Why Adolescents Are at Risk of Misusing Alcohol and Gambling

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Abstract — Aims: The aim of the study was to explore the reasons for alcohol misuse and other risk-taking behaviours in adolescence. Methods: Narrative review. Results: Vulnerable adolescents make suboptimal addictive-related choices in the period of initiation of alcohol use and gambling, which is also a period of cognitive and brain development, and in health behaviours. Hyperactive response to rewards as well as possibly hypoactive responses to punishments combined with weaknesses in the abilities to regulate strong impulses results in greater risk of alcohol and gambling misuse. Conclusion: Abnormal patterns of alcohol consumption (e.g. binge drinking) could dramatically reinforce this disequilibrium by enhancing salience for alcohol and associated information and compromising self-regulatory processes. There are some preventive and therapeutic cognitive training strategies that can strengthen willpower in adolescents.

INTRODUCTION

The paradox observed for human adolescents is that while they are stronger, faster, more resistant to disease and have better reasoning and decision-making skills than children, mortality increases 200% for them during this time (Dahl, 2001). This is notably due to difficulties in the regulation of behaviour and emotion, leading to forms of deaths by accident or suicide that should be preventable. However, considerable literature demonstrates that equal exposure to a specific psychoactive, alcohol or gambling leads to varying risk of addiction (for an important epidemiological study, see Swendsen et al., 2010). This review focuses on the weak behavioural adjustments that characterize adolescence is an experimental period with a number of exciting experiences, e.g. puberty and the time at which an individual attains a stable, independent role in society? This is a period of opportunity, during which decisions become increasingly independent of adults.

However, it is also the period of much vulnerability, with an enhanced taste for risk together with impulsive actions and decisions that can lead to serious consequences; peers influence becomes more powerful (for better or for worse) and novel experiences more attractive. Thus adolescence is an experimentation period with a number of exciting experiences, e.g. alcohol, drugs and gambling. Note that initiation is not a pathological process per se but sometimes it may evolve into severe disorders (i.e. compulsive behaviours) (Chambers et al., 2003).

Reasons for an abnormal developmental pathway in adolescence are numerous (Viner et al., 2012) and include distal (e.g. income inequality, educational status), proximal (e.g. quality of family environment, drug exposure) and individual characteristics (e.g. cognitive and brain vulnerabilities). Cognitive neuroscience has brought important insights into cognitive and neural underpinnings of adolescent behaviour (Steinberg, 2005; Ernst et al., 2006, 2011; Casey et al., 2008). Specifically, pubertal changes are concomitant with neural and cognitive changes including reward and punishment processing as well as self-regulatory mechanisms that help explain the propensity for risk taking and sensation seeking that typifies adolescent behaviour. Based upon this knowledge, developmental pathways of addictive behaviours during adolescence have been proposed (e.g. Crews and Boettiger, 2009; Silveri, 2012), with focus on the weak behavioural adjustments that characterize both adolescence and individuals who have lost control over drug use or non-drug use behaviours (e.g. Chambers et al., 2003). At the extreme, this state of ‘inflexibility’ exemplified by drug and non-drug addictive behaviours has been thought to reflect impaired ‘basic’ behavioural learning processes, poor self-regulation and impaired decision-making (e.g. Noël et al., 2013). As a consequence, ‘willpower’ may be too limited, a concept that refers to the capacity for choosing according to long-term, rather than short-term, outcomes (Bechara, 2004). In other words, one characteristic of adolescence is that the power of positive short-term consequences of choice and behaviours may exceed self-regulation capacities, which can be risky to health yet might be normal within adolescent social development (Windle et al., 2008). This paper describes individual motivational and regulatory responses thought to increase the risk of alcohol and gambling misuse in adolescents. However, individual differences that present long before the onset of puberty are also important to understand the elevation of risk (e.g. low capacities of delay of gratification in children aged 4 predict cocaine/crack use in individuals vulnerable to psychosocial maladjustment, Ayduk et al., 2000). As another example, higher levels of impulsive behaviour as early as age 3 predict aggressive behaviour and drug use in adolescence (Caspi and Silva, 1995). Thus, the quality of pre-adolescents’ cognitive and affective functioning (e.g. resisting immediate temptations) is important to take into account when investigating the addictive risk in adolescents (Romer, 2010).
ADOLESCENCE: DEFINITION

Physical growth and sexual maturation depend on functions of the hypothalamo–pituitary–gonadal axis (e.g. Romeo, 2003). These changes herald a period when many mammals become especially attracted by novelty (Steinberg and Belsky, 1996) and a tendency to move away from the safe familial nest together with increasing influence of social peers. In humans, this period of transition occurs between ages 10 and 19 but there are cultural and socioeconomic determinants too of when ‘childhood’ is deemed to end (WHO, 2003).

