adolescence can be perceived by the adolescents as part of the normative development, whereas for parents, heavy drinking may be viewed as deviant. For instance, previous research has demonstrated relationships between popularity and substance use in adolescence, indicating that youth might use alcohol to sustain their popular status (e.g., Guyll et al., 2014).

INDIVIDUAL DIFFERENCE FACTORS

Adolescence is characterized by various physical and psychological transitions, including rapid changes in brain functioning related to cognitive and social development (Choudhury et al., 2006; Crone and Dahl, 2012). Various personality traits and related cognitive processes, such as impulsivity and sensation seeking, have been related to adolescent drinking (e.g., Jurk et al., 2015). However, these 2 traits appear to follow different developmental trajectories; whereas impulsivity has been found to gradually decline with age, sensation seeking tends to peak in adolescence (Steinberg et al., 2008). In this Virtual Issue, Dougherty and colleagues (2015) compared impulsivity and risk-taking trajectories of adolescents with a positive family history of alcoholism and other drug-use disorders (FHP) with those of adolescents with a negative family history (FHN). FHP early adolescents showed greater levels of impulsivity, both with respect to delay discounting and response inhibition. Interestingly, these differences evened out by mid-adolescence. It is possible that the differences in impulsivity in early adolescence together with the general increase in risk taking augment susceptibility to an early onset of problematic alcohol use. This could be related to an earlier onset of puberty in FHP adolescents, which may be a marker of the evolutionary "live fast, die young" strategy (e.g., Gerald and Higley, 2002). Related to the possible earlier onset of puberty in FHP adolescents, recent studies have provided evidence for a peak in mesolimbic neural responses to reward in adolescence, which was related to the pubertal hormone testosterone, suggesting that puberty is indeed a driving factor in the increased response to rewards during adolescence (Braams et al., 2015; Crone and Dahl, 2012). Hence, individual differences in pubertal timing may increase or decrease one's risk, in interaction with environmental factors. On a positive note, this study suggests that the increased risk may be temporary, which may guide future interventions: preventing early onset of (heavy) drinking in this developmental window may be key in FHP individuals.

In addition to impulsivity and pubertal timing, other important risk factors for adolescent alcohol use at the individual level include changes in circadian rhythms and sleep problems (Hasler et al., 2014). Here, Wong and colleagues (2015) examined prospective relationships between poor

From Developmental Psychology (HL, RWW), University of Amsterdam, Amsterdam, the Netherlands; and Addiction, Development and Psychopathology (Adapt)-lab and Research Priority Area Yield (HL, RWW), University of Amsterdam, Amsterdam, the Netherlands.

Received for publication January 27, 2016; accepted January 27, 2016.

Reprint requests: Reinout W. Wiers, PhD, Department of Developmental Psychopathology, University of Amsterdam, Amsterdam, Noord-Holland 2051 HV, the Netherlands. Tel.: +31 20 525 6842; E-mail: r.wiers@uva.nl

DOI: 10.1111/acer.13029

sleep and substance-related problems in adolescents. Their findings demonstrated that sleep difficulties were prospectively related to a number of alcohol-related variables such as binge drinking and alcohol-related interpersonal problems. These findings are consistent with previous research from the same group in the United States (e.g., Wong and Brower, 2012) and with research in Europe (e.g., Pieters et al., 2014), indicating that sleep problems during adolescence may be a cross-cultural risk factor for alcohol use as well as for other psychological problems (Pieters et al. (2014) found sleep problems to predict both internalizing and externalizing problems). Hence, sleep problems and fatigue appear to be central symptoms, when one considers symptoms from a network perspective, which takes into account the causal interplay between symptoms. In this perspective, disorders result from the interplay between symptoms (see Borsboom and Cramer, 2013).

