

Impact of Alcohol Use on Inhibitory Control (and Vice Versa) During Adolescence and Young Adulthood: A Review

E. López-Caneda*, S. Rodríguez Holguín, F. Cadaveira, M. Corral and S. Doallo

Department of Clinical Psychology and Psychobiology, University of Santiago de Compostela, Galicia, Spain

*Corresponding author: Departamento de Psicología Clínica e Psicobiología, Facultade de Psicología, Campus Universitario Sur, E-15782 Santiago de Compostela, Galicia, Spain. Tel.: +34-8818-13915; Fax: +34-981528071; E-mail: eduardo.lopez@usc.es

Abstract — Aims: Adolescence is usually the time when individuals first drink alcohol and this has been associated with relatively weak or immature inhibitory control. This review examines the changes on brain development and inhibitory function that take place during adolescence and youth as well as the relationship between inhibitory control and alcohol use at this early age. **Methods:** Narrative review of the chief studies related to (a) the development of inhibitory control during adolescence, (b) the deficits in the inhibitory ability in alcohol use disorders and (c) the effects of acute alcohol intake and binge drinking on inhibitory control in adolescents and young adults. **Results:** Inhibitory control processes are developing during adolescence and youth. Poor inhibitory functions may predispose the individual to alcohol misuse. Likewise, acute and binge alcohol drinking may impair the inhibitory control and compromise the ability to prevent or stop behaviour related to alcohol use. **Conclusion:** Poor inhibitory control can be both the cause and the consequence of excessive alcohol use. Adolescence and young adulthood may be a particularly vulnerable period due to (a) the weak or immature inhibitory functioning typical of this stage may contribute to the inability of the individual to control alcohol use and (b) alcohol consumption *per se* may alter or interrupt the proper development of inhibitory control leading to a reduced ability to regulate alcohol intake. Further longitudinal research is needed to evaluate the interaction between inhibitory control dysfunction and alcohol use in both situations.

INTRODUCTION

Adolescence is a stage of life characterized by drug experimentation and engagement in health-risky behaviours (Spear, 2000; Dahl, 2004). In Western countries, alcohol is one of the most available and used drugs at this age (Anderson and Baumberg, 2006; Johnston *et al.*, 2009), and it currently constitutes a major public health concern (Eurobarometer, 2010; SAMHSA, 2011).

While several decades of research in adults have shown that chronic alcohol abuse is associated with major brain and cognitive impairments (e.g. Oscar-Berman and Marinkovic, 2007; Harper, 2009), the relationship between alcohol consumption and neurocognitive damage during adolescence and youth is still poorly investigated. Understanding the neurocognitive consequences of alcohol on the adolescent brain is crucial, since adolescence is a period of critical brain development and the time in which alcohol use is typically initiated.

Within the various cognitive processes affected by alcohol, the inhibitory control deserves particular consideration. Indeed, the ability to inhibit a response or action may prevent alcohol misuse, but deficits in such ability might, in turn, promote excessive alcohol consumption. Recent research indicates that acute alcohol intake, as well as heavy or binge drinking during adolescence and youth, may induce anomalies in behaviour (poor decision-making, altered impulse control, etc.) and brain functioning related to inhibition (Field *et al.*, 2007; Loeber and Duka, 2009; López-Caneda *et al.*, 2012). Likewise, an alteration in the inhibitory control may constitute a vulnerability factor for subsequent alcohol misuse and lead to an escalation or disordered regulation of alcohol intake (Norman *et al.*, 2011; Wetherill *et al.*, *in press*).

This review will focus on the definition of inhibitory control and the main experimental paradigms used to measure it, then on the core brain circuitry involved in response inhibition and its development across adolescence and early adulthood. Finally, the role of inhibitory processes as both a

determinant and a consequence of excessive alcohol use will be discussed. This review ends with some additional considerations about the relation of inhibitory control with impulsivity, as well as with other cognitive processes affected by alcohol, and with a brief discussion of the application of current knowledge to the prevention of alcohol abuse in youths.

INHIBITORY CONTROL

Inhibitory control is a core component of human behaviour. The importance of this executive function is highlighted by the broad range of psychiatric problems associated with inhibitory deficits, such as attention deficit hyperactivity disorder (Nigg, 2001), bipolar disorder (Frangou *et al.*, 2005), obsessive-compulsive disorder (OCD) (Penades *et al.*, 2007) and substance use disorder (Verdejo-García *et al.*, 2008). Although it is generally defined as the ability to withhold or suppress actions or thoughts that are inappropriate, inhibitory control is a heterogeneous construct which lacks of a simple operational definition, probably due to the multiple kinds of inhibitory processes underlying this executive function as well as the wide range of tasks used to measure it.

From an overall view, two different inhibitory functions can be distinguished: involuntary or *automatic inhibition* and voluntary or *effortful inhibition* (Nigg, 2000). The first one refers to the involuntary inhibition of attention that takes place to recently inspected locations or objects, which is traditionally known as inhibition of return (Klein, 2000). The effortful inhibition is frequently divided in *behavioural inhibition* and *interference control* (see Diamond, 2013, for a recent review).

Behavioural inhibition is the ability to suppress or stop responses that are ready to be emitted (prepotent responses) and it would comprise the motor inhibition measured by the Go/NoGo (GNG) and the stop-signal (SS) tasks (Logan, 1994). Interference control includes two other subtypes of

inhibition. The first one, *cognitive inhibition*, involves (a) the inhibition of thoughts and memories, i.e. the ability to suppress unwanted mental representations (Anderson and Levy, 2009)—usually measured by the Think/No-Think paradigm (Anderson and Green, 2001)—and (2) the inhibition of the tendency to choose smaller, immediate rewards in favour of delayed but larger rewards, also known as delayed gratification (Mischel *et al.*, 1989), which is frequently assessed by the delay discounting task (Bickel and Marsch, 2001). The second subtype of interference control refers to the inhibition of the processing of nonpertinent or irrelevant stimuli, which can be measured, for instance, by the Stroop or the Flanker tasks (Eriksen and Eriksen, 1974; MacLeod, 1991).

For the purpose of the present review, we will focus on behavioural inhibition, which has been the type of inhibitory control more investigated in studies on the relationship between alcohol and inhibitory processes. Throughout this article, when we refer to inhibitory control, we essentially refer to behavioural or response inhibition.

Given that response inhibition is typically measured by the GNG and SS paradigms, these tasks have been widely used to examine the effects of alcohol on inhibitory processes as well as the influence of inhibitory control ability on the regulation of alcohol intake. Both tasks involve rapid, repeated responses to targets, while also demanding suppression of those prepotent responses when faced with a Stop or No-Go stimulus. The main difference between them is that while the GNG task requires that individuals respond to one type of stimuli (Go) and withhold the response to the other (No-Go), the SS task demands that individuals inhibit a response that has been already initiated when a SS is presented (Fig. 1). The commission errors or false alarms (inappropriate responses to the No-Go stimulus), for the GNG task, and the time required to

stop a response once it has been initiated, for the SS task, provide the behavioural index of inhibitory control.