Puberty is associated with many changes in brain structure and function, altering drives, emotions, motivations, cognitions as well as the underpinnings of social life. Note that the relationships between the physical growth and sexual maturation that define puberty, and brain changes, remain poorly understood (Ernst et al., 2006; Blakemore et al., 2010). This paper does not further examine this issue of whether developing specific cognitive functions and sexual maturation are interconnected but note, for example, delayed or premature sexual maturation does not predict brain development (Cameron, 2004).

ADOLESCENCE AS A PERIOD OF ACCELERATED BRAIN AND COGNITIVE DEVELOPMENT

Compared with childhood, adolescence tends to be a period of higher risk taking, sensation/novelty seeking and impulsivity (e.g. Romer, 2010). Neurobiological models of adolescence have generally identified that the subcortical brain structure matures more quickly than prefrontal control regions (Casey et al., 2008). This disharmony between brain regions responsible for increased motivation and drive and those implicated in self-regulation is challenging for the adolescent and his/her necessary adjustment to social rules and personal objectives. Indeed, from developmental neuroscience, adolescence can be characterized by changes affecting the relationship between behavioural/neural systems of approach, avoidance and self-regulation. The ‘dual-systems model’ (Steinberg, 2010) and the ‘maturational imbalance theory’ (Casey et al., 2011) both posit that higher risk taking during adolescence results in a combination of heightened reward sensitivity and immature impulse control. These two systems, undergoing change during adolescence, develop along different timetables. The system deemed ‘hyperactive’ processes social and emotional information, and is localized mainly in the ventral striatum and ventromedial prefrontal cortex. The second system, viewed as ‘hypopactive’, is referred to as the ‘cognitive control’ system and is dependent on brain regions such as prefrontal, parietal and anterior cingulate cortices. Reward sensitivity follows an inverted U-shaped curve (Romer, 2010), with sensation seeking peaking during mid-adolescence, whereas cognitive control increases gradually and linearly with a peak at late adolescence.

Intuitively appealing, this dual-system approach has been recently criticized for its oversimplification, for the lack of empirical support and for the underestimation that an activated socioemotional system could be sometimes adaptive. In addition, maladaptive functioning could be the result of an activated cognitive control system (Pfeifer and Allen, 2012). Although interesting, this discussion is beyond the scope of the present review (see Strang et al., 2013 for a response to these critics).

According to the ‘triadic model’ (Ernst et al., 2006, 2009), adolescence is characterized by changes affecting the relationship between approach responses, avoidance tendencies and regulatory mechanisms. During adolescence, the approach system would be hypersensitive in comparison with the avoidance one, which would be relatively hyposensitive. The regulatory/supervisory system (the executive function) would not be strong enough to make adequate adjustments for good adaptation (i.e. by orchestrating the relative contribution of the approach and avoidant behavioural systems).

At the neural level, in association with positive emotions, the system responsible for adolescents’ goal-seeking behaviours in response to cues of reward is hyperactive (e.g. in animals, ‘adolescents’ exhibit greater locomotor sensitivity to psychostimulants: Schramm-Sapyta et al., 2004). Ventral striatum (in particular the nucleus accumbens) is thought to support reward processes and approach behaviour (Wise et al., 1992; Di Chiara, 2002). The amygdala, particularly through its basal and lateral nuclei, has been shown to mediate harm avoidance (LeDoux, 2000) and the prefrontal cortex and its role in cognitive control (e.g. Miller, 2000) would not be sufficiently developed to counteract hyperactive approach tendencies. For instance, adolescents are less sensitive to risks in the context of goal-directed action (for a review, see Ernst et al., 2006), which may suggest that their cognitive signal of harm is altered.