Moreover, as many psychiatric symptoms originate in adolescence (Paus et al., 2008), it is important to consider them in relation to adolescent alcohol use. Epidemiological studies have reported correlations between common mental disorders, such as anxiety, mood, and personality disorders, and alcohol use disorder (AUD). Here, Harford and colleagues (2015) examined psychiatric symptom clusters as risk factors for AUD in adolescence and found the same patterns as in adult populations, except for a possible moderating role of gender. Specifically, externalizing disorders were positively related to AUDs and females scoring higher on internalizing misery and lower on internalizing fear also demonstrated higher levels of AUDs. As mentioned by the authors, given the limitations of the study design, the direction of effects and underlying mechanisms could not be determined, leaving important questions for future research. One possibility for such research would be to use intensive measurement of the interplay between different symptoms, which could shed further light on developmental pathways into problem drinking (cf., Borsboom and Cramer, 2013; Shadur et al., 2015). The use of new intensive technologies could provide insight into the dynamic unfolding of risk factors for adolescent alcohol (mis)use by exploring the underlying mechanisms in natural environments. In the following paragraph, the importance of environmental factors is emphasized.

ENVIRONMENTAL FACTORS

In addition to individual risk factors, environmental factors such as the company of peers and context transitions traditionally stand out as important risk factors for the development of alcohol (mis)use. Adolescents typically do not drink alone but in the company of peers in a variety of contexts. The presence of peers strongly influences both individual drinking levels (e.g., Larsen et al., 2009; Thrul and Kuntsche, 2015) and risky drinking patterns (Kuntsche et al., 2015a). Note that although recent studies used rigorous designs, including experimental tests and event-level data in natural environments, they were conducted among young adults and not among adolescents. Also, the potential mechanisms explaining the impact of peers on drinking behavior, such as norm setting, intoxication effects, or cue reactivity, remain to be understood (for initial data on these mechanisms, see Korucuoglu et al., 2015). Here, the study by Mair and colleagues (2015) examined adolescent drinking risks in specific drinking contexts. The paper demonstrated that the social-ecological aspects of drinking in different places influence a range of alcohol-related problems. The distinction between frequent alcohol consumption and the quantity consumed appears crucial, as the 2 dimensions of drinking show different relationships with drinking contexts. Along similar lines, the study here of Burdzovic Andreas and Jackson (2015) emphasizes the importance of the transition into high school on drinking behavior. In the U.S. context, the transition into high school appears to be a critical developmental event, associated with increased social risk for excessive alcohol use beyond simple maturational factors (but note that in many other cultures, adolescents do not experience a school transition at this age). Preexisting risk factors were found to moderate alcohol use trajectories, such that children with early delinquency characteristics demonstrated a more rapid progression in alcohol use, perhaps pointing to mechanisms similar to those operating in FHP adolescents (Dougherty et al., 2015). Also in this Virtual Issue, Morean and colleagues (2014) focused on age of onset and delay to intoxication as a risk factor for adolescent alcohol use and binge drinking. Their findings indicated that, when considered simultaneously, both age of onset and rapid progression to drinking to intoxication seem to be important determinants of high-school student drinking. As noted, the importance of age of onset of use (i.e., first sip) depends strongly on culture (Rolando et al., 2012) and early drunkenness was found to be a much stronger risk factor for problem behaviors than first use (Kuntsche et al., 2013, 2015b). Along with Morean and colleagues (2014), these findings point to the importance of focusing on the transition into heavy problematic alcohol use rather than the first drink.