THE NEURAL CIRCUITRY OF RESPONSE INHIBITION

Using functional magnetic resonance imaging (fMRI), the neural substrates underlying these tasks, and therefore the inhibitory control processes, have begun to be identified. Studies in healthy population have consistently revealed a frontostriatal network involved in the inhibition of prepotent responses (Aron *et al.*, 2007; Chambers *et al.*, 2009). Within this network, the prefrontal cortex (PFC) and, particularly, the inferior frontal cortex (IFC) seem to be a critical region for successful inhibition (Konishi *et al.*, 1999; Aron *et al.*, 2004; Chikazoe *et al.*, 2009). This is also supported by neuropsychological studies, which have reported impairments in inhibitory control in subjects with IFC damage (Aron *et al.*, 2003).

Briefly, the IFC would be responsible for generating the No-Go signal which, passing through the subthalamic nucleus and the globus pallidus, would lead to the inhibition of the thalamus and, consequently, to the inhibition of motor responses in the primary motor cortex (Fig. 2) (Nambu *et al.*, 2002; Aron and Poldrack, 2006). This is clearly a very simplified model as there are other less direct inhibitory pathways (Duann *et al.*, 2009; Aron, 2011), as well as other regions such as anterior cingulate or parietal cortex (Durstun *et al.*, 2002; Watanabe *et al.*, 2002) that are involved in response inhibition.

On the other hand, neuroimaging studies have found abnormal IFC functioning linked to substance use (for reviews see Volkow and Fowler, 2000; Dom *et al.*, 2005; Feil *et al.*, 2010), including alcohol (Pfefferbaum *et al.*, 2001; Noël *et al.*, 2001;

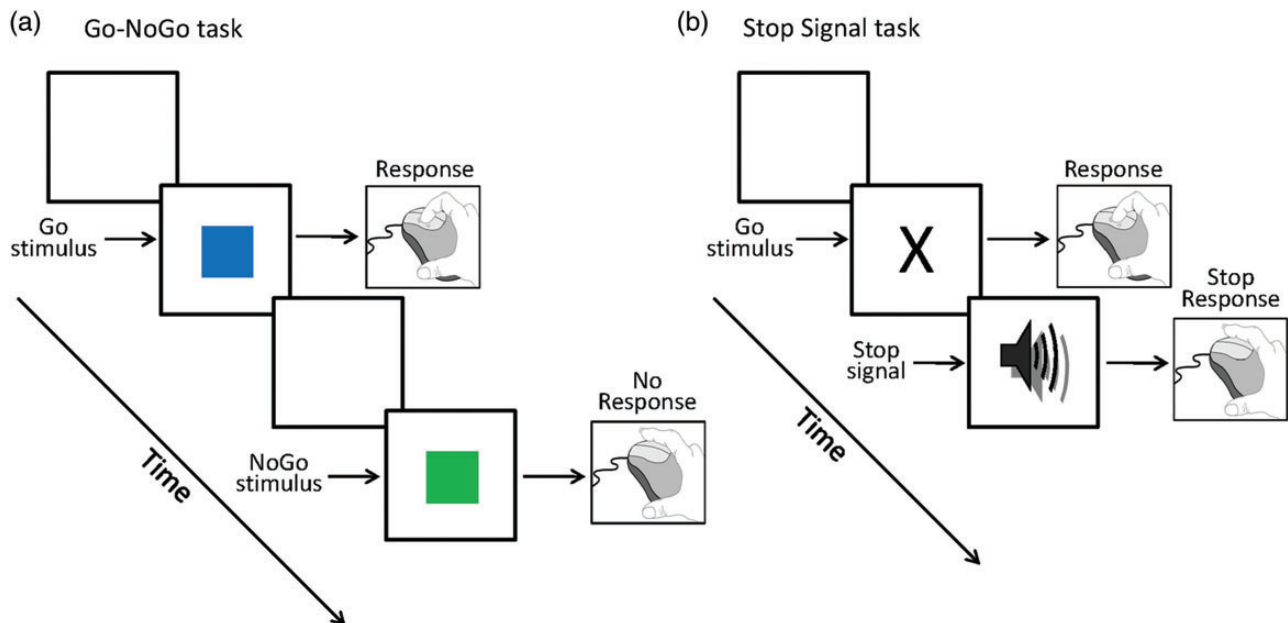


Fig. 1. Schematic of the Go/NoGo (GNG) and the stop-signal (SS) tasks. (a) In the GNG task, subjects are required to respond when a *go* stimulus (e.g. a blue square) is presented, and to inhibit or withhold their response when a *nogo* stimulus (e.g. a green square) is presented. (b) In the SS task, subjects have to respond as quickly as possible to the *go* stimuli (e.g. the X letter). During the stop condition, a stop signal (e.g. an auditory stimulus) is presented at a certain delay after the onset of the *go* stimulus and subjects must stop the already initiated motor response. (For interpretation of the references to color in this figure, the reader is referred to the web version of this article).

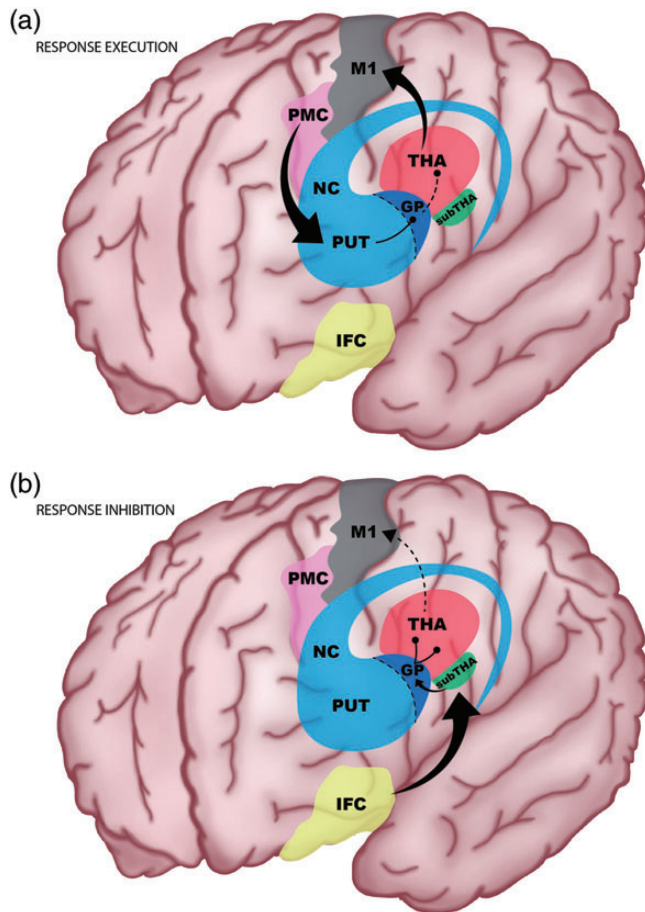


Fig. 2. Schematic representation of the neural circuitry involved in response execution and inhibition, according to Aron (2011). (a) When a motor action is initiated (e.g. moving the hand to press a button), the premotor cortex (PMC) activates the putamen (PUT), which in turn inhibits the internal segment of the globus pallidus (GP). This inhibitory projection leads to a disinhibition of the thalamus (THA), which leads to an increase in the impulses to the primary motor cortex (M1), thus resulting in response execution. (b) The inferior frontal cortex (IFC) sends a 'Stop' command through the subthalamic nucleus (SubTHA). This nucleus sends excitatory output to the GP, which results in the inhibition of large thalamic areas and hence in the inhibition of thalamocortical projections involved in hand movements, resulting in response inhibition. NC, nucleus caudatus.

