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Alcoholism: An Impulsive/Disinhibition Disorder?

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1. Introduction

In a broad sense, response inhibition represents as a useful concept to investigate impulsivity, a term referring to “behavior that is performed with little or inadequate forethought” (Evenden, 1999). Impulsivity has been studied for many decades as a trait variable of human personality that is stable within an individual and varies normatively across the healthy population (Barratt, 1995). Following the development of neuropsychology and cognitive neuroscience, impulsivity is often replaced with “disinhibition”, a term referring to the idea that top-down control mechanisms ordinarily suppress automatic or reward-driven responses that are not appropriate to the current demands (Aron, 2007).

Such a definition gives weight to the idea that alcoholism and other addictive behaviors might be the consequence of increased impulsivity, that is to say, when top-down mechanisms necessary to suppress actions, emotions and thoughts related to alcohol use are disrupted (e.g., Verdejo-Garcia et al., 2008; de Wit, 2008; Jentsch & Taylor, 1999). Throughout the present chapter, I discuss the relevance of impulsivity/disinhibition concept in order to investigate both risk factors to become alcoholics (as a trait) and acquired component of the development of alcoholism (as a state). Numerous reasons may lead to use alcohol recreationally including peer influence, personality characteristics, alcohol availability, which together tell something about how much of alcohol will be consumed. But once dependent, alcoholics persist in alcohol-taking despite awareness that their alcohol use is directly harmful to their health, their finances and their interpersonal relationships (American Psychiatric Association, 2000). Frequent unsuccessful attempts to quit drinking are a classic and this relapse phenomenon could also have something to do with deficient inhibitory control over a response that provides immediate positive consequences.

From the information-processing perspective, cognitive factors are seen as mediators involved in the development of alcoholism (e.g., Finn, 2002; Tiffany, 1990) as well as relapse (e.g., Noël et al., 2002; Bowden-Jones et al., 2005). An emerging view considers impairment of response inhibition as contributing significantly to the development of alcoholism (e.g., Lyvers, 2000) and to a variety of cognitive impairments (e.g., planning, mental flexibility)

(e.g., Noël et al., 2001). The concept of response inhibition refers to the ability to suppress responses (i.e. action, thoughts, emotions) that are inappropriate, unsafe, or no longer required, which supports flexible and goal-directed behavior in ever-changing environments (e.g., Miyake et al., 2000; Stuphorn & Schall, 2006). In everyday life, there are many examples of the importance of response inhibition, such as stopping yourself from crossing a street when a car comes around the corner without noticing you. This idea has been documented by poor performance on a variety of cognitive tasks assessing dominant response inhibition in abstinent alcoholics (e.g., Noël et al., 2001) and in children of alcoholics (e.g., Habeych et al., 2006) as well as by abnormal brain electrophysiology (e.g., Kamarajan et al., 2006) and brain metabolism (e.g., Scheinsburg et al., 2004) while performing response inhibition tasks. In addition, poor response inhibition has been demonstrated to be a predictor of problem drinking in adolescents at risk for alcoholism (e.g., Nigg et al., 2006) and maintenance of abstinence after alcohol detoxification treatment (Noël et al., 2002).

Inhibition plays a central role in theorizing about human cognition and is often regarded as a key component of executive control (e.g., Miyake et al., 2000; Nigg, 2000; Baddeley, 1996). However, inhibition may represent a family of functions rather than a single, unitary construct (Friedman & Miyake, 2004; Nigg, 2000). Indeed, according to Friedman & Miyake (2004), a distinction should be made between the inhibition of a prepotent response that implies to deliberately suppress dominant/automatic responses and the resistance to proactive interference defined as the capacity to resist to memory intrusion of information no longer relevant. In the same vein, Nigg (2000) has suggested that response inhibition may range between intentional/effortful and unintentional/automatic response inhibition. In other terms, intentional/effortful inhibition would occur on mental representations loaded in working memory whereas unintentional/automatic would prevent the intrusion of mental representations irrelevant with the current situation. In addition, within effortful inhibition, a distinction has been made between the suppression of prepotent/automatic response and the suppression of no longer relevant information loaded in working memory. In addition, choice impulsivity, as reflected by rapid temporal discounting may represent a separate impulsivity component (e.g., Verdejo-Garcia et al., 2008; de Wit, 2008).

A very important question raised by research on impulsivity/disinhibition concerns the source of these deficiencies observed in these individuals in trouble with their alcohol use. One possibility is that the repeated use of alcohol may cause a gradual attrition of behavioral self-control, plausibly mediated by structural changes in the prefrontal cortex (e.g., Bechara 2003; Goldstein & Volkow, 2002). An alternative explanation is that deficient inhibitory control may be present prior to alcohol initiation, thus acting as a predisposing factor. This vulnerability pathway has been increasingly recognized by neuroscientific models. Indeed, adolescents' brain is relatively immature on these systems responsible for reward processing, motivation and regulation of these responses (e.g., inhibition). The reasons of these individual differences in term of brain maturation are beyond the scope of this paper, but the developmental pathway of brain maturation and its failures are fascinating topics. For instance, by using a cocaine self-administration procedure, Belin and colleagues (2008) found that, in rodents, high impulsivity predicts the switch to compulsive cocaine-taking. It is likely that vulnerability and attrition ways are not mutually exclusive; poor inhibitory control prior to the onset alcohol use may lead to increase the risk to become

an addict, the excessive use of alcohol (e.g., repeated binge drinking episodes) may in turn accentuate this premorbid inhibition weakness. It is also possible that deleterious effects of alcohol are more pronounced in these individuals with poor inhibitory control.

