

Why is adolescence a key period of alcohol initiation and who is prone to develop long-term problem use?: A review of current available data

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Background: Early adolescence is a key developmental period for the initiation of alcohol use, and consumption among adolescents is characterized by drinking in high quantities. At the same time, adolescence is characterized by rapid biological transformations including dramatic changes in the brain, particularly in the prefrontal cortex and the mesocorticolimbic dopamine system.

Methods: This article begins with an overview of the unique neural and behavioural characteristics of adolescent development that predispose these individuals to seek rewards and take risks such as initiation of drinking and high levels of alcohol intake. The authors then outline important factors associated with an increased risk for developing alcohol problems in later adolescence and young adulthood. Thereafter they address causality and the complex interplay of risk factors that lead to the development of alcohol use problems in late adolescence and young adults.

Conclusions: A few recommendations for the prevention of underage drinking are presented.

Keywords: *alcohol; adolescent; brain development; risk factors; dependence*

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Alarming facts

It is well-known that rates of alcohol consumption dramatically increase during the teenage years and alcohol is by far the most widely used intoxicant among adolescents, ahead of nicotine and marijuana (Johnston et al. 2009). In all EU member states in 2000–2001 nearly all (over 90%) 15 to 16 year-old students reported having drunk alcohol at some point in their life, based on samples of 800–3000 respondents per age-group per country (Currie et al., 2004). More importantly, adolescents do not limit themselves to small doses. Indeed, adolescents drink on average more than twice as much per drinking episode as adults do (Substance Abuse and Mental Health Services Administration, 2006). In Europe, 7% of 15 to 16 year-old students report having been intoxicated already at least 20 times in their lives (Ahlström & Österberg, 2004). These rates raise several concerns: alcohol abusing adolescents are more likely to display delinquent behavior, other substance use problems, decreased academic performance (Substance Abuse &

Mental Health Services Administration, 2008), suicide and depression and unprotected sexual intercourse, placing them at risk for unplanned pregnancy and sexually transmitted diseases (Bonomo et al., 2001). Moreover, adolescence is a period associated with the highest risk for developing alcohol use disorders (AUDs; i.e., DSM-defined alcohol abuse and dependence; American Psychiatric Association, 1994) (Johnston et al., 2010). These numerous health and social consequences make the problem of alcohol consumption in adolescents a major public health concern and establishing prevention programs that focus on limiting abusive drinking during this period of life is crucial. However, for these to be adapted and successful, a deep understanding of the antecedents and aetiology of adolescent drinking behaviours is crucial. This article reviews important findings on the specificities of adolescence that (1) are thought to be responsible for alcohol initiation, use and abuse vulnerability; and (2) make this a phase during which the likelihood of developing chronic consumption increases.

On the definition of adolescence

Before addressing the subject at hand some definitions are useful. Adolescence in humans broadly denotes young people that are in their second decade of life (Hiller-Sturmhöfel & Swartzwelder, 2004). However, many researchers extend this period until 25 years of age, calling it “late adolescence” (Siegler et al., 1992). Here we adopt this latter view as many of the cited works we include in our review involved studies of adolescents studying at university, suggesting the subjects are 18–25 years of age. Because of ethical restrictions, much of the data that will be presented are from animal studies, particularly rodents, a species in which the period of adolescence is defined as 30 to 50 postnatal days (Hiller-Sturmhöfel & Swartzwelder, 2004).

Why do adolescents drink?

Is the brain at fault?

Important reasons for teenagers’ propensity to experiment and often abuse alcohol are believed to have their roots in physiological changes, and particularly those taking place at the center of the nervous system.

A brain’s construction “in progress”

Throughout its development, the human brain undergoes changes in terms of its morphology, volume, composition and function (Dekaban, 1978; Giedd et al., 1999). While earlier it was believed that brain development was completed by adolescence, it is now well established that this maturation does not end before about 25 years of age (Giedd, 2004). Moreover, given that important modifications largely occur during fetal and neonatal phases, adolescence represents another critical period of deep cortical development and remodelling (Paus et al., 2001). One of the development processes occurring at this time is *myelination*. The axons connecting brain cells become increasingly insulated with myelin, which speeds up the relay of electric impulses within the brain and thus the flow of information along axonal pathways (Benes et al., 1994; Salami et al., 2003). This permits the emergence of networks involving distant brain regions, replacing what were more local influences, and it thus supports the maturation of decision-making, impulse control, and emotional regulation (e.g. Rubia et al., 2007). Another process is called the *pruning process* and consists of a synaptic refinement (Giedd, 2004). During this sculpting phase, unused or rarely activated connections are removed and room is made for more efficient wiring, which assures more efficient information processing. Pruning is also essential for the construction of longer chains of neurons, which are necessary for complex decision-making (Winters & Arria, 2011). These development processes affect two important regions. First is the prefrontal cortex (PFC) (e.g. Crews et al., 2007; Spear, 2007): its volume is reduced