Li *et al.*, 2009). Adolescence also appears to be associated with a particular IFC functioning which has been related to the maturational changes that take place during this period (see section below).

INHIBITORY CONTROL DEVELOPMENT DURING ADOLESCENCE

Basic cognitive processes are already well established in childhood. However, more complex cognitive functions, such as inhibitory control, undergo a substantial refinement during adolescence. Thus, although the ability to inhibit a response is already present in infancy and childhood (Diamond and Goldman-Rakic, 1989; Jones *et al.*, 2003), it is during adolescence when this ability becomes more efficient (Tamm *et al.*, 2002; Marsh *et al.*, 2006; Luna, 2009). A number of studies show that inhibitory control improves with age, as

demonstrated by the higher speed (reduction in reaction times) (Williams *et al.*, 1999; Band *et al.*, 2000), and better performance (lower commission error rates) (Casey and Trainor, 1997; Jonkman, 2006) in response inhibition across development.

This more efficient inhibitory control appears to be related to the anatomical and functional changes that take place in the PFC throughout adolescence and youth (Luna *et al.*, 2004). Important brain maturational changes such as myelination or synaptic pruning/reorganization continue well into late adolescence and early adulthood, being the PFC the last region to reach maturity (Giedd *et al.*, 1999; Gogtay *et al.*, 2004; Lenroot and Giedd, 2006). Both myelination and synaptic reorganization have been associated with an improvement in neural networks functioning as well as with increased neuronal and behavioural efficiency (Casey *et al.*, 2005; Spear, 2010).

Similarly, fMRI studies have shown that inhibitory function develops in association with changes in PFC activity. Although the relationship between behavioural performance in inhibitory tasks and greater or lesser fMRI activation is still controversial (Bunge and Wright, 2007; Luna *et al.*, 2010), several studies have reported greater IFC activity during inhibition in children compared with adolescents, and in adolescents compared with adults. This progressive reduction in prefrontal activation has been associated with better inhibitory control performance (Casey *et al.*, 1997; Somerville *et al.*, 2011). These findings support the notion that an immature brain (such as the adolescent brain) displays greater and less efficient prefrontal activation as well as poorer performance related to inhibition than a mature (and, therefore, adult) brain.

The adolescent brain, and particularly the PFC, appears to be especially sensitive to the harmful effects of alcohol as compared with the adult brain (Crews *et al.*, 2000; Spear, 2013), probably due to these maturational changes (Giedd *et al.*, 1999; Lebel and Beaulieu, 2011). This special sensitivity along with the relative developmental delay in inhibitory control could involve, on one hand, a greater propensity to excessive alcohol consumption (due to the reduced ability to prevent or stop behaviours related to alcohol use) and, on the other, a greater vulnerability of inhibitory mechanisms to the harmful effects of alcohol (due to the alteration or interruption in the normal development of inhibitory function).

POOR INHIBITORY CONTROL AND PROPENSITY TO ALCOHOL INTAKE DURING ADOLESCENCE

The immaturity of brain functioning underlying inhibitory control during adolescence appears to be linked to the peak onset of substance abuse observed through this period (Steinberg, 2008). Adolescence constitutes a stage of special risk for drug use initiation and the development of substance dependence (Rohde *et al.*, 2001; Hardin and Ernst, 2009), as well as for the emergence of psychiatric disorders related to disinhibitory behaviours such as conduct disorders or OCD (Zoccolillo, 1993; Pauls *et al.*, 1995).

Ineffective response inhibition may render individuals more vulnerable to develop addictive behaviours (Perry and Carroll, 2008). In this sense, Goldstein and Volkow (2002) proposed that drug addiction is a 'syndrome of impaired response inhibition', in which deficits in inhibitory control, along with an increased salience of drug-related stimuli (e.g. alcoholic drinks), would contribute to the inability to control the drug

use (Goldstein and Volkow, 2002; see also Jentsch and Taylor, 1999; Robinson and Berridge, 2003; Wiers *et al.*, 2007).

Consistent with this hypothesis, several studies have associated a weak inhibitory control with alcohol use during adolescence and youth. For example, Henges and Marczinski (2012) observed that failures to inhibit a response, as measured by the cued GNG task (Miller *et al.*, 1991), predicted the binge use of alcohol in young social drinkers. Other authors have reported that poor inhibitory control, as measured with the SS task, is associated with alcohol-use-related problems as well as with risk of alcohol dependence in adolescents (Nigg *et al.*, 2006; Rubio *et al.*, 2008).

Studies of offspring of alcoholics have shown a lower response in the inhibition performance in subjects with family history of alcohol use disorders (Nigg *et al.*, 2004; Schweinsburg *et al.*, 2004). Consistent with this, children and adolescents with a positive family history for alcohol use disorders also show anomalies in the anatomical and functional structure of some regions involved in inhibitory control (Schweinsburg *et al.*, 2004; Hill *et al.*, 2009; Heitzeg *et al.*, 2010). These anomalies might predispose the children to develop alcohol misuse during adolescence (Norman *et al.*, 2011). Accordingly, a recent longitudinal study conducted by Wetherill and Colleagues showed that adolescents who later became heavy drinkers displayed less activation of inhibitory circuitry during a GNG task than age-matched controls, which was indicative of neural vulnerabilities prior to the onset of alcohol use. After becoming heavy drinkers, adolescents showed more activation during response inhibition than controls, indicating that heavy drinking *per se* may lead to additional alterations in brain functioning related to inhibitory control (Wetherill *et al.*, in press).

Finally, weak inhibitory control has also been proposed as a general vulnerability factor for addictive behaviours, including alcohol use disorder (Goldstein and Volkow, 2002, 2011; Brewer and Potenza, 2008).

Although reduced inhibitory ability may play a major causal role in development of alcohol misuse or heavy drinking, conversely an impairment of the inhibitory control can be directly caused by heavy alcohol consumption. As described below, alcohol might compromise brain regions responsible for successful inhibition, thus reducing the ability to withhold a response. Therefore, not only a weak response inhibition may increase or encourage alcohol consumption, but also alcohol drinking may produce a weakening of inhibitory control, leading to a lower ability to stop alcohol consumption.

CONSEQUENCES OF ALCOHOL INTAKE ON INHIBITORY CONTROL

There are two main lines of research in the study of alcohol effects on inhibitory control: (a) the study of the acute effects of alcohol on response inhibition, where subjects execute different cognitive tasks or neuropsychological testing under the influence of certain doses of alcohol and (b) the study of the consequences of heavy or binge alcohol drinking on the inhibitory ability.

Acute effects of alcohol

Studies of the acute effects of alcohol support the hypothesis that alcohol disrupts (reduces) inhibitory control ability (Reynolds *et al.*, 2006; Ostling and Fillmore, 2010).

Specifically, adolescents and youths exposed to different doses of alcohol exhibit poor performance in a variety of response inhibition tasks. For instance, using SS and GNG tasks, several studies have demonstrated that moderate to high doses of alcohol (leading to blood alcohol contents ~0.06–0.09%) impair inhibitory control in young healthy subjects (Fillmore and Vogel-Sprott, 1999; Easdon and Vogel-Sprott, 2000; Marczinski and Fillmore, 2003; Rose and Duka, 2007, 2008; Loeber and Duka, 2009). Interestingly, it has been found that although moderate doses of alcohol impair the ability to suppress a response, they do not affect the ability to execute a response, which appears to be indicative of a specific disruption of the inhibitory mechanisms (Field *et al.*, 2010). Accordingly, the alcohol-seeking behaviour might remain intact, whereas the ability to inhibit this impulse and to control alcohol use might be compromised (Leeman *et al.*, 2012).