2. Alcoholism: An impulsive/disinhibition disorder?

2.1 Impulsivity measured by self-report questionnaires

Cognitive-motivational theory of personality vulnerability to alcoholism describes impulsivity/novelty seeking and sensation/excitement seeking as fundamental personality dimensions that are distinguished in terms of the motivation, emotional, and cognitive processes that mediate or moderate vulnerability to alcohol use disorders (for a review, see Finn, 2002). For instance, a substantial body of research emphasised that exaggerated levels of novelty-seeking, which is highly correlated with impulsivity and aggressivity (e.g., Finn et al., 2002) and of excitement-seeking mediate alcohol use disorders (for reviews, see Mulder, 2002; Finn, 2002). In young alcoholics, high levels of disinhibited and appetitive personality traits, such as impulsivity, boredom susceptibility, thrill and adventure seeking, excitement-seeking (Finn et al., 2002; von Knorring et al., 1985); novelty-seeking (Finn et al., 2002); and aggressiveness (Babor et al., 1992; von Knorring et al., 1987) were reported. High impulsivity sub-scale of novelty seeking, which reflects poor control of appetitive and aggressive impulses, difficulties delaying gratification, acting without thinking and increased activity and assessed at 3 years of age predict the development of alcohol abuse in early adulthood (Kirisici et al., 2007).

Sensation seeking, as defined as a strong need for varied, novel, and stimulation experiences, and willingness to take risks for the sake of such experiences (Zuckerman, 1979), is another of the personality traits associated with high levels of alcohol and drug use (Andrucci et al., 1989).

Although useful, the questionnaire-based methodology introduces a number of caveats in the context of alcoholic population. The most meaningful may be that impulsivity itself could directly interfere with the completion of the questionnaires themselves, such that an impulsive subject may give less consideration to responses than a non-impulsive subject, the former having possibly less insight capacities than the latter. In addition, self-report questionnaires are susceptible to be highly dependent to social desirability that may naturally differ between alcoholic inpatients and healthy participants.

For all these reasons, direct measurement of inhibitory control processes using laboratory tasks has considerably developed over the past decade.

2.2 Impulsivity measured by laboratory tasks

2.2.1 Chronic effects of alcohol on response inhibition

One of first elements of impulsivity is acting without thinking, which may be operationalized as poor behavior inhibition in a variety of rapid stimulus-discrimination tasks. In this category of tasks, participants are required to respond to target stimuli and not to non-target ones. For instance, on a task consisting to click a button when presented with a five-digit number he or she thought was identical to the preceding number, alcohol-

dependent patients made more commission errors, thus indicating that these participants are more impulsive than controls (Bjork et al., 2004). In line with this idea, patients also had faster response times to target, which were inversely correlated with error rates across all subjects, which seemed to point to a 'fast-guess' mechanism of impulsive response. On a go/no-go task for which participants had to respond to a target by pressing a button as quickly as possible (go trials) and withholding their response when a non-target displayed (no-go trials), authors found that (1) the statistical difference between the No-Go and Go conditions was more robust in controls than in alcoholics; (2) relatively less anteriorization of current source density polarity in alcoholics during No-go processing indicating an impaired/decreased frontal lobe contribution. Interestingly, in comparison with patients with late onset of problem drinking and no problem-drinking parent, those alcoholics with earlier age of problem drinking and who reported a problem-drinking father (type 2-like alcohol dependence according to Cloningers' typology) demonstrated faster response latencies and more responses to non-target stimuli in the prepotent motor-response task. Regarding the relationship between these impulsivity measures and clinical indices of alcoholism, the age of onset of alcohol use/heavy drinking and measures of alcohol severity (for a review of the question, see Verdejo-Garia et al., 2008).

A deficit of inhibitory control has been identified consistently as a feature of dependence to alcohol. For instance, in a previous study (Noël et al., 2001), the re-examination of the 'frontal lobe vulnerability' hypothesis of alcoholism with tasks designed to assess separately non-executive and specific executive operations (which proved to be sensitive to frontal dysfunction) highlighted impaired intentional inhibition in recently detoxified alcoholics. The theoretical framework on which this study was based is the control to action model developed by Norman and Shallice (2000) in which two control to action mechanisms are distinguished. The first, contention scheduling, is involved in routine situations in which actions are triggered automatically. The second, the Supervisory Attentional System, (SAS) is needed in situations where the routine selection of action is unsatisfactory, and they conceived it as carrying out a variety of processes allowing the genesis of plans and willed actions. Reflecting this two control to action processes model, the Hayling task assesses the capacity to both activate a habitual response and to suppress (inhibit) this response (Burgess & Shallice, 1996). The test consisted of two sections (A and B) of 15 sentences each read aloud by the experimenter, in which the last word was missing. In section A (initiation/automatic) subjects were asked to give the word that made sense, which contrasts to the section B (inhibition), in which participants were asked to give a word that made no sense at all in the context of the sentence. On this task, non-amnesic alcoholics were as fast and accurate as their controls to produce the expected words but slower and less accurate when the expected word was to be suppressed. As suggested by a PET study (Collette et al., 2001), bilateral median frontal activation occurs during section B of the Hayling test, thus suggesting that alcoholics' inhibition deficits might be due to frontal lobe abnormalities.

Other results of this study were consistent with the existence of an inhibition deficit. In the Trail-Making test, alcoholics were slower than controls on the section B but not on the section A. Similarly, they showed poor performance in the alternate fluency task. Finally, patients spent more time to complete the flexibility condition of the Stroop test. The trail B requires inhibiting current realization strategy (1, 2, 3...) to switch between numbers and

letters (1A, 2B, 3C...). Performance in the alternate fluency task requires, notably, that subjects inhibit one search strategy to switch to another. In the Stroop test, the flexibility condition requires the subject to switch between two rules alternatively, that is to inhibit the current rule.

On go/no-go paradigms, alcoholics made more commission errors, thus indicating that they are less efficient suppressing the most common motor action to press key in presence of a target (Bjork et al., 2004; Kamarajan et al., 2005). On the stop signal task, Goudriaan and colleagues (2006) found increased stop signal reaction time in alcoholic patients, which indicates weaker inhibition efficiency.