Increased reward sensitivity has been demonstrated by investigating inter-temporal decision-making (Steinberg et al., 2009). This literature indicates that teenagers’ preference for smaller immediate rewards over larger delayed rewards is mainly due to increased activation of the ventral striatum, orbitofrontal cortex and medial prefrontal cortex, presumably because immediate rewards are especially emotionally arousing (‘consider the difference between how you might feel if a crisp 100 euros bill were held in front of you and being told that you will receive 150 euros in 2 months’).

In addition, there is evidence showing that prepotent response inhibition systems are less mature in youths than in older adults. Firstly, pattern of prefrontal activation during performance inhibition in youths is more diffuse compared with adults (Casey et al., 2000). Secondly, adolescents perform worse than adults on a number of cognitive tasks probing the capacity to suppress dominant responses (e.g. Stroop, go/no-go or antisaccade tasks) (Casey et al., 2000). Thirdly, the protracted pruning of the prefrontal cortex, resulting in thinning of cortical grey matter, represents growing frontal control over behaviour, the absence of which is associated with impulsive actions and disadvantageous decision (Romer, 2010). Structural brain imaging shows slower cortical thinning and white matter growth in dorsal and frontal brain areas than in ventral and occipital areas during and beyond the adolescent period (Gogtay et al., 2004). Finally, the structural and functional connectivity among these neural systems evolves during adolescence (for review, see Lewis et al., 2004).

Of note, Bjork and collaborators (2004) reported data that could be interpreted as hyposensitivity of ventral striatum/dopamine regions in adolescence. Indeed, they found less activation in this region for a similar level of reward-related performance compared with adults during motivation to act. Interestingly, the context represents a major moderator of the relationship between cognitive and brain systems and risk taking (Gardner and Steinberg, 2005). Indeed, those
adolescents who performed a video driving game in the presence of peers take more risks than those who were alone and peer influence had no effect on adult and less effect on young adults. Consistently with most theories of adolescence, increased attention paid to the magnitude of potential rewards (i.e. reward salience) is intrinsically present at puberty and could still be enhanced due to their development of social brain (through peer influence).

A neurocognitive approach to decision-making in adolescents (Van Duijvenvoorde and Crone, 2013) has brought together domains of psychology, economy, neuroscience and computational science to investigate how people make decisions. In an attempt to break down adolescents’ assumed taste of risk into its constitutive components (gain sensitivity, sensitivity to known and unknown outcomes distribution), Tymula et al. (2012) showed that in comparison with adults, adolescents were more prone to accept offers in ambiguous conditions, that is, when the likelihood of winning and losing is unknown, reflecting a higher tolerance of the unknown. Although the advantage in terms of learning opportunities is clear, being comfortable with uncertainty could also render adolescents less efficient at making advantageous decisions in their everyday life. Indeed, in some cases in which information is missing, a good decision could be to gain additional information (e.g. by engaging more cognitive resources) instead of making a firm choice, which could be less likely in individuals with low sensitivity to uncertainty.

**ADOLESCENCE AND COOL/HOT EXECUTIVE FUNCTIONING**

Executive functions (EFs) could be understood as a variety of cognitive ability allowing the conscious control of thought, emotion and action. People differ in their ability to regulate thoughts and actions (or ‘willpower’). Abilities that fall under the rubric of executive functioning include planning, abstract reasoning, mental flexibility (i.e. switching between tasks or mental sets), manipulation of information in working memory (WM) (e.g. constant updating content stored in WM), decision-making and suppression of dominant/automatic response (e.g. Bechara and Van Der Linden, 2005). Recent theories about the nature and organization of individual differences in EFs strengthen the idea that EFs show both ‘unity’ and ‘diversity’ (Miyake and Friedman, 2012). Indeed, common EF is about one’s ability to actively maintain task goals and goal-related information and use this information to effectively bias lower-level processing (i.e. through inhibition). Specific contributions reflect flexibility (i.e. ease to transitioning to new task-set representations) and updating-specific components such as hypothetically effective gating of information and controlled retrieval from long-term memory. An important contribution to the understanding of the relationship between behavioural disinhibition and response inhibition arises from a twin study showing that at 12 and 17 years of age, behavioural disinhibition ‘was more closely related to response inhibition than to other EFs (WM updating and task-set shifting), and this relationship was primarily genetic in origin’ (Young et al., 2009). EFs are characterized by both their precocity and their stability (Miyake and Friedman, 2012). Indeed, when children were shown an attractive toy with an instruction not to touch it for 30 s, inter-individual differences at age 3 were still evident at age 17 (Friedman et al., 2011).