Another, particularly relevant study showed that impairments in inhibitory control after a moderate dose of alcohol are most pronounced in binge drinkers than in non-binge drinker subjects (Marczinski *et al.*, 2007). This study indicates that individuals who binge drink alcohol can be particularly sensitive to the acute alcohol effects on response inhibition, such that when they become intoxicated, they are less able to refrain from the impulse or desire to consume more alcohol, leading to further binge drinking (BD). This finding is consistent with a study conducted by Weafer and Fillmore (2008), who reported that greater impairment of inhibitory control from alcohol predicted increased *ad libitum* drinking in young social drinkers.

Research examining the neural correlates of acute effects of alcohol also suggests that moderate-to-high doses of alcohol induce abnormalities on brain functioning involved in inhibitory control in adults (Ridderinkhof *et al.*, 2002; Easdon *et al.*, 2005). However, to our knowledge, only one study has assessed the electrophysiological patterns of response inhibition during alcohol intoxication in young people (Euser and Franken, 2012). In this study, moderate doses of alcohol not only decreased performance in an emotional GNG task, but also altered the components of the event-related potentials (ERPs) related to inhibitory control (N2-NoGo and P3-NoGo) (see, e.g. Falkenstein *et al.*, 1999; Kok *et al.*, 2004, for information about these components). These results were interpreted as indicating that those youths under the effects of alcohol need to activate more cognitive resources during the inhibition process (Euser and Franken, 2012).

Despite the well-established view that alcohol impairs inhibitory control, to date only one fMRI study has assessed the effect of acute alcohol ingestion in young people during response inhibition (Schuckit *et al.*, 2012). In line with other studies that considered jointly young and adult participants (Anderson *et al.*, 2011; Nikolaou *et al.*, 2013), alcohol decreased activity of regions involved in inhibitory control (such as pre-frontal and cingulate regions) during a SS task in young people, specifically in those with a low response to alcohol and, therefore, with higher risk of problem drinking (Schuckit *et al.*, 2012). However, additional research in this field is needed to clarify the impact of acute alcohol consumption on brain functioning related to response inhibition at this young age.

Effects of heavy/binge alcohol drinking

BD or heavy episodic drinking, i.e. the consumption of large amounts of alcohol in a short time followed by periods of

abstinence (NIAAA, 2004; Courtney and Polich, 2009), has been related to neurocognitive impairments in adolescents and young people (e.g. Heffernan *et al.*, 2010; Squeglia *et al.*, 2012; López-Caneda *et al.*, 2013; Mota *et al.*, 2013; see also Hermens *et al.*, 2013; Jacobus and Tapert, 2013, for recent reviews). Studies about inhibitory control, although still rare, have reported that BD is associated with abnormalities in brain function and behavioural performance related to response inhibition. In this sense, neuropsychological studies have shown poor performance in several tasks assessing inhibitory processes in youths with a BD pattern (Townshend and Duka, 2005; Nederkoom *et al.*, 2009; Scaife and Duka, 2009). For instance, Townshend and Duka (2005) observed that young BD women had more difficulties to inhibit their response to alerting stimuli in a vigilance task than controls, which was interpreted as a sign of a deficit in the frontal inhibitory control. More recently, Nederkoom *et al.* (2009) reported an increased SS reaction time also in young BD women, indicating again a poor response inhibition in this population. Although these data are suggestive of a greater vulnerability in females to the neurotoxic effects of alcohol on inhibitory control, which is consistent with the stronger structural and functional impairments observed in women with an alcohol use disorder (Caldwell *et al.*, 2005; Medina *et al.*, 2008), additional research is required to test this hypothesis.

The three electrophysiological studies that to date have examined the effects of alcohol on inhibitory processes in young binge or heavy drinkers have shown anomalies both in the latency (Petit *et al.*, 2012) and the amplitude (López-Caneda *et al.*, 2012; Smith and Mattick, *in press*) of the NoGo-P3 component of the ERPs. In the study by Petit *et al.* (2012), heavy social drinkers showed delayed latencies of NoGo-P3 in an alcohol-related context, which was considered as an index of prioritizing processing related to alcohol that led to poorer inhibitory performance. In a recent follow-up study by our research group (López-Caneda *et al.*, 2012), a greater NoGo-P3 was observed in young binge drinkers, which was associated with a hyperactivation in the right IFC. These results were interpreted as indicative of the activation of additional neural resources to compensate emerging functional alterations in the regions engaged in response inhibition, which would allow binge drinkers to perform an efficient inhibitory control. Finally, the study conducted by Smith and Mattick (*in press*) showed longer SS reaction time in young female heavy drinkers than in female controls as well as larger P3 increase for successful compared with failed inhibition trials in female heavy drinkers. Following the authors, these results were indicative that females who regularly drink heavily needed longer time and greater cognitive effort to inhibit the response correctly.

On the other hand, to our knowledge, the only neuroimaging study examining the response inhibition in adolescent binge drinkers is the one conducted by Wetherill *et al.* (*in press*), which reported anomalies in the functioning of inhibitory circuitry before and after the onset of heavy alcohol use (see previous section). Another study in adolescents binge drinkers assessed the neural correlates of the Iowa Gambling Task (IGT), a decision-making task that can be considered a measure of cognitive inhibition (Verdejo-García *et al.*, 2008). In this study, Xiao *et al.* (2013) reported that adolescents with a BD pattern displayed a poor decision-making as well as a higher activity in the neural circuitry involved in emotional

and incentive-related behaviours (the amygdala and the insula). According to the authors, the hyperreactivity of this neural system could entail difficulties to inhibit the desire to consume alcohol.

Taken together, research on the effects of acute and binge alcohol drinking suggests that alcohol consumption might lead to a 'snowball effect' by which the acute effects of alcohol on inhibitory control would promote a continuous auto-administration of the substance which, in turn, would contribute to the deterioration of the inhibitory control system.

ADDITIONAL CONSIDERATIONS

Inhibitory control and impulsivity

Impulsivity is a psychological construct closely linked to inhibitory control. This term includes those behaviours that are risky, poorly planned, and that entail undesirable or negative consequences (e.g. Evenden, 1999; Mitchell, 2004). Within neuropsychology and cognitive neuroscience, impulsivity is often associated with *disinhibition*, and it is thought to arise from an impairment of inhibitory control (Enticott *et al.*, 2006; Lawrence *et al.*, 2009). Impulsivity, similar to inhibitory control, plays a major role in alcohol-related disorders, as is demonstrated by the fact that (a) it predicts early onset drinking age and development of heavy drinking and alcohol dependence in young adults (Ernst *et al.*, 2006); (b) different impulsivity dimensions are positively correlated with increased alcohol use and with alcohol-related problems (Dom *et al.*, 2006; Hittner and Swickert, 2006; Cyders *et al.*, 2008) and (c) alcohol-dependent subjects display high scores on impulsivity measures (Whiteside and Lynam, 2003; Mitchell *et al.*, 2005; Fox *et al.*, 2008). In the same way, several studies have suggested that excessive alcohol consumption in adolescents and youths is linked to the increased impulsivity during this period (Carlson *et al.*, 2010; Moreno *et al.*, 2012). A decline in this trait that usually takes place over the 18–25 age range has been related to decrease in alcohol use (Littlefield *et al.*, 2010).