In contrast to the inhibition of prepotent response for which responses to be inhibited are strongly automatic, the suppression of no longer relevant mental contents (cognitive inhibition) is appropriately assessed by the directed-forgetting procedure. In this procedure (Andrés et al., 2004), memory performance of letter trigrams in three conditions is compared: presented alone (*single-item* condition); followed by a second trigram to be recalled (*double-item* condition); followed by a second trigram to be forgotten (*directed-forgetting* condition). In addition, participants are instructed to perform a distracter task, thus requiring simultaneous maintenance and processing of information. Therefore, low performance in directed-forgetting would reflect impaired ability to inhibit a mental content held in working memory. In alcoholism, this type of inhibition could be of great importance; difficulty suppressing repetitive thoughts about drinking and drinking expectations might represent the core of a craving episode for alcohol (May et al., 2004). In a recent article (Noël et al., 2009), we examined 3-4 weeks abstinent alcoholic's ability to inhibit irrelevant information in working memory by the mean of a directed-forgetting procedure. Results showed that despite similar performances between groups in the *double-item* (interference) condition of the task, alcoholic participants did not improved their performance in the *directed-forgetting* condition relatively to the *double-item* condition, whereas control participants did. In addition, we also highlighted that alcoholics were more sensitive to intrusion errors in the *directed-forgetting* condition. Finally, we found that the inhibition score (measured by the difference in recall performance between the single-trigram and directed-forgetting conditions) was positively correlated with the duration of alcoholism.

These findings are interesting because they complete previous works showing prepotent response inhibition (e.g., Noël et al., 2001; Gaudriaan et al., 2005). Indeed, abstinent alcoholics exhibited poor performance on a variety of dominant response inhibition tasks (e.g., Stop Signal task, Goudriaan et al., 2006; Hayling task, Noël et al., 2001). As shown by Friedman and Miyake (2004) in the first study attempting to empirically evaluate proposed taxonomy of inhibition-related functions, inhibition of dominant response and inhibition of proactive interference may be considered as distinct processes. Therefore, alcoholism would be associated with deficit on those two types of inhibition. However, each of inhibition deficits could be involved in separate aspects of the development and the maintenance of alcoholism. For instance, in Obsessive Compulsive Disorder (OCD), compulsions symptoms may be related with failures in behavioral inhibitory processes leading to repetitive stereotyped behaviours (e.g., ritualistic checking behaviour) whereas obsessions may be related with failures in cognitive inhibitory processes resulting in frequent intrusive thoughts and ideas entering into consciousness (e.g., mental rituals) (Chamberlain et al., 2005). In the same vein, we hypothesized that prepotent response inhibition could prevent

alcoholics in resisting to automatically triggered alcohol-related behaviours (i.e., to take a drink) (e.g., Goldstein & Volkow, 2002; Whiteside & Lynam, 2003) whereas inhibition of proactive interference deficit observed on the directed-forgetting procedure in our experiment may lead to enhance occurrence of irrelevant and/or intrusive alcohol-related thoughts. In turn, when alcohol-related representation break through into awareness (being loaded into working memory) and experienced as a craving episode for alcohol (e.g., May et al., 2004), alcoholics would also be in trouble to suppress them and resist drinking because of an impairment to inhibit dominant response. It is obvious that this model remains largely speculative and that further investigations are needed to investigate the relationship between clinical phenomena characterizing alcoholism and different types of cognitive inhibition.

In a recent research (Noël et al., unpublished data), we aimed to reexamine the disinhibitory hypothesis of alcoholism in light of the model proposed by Friedman and Miyake (2004) and this in using several response inhibition tasks tapping into both the automatic suppression of proactive interference and the intentional inhibition of dominant response. One proactive interference inhibition task was Brown-Peterson variant in which participants had to learn four lists of eight words each. The first three lists were taken from the same semantic category, thus generating proactive interference to-be-inhibited for better performance. As an example of intentional prepotent response inhibition task, the antisaccade task (adapter from Roberts et al., 1994) assesses the capacity to minimize the reflexive response (proactive saccade) of looking at the initial cue. Our main finding was that, compared to non-alcoholics, patients had poor performance on cognitive tasks requiring the inhibition of prepotent response. In contrast, alcoholics performed normally on tasks exploring the resistance (inhibition) to proactive interference. The second major finding was that we found a relationship between inhibition of dominant response and alcoholics' greater tendency to act impulsively in particular when facing with their negative feelings.

An intriguing and important question remaining to be clarified is the relationship between enhanced attention for alcohol cues (cognitive bias) and impaired prepotent response inhibition (cognitive deficit). Studies having used the alcohol Stroop task did not report difference between light and heavy drinkers (Sharma et al., 2001) and between alcoholics and healthy participants (Lusher et al., 2004) in terms of the number of errors made when words are related to alcohol. One reason for the absence of cognitive disinhibition in the alcohol Stroop task is that both problematic users of alcohol and healthy participants made very few errors, thus reflecting a ceiling effect. Another limitation of the Stroop task is the questionable nature of inhibitory; whereas the Stroop task has generally been considered as examining resistance to interference (Nigg, 2000), it might also be viewed as taxing mechanisms of inhibitory control, i.e., the suppression of pre-potent responses (i.e., to read the alcohol related words rather than the color). In order to overcome these limitations, we designed an alcohol version of a go/no-go paradigm (the Alcohol Shifting task), which examines distinctly motor response inhibition, shifting of attention and the influence of alcohol-related stimuli's processing on these functions (Noël et al., 2005). We hypothesized that alcoholic subjects exhibit impairments in tasks requiring inhibitory control, as well as shifting. The aim was to test the ability of alcoholics to discriminate between alcohol-related and neutral words. Sometimes, the alcohol-related words were the targets for the "go"