(Sowell et al., 1999, 2001; van Eden et al., 1990) and its neural circuits and connectivity are refined (e.g. Huttenlocher, 1984; Rosenberg & Lewis, 1994). The changes in this region are very important as they underlie the acquisition of executive functions (e.g., response inhibition, attention, working memory) (Casey et al., 2000; Paus, 2005). Second, the pattern of dopamine (DA) production and utilization is modified. The mesocortico- limbic brain regions, including the striatum and the nucleus accumbens (constituting the reward system), which receive input from the neurotransmitter DA also undergo pronounced developmental changes (Spear, 2009).

The question that arises is: Does this phase of brain maturation make the brain vulnerable to alcohol use?

Alcohol initiation and abuse during adolescence must be reframed as part of the typical reward-seeking and risk-taking behaviors that characterize this period of life (Crews et al., 2007). As many parents of adolescents have observed, young people are masters at identifying novel and highly stimulating experiences as well as at taking risks to engage in them (Zuckerman, 1979). This is what scientists call “sensation seeking” (Hittner & Swickert, 2006). Teens are also “risk-takers”; with few inhibitions they make risky decisions in response to cues for potential gain, despite the possibility of negative consequences (Fowles, 1980; Zuckerman, 1983). Although these transient personality traits may be normative and functionally adaptive as the adolescent strives for independence from adults, such attitudes may also lead to some adverse consequences such as initiation of drug use. Indeed, these two disinhibitory traits have been associated (among others) with emergent alcohol use (e.g., Sher, et al., 2000; Zuckerman & Kuhlman, 2000; Lejuez et al., 2002; Lejuez et al., 2007), early onset, use frequency (e.g., Donohew et al., 1999; Earleywine & Finn, 1991) and higher rates of alcohol consumption in 14–22 year olds (Romer & Hennessy, 2007). Adolescents’ proclivity toward risk-taking behaviour such as alcohol use might be explained by several elements characterizing this period of high neuroplasticity in youth.

A central factor is the *desynchronised maturation of brain regions*: the shaping process indeed seems to occur following a specific trajectory, i.e. from the back to the front of the brain (Gogtay et al., 2004). While children have equally immature limbic and prefrontal regions and adults have acquired both fully developed systems, the adolescent phase is characterised by an asynchrony in the maturation of the two regions. Indeed, bottom-up limbic regions underlying emotional and incentive processing develop earlier than top-down prefrontal regions, which are implicated in refinement of reasoning, goal and priority setting, behavioral control and evaluating long and short term rewards (e.g. Bava & Tapert, 2010). A growing child must wait until the end of adolescence or early adulthood for these latter regions to be fully

developed. This imbalance in the development of reward and control systems has been suggested to be the cause of the enhanced responsivity to incentives and risky behaviours observed in adolescents (Casey et al. 2008; Ernst et al. 2006; Somerville et al. 2010; Steinberg et al. 2008). This situation led Dr. Walsh to describe the teenage brain as “*a fully functional car accelerator, but in which the brakes have not been installed yet*” (Walsh, 2004, p. 72). This perspective suggests that the more mature limbic system overrules the prefrontal system and drives choices towards the pursuit of immediate gratification, rather than long-term gains, resulting in poor decisions (Bava & Tapert, 2010).