Although impulsivity and inhibitory control are related, they can make unique contributions to alcohol use (Leeman *et al.*, 2012) and both constructs should be taken into account in studies examining the individual's ability to control alcohol use.

Alcohol also affects other related cognitive processes

Although this review is focused on the inhibitory control impairment induced by alcohol use, it is important to note that alcohol may also indirectly affect the inhibitory system. Other cognitive processes that interact with inhibitory control, such as working memory, are also affected by alcohol consumption. For instance, a study conducted by Finn *et al.* (1999) showed that young subjects with low working memory capacity were more susceptible to the effects of alcohol on impulsive behaviour, suggesting that alcohol reduced the ability of working memory to modulate response inhibition. Alcohol might thus affect inhibitory control via (a) weakening the inhibitory system or (b) decreasing working memory capacity (Vogel-Sprott *et al.*, 2001).

Alcohol, inhibition and gender

Another important moderator of the relationship between alcohol and inhibitory control is the gender. In this sense, it has been observed that while men display greater disruption of inhibitory control when receiving acute doses of alcohol than women (Fillmore and Weafer, 2004), the effects of frequent or binge alcohol drinking on response inhibition appear to be greater in females compared with males (Townshend and Duka, 2005; Nederkooft *et al.*, 2009). However, the neuro-cognitive results relating to gender and alcohol consumption in non-clinical populations are still scarce and inconsistent; further research is therefore needed.

Potential clinical implications

Given that alcohol misuse is associated with deterioration of inhibitory control skills, response inhibition training could theoretically improve inhibitory control and, consequently, lead to a decrease of alcohol intake (Houben *et al.*, 2011; Jones *et al.*, 2011). Houben *et al.* (2011) demonstrated, in a recent study, that young heavy drinkers trained to withhold a response to alcohol-related stimuli during a GNG task consumed significantly less alcohol in the week following the training. This finding, although needs to be replicated and validated for longer periods, suggests that the strengthening of response inhibition may be a useful intervention strategy for reducing alcohol use. It also underlines the importance of inhibitory control mechanisms on alcohol drinking behaviour as well as the usefulness of the early detection of response inhibition problems in alcohol use disorders prevention programmes.

CONCLUSIONS

Adolescence is a stage of life frequently associated with an early onset of alcohol use. It is also characterized by a weak inhibitory control due to the immaturity of the brain circuitry supporting this executive function. These reduced inhibitory skills consequently affect the ability to control the alcohol intake. Inhibitory control processes, in particular the behavioural inhibition, may equally be the cause and the consequence of excessive alcohol use. In fact, not only a weak response inhibition may lead to alcohol consumption, but drinking alcohol, in turn, may entail a weakening of the inhibitory control, leading to a lower ability to stop alcohol consumption. In this review, we have highlighted the main studies examining the relationship between inhibitory control and alcohol use in adolescents and young adults. Nevertheless, much further research is required to clarify how the excessive alcohol consumption may induce deficits in inhibitory control or how inhibitory control disruptions may constitute a vulnerability factor for alcohol misuse. The cross-sectional nature of most of the studies exploring neurocognitive functioning in young and adolescent binge drinkers makes it difficult to establish this relationship, so longitudinal studies are needed to evaluate the extent of the interaction between the inhibitory control dysfunction and alcohol use in both directions, as a vulnerability factor and as an effect of excessive drinking. Another major challenge would be to design prevention and treatment programmes that systematically integrate this growing body of knowledge.

Acknowledgements — The authors thank illustrator Carlos Rodríguez Brea for his help in the design of figures.

Funding — The study was supported by grants from the Spanish *Ministerio de Ciencia e Innovación* (PSI2011-22575) and the *Ministerio de Sanidad y Política Social, Plan Nacional sobre Drogas* (exp 2010/134). Eduardo López-Caneda was supported by the FPU program (AP2008-03433) of the Spanish *Ministerio de Educación*, and Sonia Doallo was supported by a postdoctoral contract from the Isidro Parga Pondal program (Xunta de Galicia, Spain).

Conflict of interest statement. None declared.

REFERENCES

- Anderson P, Baumberg B. (2006) *Alcohol in Europe*. London: Institute of Alcohol Studies.
- Anderson MC, Green C. (2001) Suppressing unwanted memories by executive control. *Nature* **410**:366–9.
- Anderson MC, Levy B. (2009) Suppressing unwanted memories. *Curr Dir Psychol Sci* **18**:189–94.
- Anderson BM, Stevens MC, Meda SA *et al.* (2011) Functional imaging of cognitive control during acute alcohol intoxication. *Alcohol Clin Exp Res* **35**:156–65.
- Aron AR. (2011) From reactive to proactive and selective control: developing a richer model for stopping inappropriate responses. *Biol Psychiatry* **69**:e55–68.
- Aron AR, Poldrack RA. (2006) Cortical and subcortical contributions to Stop signal response inhibition: role of the subthalamic nucleus. *J Neurosci* **26**:2424–33.
- Aron AR, Fletcher PC, Bullmore ET *et al.* (2003) Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nat Neurosci* **6**:115–6.
- Aron AR, Robbins TW, Poldrack RA. (2004) Inhibition and the right inferior frontal cortex. *Trends Cogn Sci* **8**:170–7.
- Aron AR, Behrens TE, Smith S *et al.* (2007) Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *J Neurosci* **27**:3743–52.
- Band GPH, van der Molen MW, Overtom CCE *et al.* (2000) The ability to activate and inhibit speeded responses: separate developmental trends. *J Exp Child Psychol* **75**:263–90.
- Bickel WK, Marsch LA. (2001) Toward a behavioral economic understanding of drug dependence: delay discounting processes. *Addiction* **96**:73–86.
- Brewer JA, Potenza MN. (2008) The neurobiology and genetics of impulse control disorders: relationships to drug addictions. *Biochem Pharmacol* **75**:63–75.
- Bunge SA, Wright SB. (2007) Neurodevelopmental changes in working memory and cognitive control. *Curr Opin Neurobiol* **17**:243–50.
- Caldwell LC, Schweinsburg AD, Nagel BJ *et al.* (2005) Gender and adolescent alcohol use disorders on BOLD (blood oxygen level dependent) response to spatial working memory. *Alcohol Alcohol* **40**:194–200.
- Carlson SR, Johnson SC, Jacobs PC. (2010) Disinhibited characteristics and binge drinking among university student drinkers. *Addict Behav* **35**:242–51.
- Casey BJ, Trainor R, Orendi JL *et al.* (1997) A pediatric functional MRI study of prefrontal activation during performance of a Go-No-Go task. *J Cogn Neurosci* **9**:835–47.
- Casey BJ, Galvan A, Hare TA. (2005) Changes in cerebral functional organization during cognitive development. *Curr Opin Neurobiol* **15**:239–44.
- Chambers CD, Garavan H, Bellgrove MA. (2009) Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neurosci Biobehav Rev* **33**:631–46.
- Chikazoe J, Jimura K, Asari T *et al.* (2009) Functional dissociation in right inferior frontal cortex during performance of go/no-go task. *Cereb Cortex* **19**:146–52.
- Courtney KE, Polich J. (2009) Binge drinking in young adults: data, definitions, and determinants. *Psychol Bull* **135**:142–56.
- Crews FT, Braun CJ, Hoplight B *et al.* (2000) Binge ethanol consumption causes differential brain damage in young adolescent rats compared with adult rats. *Alcohol Clin Exp Res* **24**:1712–23.