response, with neutral words as distracters, sometimes the reverse. Several shifts in the type of the target occurred during the task. More precisely, in our go/ no-go task, words are briefly displayed, one by one, in the center of the screen. Half of the words are targets and half are distracters. Subjects are instructed to respond to targets by pressing the space bar as quickly as possible, but not respond to distracters. Words are presented for 500ms, with an inter-stimulus interval of 900 ms. A 500 ms/450 Hz tone sounds for each false alarm (i.e., a response to a distracter), but not for omissions (i.e., failures to respond to a target). The task comprises two practice blocks followed by eight test blocks of 18 stimuli each composed of nine 'neutral' (N) and nine 'alcohol related' words (A). In each block, either neutral or alcohol related words are specified as targets, with targets for the 10 blocks presented either in the order NNAANNAANN or AANNAANNAA. Due to this arrangement, four test blocks are 'non-shift' blocks, where subjects must continue responding to stimuli in the same way. Four test blocks, however, are 'shift' blocks, where subjects must begin responding to stimuli, which had been distracters, and cease responding to stimuli, which had been targets. These results demonstrate that alcoholics exhibit a basic prepotent response inhibition deficit accentuated when the response to be suppressed is related to alcohol (Noël et al., 2007). Increased impulsivity by alcohol cues observed at the end of a detoxification treatment in ALC might have some clinical implications. Indeed, alcohol-drinking practice in individuals suffering from alcoholism can be viewed as encompassing stimulus-driven automatic behaviors (e.g., Tiffany, 1990). Besides, the intensity of the alcohol-related response may be stronger because of the behavioral sensitization phenomenon described by Robinson and Berridge (2003). In these circumstances, moderating or stopping alcohol drinking might require the inhibition of a prepotent response. The present findings show that the response inhibition deficit seen in ALC is more pronounced when a response associated with alcohol-related stimuli is to be suppressed. Thus, psychopharmacological and psychological strategies consisting to improve the prepotent response inhibition capacities would be fruitful for attenuating the severity of alcoholism and to prevent alcohol relapse.

2.2.2 Sensitivity to delay discounting

On a delay discounting task, subjects are given choices between a small, sooner reward and a larger, delay reward. Traditionally, the outcomes of a series of such choices are used to estimate the present subjective value of a delayed reward as a function of delay time, yielding hyperbolic temporal discount curves (Mazur, 1987; Rachlin, 2000). On this task, the temporal discounting functions are significantly different between groups with alcoholics demonstrating steeper discounting curves (Mitchell et al., 2005). This tendency to discount delayed rewards was positively correlated with subjective reports of both alcohol addiction severity and impulsivity (as assessed by the Barratt Impulsivity Scale). Interestingly, in the same study, alcoholic patients did not differ on motor impulsivity, which means that their inabilities to delay gratification and to inhibit a prepotent response are dissociated. In a study comparing early-onset alcoholics (EOAs) and late-onset alcoholics (LOAs) on sensitivity to delay discounting task (Dom et al., 2006), EOAs had higher discount rates than both the non-substance-abusing subjects and the LOAs, with these two groups performing similarly. This differentiation between EOAs and LOAs in terms of impulsive decision making emphasized the heterogeneity of individuals with alcoholism on the one hand and the existence of distinct pathways leading to alcoholism on the other. Both research and

treatment programs should take into account the existence and differences observed in the two alcoholism subtypes.

2.3 Acute effect of alcohol on cognitive inhibitory processes

It is now widely accepted that variable doses of alcohol can affect reaction times (RTs) (Holloway, 1995). Indeed, 80% of the 23 RT studies observed impaired (slowed) RT at different blood alcohol concentrations (BACs). Actually, results may depend considerably on the complexity level of the tasks used, which raises the question as to whether specific cognitive mechanisms are impaired by moderate doses of alcohol. There is a huge difference in terms of the interference of small doses of alcohol on RT between such very simple tasks as pressing a key as quickly as possible when a cross displays on the centre of a computer screen and more complex tasks, such as pressing the same key only when a target symbol appears among distractors, which requires the participants to withhold the response (Holloway, 1995). When compared according to task complexity, RTs on complex tasks are impaired at lower BACs than RTs on simple tasks (Mitchell, 1985). Since the motor execution is similar for both tasks, pre-motor RTs (i.e., cognitive processes) are likely to be more affected by acute effects of alcohol than are the motor functions. Recent findings have in fact supported this idea. Indeed, one study examined directly the possibility that moderate BACs may impair cognitive processes before disturbing motor functions (Hernandez et al., 2006). To do so, RT to the presentation of a stimulus or to the omission of a regularly occurring visual, auditory, or tactile stimulus was fractionated into independent premotor (cognitive) and motor (movement) components. The main finding was that rising BACs slowed premotor RT and had no detectable effect on motor reaction time, thus indicating that moderate doses of alcohol affect cognitive processing more than motor execution (Hernandez et al., 2006).

Alcohol is known for its acute “disinhibiting” effects on behaviour, which may be the consequence of impaired basic cognitive inhibitory mechanisms that normally serve to suppress inappropriate behaviour (Fillmore, 2003). It has been shown to induce perseveration in an attentional set-shifting task, namely the Wisconsin Card Sorting task, and to disrupt inhibition of prepotent behaviour in “Stop-Signal” tasks (Mulvihill et al., 1997). For instance, the cued Go/No-Go reaction time task models behavioural control as the ability to activate a response to a Go-signal quickly and suddenly inhibits a response when a stop-signal occurs (Logan & Cowan, 1984; Logan, 1994). On these types of tasks, alcohol produces dose-dependent impairment on both execution (Go response) and motor inhibition (No-Go response) (Marczinski, Abrams, Van Selst, & Fillmore, 2005). Accordingly, the “No-Go P3” event-related potential (ERP) has been identified as one of the markers for response inhibition (Smith et al., 2006). In alcoholic subjects, a decreased amplitude and a delayed latency of this P3 component to task-relevant target (Go) stimuli has been widely observed, particularly over parietal regions (e.g., Begleiter et al., 1984). Other studies (e.g., Kamarajan et al., 2005) have documented not only low amplitude P3b components to target stimuli, but also reduced frontally distributed P3 amplitudes to No-Go stimuli. These deficits observed in both Go and No-Go conditions suggest that both response activation and response inhibition are dysfunctional in alcoholic individuals. Furthermore, while normal controls manifest their largest P3b amplitudes in response to targets over parietal regions of the scalp, and their largest P3a amplitudes in response to rare non-targets over frontal regions, alcoholics manifest

poor differentiation (i.e. similar low-amplitude P3s) between task conditions (Kamarajan et al., 2005). Assessing the amplitude and topographic features of ERPs and Current Source Density (CSD) in a Go/No-Go task, Kamarajan et al. (2005) also found less anteriorization of CSD polarity in alcoholics during the No-Go processing. The reduced No-Go P3 along with the less anteriorized CSD topography during the No-Go condition suggests poor inhibitory control in alcoholics, perhaps reflecting underlying central nervous system hyperexcitability (Begleiter & Porjesz, 1999).