In addition, the nature of EFs must be embedded within a specific task context (the target EF has something to operate on) (Miyake and Friedman, 2012). Critically, a distinction has been made between EFs operating on unemotional information such as abstract decontextualized reasoning (cool EF) and the regulation of affective and motivational processes (hot EF) (Zelazo and Müller, 2002). It has been demonstrated that the inhibition of a response associated with incentive-related cues (emotionally charged or reinforcing contexts) is still more difficult to perform (Somerville et al., 2011).

Interestingly, more closely related to impairments in social and emotional functioning than cool EFs (Hongwanishkul et al., 2005; Giancola et al., 2012), hot EFs are required to control emotional reaction and inhibit basic impulses that make possible further elaborated decontextualized problem-solving abilities (Sonuga-Barke et al., 2002). As early as 3–4 years of age, personal salience of the rewards compromises the implementation of the appropriate top-down control necessary for delaying gratification (Prencipe and Zelazo, 2005).

The distinction between hot and cool EFs is particularly relevant in the context of adolescence, that is, when a new set of challenging emotional experiences may undermine emerging self-control. Taken together, there have been indications that hot EFs develop slower relative to cool EFs during adolescence. For example, on a delay-discounting task (Madden et al., 1997) in which participants were instructed to decide between smaller immediate and larger delayed rewards, adolescents younger than 16 years of age tended to discount future reward more steeply than either older adolescents or adults (Steinberg et al., 2009). Another well-known task, The Iowa Gambling Task (IGT; Bechara et al., 1994, 1997), which was initially developed to investigate the decision-making defects of neurological patients in real-life, has been shown to tap into aspects of decision-making that are influenced by affect and emotions (Bechara, 2004). In this task, some decks of cards are disadvantageous because they yield higher immediate rewards but unpredictable and larger delayed punishments, while the advantageous decks provide lower immediate gains but unexpected and smaller future losses (Bechara et al., 1994). In contrast to the typical linear development of EFs (Stroop task, digit span task), affective decision-making abilities progressed in a J-shaped curve (Prencipe et al., 2011; Smith et al., 2012): younger, more developmentally naïve children performed better on the IGT than older, early adolescent individuals, with performance becoming advantageous again towards the end of the teenage years.

This immature period of brain regions involved in the hot executive system (e.g. orbitofrontal cortex and ventromedial prefrontal cortex) perhaps explains why adolescents are often capable of understanding the risks and consequences of their actions (good explicit knowledge), despite making disadvantageous decisions, which suggests that cognitive control functions are already in place, whereas affective control is still developing (Steinberg, 2005).

**ADOLESCENCE AND ALCOHOL USE**

The proportion of 15- to 16-year-old European teenagers who report being drunk at some time in their lives ranges from 36%

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in Portugal to 89% in Denmark (EMCDDA, 2003). Concern is growing about increased prevalence of drunkenness and ‘binge’ use of alcohol for recreational purposes.

In Europe, whereas the overall consumption of alcohol has decreased in young people over the last decades, patterns of ‘binge’ or ‘heavy drinking’ increase (EMCDDA, 2003). Heavy drinking, as defined as the consumption of more than five drinks for adult men and four for adult women over a 2-h period (US ‘drinks’, i.e. 13 g ethanol, which results in a blood alcohol concentration of 0.08% or greater, NIAAA, 2004) in the USA, climbs from 1% at age 12 to 30.4% at age 18 and even around 50% at ages 21 and 22 (SAMHSA, 2010). These levels are higher for males and females in their first year of college in the USA (White et al., 2006). Earlier age of onset of drinking alcohol predicts later abnormal alcohol consumption: whereas the onset of alcohol use at age 17 is associated with a lifetime prevalence of alcohol dependence of 28%, which rises to 38% when the onset of alcohol use was before age of 15 (Hingson et al., 2006).

Weak executive functioning/decision-making and alcohol misuse in adolescence

A growing body of literature demonstrates alterations in cognitive performance engaging the frontal networks and hippocampus in adolescent and young adults with alcohol-use disorders (e.g. Tapert et al., 2004) and with binge drinking (e.g. Schweinsburg et al., 2010; Squeglia et al., 2012). In rodents, acute exposure to the equivalent of two beers in a human disrupts NMDA receptor-mediated long-term potentiation in the adolescent but not adult hippocampus (see review by Zeigler et al., 2005).