- Cyders MA, Flory K, Rainer S *et al.* (2008) The role of personality dispositions to risky behavior in predicting first-year college drinking. *Addiction* **104**:193–202.
- Dahl RE. (2004) Adolescent brain development: a period of vulnerabilities and opportunities. Keynote address. *Ann N Y Acad Sci* **1021**:1–22.
- Diamond A. (2013) Executive functions. *Annu Rev Psychol* **64**:135–68.
- Diamond A, Goldman-Rakic PS. (1989) Comparison of human infants and rhesus monkeys on Piaget's AB task: evidence for dependence on dorsolateral prefrontal cortex. *Exp Brain Res* **74**:24–40.
- Dom G, Sabbe B, Hulstijn W *et al.* (2005) Substance use disorders and the orbitofrontal cortex: systematic review of behavioural decision-making and neuroimaging studies. *Br J Psychiatry* **187**:209–20.
- Dom G, Hulstijn W, Sabbe B. (2006) Differences in impulsivity and sensation seeking between early- and late-onset alcoholics. *Addict Behav* **117**:2030–9.
- Duann JR, Ide JS, Luo X *et al.* (2009) Functional connectivity delineates distinct roles of the inferior frontal cortex and pre-supplementary motor area in stop signal inhibition. *J Neurosci* **29**:10171–9.
- Durston S, Thomas KM, Worden MS *et al.* (2002) The effect of preceding context on inhibition: an event-related fMRI study. *Neuroimage* **16**:449–53.
- Easdon CM, Vogel-Sprott M. (2000) Alcohol and behavioural control: impaired response inhibition and flexibility in social drinkers. *Exp Clin Psychopharmacol* **8**:387–94.
- Easdon CM, Izenberg A, Marmilio ML *et al.* (2005) Alcohol consumption impairs stimulus- and error-related processing during a Go/No-Go Task. *Brain Res Cogn Brain Res* **25**:873–83.
- Enticott PG, Ogloff JRP, Bradshaw JL. (2006) Associations between laboratory measures of executive inhibitory control and self-reported impulsivity. *Pers Individ Dif* **41**:285–94.
- Eriksen BA, Eriksen CW. (1974) Effects of noise letters upon the identification of a target letter in a nonsearch task. *Percept Psychophys* **16**:143–49.
- Ernst M, Luckenbaugh D, Moolchan E *et al.* (2006) Behavioral predictors of substance-use initiation in adolescence with and without attention deficit/hyperactivity disorder. *Pediatrics* **117**:2030–9.
- Eurobarometer S. (2010) *EU Citizens' Attitudes Towards Alcohol*. Brussels: European Commission.
- Euser AS, Franken IH. (2012) Alcohol affects the emotional modulation of cognitive control: an event-related brain potential study. *Psychopharmacology* **222**:459–76.
- Evenden J. (1999) Impulsivity: a discussion of clinical and experimental findings. *J Psychopharmacol* **13**:180–92.
- Falkenstein M, Hoormann J, Hohnsbein J. (1999) ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychol* **101**:267–91.
- Feil J, Sheppard D, Fitzgerald PB *et al.* (2010) Addiction, compulsive drug seeking, and the role of frontostriatal mechanisms in regulating inhibitory control. *Neurosci Biobehav Rev* **35**:248–75.
- Field M, Christiansen P, Cole J *et al.* (2007) Delay discounting and the alcohol Stroop in heavy drinking adolescents. *Addiction* **102**:579–86.
- Field M, Wiers RW, Christiansen P *et al.* (2010) Acute alcohol effects on inhibitory control and implicit cognition: implications for loss of control over drinking. *Alcohol Clin Exp Res* **34**:1346–52.
- Fillmore MT, Vogel-Sprott M. (1999) An alcohol model of impaired inhibitory control and its treatment in humans. *Exp Clin Psychopharmacol* **7**:49–55.
- Fillmore MT, Weafer J. (2004) Alcohol impairment of behavior in men and women. *Addiction* **99**:1237–46.
- Finn PR, Justus A, Mazas C *et al.* (1999) Working memory, executive processes and the effects of alcohol on Go/No-Go learning: testing a model of behavioral regulation and impulsivity. *Psychopharmacology* **146**:465–72.
- Fox HC, Hong KA, Sinha R. (2008) Difficulties in emotion regulation and impulse control in recently abstinent alcoholics compared with social drinkers. *Addict Behav* **33**:388–94.
- Frangou S, Haldane M, Roddy D *et al.* (2005) Evidence for deficit in tasks of ventral, but not dorsal, prefrontal executive function as an endophenotypic marker for bipolar disorder. *Biol Psychiatry* **58**:838–9.
- Giedd JN, Blumenthal J, Jeffries NO *et al.* (1999) Brain development during childhood and adolescence: a longitudinal MRI study. *Nat Neurosci* **2**:861–3.
- Gogtay N, Giedd JN, Lusk L *et al.* (2004) Dynamic mapping of human cortical development during childhood through early adulthood. *Proc Natl Acad Sci USA* **101**:8174–9.
- Goldstein RZ, Volkow ND. (2002) Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *Am J Psychiatry* **159**:1642–52.
- Goldstein RZ, Volkow ND. (2011) Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nat Rev Neurosci* **12**:652–69.
- Hardin MG, Ernst M. (2009) Functional brain imaging of development-related risk and vulnerability for substance use in adolescents. *J Addict Med* **3**:47–54.
- Harper C. (2009) The neuropathology of alcohol-related brain damage. *Alcohol Alcohol* **44**:136–40.
- Heffernan T, Clark R, Bartholomew J *et al.* (2010) Does binge drinking in teenagers affect their everyday prospective memory? *Drug Alcohol Depend* **109**:73–8.
- Heitzeg MM, Nigg JT, Yau WY *et al.* (2010) Striatal dysfunction marks preexisting risk and medial prefrontal dysfunction is related to problem drinking in children of alcoholics. *Biol Psychiatry* **68**:287–95.
- Henges AL, Marcinski CA. (2012) Impulsivity and alcohol consumption in young social drinkers. *Addict Behav* **37**:217–20.
- Hermens DF, Lagopoulos J, Tobias-Webb J *et al.* (2013) Pathways to alcohol-induced brain impairment in young people: a review. *Cortex* **49**:3–17.
- Hill SY, Wang S, Kostelnik B *et al.* (2009) Disruption of orbitofrontal cortex laterality in offspring from multiplex alcohol dependence families. *Biol Psychiatry* **65**:129–36.
- Hittner J, Swickert R. (2006) Sensation seeking and alcohol use: a meta-analytic review. *Addict Behav* **31**:1383–401.
- Houben K, Nederkoorn C, Wiers RW *et al.* (2011) Resisting temptation: decreasing alcohol-related affect and drinking behavior by training response inhibition. *Drug Alcohol Depend* **116**:132–6.
- Jacobus J, Tapert SF. (2013) Neurotoxic effects of alcohol in adolescence. *Annu Rev Clin Psychol* **9**:703–21.
- Jentsch JD, Taylor JR. (1999) Impulsivity resulting from frontostriatal dysfunction in drug abuse: implications for the control of behavior by reward-related stimuli. *Psychopharmacology* **146**:373–90.
- Johnston LD, O'Malley PM, Bachman JG *et al.* (2009) *Monitoring the Future National Results on Adolescent Drug Use: Overview of Key Findings, 2008*. Bethesda, MD: National Institute on Drug Abuse.
- Jones LB, Rothbart MK, Posner MI. (2003) Development of executive attention in preschool children. *Dev Sci* **6**:498–504.
- Jones A, Guerrieri R, Fernie G *et al.* (2011) The effects of priming restrained versus disinhibited behaviour on alcohol-seeking in social drinkers. *Drug Alcohol Depend* **113**:55–61.
- Jonkman LM. (2006) The development of preparation, conflict monitoring and inhibition from early childhood to young adulthood: a Go/Nogo ERP study. *Brain Res* **1097**:181–93.
- Klein RM. (2000) Inhibition of return. *Trends Cogn Sci* **4**:138–47.
- Kok A, Ramautar JR, De Ruiter MB *et al.* (2004) ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology* **41**:9–20.
- Konishi S, Nakajima K, Uchida I *et al.* (1999) Common inhibitory mechanism in human inferior prefrontal cortex revealed by event-related functional MRI. *Brain* **122**:981–91.
- Lawrence AJ, Luty J, Bogdan NA *et al.* (2009) Impulsivity and response inhibition in alcohol dependence and problem gambling. *Psychopharmacology* **207**:163–72.
- Lebel C, Beaulieu C. (2011) Longitudinal development of human brain wiring continues from childhood into adulthood. *J Neurosci* **31**:10937–47.
- Leeman RF, Patock-Peckham JA, Potenza MN. (2012) Impaired control over alcohol use: An under-addressed risk factor for problem drinking in young adults? *Exp Clin Psychopharmacol* **20**:92–106.