Indeed, the inhibitory aspects of behavioural control are more vulnerable to the acute effects of alcohol than the activational aspects (e.g., Abroms, Fillmore, & Marczyński, 2003).

Research in cognition has shown that learned information can be retained and guides behaviour consciously or unconsciously (e.g., Norman & Shallice, 1986). Research investigating the respective influence of alcohol on controlled processes and automatic processes has shown that intentional processes are impaired by alcohol, whereas automatic processes are essentially unchanged (Holloway, 1995; Fillmore, 2007). The same distinction between intentional and automatic processes has been made for cognitive response inhibition (Friedman & Miyake, 2004). Intentional response inhibition is under the control of the individual, and operates at the level of awareness (e.g., trying not to press the spacebar in the presence of a distractor on a Go/No-Go task). By contrast, automatic response inhibition occurs below the consciousness threshold in a reflexive manner evoked for instance by the presence of previously learned but no longer relevant information (e.g., negative priming tasks).

Several studies have shown that cognitive response inhibition that depends on control/intention is more vulnerable to the impairing effects of alcohol than response inhibition dependent upon automatic processes (Fillmore & Vogel-Sprott, 2006; Fillmore, 2007; Abroms, Gottlob & Fillmore, 2006). For instance, Abroms et al., (2006) used a delayed ocular response task and a saccadic interference task, which are similar in their response requirements (the ability to execute a saccade to a target location) but different in the nature of the inhibitory mechanism implicated (intentional vs. automatic). Indeed, on the delayed ocular response task (Ross, Hommer, Breiger, Varley, & Radant, 1994), people were required to inhibit intentionally their tendency to make a reflexive saccade toward the sudden appearance of a visual stimulus on a computer screen. Indeed, while participants attended to the fixation point, a bright target stimulus was presented in the periphery, which normally causes a reflexive saccade, which the participant needs to inhibit while maintaining their gaze on the fixation point until it disappears. Automatic inhibition was assessed by the saccadic interference task (Reingold & Stampe, 2002), which measures the ability of a subject to execute a saccade in the presence of an irrelevant, interfering stimulus. The rationale for this task is that it takes longer to execute a saccade to targets on trials with a distractor compared with trials with no distractor, because the distractor interferes with the generation of the saccade by compromising its programming in the superior colliculus. Also, automatic inhibitory processes located in this region are reflexively executed to suppress this interference. Results indicated that moderate doses of alcohol (i.e., 0.45 g/kg of body mass, that is, around 3 regular beers for a participant weighing 70 kg) impaired the intentional but not the automatic inhibition (Abroms et al., 2006). Moreover, this impairment was quite pronounced; the number of controlled/intentional inhibition errors under the highest dose (0.65 g/kg) was nearly three times greater than in response to placebo.

The deleterious power of moderate doses of alcohol on response inhibition and increased impulsivity could be responsible for other cognitive impairments (Weissenborn & Duka, 2003). For instance, on a planning task, the Tower of London (Shallice, 1982), subjects are presented with two sets of three coloured 'balls', one in the top half of the screen and one in the bottom. They are instructed to move the balls in the bottom array so that they match the pattern in the top array, which requires making and executing plans. On this task, alcohol decreased the thinking time prior to initiating a solution, which may reflect greater impulsivity (Weissenborn & Duka, 2003). But alcohol also impacted planning not only by decreasing the number of correct trials to solve the Tower of London task, but also by increasing the time spent thinking about moves once a solution had been initiated. It is reasonable to assume that, in the absence of alcohol-impaired spatial working memory, acute doses of alcohol dramatically disrupt planning capacities notably by decreasing intentional/controlled response inhibition.

The relationship between acute effects of alcohol and disinhibition is likely to be moderated by numerous cognitive and affective factors (Finn et al., 1999; Ernst et al., 2006; Dom et al., 2006; Hittner & Swickert, 2006). For instance, executive functions other than inhibition-related ones could impact the relationship between acute effects of alcohol and response inhibition. Indeed, only subjects with low capacity to manipulate information stored in working memory (as assessed by counting digits backwards) showed alcohol-induced motor inhibitory control as attested by a greater number of false alarms in a Go/No-Go task after they ingested moderate doses of alcohol (Finn et al., 1999).

Another main moderator of the relationship between acute effects of alcohol and response inhibition would be some personality characteristics (Fillmore et al., 2008; Ray et al., 2006). This line of research has been justified by growing evidence that impulsivity might play an important causal role in problem drinking (Lacono et al., 1999). For instance, longitudinal studies of children and adolescents have shown that impulsivity predicts early onset drinking age and development of heavy drinking and alcohol dependence in young adults (Ernst et al., 2006). In particular, sensation-seeking, considered as a neurobiologically based tendency to seek novel, complex, intense sensations was positively correlated with increased alcohol use and alcohol-related problems (Dom et al., 2006; Hittner & Swickert, 2006). This greater risk to experience alcohol and other substance abuse may be that these individuals are more responsive to the rewarding effects of alcohol as expressed by its more subjective stimulant-like effects (Ray et al., 2006). Interestingly, a study also found that those non-alcoholic students high on sensation-seeking demonstrated increased sensitivity to the subjective rewarding effects of alcohol and also less motor inhibition than those low on sensation-seeking (Fillmore et al., 2008).

3. Conclusion

The present article emphasized the relevance of impulsivity/disinhibition for investigating both predisposing and developmental factors leading to alcohol-related disorders. The association between response inhibition weaknesses prior to the onset of alcohol use with the disinhibitory acute effects of alcohol and its deleterious chronic effects may dramatically improve the likelihood to loss control over alcohol use.

However, one main problem with the use of one or two cognitive tasks is that the construct validities of most commonly used inhibition tasks are not well established. For instance, go/no-go and stop-signal paradigms are generally considered as investigating a same construct, namely intentional prepotent motor response inhibition. However, an important contribution was that these tasks can both rely on both bottom-up control and top-down control (Verbruggen & Logan, 2008). Automatic and controlled inhibition can work together to guide goal-directed behavior. Indeed, when a no-go stimulus (in the go/no-go paradigm) or a stimulus that was previously accompanied by a stop signal (in the stop-signal paradigm) is repeated, the stop goal is activated through the retrieval of stimulus-stop associations, and it suppresses automatically the go response. In other terms, a stopping action might be reflecting either the need for top-down executive control processes or the need for bottom-up automatic processes. One way to overcome the fact that an entire task does not tap into one single psychological construct may be to perform analyses within a single task to extract the relative contribution of automatic versus intentional inhibition to the interruption of an action. Then, it will be easy to perform between group comparisons with dependent variables assessing either automatic or intentional response inhibition.