Developmental changes in the ventral striatum during adolescence are also believed to be responsible for heightened reward-driven behaviours. The striatum is indeed widely considered to be the region in the brain that is implicated in the evaluation of rewards (e.g., Schultz, 1998; Montague et al., 2004) and its non-linear developmental route peaks in mid-adolescence. Neurobiological evidence from both rodent and human studies indicates that the remodelling of the striatum that occurs around this period is linked to increased sensitivity to rewarding stimuli (e.g. Doremus-Fitzwater et al., 2010; Ernst et al., 2005; Galvan et al., 2006; Van Leijenhorst et al., 2010). Two opposing theories of reward processing in the striatum during adolescence have been proposed. One hypothesis posits that adolescent reward-seeking and risk-taking might result from a relative hypo-responsivity of the striatum during adolescence (Blum et al., 1996, 2000; Bjork et al., 2004), such that adolescents generally attain less positive feelings from rewarding stimuli, leaving them in need of heightened reward-seeking behaviors to achieve the same activation as adults (Spear, 2000). The opposing view suggests that during adolescence the striatal reward system is disproportionately hyper-responsive, which subsequently results in greater reward-related behaviors (Chambers et al., 2003). While evidence for both hypotheses has been reported, the field has generally converged on this latter hypothesis, given the relatively greater amount of evidence to support it (Ernst et al., 2009; Somerville et al., 2009). However, it is important to consider some plausible explanations for the disparate results. Together, data from different works on dopamine specificities in adolescents compared with adults (e.g. Teicher et al., 1993; Andersen et al., 1997; Stamford, 1989; Andersen and Gazzara, 1993; Laviola et al., 2001, 2003; Bolanos et al. 1998) lead to the conclusion that adolescents actually have lower basal rates of dopamine release, which would lead them to seek greater stimulation (rewards) to increase dopamine release. On the other hand, their storage pool would be more important, and under stimulation conditions their ability to release dopamine (partly due to an increased number of dopamine receptors) would be greater than

that of adults. Thus, once stimulated by rewarding experiences, the level of dopamine released would be more generous (Laviola et al., 2003), and in turn would conduce to a reinforcing feedback cycle, promoting more reward-seeking actions (Galvan, 2010).

A brain like no others

Besides the particularities in brain maturation, other biological factors might direct decisions on alcohol use and abuse during the teenage years, namely, differences in *alcohol sensitivity*. Alcohol sensitivity refers to the amount of alcohol an individual must absorb to experience the effects of alcohol (O’Neill et al., 2002; Bartholow et al., 2003): low-sensitivity (LS) individuals are less sensitive to the subjective alcohol-related sensations or impairments than are high-sensitivity (HS) individuals. Much of what is known about alcohol susceptibility in adolescence is from rat studies. These suggest that adolescents require more alcohol than their older fellows to experience the negative consequences of alcohol, such as disruption of motor function (White et al., 2002), sedation (Moy et al., 1998; Little et al., 1996), social impairment (e.g., Varlinskaya & Spear, 2006), and withdrawal symptoms/hangover effects (Acheson et al., 1999). This lower sensitivity to undesired effects of alcohol, which serve as cues to limit intake, indicates that more drinking is necessary to elicit a signal that it is time to stop. Although ethical concerns today exclude replicating such an experiment on humans, in one older study, authors Behar et al. (1983) observed only limited behavioural changes occurring in children aged 8 to 15, after having received a dose of alcohol that had been intoxicating in an adult population. On the other hand, it has been shown that adolescent rats are more sensitive to some positive effects, i.e. the social disinhibition effect of alcohol when compared with adults (Spear et al., 2002). This means that intoxication procures greater “social comfort” in adolescent rats than in adults. These enhanced appetitive and attenuated aversive sensitivities in adolescents in contrast to adults, could then also explain the propensity of adolescents to drink more than other age groups.

In conclusion, adolescence is associated with a greater propensity for alcohol use and abuse, which is part of the typical tendency of this age group to undertake risky and reckless behaviours. Important reasons given for this vulnerability are centered on changes occurring in the brain around the time of puberty. The most critical changes were reviewed above. However, the direct impact of the brain on adolescent behaviour must be considered with caution (Giedd, 2004). Social and cultural factors should be taken into account while considering human behaviour. Below we will review other important factors underlying alcohol use and abuse among adolescents. Given the considerable volume of factors implicated in

this issue, this review is not exhaustive. Our focus here is on presenting some factors to provide part of the picture toward in an effort to better understand alcohol consumption within this young population.

Positive alcohol expectancies

Expectancies are defined as a “probability held by the individual that a particular reinforcement will occur as a function of a specific behaviour” (Wiers et al., 1997). The role of beliefs about the positive or negative behavioural, emotional and cognitive effects of alcohol intake has been demonstrated for alcohol use (Ajzen, 1991; Brown et al., 1985). The belief that alcohol increases sexual, physical and social pleasure, and enhances social assertiveness and relaxation is likely due not only to the individual’s own experience with the substance, but also to observations of others’ attitudes while drinking, as well as exposure to advertising (Brown et al., 1987). If exposure to these episodes takes place during childhood and begins influencing beliefs, it has been shown that positive alcohol expectancies increase and culminate during adolescence (Miller et al., 1990). Before the age of 9 children usually regard alcohol negatively and think drinking is bad, while by about age 13 a progressive shift occurs in their expectancies; they become more positive (Dunn & Goldman, 1996, 1998). However, these expectancies then stagnate and begin to wane in adulthood (Miller et al., 1990, Sher et al., 1996). Positive expectancy of alcohol’s effects appears to be a robust predictor of the initiation of drinking, intention to drink, and drinking rates among both middle school (Christiansen et al., 1989) and college students (Stacy et al., 1990) and drinking among individuals under 35 years of age (Leigh & Stacy, 2004).