Impaired executive functioning (Moss et al., 1994; Giancola and Tarter, 1999) and deficits in language (Moss et al., 1994) have also been reported. Maladaptive trajectories such as poor occupational functioning were linked to poorer visuospatial processing, verbal and working memory, and attention (Hanson et al., 2011).

WM has been recognized as a key concept for the understanding of alcohol misuse at adolescence (Giancola and Tarter, 1999; Nigg et al., 2006; Romer et al., 2009; Khurana et al., 2013). By definition, WM is a system required for the simultaneous storage and processing of information, hence supporting human thought processes by providing an interface between perception, long-term memory and action (Baddeley, 2003). For instance, after several potential confounding variables were controlled (e.g. anxiety), adolescent binge drinkers compared with non-binge drinkers had lower performance on visuospatial tasks requiring manipulation of information stored in WM (Sher et al., 1997). In a prospective study (Khurana et al., 2013), pre-existing weakness in WM predicted both concurrent alcohol use as well as the increased frequency of use over a 4-year follow-up period in a community-based sample or early adolescents. Furthermore, two forms of impulsivity, delay discounting and acting without thinking, were an underlying weakness in updating their triadic model for adolescence; Ernst and colleagues (2011) emphasized that risk of the onset of psychopathologies such as anxiety, depression and addiction during this developmental period may be due to enhanced automatic stimulus-driven attention (as opposed to goal-driven attention requiring WM-related functions such as inhibition and goal-maintenance) that could lead to higher impulsiveness in youths.

Better general EFs are associated with less behavioural disinhibition, a concept underlying a variety of externalizing behaviour problems seen in Attention Deficit Hyperactive Disorder (ADHD), conduct disorder and substance use (e.g. Barkley, 1997). Independently of ADHD and conduct disorder status, impaired response inhibition ability predicted the onset of alcohol-related problems and illicit drug use in adolescents (Nigg et al., 2006). In addition, weakened executive functioning moderated the relationship between alcohol use and aggression in a sample of university students (Giancola et al., 2012). On a task assessing aggressive tendencies while breath alcohol concentration was moderately high, participants with lower EFs manifest more aggression.

Regarding decision-making under uncertainty, performance on the IGT (Bechara et al., 1994) in adolescents has brought important insights about the relationship between hot EFs and alcohol-use disorders (e.g. Johnson et al., 2008). After controlling for WM capacities, performance on the IGT predicted binge drinking (Johnson et al., 2008). Closer inspection of these results revealed that binge drinkers had acquired some awareness and gut feeling about the relative payoffs of good and bad decks in the early trials but that due to hypersensitivity to reward, they took too many risks in the latter trials, that is to say, once decisions are made under risk. The follow-up phase of this study showed that better IGT scores predicted fewer drinking problems and fewer drinks 1 year later after controlling for demographic variables, previous drinking behaviours, WM and impulsivity (Xiao et al., 2009). Taken together, an analogy could be made between failure to learn from repeated mistakes on the IGT and disadvantageous decision-making underlying risky behaviours such as repeated binge drinking episodes with harms.

Cognitive deficits and alcohol use: what comes first?

It remains difficult to know whether these abnormalities are a direct result of the toxic effects of alcohol on the brain (i.e. the consequence of use) or whether pre-existing brain conditions (antecedent to the initiation of use) are present that differentiate those who begin using at a young age form those who do not (Silveri, 2012).

The ‘chicken or the egg’ question has been addressed in different ways. For instance, in adolescent girls more drinking days in the past year predicted a greater reduction in performance on visuospatial memory tasks, whereas in boys, more hangover symptoms in the year before follow-up testing predicted worsening of sustained attention (Squeglia et al., 2009). Such results might suggest a neurotoxic effect of ethanol or a harmful effect on neurons of repeated, albeit mild, ethanol withdrawal, which could interfere with brain maturation. However, despite increased frequency of alcohol use over a 4-year follow-up period in a community-based sample of early adolescents (Khurana et al., 2013), WM among participants remained stable over the 4-year period suggesting that the detrimental influence of increased drinking frequency on WM is marginal between early and mid-adolescence.