Another way to draw more robust conclusions about the failure of alcoholics to inhibit response could be based on the methodology proposed by Friedmann and Miyake (2004). Indeed, a latent variable analysis makes it possible to “extract” what is common among the tasks selected to tap a putative executive function and use that “purer” latent variable to examine how different executive functions relate to one another. By doing this, these authors showed that prepotent response inhibition and resistance to distractor interference were closely related, but both were unrelated to resistance to proactive interference (Friedman & Miyake, 2004). By using inhibition tasks selected from this theoretical framework, we found that recently detoxified alcoholics exhibit a massive prepotent response inhibition disruption despite preserved bottom up/automatic inhibition (Noël et al., unpublished data).

Another promising avenue of research refers to the notion of “inhibition biases” that reflects performance decreasing while information to-be-inhibited is associated with alcoholism. It is the case when the action suppression associated with alcohol-related words results in the increasing of the number of commission errors in alcoholics compared to non-alcoholics (e.g., Noël et al., 2007). Thus, the investigation of “inhibition deficits” (i.e., disruption of inhibition regardless the kind of information processes) could be advantageously complement with research focused on “inhibition biases”.

Finally, it has been more and more obvious that alcoholism is a heterogeneous disease. As a striking example, we reviewed in this chapter that individuals with early onset of alcoholism (EOAs) are generally more impulsive than those with late onset of alcoholism (LOAs). Dom's studies have robustly showed that EOAs had higher discount rates than both the non-substance-abusing subjects and the LOAs, with these two groups performing similarly. This differentiation between EOAs and LOAs in terms of impulsive decision making emphasized the heterogeneity of individuals with alcoholism on the one hand and the existence of distinct pathways leading to alcoholism on the other.

Taken together, it is highly recommendable that research on impulsive decision and behavior should be intensified within more robust theoretical frameworks and updated methodologies. Further understanding of psychological and neurobiological underpinnings

of inhibitory control offer obvious promise for improving pharmacological and psychological treatment for individuals with alcoholism.

4. References

- Abroms, B. D., Fillmore, M. T., & Marcinski, C. A. (2003). Alcohol-induced impairment of behavioral control: effects on the alteration and suppression of prepotent responses. *Journal of Studies on Alcohol*, 64(5), 687-695.
- Abroms, B. D., Gottlob, L. R., & Fillmore, M. T. (2006). Alcohol effects on inhibitory control of attention: distinguishing between intentional and automatic mechanisms. *Psychopharmacology*, 188(3), 324-334. doi:10.1007/s00213-006-0524-y
- Andrés, P., Van der Linden, M., & Parmentier, F. B. R. (2004). Directed forgetting in working memory: age-related differences. *Memory*, 12(2), 248-256. doi:10.1080/09658210244000612
- Andrussi, G. L., Archer, R. P., Pancoast, D. L., & Gordon, R. A. (1989). The relationship of MMPI and Sensation Seeking Scales to adolescent drug use. *Journal of Personality Assessment*, 53(2), 253-266. doi:10.1207/s15327752jpa5302_4
- Aron, A. R. (2007). The neural basis of inhibition in cognitive control. *The Neuroscientist: A Review Journal Bringing Neurobiology, Neurology and Psychiatry*, 13(3), 214-228. doi:10.1177/1073858407299288
- Babor, T. F., Hofmann, M., DelBoca, F. K., Hesselbrock, V., Meyer, R. E., Dolinsky, Z. S., & Rounsaville, B. (1992). Types of alcoholics, I. Evidence for an empirically derived typology based on indicators of vulnerability and severity. *Archives of General Psychiatry*, 49(8), 599-608.
- Bechara, A. (2003). Risky business: emotion, decision-making, and addiction. *Journal of Gambling Studies*, 19(1), 23-51.
- Begleiter, H., & Porjesz, B. (1999). What is inherited in the predisposition toward alcoholism? A proposed model. *Alcoholism, Clinical and Experimental Research*, 23(7), 1125-1135.
- Belin, D., Mar, A. C., Dalley, J. W., Robbins, T. W., & Everitt, B. J. (2008). High impulsivity predicts the switch to compulsive cocaine-taking. *Science*, 320(5881), 1352-1355. doi:10.1126/science.1158136
- Bjork, J. M., Hommer, D. W., Grant, S. J., & Danube, C. (2004). Impulsivity in abstinent alcohol-dependent patients: relation to control subjects and type 1-/type 2-like traits. *Alcohol*, 34(2-3), 133-150.
- Bowden-Jones, H., McPhillips, M., Rogers, R., Hutton, S., & Joyce, E. (2005). Risk-taking on tests sensitive to ventromedial prefrontal cortex dysfunction predicts early relapse in alcohol dependency: a pilot study. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 17(3), 417-420. doi:10.1176/appi.neuropsych.17.3.417
- Burgess, P. W., & Shallice, T. (1996). Response suppression, initiation and strategy use following frontal lobe lesions. *Neuropsychologia*, 34(4), 263-272.
- Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience and Biobehavioral Reviews*, 29(3), 399-419. doi:10.1016/j.neubiorev.2004.11.006