Peer influence and normative perceptions

Descriptive normative perceptions are defined as people’s perceptions of the way the majority of people behave in given situations (Borsari & Carey, 2001; Lapinski & Rimal, 2005). Adolescents are highly vulnerable to social influences (Kandel et al., 1987); they tend to copy what their friends do to feel accepted (Terry-McElrath et al., 2009) even though they know their parents would disapprove of their behaviors (Steinberg, 2008). Descriptive norms are claimed to represent the most important predictors of adolescent risk behaviour (Beal et al., 2001; D’Amico & McCarthy, 2006). Affiliation with deviant or substance-using peers is linked to a higher risk of engaging in substance-use, delinquency and risk-taking behaviours (Gardner & Steinberg 2005). The problem is that youth often overestimate levels of their peers’ substance use, and as this perception has a high impact on their own behavior, they are more likely to overuse substances (Edwards et al., 2008). Thus, although greater autonomy and affiliation with peers have many positive consequences, peers have a specific and highly significant role in adolescence when it

comes to risk taking behaviours such as alcohol use, which in turn lead to negative consequences. Moreover, when making risk decisions, younger adolescents often take into account the normative behavior of older peers (Kinsman et al., 1998). It has been shown that adolescents who have older peers face an increased risk for alcohol and other substance use than adolescents who do not associate with older peer groups (Grant et al., 1998).

A recent study took a special interest in Facebook, a social networking site that currently counts more than 500 million active users world-wide (Zuckerberg, 2008), and which has seen a 149% increase in new teen (12–17 years) members over the last two years (Corbett, 2010), as a potential factor in influencing normative perceptions in youth. Indeed, based on the content of the photos and comments posted on profiles, adolescents may develop a perception of normative alcohol use among peers of varying ages, including older peers. The study showed that adolescents who view Facebook profiles featuring older peers drinking alcohol as normative, reported more positive affective attitudes toward alcohol use and pictures of alcohol users, a greater drive to use alcohol, a lower perception of vulnerability to the adverse effects of alcohol, and greater perceived norms of alcohol use when compared with adolescents who viewed profiles that did not depict alcohol use as normative.

Why are some adolescents more at risk of addiction?

If alcohol experimentation and use appear to be almost an inevitable milestone from childhood to adulthood, different drinking patterns have been identified over time once drinking begins. Most adolescents drop their interest in alcohol as they begin to take on adult responsibilities such as marriage, parenthood, employment, incompatible with continued alcohol abuse (e.g. Muthen & Muthen, 2000; Wood et al., 2000) while others do not. However, research also shows that drug use during adolescence may boost a young person’s risk for developing a substance use disorder later in life (Casey et al., 2008). What specificities make some young people at higher risk for chronic alcohol use? Epidemiological studies have provided insight into some factors that would explain the difference between young recreational users and those at risk of developing alcohol use disorders (AUDs). Wide ranges of explicative elements for the development of alcohol problems from adolescence to adulthood have been proposed. Although it is difficult to predict which individuals will develop serious problems and who will not, some of these factors are reviewed below.

Drinking motives

When you ask adolescents why they drink, most of them report drinking for social motives or enjoyment. Some report enhancement motives and only a few talk about

copied as a reason (Kuntsche et al., 2005). For example, in an Argentinian study 80% of 13 to 18 year olds reported drinking for enjoyment while only 7% stated to improve a bad mood, 4.6% to be accepted by peers, and 1% to relax or to escape boredom (Jerez & Coviello, 1998). In another study conducted among 14 to 16 year olds in the UK, 94.4% explained that their heavy drinking was to make nights out more pleasurable (Plant et al., 1990). In a Canadian study, most college students talked about enjoying the taste, partying and being social, while only 2.1% mentioned forgetting worries or feeling less shy (Kairouz et al., 2002). While social motives appear to be associated with moderate alcohol use, enhancement is linked to heavy drinking, and coping motives are associated with alcohol dependence later in adulthood (Carpenter & Hasin, 1998a, 1998b; Carpenter & Hasin, 1999). Indeed, it has been suggested that drinking to cope with problems is effective in the short term but leads to adverse long-term consequences as it also raises discordance as negative issues have not been adequately dealt with (Cooper et al., 1995; Kassel et al., 2000).