GENE × ENVIRONMENT

While clearly both individual and environmental factors are important in understanding the development of adolescent alcohol use and misuse, an important area of research concerns their interplay. The past decade has seen an abundance of empirical research examining gene × environment interactions (G × E). For instance, an early European finding suggested that young adults who carried the dopamine receptor D4 variable number tandem repeat 7-repeat allele were more prone to the influence of other people's drinking than young adults without that specific variant (Larsen et al., 2010), a finding recently replicated in the United States (Creswell et al., 2012). Here, Olfson and colleagues (2014) examined the interaction between peer drinking and variation in ADH1B, the minor allele of which is a protective genetic factor. They found that among adolescents who

reported that most of their best friends did not drink, the ADH1B variant had a protective effect on drinking, but the effect was reduced among those who reported that most of their best friends were drinkers. A related line of research focused on parental monitoring and alcohol-related rules as environmental factors interacting with genetics (e.g., van der Zwaluw et al., 2014). Despite these important findings, much G × E research still needs to be carried out. Such efforts will need to address the low statistical power and suboptimal measurement of environmental factors that often plague G × E research. Genetically informed studies in which interventions are used to experimentally manipulate environmental factors (e.g., Bakermans-Kranenburg et al., 2008) provide a promising approach to address these problems.

WAYS AHEAD AND PREVENTION

While the selected papers for this Virtual Issue provide interesting samples of current research into adolescent drinking, some limitations should be noted. First, all selected papers included U.S. adolescents, which limits the generalizability to other cultures. Second, while *Alcoholism: Clinical and Experimental Research* publishes both animal and human research, no animal research has been included here (for a recent review, see Spear and Swartzwelder, 2014). Similarly, the collection does not include cognitive neuroscience studies of alcohol's effects on the developing human brain (for a recent review, see Ewing et al., 2014). Although both these lines of research point to detrimental effects of alcohol on the developing brain, with some evidence pointing at stronger effects in females, a recent large prospective study did not find effects of years of binge drinking on a range of neuropsychological measures (Boelema et al., 2015). In a recent review, it was concluded that the evidence is stronger that adolescent alcohol use increases relatively automatic appetitive responses to alcohol than that it impairs the further development of control processes, but that the effect of these appetitive processes on subsequent drinking behavior appears to be stronger in individuals who, at the outset, have relatively weak control functions (Wiers et al., 2015). Hence, further integration and cross-validation of animal research, cognitive neuroscience, and behavioral research into the development of adolescent alcohol use and misuse are warranted. Such efforts may provide important windows of opportunity for interventions that target key processes, such as sleep (e.g., Hendricks et al., 2014), parental rule setting (e.g., Koning et al., 2011), or individual risk factors related to personality (Conrod et al., 2010). Importantly, interventions are not only of practical value but can also be seen as experimental manipulations to test differential susceptibility to environmental factors, for example, G × E predictions. Clearly, the area of research covered in this Virtual Issue is of great importance, both to answer basic scientific questions and because of its substantial clinical and societal implications.

REFERENCES

Bakermans-Kranenburg MJ, Van IJzendoorn MH, Pijlman FT, Mesman J, Juffer F (2008) Experimental evidence for differential susceptibility: dopamine D4 receptor polymorphism (DRD4 VNTR) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. Dev Psychol 44:293–300.

Belsky J, Jonassaint C, Pluess M, Stanton M, Brummett B, Williams R (2009) Vulnerability genes or plasticity genes? Mol Psychiatry 14:746–754.

Boelema SR, Harakeh Z, van Zandvoort MJ, Reijneveld SA, Verhulst FC, Ormel J, Vollebergh WAM (2015) Adolescent heavy drinking does not affect maturation of basic executive functioning: longitudinal findings from the TRAILS Study. PLoS One 10:e0139186.

Borsboom D, Cramer AOJ (2013) Network analysis: an integrative approach to the structure of psychopathology. Annu Rev Clin Psychol 9:91–121.

Braams BR, van Duijvenvoorde ACK, Peper JS, Crone EA (2015) Longitudinal changes in adolescent risk-taking: a comprehensive study of neural responses to rewards, pubertal development, and risk-taking behavior. J Neurosci 35:7226–7238.

*Burdzovic Andreas J, Jackson KM (2015) Adolescent alcohol use before and after the high school transition. Alcohol Clin Exp Res 39:1034–1041.