- Lenroot RK, Giedd JN. (2006) Brain development in children and adolescents: insights from anatomical magnetic resonance imaging. *Neurosci Biobehav Rev* **30**:718–29.
- Li CSR, Luo X, Yan P *et al.* (2009) Altered impulse control in alcohol dependence: neural measures of stop signal performance. *Alcohol Clin Exp Res* **33**:740–50.
- Littlefield AK, Sher KJ, Steinley D. (2010) Developmental trajectories of impulsivity and their association with alcohol use and related outcomes during emerging and young adulthood I. *Alcohol Clin Exp Res* **34**:1409–16.
- Loeber S, Duka T. (2009) Acute alcohol impairs conditioning of a behavioural reward-seeking response and inhibitory control processes—implications for addictive disorders. *Addiction* **104**:2013–22.
- Logan G. (1994) On the ability to inhibit thought and action: a users' guide to the stop signal paradigm. In Dagenbach D, Carr TH (eds), *Inhibitory Processes in Attention, Memory and Language*. San Diego, CA: Academic Press, 189–239.
- López-Caneda E, Cadaveira F, Crego A *et al.* (2012) Hyperactivation of right inferior frontal cortex in young binge drinkers during response inhibition: a follow-up study. *Addiction* **107**:1796–808.
- López-Caneda E, Cadaveira F, Crego A *et al.* (2013) Effects of a persistent binge drinking pattern of alcohol consumption in young people: A follow-up study using event-related potentials. *Alcohol Alcohol* **48**:464–71.
- Luna B. (2009) Developmental changes in cognitive control through adolescence. *Adv Child Dev Behav* **37**:233–78.
- Luna B, Garver KE, Urban TA *et al.* (2004) Maturation of cognitive processes from late childhood to adulthood. *Child Dev* **75**:1357–72.
- Luna B, Padmanabhan A, O'Hearn K. (2010) What has fMRI told us about the development of cognitive control through adolescence? *Brain Cogn* **72**:101–13.
- MacLeod CM. (1991) Half a century of research on the Stroop effect: an integrative review. *Psychol Bull* **109**:163–203.
- Marczinski CA, Fillmore MT. (2003) Dissociative antagonistic effects of caffeine on alcohol-induced impairment of behavioral control. *Exp Clin Psychopharmacol* **11**:228–36.
- Marczinski CA, Combs SW, Fillmore MT. (2007) Increased sensitivity to the disinhibiting effects of alcohol in binge drinkers. *Psychol Addict Behav* **21**:346–54.
- Marsh R, Zhu H, Schultz RT *et al.* (2006) A developmental fMRI study of self-regulatory control. *Hum Brain Mapp* **27**:848–63.
- Medina KL, McQueeney T, Nagel BJ *et al.* (2008) Prefrontal cortex volumes in adolescents with alcohol use disorders: unique gender effects. *Alcohol Clin Exp Res* **32**:386–94.
- Miller J, Schaffer R, Hackley SA. (1991) Effects of preliminary information in a go versus no-go task. *Acta Psychol* **76**:241–92.
- Mischel W, Shoda Y, Rodríguez ML. (1989) Delay of gratification in children. *Science* **244**:933–38.
- Mitchell SH. (2004) Measuring impulsivity and modeling its association with cigarette smoking. *Behav Cogn Neurosci Rev* **3**:261–75.
- Mitchell JM, Fields HL, D'Esposito M *et al.* (2005) Impulsive responding in alcoholics. *Alcohol Clin Exp Res* **29**:2158–69.
- Moreno M, Estevez AF, Zaldivar F *et al.* (2012) Impulsivity differences in recreational cannabis users and binge drinkers in a university population. *Drug Alcohol Depend* **124**:355–62.
- Mota N, Parada M, Crego A *et al.* (2013) Binge drinking trajectory and neuropsychological functioning among university students: A longitudinal study. *Drug Alcohol Depend* **1**:108–14.
- Nambu A, Tokuno H, Takada M. (2002) Functional significance of the corticostriatal-pallidal 'hyperdirect' pathway. *Neurosci Res* **43**:111–7.
- National Institute of Alcohol Abuse and Alcoholism. (2004) NIAAA council approves definition of binge drinking. NIAAA Newsletter, No. 3, p. 3.
- Nederkoorn C, Baltus M, Guerrieri R *et al.* (2009) Heavy drinking is associated with deficient response inhibition in women but not in men. *Pharmacol Biochem Behav* **93**:331–6.
- Nigg JT. (2000) On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and working inhibition taxonomy. *Psychol Bull* **126**:220–46.
- Nigg JT. (2001) Is ADHD an inhibitory disorder? *Psychol Bull* **127**:571–98.
- Nigg JT, Glass JM, Wong MM *et al.* (2004) Neuropsychological executive functioning in children at elevated risk for alcoholism: findings in early adolescence. *J Abnorm Psychol* **113**:302–14.
- Nigg JT, Wong MM, Martel MM *et al.* (2006) Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *J Am Acad Child Adolesc Psychiatry* **45**:468–75.
- Nikolaou K, Critchley H, Duka T. (2013) Alcohol affects neuronal substrates of response inhibition but not of perceptual processing of stimuli signalling a stop response. *PLoS One* **8**:e76649.
- Noël X, Paternot J, Van der Linden M *et al.* (2001) Correlation between inhibition, working memory and delimited frontal area blood flow measure by 99mTc-Bicisate SPECT in alcohol-dependent patients. *Alcohol Alcohol* **36**:556–63.
- Norman AL, Pulido C, Squeglia LM *et al.* (2011) Neural activation during inhibition predicts initiation of substance use in adolescence. *Drug Alcohol Depend* **119**:216–23.
- Oscar-Berman M, Marinkovic K. (2007) Alcohol: effects on neurobehavioral functions and the brain. *Neuropsychol Rev* **17**:239–57.
- Ostling EW, Fillmore MT. (2010) Tolerance to the impairing effects of alcohol on the inhibition and activation of behavior. *Psychopharmacology* **212**:465–73.
- Pauls DL, Alsobrook JP II, Phil M *et al.* (1995) A family study of obsessive-compulsive disorder. *Am J Psychiatry* **152**:76–84.
- Penades R, Catalan R, Rubia K *et al.* (2007) Impaired response inhibition in obsessive compulsive disorder. *Eur Psychiatry* **22**:404–10.
- Perry JL, Carroll ME. (2008) The role of impulsive behavior in drug abuse. *Psychopharmacology* **200**:1–26.
- Petit G, Kornreich C, Noël X *et al.* (2012) Alcohol-related context modulates performance of social drinkers in a visual Go/No-Go task: a preliminary assessment of event-related potentials. *PLoS One* **7**:e37466.
- Pfefferbaum A, Desmond JE, Galloway C *et al.* (2001) Reorganization of frontal systems used by alcoholics for spatial working memory: an fMRI study. *Neuroimage* **14**:7–20.
- Reynolds B, Richards JB, de Wit H. (2006) Acute-alcohol effects on the Experiential Discounting Task (EDT) and a question-based measure of delay discounting. *Pharmacol Biochem Behav* **83**:194–202.
- Ridderinkhof KR, de Vlugt Y, Bramlage A *et al.* (2002) Alcohol consumption impairs detection of performance errors in mediofrontal cortex. *Science* **298**:2209–11.
- Robinson TE, Berridge KC. (2003) Addiction. *Annu Rev Psychol* **54**:25–53.
- Rohde P, Lewinsohn PM, Kahler CW *et al.* (2001) Natural course of alcohol use disorders from adolescence to young adulthood. *J Am Acad Child Adolesc Psychiatry* **40**:83–90.
- Rose AK, Duka T. (2007) The influence of alcohol on basic motoric and cognitive disinhibition. *Alcohol Alcohol* **42**:544–51.
- Rose AK, Duka T. (2008) Effects of alcohol on inhibitory processes. *Behav Pharmacol* **19**:284–91.
- Rubio G, Jiménez M, Rodríguez-Jiménez R *et al.* (2008) The role of behavioral impulsivity in the development of alcohol dependence: a 4-year follow-up study. *Alcohol Clin Exp Res* **32**:681–7.
- Scaife JC, Duka T. (2009) Behavioural measures of frontal lobe function in a population of young social drinkers with binge drinking pattern. *Pharmacol Biochem Behav* **93**:354–62.
- Schuckit MA, Tapert S, Matthews SC *et al.* (2012) fMRI differences between subjects with low and high responses to alcohol during a stop signal task. *Alcohol Clin Exp Res* **36**:130–40.
- Schweinsburg AD, Paulus MP, Barlett VC *et al.* (2004) An FMRI study of response inhibition in youths with a family history of alcoholism. *Ann N Y Acad Sci* **1021**:391–4.
- Smith JL, Mattick RP. (in press) Evidence of deficits in behavioural inhibition and performance monitoring in young female heavy drinkers. *Drug Alcohol Depend*.
- Somerville LH, Hare T, Casey BJ. (2011) Frontostriatal maturation predicts cognitive control failure to appetitive cues in adolescents. *J Cogn Neurosci* **23**:2123–34.
- Spear L. (2000) The adolescent brain and age-related behavioral manifestations. *Neurosci Biobehav Rev* **24**:417–63.
- Spear L. (2010) The behavioral neuroscience of adolescence. WW Norton & Company.
- Spear L. (2013) The teenage brain. Adolescents and alcohol. *Curr Dir Psychol Sci* **22**:152–7.