Family history: the genetic component

Research has consistently provided evidence that a positive family history is a strong (and perhaps the most robust) predictor of alcoholism risk (e.g., McGue, 1999). Biological children of alcoholics are about 3 to 5 times more inclined to develop alcohol related problems than the biological children of non-alcoholics (e.g. Cotton 1979; Finn et al. 1990; Lieb et al. 2002), with a particularly high level of risk for multigenerational (Hill et al. 2000; Peterson et al. 1992) or dense (Dawson & Grant 1998) family history. Moreover, the role of age in heritability seems to be very important. Research specifically addressing heritable components in the emergence of AUDs during adolescence have found that although the initiation of alcohol use and social drinking were only modestly related to genetic risk in this age range, problematic use appears to be very much heritable (Hopfer et al., 2005; Rhee et al., 2003; Pagan et al., 2006). Fowler et al. (2007) similarly reported that about 30% of the variance in initiation was due to genetic influences while they accounted for 50–60% in the progression of alcohol use. Furthermore, it has been shown that the genetic contribution to problem drinking in youths is limited in mid-adolescence, but that the biological risk increases as teens mature into late adolescence (Rose et al., 2001).

Conduct disorders

It has been shown that conduct problems during childhood predict alcohol-related problems among adolescents and young adults (Kuperman et al., 2005; Johnson et al., 1995). Such issues are comorbid with ADHD and when combined they pose a high risk for SUD, although this risk is halved for treated youth (Wilens

et al. 2003). Further, the severity of the subtype of conduct or antisocial behaviour is correlated with the severity of the substance dependence problems (Cottler et al., 1995; Bucholz et al., 2000).

Individual differences in cognitive control

Impulsivity is an established risk factor for alcoholism. Inhibition, the capacity to suppress inappropriate or impulsive behaviours, matures across childhood (Diamond & Goldman-Rakic, 1989) and continues to develop during adolescence (Luna et al., 2001, 2004). The increase in cognitive control with age has been displayed using several diverse inhibitory tasks as go/no-go (Luciana & Nelson, 1998), Stroop (Tipper et al., 1989), stop signal (Greenberg & Waldman, 1993; Ridderinkhof et al., 1999; Williams et al., 1999), and antisaccade tasks (Fischer & Weber, 1998; Fukushima et al., 2000; Luna et al., 2004; Munoz et al., 1998). Adolescents perform better than children, but still not as well as young adults in these tasks (e.g. go/nogo: Casey et al., 1997; Jonkman, 2006; antisaccade: Fischer & Weber, 1998; Fukushima et al., 2000; Luna et al., 2004; Munoz et al., 1998). As noted above, these changes in control abilities develop in parallel with changes in prefrontal region activity (Booth et al., 2003; Casey et al., 1997; Velanova et al., 2008) and occur later in the development process. However, differences do exist in this development. Indeed, studies have shown that adolescents at risk for substance use disorders (SUD) display decreased activation in frontal and parietal regions (Schweinsburg et al., 2004a) and less overall frontal activation (McNamee et al., 2008; Schweinsburg et al., 2004b) in inhibition paradigms. Norman et al. (2011) showed that youth who progress into heavy use of alcohol show less activation in frontal and temporal brain regions during inhibition than those who remain non- to minimal users throughout adolescence. In their work, Petit et al. (2012) showed that heavy drinking adolescents, who were considered at risk for developing alcohol misuse, had poorer inhibitory performance when compared with moderate drinking adolescents. These results thus support the hypothesis that abnormal inhibition indices may antedate and predict future involvement problems with alcohol.

Personality Traits: Sensation seeking and risk taking

Besides impulsivity, we have identified two other disinhibitory traits, sensation seeking and risk-taking, which have also been linked to alcohol abuse (e.g., Sher, et al., 2000; Zuckerman & Kuhlman, 2000). Although adolescence has been described as a period of heightened reward seeking and risk-taking behaviors (Spear, 2000; Gardner & Steinberg, 2005), this does not mean that all adolescents are high reward seekers (Galvan et al., 2010). Individual differences in taking risks also exist (Benthin et al., 1993). Initial high levels of sensation seeking assessed at baseline in early adolescence have