Choudhury S, Blakemore SJ, Charman T (2006) Social cognitive development during adolescence. Soc Cogn Affect Neurosci 1:165–174.

Conrod PJ, Castellanos-Ryan N, Strang J (2010) Brief, personality-targeted coping skills interventions and survival as a non-drug user over a 2-year period during adolescence. Arch Gen Psychiatry 67:85–93.

Creswell KG, Sayette MA, Manuck SB, Ferrell RE, Hill SY, Dimoff JD (2012) DRD4 polymorphism moderates the effect of alcohol consumption on social bonding. PLoS One 7:e28914.

Crone EA, Dahl RE (2012) Understanding adolescence as a period of social-affective engagement and goal flexibility. Nat Rev Neurosci 13:636–650.

*Dougherty DM, Lake SL, Mathias CW, Ryan SR, Bray BC, Charles NE, Acheson A (2015) Behavioral impulsivity and risk-taking trajectories across early adolescence in youths with and without family histories of alcohol and other drug use disorders. Alcohol Clin Exp Res 39:1501–1509.

Ewing SWF, Sakhardande A, Blakemore SJ (2014) The effect of alcohol consumption on the adolescent brain: a systematic review of MRI and fMRI studies of alcohol-using youth. Neuroimage Clin 5:420–437.

Gerald MS, Higley JD (2002) Evolutionary underpinnings of excessive alcohol consumption. Addiction 97:415–425.

Guyll M, Madon S, Spoth R, Lannin DG (2014) Popularity as a predictor of early alcohol use and moderator of other risk processes. J Stud Alcohol Drugs 75:919–992.

*Harford TC, Yi HY, Chen CM, Grant BF (2015) Psychiatric symptom clusters as risk factors for alcohol use disorders in adolescence: a national study. Alcohol Clin Exp Res 39:1174–1185.

Hasler BP, Soehner AM, Clark DB (2014) Circadian rhythms and risk for substance use disorders in adolescence. Curr Opin Psychiatry 27:460–466.

Hendricks MC, Ward CM, Grodin LK, Slifer KJ (2014) Multicomponent cognitive-behavioural intervention to improve sleep in adolescents: a multiple baseline design. Behav Cogn Psychother 42:368–373.

Jurk S, Kuitunen-Paul S, Kroemer NB, Artiges E, Banaschewski T, Bokde AL, Büchel C, Conrod P, Fauth-Bühler M, Flor H, Frouin V, Gallinat J, Garavan H, Heinz A, Mann KF, Nees F, Paus T, Pausova Z, Poustka L, Rietschel M, Schumann G, Struve M, Smolka MN (2015) Personality and substance use: psychometric evaluation and validation of the substance use risk profile scale (SURPS) in English, Irish, French, and German adolescents. Alcohol Clin Exp Res 39:2234–2248.

Koning IM, van den Eijnden RJ, Engels RC, Verdurmen JE, Vollebergh WA (2011) Why target early adolescents and parents in alcohol prevention? The mediating effects of self-control, rules and attitudes about alcohol use. Addiction 106:538–546.

Korucuoglu O, Gladwin TE, Wiers RW (2015) Alcohol-induced changes in conflict monitoring and error detection as predictors of alcohol use in late adolescence. Neuropsychopharmacology 40:614–621.

Kuntsche E, Otten R, Labhart F (2015a) Identifying risky drinking patterns over the course of Saturday evenings: an event-level study. Psychol Addict Behav 29:744–752.

Kuntsche E, Rossow I, Engels R, Kuntsche S (2015b) Is 'age at first drink' a useful concept in alcohol research and prevention? We doubt that. Addiction doi:10.1111/add.12980 [Epub ahead of print].

Kuntsche E, Rossow I, Simons-Morton B, Ter Bogt T, Kokkevi A, Godeau E (2013) Not early drinking but early drunkenness is a risk factor for problem behaviors among adolescents from 38 European and North American countries. Alcohol Clin Exp Res 37:308–314.

