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GUEST EDITORIAL

Binge drinking and the differential influence of ethanol on cognitive control subprocesses: a novel field of neurotoxicology

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Alcohol is a common drug of abuse and the adverse effects of chronic overconsumption are well known (e.g., Fadda and Rossetti 1998). However, drinking patterns and habits have started to change drastically, especially among younger people in Western cultures (Jernigan 2001; Montgomery et al. 2012). In this context, the term "binge drinking" describes a drinking pattern that is characterized by the consumption of alcoholic beverages with the primary intention of reaching a marked intoxication (as characterized by a blood alcohol concentration of at least 0.8 ‰). For adult males, it takes at least five standard drinks, and for adult females, it takes at least four drinks to achieve this level of intoxication (Crabbe et al. 2011). The most striking differences between binge drinking and "traditional" alcoholism are the irregularity of alcohol intake and the young age of consumers (Hermens et al. 2013). Since the prefrontal cortex (PFC) is subject to development and maturation during adolescence and emerging adulthood, binge drinking until the age of 24 probably poses a bigger harm to proper executive functioning than it does in later stages of life (Hermens et al. 2013).

To better understand the effects of binge drinking, one must take a closer look at the acute neurobiological changes it induces. During acute alcohol intoxication, several neurotransmitter systems are subject to changes. While glutamatergic neurotransmission is downregulated, signaling via GABA, dopamine, serotonin, and opioid peptides becomes enhanced (Clapp et al. 2008). Even though this list of changes is not comprehensive, it suggests two main mechanisms via which alcohol intoxication can alter behavior and cognition: Globally speaking, one would expect an unspecific dysfunction of higher-order cognition due to the reduction in a mainly excitatory transmitter (glutamate) and the boosting of a mainly inhibitory transmitter (GABA). More specifically, one would expect changes in executive functions. Executive functions that can be altered by alcohol comprise response inhibition, task selection, task switching, and response monitoring (Goldstein and Volkow 2011). There are many studies and reviews substantiating a decline of executive functioning in long-term alcohol abuse (e.g., Oscar-Berman 2012), and more recent studies have started to demonstrate similar effects in frequent binge drinkers, although to a lesser extent and with more mixed results (see Montgomery et al. 2012). Yet, there is a marked scarcity of studies investigating the acute effects of a heavy (binge-like) intoxication. The shortage of such studies poses a problem when it comes to logically linking acute to long-term consequences of increased alcohol consumption in the form of binge drinking.

Hence, very little is known about whether behavioral (executive) deficits observed during a pronounced intoxication are the result of a global and unspecific "dampening" of cognitive functions or rather the consequence of deficits in a distinct subset of cognitive subprocesses contributing to behavior. Executive functions are mediated via loops interconnecting the PFC and the basal ganglia (which have been shown to heavily depend the proper functioning of GABAergic and dopaminergic signaling; see Plenz 2003; Beste et al. 2009). The high complexity of these fronto-striatal loops, the fact that different cognitive functions rely on the integrity of different transmitter systems, and the finding that cognitive subprocesses can be differentiated using experimental psychological and neurophysiological

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techniques (e.g., Beste et al. 2010a, b, 2011; Barnes et al. 2011) would, however, suggest specific changes.

In order to start answering these questions, we recently investigated the acute effects of binge-like ethanol intoxication on task switching in young healthy humans. We were able to demonstrate specific changes in both behavioral and neurophysiologic measures (Stock et al. 2013), underpinning the assumption that not all cognitive functions and (sub)processes are equally altered by ethanol intoxication. This finding raises the important question of whether long-term abuse produces deficits in the same cognitive subprocesses as acute intoxication. Closing this knowledge gap would greatly promote our understanding of how the deficits associated with long-term alcohol abuse and/or long-term binge drinking develop and how they are maintained (compare Hermens et al. 2013). Therefore, a major future challenge in this area of research is to investigate the development of alcohol-related deficits via elaborated comparisons of the behavioral and cognitive changes observed in acute intoxication (in healthy individuals), acute withdrawal, regular binge drinking, and alcohol abuse. In this context, it is also of interest to see whether cognitive subprocesses differ in the fashion of a dose–effect relationship: Does their deterioration linearly follow increases in blood alcohol concentrations, or do they decouple from such a relationship (depending on the neurobiological systems mediating these cognitive subprocesses)?

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