been associated prospectively with alcohol use trajectories, characterized by quicker consumption intensification and persistence of use further into later adolescence and early adulthood (Bennett et al., 1999; Crawford et al., 2003). Measures of great risk taking traits at baseline assessment have been linked to more severe latent courses of alcohol use over time from early adolescence (Colder et al., 2002). Furthermore, looking specifically at individual changes in disinhibitory constructs on alcohol use in early adolescents, MacPherson et al. (2010) showed that it is not simply initial high levels of sensation seeking and risk taking that are responsible for alcohol use, but rather an intensification of both traits that forecast a greater likelihood of future abuse. Galvan et al. (2007) demonstrated that individual differences exist in the dopaminergic mesolimbic circuitry implicated in reward processing and that this relates to the propensity to engage in risky behaviours across development. Specifically, they showed that individuals more inclined to report a higher frequency of risky behaviours in “real-life” were also those with the more important ventral striatum recruitment as measured in laboratory. Together, these findings suggest that although adolescents as a group are considered to be risk-takers, some adolescents with particularly elevated novelty and sensation-seeking traits, due to neural changes, are more at risk than others for engaging in and later pursuing risky behaviors, like drug abuse.

Gender

While a decrease in alcohol use is typically observed in the late 20s, men demonstrate a higher peak in consumption levels when compared with women (Casswell et al., 2003; Muthen & Muthen, 2000). Thus, while gender differences in alcohol use are minimal during adolescence (Young et al., 2002; Hicks et al., 2007; Johnston et al., 2008), boys who abuse alcohol are more at risk of developing alcoholism as adults (e.g. Prescott, 2001; SAMHSA, 2008; Sher et al., 2005). A recent work (Petit et al., 2013) supports this fact and suggests that differences in alcohol cue reactivity (that could be due to differential DA release) may partly explain the greater vulnerability of males.

Patterns of drinking

Early initiation

As most adolescents begin experimenting with alcohol at the beginning of puberty (DeWit et al., 2000; Faden, 2006), the initiation of alcohol use among adolescents takes place at an early age. In Europe, students initiate use on average just after 12½ years of age (Currie et al., 2004). At the same time, many prospective and retrospective human studies have revealed that early onset of alcohol intake constitutes a risk factor for later alcohol abuse or addiction (e.g. Grant & Dawson, 1997; DeWit et al., 2000; Grant et al., 2001; Warner & White, 2003; York et al., 2004; Pitkanen et al., 2005). For example,

Grant & Dawson (1997) showed that out of those who initiated drinking at age 14 or younger, 40% developed alcohol dependence, while only 10% of those who began drinking at age 20 or older did so. They demonstrated a relationship between decreasing age of onset and risk of alcohol dependence. They observed a 14% reduction in risk of future adult-onset substance use disorders for each year onset of drinking was delayed. Results from the 2008 U.S. survey on drug use and health showed that individuals who started using alcohol at age 14 or younger were five times more at risk of lifetime alcohol use disorder as compared with those who first consumed alcohol after 21 years of age (SAMSHA 2009).

Binge drinking

Binge drinking is the consumption of at least 4 (for women) and 5 (for men) alcoholic drinks (10 g of pure ethanol) on one occasion at least once in the last two weeks and often with the aim of becoming drunk (Stolle et al., 2009). The drinking style of adolescents and young adults is characterized as binge drinking; heavy drinking and drinking to intoxication are highly prevalent (Currie et al. 2004; Hibell et al. 2004; Kuntsche et al. 2004). For example, in Europe 15 to 16 year olds reported that the average amount of alcohol drunk on the last drinking occasion was six drinks (Hibell et al., 2004). Approximately 39% of 15–16 year olds report binge drinking in the past month (Hibell et al., 2012). Moreover, frequent early binge drinking constitutes another risk factor for the development of alcohol consumption disorders (e.g. McCarty et al., 2004; Maurage et al., 2013a; Maurage et al., 2013b; Campanella et al., 2013).

Discussion: The chicken and egg problem

A consideration of factors associated with alcohol dependence inevitably leads to a “chicken and egg” question, or a debate over “what came first”. It is indeed possible that the factors we highlighted above cannot in fact be properly considered as risk factors for developing alcohol abuse or dependence. To be considered as a risk factor, a variable must be associated with the disorder, and antedate the onset of the disorder (Mrazek & Haggerty, 1994). Collecting longitudinal data (Donovan, 2004) (i.e. collected by following an individual respondent over a course of time) is necessary to determine the status of a variable as a risk factor. Some of the factors we reviewed have been identified through the use of such methodologies, including cross-lagged design models, which allow us to elucidate the directionality of disorders. Many of these disorders have been shown to predate substance-misuse, and thus can be considered risk-factors. For example, studies have confirmed that antisocial behaviors predate adolescent alcohol misuse (Young et al., 2008). Other studies found that conduct problems and risk taking are predictive factors of alcohol use and alcohol-