Larsen H, Engels RCME, Granic I, Overbeek G (2009) An experimental study on imitation of alcohol consumption in same-sex dyads. Alcohol Alcohol 44:250–255.

Larsen H, van der Zwaluw C, Overbeek G, Granic I, Franke B, Engels RCME (2010) A variable-number of-tandem-repeats polymorphism in the dopamine D4 receptor gene affects social adaptation of alcohol use: investigation of a gene-environment interaction. Psychol Sci 21:1064–1068.

*Mair C, Lipperman-Kreda S, Gruenewald PJ, Bersamin M, Grube JW (2015) Adolescent drinking risks associated with specific drinking contexts. Alcohol Clin Exp Res 39:1705–1711.

*Morean ME, Kong G, Camenga DR, Cavallo DA, Connell C, Krishnan-Sarin S (2014) First drink to first drunk: age of onset and delay to intoxication are associated with adolescent alcohol use and binge drinking. Alcohol Clin Exp Res 38:2615–2621.

*Olfson E, Edenberg HJ, Nurnberger J Jr, Agrawal A, Bucholz KK, Almasy LA, Chorlian D, Dick DM, Hesselbrock VM, Kramer JR, Kuperman S, Porjesz B, Schuckit MA, Tischfield JA, Wang JC, Wetherill L, Foroud TM, Rice J, Goate A, Bierut LJ (2014) An ADH1B variant and peer drinking in progression to adolescent drinking milestones: evidence of a gene-by-environment interaction. Alcohol Clin Exp Res 38:2541–2549.

Paus T, Keshavan M, Giedd JN (2008) Why do many psychiatric disorders emerge during adolescence? Nat Rev Neurosci 9:947–957.

Pieters S, Burk WJ, Van der Vorst H, Dahl RE, Wiers RW, Engels RCME (2014) Prospective relationships between sleep problems and substance use, internalizing and externalizing problems. J Youth Adolesc 44:379–388.

Rolando S, Beccaria F, Tigerstedt C, Törrönen J (2012) First drink: what does it mean? The alcohol socialization process in different drinking cultures. Drugs Educ Prev Policy 19:201–212.

Shadur JM, Hussong AM, Maleeha HM (2015) Negative affect variability and adolescent self-medication: the role of the peer context. Drug Alcohol Rev 34:571–580.

Spear LP, Swartzwelder HS (2014) Adolescent alcohol exposure and persistence of adolescent-typical phenotypes into adulthood: a mini-review. Neurosci Biobehav Rev 45:1–8.

Steinberg L (2008) A social neuroscience perspective on adolescent risk-taking. Dev Rev 28:78–106.

Steinberg L, Albert D, Cauffman E, Banich M, Graham S, Woolard J (2008) Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: evidence for a dual systems model. Dev Psychol 44:1764–1778.

Thrul J, Kuntsche E (2015) The impact of friends on young adults' drinking over the course of the evening—an event-level analysis. Addiction 110:619–626.

Wiers RW, Boelema SR, Nikolaou K, Gladwin TE (2015) On the development of implicit and control processes in relation to substance use in adolescence. Curr Addict Rep 2:141–155.

Wong MM, Brower KJ (2012) The prospective relationship between sleep problems and suicidal behavior in the National Longitudinal Study of Adolescent Health. J Psychiatr Res 46:953–959.

*Wong MM, Robertson GC, Dyson RB (2015) Prospective relationship between poor sleep and substance-related problems in a national sample of adolescents. Alcohol Clin Exp Res 39:355–362.

van der Zwaluw C, Engels RCME (2009) Gene-environment interactions and alcohol use and dependence: current status and future challenges. Addiction 104:907–914.

van der Zwaluw CS, Otten R, Kleinjan M, Engels RCME (2014) Different trajectories of adolescent alcohol use: testing gene–environment interactions. Alcohol Clin Exp Res 38:704–712.

(* in this Virtual Issue).