Another way to address this issue was to compare adolescents who have no or minimal alcohol exposure, but who have a positive family history for alcoholism (FH+), to age-matched
adolescents who have a negative family history (FH−) for alcoholism. The FH+ group is an ideal genetic-risk model, as a positive history of alcoholism is associated with an earlier onset and higher magnitude of use as well as with a higher prevalence of alcohol-use disorders in adolescents and young adults. The neuropsychological literature found mixed results regarding cognitive impairments in FH+ prior to the initiation of alcohol consumption. Evidence shows that FH+ youths have deficits in abstract reasoning and planning and have lower IQ scores, poorer academic performance and slower trajectories in cognitive improvement at 1-year follow-up when compared with FH− youths (Silveri et al., 2004). But other studies failed to document differences between FH+ and FH− youths. Interestingly, deficits in FH+ youths were predominantly found in children of antisocial alcoholics (Poon et al., 2000), which may constitute a critical and poorly controlled factor to explain discrepancy across studies. In addition, the number of relatives determining the criteria for a positive family history of substance (a single parent in some studies, numerous in others) is also likely to explain differences across studies, i.e. a more extensive family history of alcoholism may indicate a greater genetic susceptibility than a lesser family loading.

From a neurobiological perspective, FH+ youths exhibit smaller overall total brain volumes and less inhibitory frontal activation during the performance of a Go/No-Go functional magnetic resonance imaging task, than their FH− comparison subjects (Schweinsburg et al., 2004). At the structural brain level, information processing speed was correlated significantly with white matter volume in FH− females only (Silveri et al., 2008), which may indicate that subtle abnormal cognitive/brain relationship could represent a risk factor for substance abuse in female adolescents who have not yet initiated drug use.

Many reports support the idea that higher impulsivity, which could be defined as ‘actions that are poorly conceived, prematurely expressed, unduly risky or inappropriate to the situation and that often result in undesirable consequences’ (Durana and Barnes, 1993), elevates the risk of addiction in young population. For instance, lower rates of development of behavioural self-control strongly and specifically predict early initiation of drug use at 14 years of age and higher number of drug-related problems at 17 years of age (Wong et al., 2006). In line with this result, impulsive/hyperactive symptoms of ADHD in children were the stronger predictors in a prospective study for initiation of tobacco, alcohol and illicit drug use at 14 (Elkins et al., 2007). In another group with genetic risk of addiction, the adolescent offspring of substance-use disorder parents showed elevated prevalence of alcohol dependency and stimulant use (Kendler et al., 2002). This relationship is hypothesized to be due to impulsivity-related endophenotype (Verdejo-García et al., 2008). In the same vein, impulsivity measures (personality dimension of constraint) during adolescence also significantly predict problem gambling behaviour at a follow-up assessment (e.g. Slutske et al., 2005).

In summary, during childhood and adolescence, decrements in cognitive abilities, associated with familial history of substance abuse (and antisocial personality component and multiplex family history of alcoholism), may be present prior to cognitive impairments that result from early initiation and continued use of alcohol and drugs.

ADOLESCENCE AND GAMBLING

Within an environment where gambling is becoming more accessible and normalized, increasing numbers of underaged youths are participating in these activities. Among those who gamble (between 70 and 90% of adolescents, Blinn-Pike et al., 2010), 3–8% are problem gamblers (two to four times higher than in adults) and 10–15% are at-risk to become pathological (Derevensky et al., 2007). Poor school performance, criminality and family problem are the most currently reported consequences of problem gambling in teens. A number of factors have been put forward to increase the risk of maladjustment to gambling offer in young populations: cognitive, social and dispositional (Donati et al., 2013).

With regard to cognitive factors, it is noteworthy that adolescents’ knowledge of mathematical principles or gambling odds is similar between problem and non-problem gamblers (Derevensky et al., 2007). Nonetheless, problem and distinct to non-problem gamblers in terms of a number of erroneous beliefs centre on the topic of independence of random events, such as the gambler’s fallacy (Tversky, 1974). For instance, it is the case when after a long sequence of Black, the likelihood of Red could be judged higher on the next spin. Social factors include parents’ gambling frequency and the severity of the parents’ gambling problems (Vachon et al., 2004) and peer influence (e.g. having friends who gamble increases the risk of gambling, Hardoon et al., 2004). Of note, higher levels of parental monitoring are associated with lower levels of adolescent gambling behaviours but only for youths whose parents do not gamble (Vachon et al., 2004). Regarding dispositional factors, impulsivity (acting without thinking and the inability to delay gratification and sensation seeking) and depression are key dimensions of problem gambling in adolescents, as evidenced by correlational (e.g. Turner et al., 2008) and longitudinal (Lee et al., 2011; Liu et al., 2013) studies. For instance, male youths being in the high impulsivity trajectory doubled the odds of meeting criteria for at-risk or problem gambling (Liu et al., 2013). In other words, gambling in youths could be a means of coping with stress, avoiding or escaping from problems, especially in adolescents with poor or maladaptive coping skills and impulsive tendencies.