related problems among adolescents and young adults (Ohannessian & Hesselbrock, 2008a, b; Kuperman et al., 2005; Johnson et al., 1995). Finally, Norman et al. (2011) showed that aberrant inhibitory networks in youth who progress into heavy use of alcohol are present before the onset of use. However, other findings we reviewed are based on cross-sectional data (i.e. collected from participants at one point in time) and as such we can establish that a variable is associated with the alcoholism variable, but cannot determine the direction of influence between the two variables. It remains unclear whether some of the factors we cited preceded alcoholism or were a consequence of it. Moreover, bi-directional effects are also possible in some contexts (Young et al., 2008). For example, it has been shown that early drug users are more prone to develop mental health problems, like anxiety, depression, and conduct disorder, which further heighten probabilities of developing regular and problematic consumption of alcohol (Teesson et al., 2005). Furthermore, although these studies provide insight into directionality, they do not discern how these disorders relate, or the mechanisms that are involved. Some studies have also suggested that the disorders associated with and adolescent substance use share a common underlying etiology, which might account for a proportion of the comorbidity between them (Castellanos-Ryan & Conrod, 2012).

Another debate concerns the alcohol initiation factor. Two main hypotheses have been proposed to explain the relationship between early alcohol use and later dependence. First, it has been suggested that exposure to alcohol during adolescence might disrupt critical developmental processes occurring at that time (e.g. decision making), and therefore lead to problems with alcohol later on. This idea is based on the finding that heavy drinking during early and mid-adolescence is associated with and could be the cause of (still to be evidenced) neuropsychological deficits like memory problems (e.g. Brown et al. 2000). The other explanation for the early exposure effect could simply be that early intake of alcohol has to be considered as a marker, not as a precursor for an abusive disorder later in life. Several works have indeed shown that other risk factors like family history of alcoholism (McGue et al., 2001b), high tendency to seek novel situations (Baumrind 1987), disinhibited behaviour and psychopathology enhance the probability of early drug initiation (Tarter et al. 1999; Franken & Hendriks 2000; McGue et al., 2001a, b). For example, an adolescent that has an early alcohol debut may end up developing alcoholism because he or she actually has a high tendency to seek out new experiences (Spear, 2003). From this perspective, McGue et al. (2001b) have shown that individuals who first drink at an early age also demonstrate heightened degrees of disinhibitory behaviour and psychopathology *before* they first experiment with alcohol. Other studies

(e.g. Westling et al., 2008; Downing & Bellis, 2009) also contribute evidence that early age of first drink may not be the cause of subsequent alcohol problems. They found that early pubertal onset (associated with alcoholism risk; Downing & Bellis, 2009) predicts consumption of alcohol and intoxication at an earlier age. Two twin studies aiming at resolving this causality question, albeit by studying different substances, have resulted in conflicting outcomes. One large study examining the risk for alcohol abuse and dependence reported that age of onset was correlated with but was not causal in the development of alcohol use disorders (Prescott & Kendler 1999). In contrast, a smaller study of twins who were discordant for early-onset marijuana use, reported that age of onset was causal in the development of later drug use and abuse problems (Lynskey et al. 2003). The question of causality is thus still to be determined. Consequently, the assumption that one can prevent alcohol dependence or other alcohol-related problems later in life by keeping young people away from drinking until age 21 or older sounds utopian.

Conclusion

The period of adolescence is associated with a greater propensity for alcohol use and abuse, which is part of the typical tendency of this age group to engage in risky and reckless behaviours. Relatively new research, supported by sophisticated brain imaging technologies, has documented reasons for this vulnerability, locating these in changes occurring in the brain around the time of puberty. As the brain cannot be blamed for the whole story, we presented above two characteristics constituting important (but not exhaustive) factors that shape adolescence and which play a role in increasing alcohol use/abuse at this age. These help us to understand why a situation with short-term reward or positive outcome possibilities perception, along with peer pressure and the opportunity to consume alcohol as a way to enhance assertiveness, may submerge the still-maturing “brake” circuitry of an adolescent via the “accelerator” region, thus weakening his capacity to make thoughtful decisions (Reyna & Farley, 2006), and pushing him to accept drinking alcohol. Moreover, his high tolerance and high sensitivity to the positive effects of the substance could then incite him to consume more than reasonable quantities.