- Squeglia LM, Pulido C, Wetherill RR *et al.* (2012) Brain response to working memory over three years of adolescence: influence of initiating heavy drinking. *J Stud Alcohol Drugs* **73**:749–60.
- Steinberg L. (2008) A social neuroscience perspective on adolescent risk-taking. *Dev Rev* **28**:78–106.
- Substance Abuse and Mental Health Services Administration (SAMHSA). (2011) *Results from the 2010 National Survey on Drug Use and Health: Summary of National Findings*. NSDUD Series H-41, HHS Publication No. (SMA) 11-4658. In: Office of Applied Studies. Rockville, MD.
- Tamm L, Menon V, Reiss AL. (2002) Maturation of brain function associated with response inhibition. *J Am Acad Child Adolesc Psychiatry* **41**:1231–8.
- Townshend JM, Duka T. (2005) Binge drinking, cognitive performance and mood in a population of young social drinkers. *Alcohol Clin Exp Res* **29**:317–25.
- Verdejo-García A, Lawrence AJ, Clark L. (2008) Impulsivity as a vulnerability marker for substance-use disorders: review of findings from high-risk research, problem gamblers and genetic association studies. *Neurosci Biobehav Rev* **32**:777–810.
- Vogel-Sprott M, Easdon C, Fillmore M *et al.* (2001) Alcohol and behavioral control: cognitive and neural mechanisms. *Alcohol Clin Exp Res* **25**:117–21.
- Volkow ND, Fowler JS. (2000) Addiction, a disease of compulsion and drive: involvement of the orbitofrontal cortex. *Cereb Cortex* **10**:318–25.
- Watanabe J, Sugiura M, Sato K *et al.* (2002) The human prefrontal and parietal association cortices are involved in NO-GO performances: an event-related fMRI study. *Neuroimage* **17**:1207–16.
- Weafer J, Fillmore MT. (2008) Individual differences in acute alcohol impairment of inhibitory control predict *ad libitum* alcohol consumption. *Psychopharmacology* **201**:315–24.
- Wetherill RR, Squeglia LM, Yang TT *et al.* (in press) A longitudinal examination of adolescent response inhibition: neural differences before and after the initiation of heavy drinking. *Psychopharmacology*.
- Whiteside SP, Lynam DR. (2003) Understanding the role of impulsivity and externalizing psychopathology in alcohol abuse: application of the UPPS impulsive behavior scale. *Exp Clin Psychopharmacol* **11**:210–7.
- Wiers RW, Bartholow BD, van den Wildenberg E *et al.* (2007) Automatic and controlled processes and the development of addictive behaviors in adolescents: a review and a model. *Pharmacol Biochem Behav* **86**:263–83.
- Williams BR, Ponesse JS, Schachar RJ *et al.* (1999) Development of inhibitory control across the life span. *Dev Psychol* **35**:205–13.
- Xiao L, Bechara A, Gong Q *et al.* (2013) Abnormal affective decision making revealed in adolescent binge drinkers using a functional magnetic resonance imaging study. *Psychol Addict Behav* **27**:443–54.
- Zoccolillo M. (1993) Gender and the development of conduct disorder. *Dev Psychopathol* **5**:65–78.