In addition, gender is also an important variable, with peer influence, gambler’s fallacy and impulsivity contributing less in females than in males to teenagers’ problem gambling (Donati et al., 2013).

CLINICAL IMPLICATIONS AND PERSPECTIVES

This review points to the usefulness of WM training as a critical intervention strategy to reduce early alcohol use and gambling and to enhance self-control abilities (Hofmann et al., 2012).

Various components of WM can be improved through training (Klingberg, 2010) and are thought to help to curb impulsive drinking in hazardous college drinkers (for a recent review, see Houben et al., 2011a,b; Jones et al., 2011; Bates et al., 2013) and reduce problematic eating behaviour (Houben et al., 2011a,b). For instance, adapted versions of the WM and control tasks designed by Klingberg et al. (2002) have been used for the purposes of training working memory: a visuospatial WM task, a backward digit span task and a letter span
task with the difficulty level being automatically adjusted on a trial-by-trial basis. Initially, each task involves sequences of three items, the length of the sequences increases and decreases according to participants’ performance. By contrast, in the control condition, the difficulty levels of the WM tasks are not adjusted, remaining at the initial, easy level throughout each task. Critically, studies on training-induced improvements indicated transfer improvement in WM tasks that were not part of the training programme and higher BOLD activity in task-relevant areas (Klingberg, 2010). In addition to this transfer, a recent study emphasized another transfer from neurocognitive training on WM to decreased delay discounting in a sample of adults in treatment for stimulant use (Bickel et al., 2011).

In the Stop-Signal task, Jones et al. (2011) primed either disinhibited behaviour (by emphasizing rapid responding at the expense of successful inhibition) or restrained behaviours (by emphasizing successful inhibition at the expense of speed). They found that subjects in the ‘disinhibition group’ increased their consumption of beer during the taste test compared with the ‘inhibited group’. Similar results have been found when comparing alcohol consumption during a taste phase after experimentally building an association between ‘go’ response and alcohol cues (by using an alcohol version of go/no-go task) compared with the association between ‘no-go’ response and alcohol cues (Houben et al., 2011a,b). These findings have led to the ideas that (a) ‘motor inhibition’ shares mechanisms with alcohol use, at least in social drinkers and (b) improving motor inhibitory control might result in decreasing alcohol use.

Undoubtedly, the avenue of research investigating positive transfer of WM and inhibition training on everyday behaviour should echo the need of more efficient intervention to prevent alcohol and gambling misuse in vulnerable adolescents.

CONCLUSION

Adolescence is associated with a variety of brain and cognitive changes altering the way individuals experience novel situations and peer relationship. According to their pre-adolescent existing capacities to refrain themselves by operating several forms of EFs (e.g. inhibition), this taste for uncertainty might dramatically increase the risk of drug and non-drug addictive behaviours. Research on decision-making has led to the idea that, despite similar perception of risk and explicit knowledge about its consequences, adolescents’ decisions are more biased by short-term consequences, which could cause mal-adaptation due to the occurrence of delayed negative consequences. During this developmental period, abnormal drinking patterns (e.g. binge drinking) could have a deleterious impact on EFs, thus increasing the risk of addiction in not enough self-refrained adolescents. In addition, repeated heavy alcohol use could enhance the salience of alcohol cues, thus making the engagement in alcohol use still more difficult to control (Robinson and Berridge, 2003).

Cognitive training procedures aiming at boosting supervisory systems are viewed as a key resource for prevention and treatment of alcohol and gambling abuse in adolescents. This goal will require better identification of individual cognitive components reflecting poor decision-making of abnormal pathways in adolescence.
Adolescence, alcohol and gambling