- Collette, F., Van der Linden, M., Delfiore, G., Degueldre, C., Luxen, A., & Salmon, E. (2001). The functional anatomy of inhibition processes investigated with the Hayling task. *NeuroImage*, 14(2), 258-267. doi:10.1006/nimg.2001.0846
- de Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: a review of underlying processes. *Addiction Biology*, 14(1), 22-31. doi:10.1111/j.1369-1600.2008.00129.x
- Dom, G., D'haene, P., Hulstijn, W., & Sabbe, B. (2006). Impulsivity in abstinent early- and late-onset alcoholics: differences in self-report measures and a discounting task. *Addiction*, 101(1), 50-59. doi:10.1111/j.1360-0443.2005.01270.x
- Ernst, M., Luckenbaugh, D. A., Moolchan, E. T., Leff, M. K., Allen, R., Eshel, N., London, E. D., et al. (2006). Behavioral predictors of substance-use initiation in adolescents with and without attention-deficit/hyperactivity disorder. *Pediatrics*, 117(6), 2030-2039. doi:10.1542/peds.2005-0704
- Evenden, J. L. (1999). Varieties of impulsivity. *Psychopharmacology*, 146(4), 348-361.
- Fillmore, M. T. (2003). Drug abuse as a problem of impaired control: current approaches and findings. *Behavioral and Cognitive Neuroscience Reviews*, 2(3), 179-197. doi:10.1177/1534582303257007
- Fillmore, M. T. (2007). Acute alcohol-induced impairment of cognitive functions: past and present findings. *International Journal on Disability and Human Development*, 6, 115-125.
- Fillmore, M. T., Ostling, E. W., Martin, C. A., & Kelly, T. H. (2009). Acute effects of alcohol on inhibitory control and information processing in high and low sensation-seekers. *Drug and Alcohol Dependence*, 100(1-2), 91-99. doi:10.1016/j.drugalcdep.2008.09.007
- Finn, P R, Justus, A., Mazas, C., & Steinmetz, J. E. (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(4), 465-472.
- Finn, Peter R. (2002). Motivation, working memory, and decision making: a cognitive-motivational theory of personality vulnerability to alcoholism. *Behavioral and Cognitive Neuroscience Reviews*, 1(3), 183-205.
- Friedman, Naomi P, & Miyake, A. (2004). The relations among inhibition and interference control functions: a latent-variable analysis. *Journal of Experimental Psychology. General*, 133(1), 101-135. doi:10.1037/0096-3445.133.1.101
- Gazzaniga, M. (2000). *Cognitive neuroscience : a reader*. Malden Mass.: Blackwell.
- Goldstein, R. Z., & Volkow, N. D. (2002). Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *The American Journal of Psychiatry*, 159(10), 1642-1652.
- Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & van den Brink, W. (2005). Decision making in pathological gambling: a comparison between pathological gamblers, alcohol dependents, persons with Tourette syndrome, and normal controls. *Brain Research. Cognitive Brain Research*, 23(1), 137-151. doi:10.1016/j.cogbrainres.2005.01.017
- Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & van den Brink, W. (2006). Neurocognitive functions in pathological gambling: a comparison with alcohol dependence, Tourette syndrome and normal controls. *Addiction*, 101(4), 534-547. doi:10.1111/j.1360-0443.2006.01380.x

- Habeych, M. E., Folan, M. M., Luna, B., & Tarter, R. E. (2006). Impaired oculomotor response inhibition in children of alcoholics: The role of attention deficit hyperactivity disorder. *Drug and Alcohol Dependence*, 82(1), 11-17. doi:10.1016/j.drugalcdep.2005.07.012
- Hernández, O. H., Vogel-Sprott, M., Huchín-Ramírez, T. C., & Aké-Estrada, F. (2006). Acute dose of alcohol affects cognitive components of reaction time to an omitted stimulus: differences among sensory systems. *Psychopharmacology*, 184(1), 75-81. doi:10.1007/s00213-005-0237-7
- Hittner, J. B., & Swickert, R. (2006). Sensation seeking and alcohol use: a meta-analytic review. *Addictive Behaviors*, 31(8), 1383-1401. doi:10.1016/j.addbeh.2005.11.004
- Holloway, F. (s. d.). Low-dose alcohol effects on human behavior and performance. *Alcohol, Drugs and Driving*, 11(1), 39-56.
- Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral disinhibition and the development of substance-use disorders: findings from the Minnesota Twin Family Study. *Development and Psychopathology*, 11(4), 869-900.
- Jentsch, J. D., & Taylor, J. R. (1999). Impulsivity resulting from frontostriatal dysfunction in drug abuse: implications for the control of behavior by reward-related stimuli. *Psychopharmacology*, 146(4), 373-390.
- Kamarajan, C., Porjesz, B., Jones, K., Chorlian, D., Padmanabhapillai, A., Rangaswamy, M., Stimus, A., et al. (2006). Event-related oscillations in offspring of alcoholics: neurocognitive disinhibition as a risk for alcoholism. *Biological Psychiatry*, 59(7), 625-634. doi:10.1016/j.biopsych.2005.08.017
- Kirisci, L., Tarter, R. E., Reynolds, M., & Vanyukov, M. (2006). Individual differences in childhood neurobehavior disinhibition predict decision to desist substance use during adolescence and substance use disorder in young adulthood: a prospective study. *Addictive Behaviors*, 31(4), 686-696. doi:10.1016/j.addbeh.2005.05.049
- Logan, G D, Cowan, W. B., & Davis, K. A. (1984). On the ability to inhibit simple and choice reaction time responses: a model and a method. *Journal of Experimental Psychology. Human Perception and Performance*, 10(2), 276-291.
- Logan, Gordon D. (1994). On the ability to inhibit thought and action: A user's guide to stop signal paradigm. *Inhibitory processes in attention, memory and language* (Academic Press., p. 189-239). San Diego, CA: D. Dagenbach, T.H. Carr.
- Lusher, J., Chandler, C., & Ball, D. (2004). Alcohol dependence and the alcohol Stroop paradigm: evidence and issues. *Drug and Alcohol Dependence*, 75(3), 225-231. doi:10.1016/j.drugalcdep.2004.03.004
- Lyvers, M. (2000). « Loss of control » in alcoholism and drug addiction: a neuroscientific interpretation. *Experimental and Clinical Psychopharmacology*, 8(2), 225-249.
- Marczinski, C. A., Abroms, B. D., Van Selst, M., & Fillmore, M. T. (2005). Alcohol-induced impairment of behavioral control: differential effects on engaging vs. disengaging responses. *Psychopharmacology*, 182(3), 452-459. doi:10.1007/s00213-005-0116-2
- May, J., Andrade, J., Panabokke, N., & Kavanagh, D. (2004). Images of desire: cognitive models of craving. *Memory*, 12(4), 447-461. doi:10.1080/09658210444000061
- Mazur, J. (1987). An adjusting procedure for studying delayed reinforcement. The effect of delay and of intervening events on reinforcement value, In: *The effect of delay and of intervening events on reinforcement value, Quantitative analyses of behavior*, M.L.