Given that adolescents typically “grow out of” drinking during their mid-twenties, we presented several putative susceptibility factors that singly or in combination might contribute to the development of pathological alcohol involvement during this sensitive developmental period. If the factors we reviewed have been particularly associated with alcohol problems, they constitute only a sample of them. For example, neuropsychological deficiencies, weak social support, stressful life events, childhood mistreatment, alcohol availability (Clark & Winters, 2002), early

pubertal onset (Downing and Bellis, 2009) and family violence (Enoch, 2006) also contribute to the likelihood of involvement with substance problems.

We explained above that the relationship between the use of alcohol and the factors cited is intricate, as consumption could actually be a consequence, rather than the cause. We also discussed whether early onset of alcohol use should be considered as a precursor for later alcohol problems, or as a marker. In fact, debuting with drugs or developing a drug dependence are complex behaviours that should be reframed; individual genes, the environment and the dynamic interaction between them have to be taken into account in seeking to understand them. The finding of a high correlation between the genetic risk for alcohol use and friendships based on substance-use (Fowler et al., 2007) illustrates this complex interaction of genetic and environmental variables in explaining teen alcohol use. Dick et al. (2007a, b) have also demonstrated that besides friends' alcohol use, genetic influences on adolescent substance use were enhanced in environments characterized by lower parental monitoring. In the same vein, sons of alcoholic fathers characteristically show attenuated sensitivity to the intoxicating and aversive effects of alcohol (e.g. Begleiter & Porjesz, 1999; Schuckit, 1994). Vulnerability factors can also trigger one another. For example, it has been shown that under controlled behaviour increases conduct problems and positive expectancies for alcohol consumption (Ohannessian & Hesselbrock, 2008; Sher et al., 1991). Let us imagine a 13 year-old boy going through normative adolescence-linked biological and physiological changes. These body modifications incite him to engage in risk-taking behavior, such as trying alcohol. His first positive experimentation with alcohol leads him to spend time with other peers who like alcohol, leading to more frequent use of alcohol inciting additional episodes. His particular sensitivity, combined with the inciting environment he frequents, combined with weak parental monitoring, leads him to drink more and more. Continued drinking causes conduct disorders, which in turn lead him to continue to drink and to alcohol dependence. In this way, a juvenile alcohol use screenplay may be the start of a developmental route, leading to abuse and dependence. Let us imagine a second young boy who is also experiencing changes at adolescence, but for whom a different outcome emerges. The only conclusion we can draw is that cumulative risk factors for a young adolescent confronted with alcohol experimentation will elevate his risk for alcohol use disorders (Warner & White 2003).

Perspectives

In conclusion, we can confirm that adolescents are more risk of alcohol use, abuse and dependence than other age groups. A complete understanding of the causes requires

further investigation, and this is clearly an important field to explore. Indeed, a body of evidence indicates that adolescents are extraordinarily sensitive to the effects of drugs (Spear, 2002). Adolescent alcohol use has a negative impact on brain maturation (e.g. morphological changes; Medina et al. 2008; Nagel et al. 2005; anisotropic differences in white matter; De Bellis et al. 2008), leaves irreversible sequelae on cognitive functioning (e.g. attention, visuospatial functioning, learning, retrieval of verbal and nonverbal information; Brown et al. 2000; Medina et al. 2007; Tapert & Brown 1999, 2000; Tapert et al. 2002) and significantly heightens susceptibility for developing a substance use disorder. Further research should integrate the great number of neural, genetic, and behavioural components via cross-modal analyses to determine biomarkers for alcohol problems in youth. In the mean time, setting up prevention programs to promote an alcohol-free lifestyle and providing treatment for those youth who are already abusing alcohol is a matter of urgency. More importantly, we must take into account and integrate scientific discoveries on brain development when constructing these programs for them to have a chance of succeeding. This means that one must be well aware that adolescents take risks in large part because of normative biological processes, and that the tendency to engage in risk taking is not only negative; it may be regarded positively as it promotes personal growth (Galvan et al., 2007). Given that their brains are programmed to engage in risk taking, speaking against this inclination by teaching teens about negative outcomes, is consequently useless (Winters & Arria, 2011). Rather, efforts should take account of what is happening and, for example, work with adolescents on decision-making aptitudes, to make up for their "brake systems". This might help them to have greater control, particularly when they are in a situation that "challenges" the state of their brain (i.e. in emotional and arousing situations with a high degree of peer influence) (Winters & Arria, 2011).

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