- Commons, J.E. Mazur, J.A. Nevin, H. Rachlin (Ed.), 55-73, Lawrence Erlbaum Associates, Hillsdale, NJ, England.
- Mitchell, J. M., Fields, H. L., D'Esposito, M., & Boettiger, C. A. (2005). Impulsive responding in alcoholics. *Alcoholism, Clinical and Experimental Research*, 29(12), 2158-2169.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex « Frontal Lobe » tasks: a latent variable analysis. *Cognitive Psychology*, 41(1), 49-100. doi:10.1006/cogp.1999.0734
- Mulder, R. T. (2002). Alcoholism and personality. *The Australian and New Zealand Journal of Psychiatry*, 36(1), 44-52.
- Mulvihill, L. E., Skilling, T. A., & Vogel-Sprott, M. (1997). Alcohol and the ability to inhibit behavior in men and women. *Journal of Studies on Alcohol*, 58(6), 600-605.
- Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin*, 126(2), 220-246.
- Nigg, J. T., Wong, M. M., Martel, M. M., Jester, J. M., Puttler, L. I., Glass, J. M., Adams, K. M., 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. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(4), 468-475. doi:10.1097/01.chi.0000199028.76452.a9
- Noël, X., Van der Linden, M., Schmidt, N., Sferrazza, R., Hanak, C., Le Bon, O., De Mol, J., et al. (2001). Supervisory attentional system in nonamnesic alcoholic men. *Archives of General Psychiatry*, 58(12), 1152-1158.
- Noël, X., Billieux, J., Van der Linden, M., Dan, B., Hanak, C., de Bournonville, S., Baurain, C., & Verbanck, P. (2009). Impaired inhibition of proactive interference in abstinent individuals with alcoholism. *Journal of Clinical and Experimental Neuropsychology*, 31(1), 57-64. doi:10.1080/13803390801982726
- Noël, X., Sferrazza, R., Van Der Linden, M., Paternot, J., Verhas, M., Hanak, C., Pelc, I., et al. (2002). Contribution of frontal cerebral blood flow measured by (99m)Tc-Bicisate spect and executive function deficits to predicting treatment outcome in alcohol-dependent patients. *Alcohol and Alcoholism*, 37(4), 347-354.
- Noël, X., Van der Linden, M., d'Acromont, M., Bechara, A., Dan, B., Hanak, C., & Verbanck, P. (2007). Alcohol cues increase cognitive impulsivity in individuals with alcoholism. *Psychopharmacology*, 192(2), 291-298. doi:10.1007/s00213-006-0695-6
- Noël, X., Van der Linden, M., d'Acromont, M., Colmant, M., Hanak, C., Pelc, I., Verbanck, P., et al. (2005). Cognitive biases toward alcohol-related words and executive deficits in polysubstance abusers with alcoholism. *Addiction*, 100(9), 1302-1309. doi:10.1111/j.1360-0443.2005.01125.x
- Norman, D. A., & Shallice, T. (1986). Attention to action: Willed and automatic control of behaviour, In: *Consciousness and Self-Regulation: Advances in Research and Theory*, R.J. Davidson, G.E. Schwartz, D. Shapiro (Ed.), 1-18, Plenum Press, New York.
- Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the barratt impulsiveness scale. *Journal of Clinical Psychology*, 51, 768-774. doi:10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1
- Ray, L. A., McGeary, J., Marshall, E., & Hutchison, K. E. (2006). Risk factors for alcohol misuse: examining heart rate reactivity to alcohol, alcohol sensitivity, and

- personality constructs. *Addictive Behaviors*, 31(11), 1959-1973. doi:10.1016/j.addbeh.2006.01.010
- Reingold, E. M., & Stampe, D. M. (2002). Saccadic inhibition in voluntary and reflexive saccades. *Journal of Cognitive Neuroscience*, 14(3), 371-388. doi:10.1162/089892902317361903
- Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology*, 54, 25-53. doi:10.1146/annurev.psych.54.101601.145237
- Ross, R. G., Hommer, D., Breiger, D., Varley, C., & Radant, A. (1994). Eye movement task related to frontal lobe functioning in children with attention deficit disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(6), 869-874. doi:10.1097/00004583-199407000-00013
- Schweinsburg, A. D., Paulus, M. P., Barlett, V. C., Killeen, L. A., Caldwell, L. C., Pulido, C., Brown, S. A., et al. (2004). An FMRI study of response inhibition in youths with a family history of alcoholism. *Annals of the New York Academy of Sciences*, 1021, 391-394. doi:10.1196/annals.1308.050
- Sharma, D., Albery, I. P., & Cook, C. (2001). Selective attentional bias to alcohol related stimuli in problem drinkers and non-problem drinkers. *Addiction*, 96(2), 285-295. doi:10.1080/09652140020021026
- Stuphorn, V., & Schall, J. D. (2006). Executive control of countermanding saccades by the supplementary eye field. *Nature Neuroscience*, 9(7), 925-931. doi:10.1038/nn1714
- Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: role of automatic and nonautomatic processes. *Psychological Review*, 97(2), 147-168.
- Verbruggen, F., & Logan, G. D. (2008). Automatic and controlled response inhibition: associative learning in the go/no-go and stop-signal paradigms. *Journal of Experimental Psychology. General*, 137(4), 649-672. doi:10.1037/a0013170
- Verdejo-García, A., Lawrence, A. J., & 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. *Neuroscience and Biobehavioral Reviews*, 32(4), 777-810. doi:10.1016/j.neubiorev.2007.11.003
- von Knorring, L., Palm, U., & Andersson, H. E. (1985). Relationship between treatment outcome and subtype of alcoholism in men. *Journal of Studies on Alcohol*, 46(5), 388-391.
- von Knorring, L., von Knorring, A. L., Smigan, L., Lindberg, U., & Edholm, M. (1987). Personality traits in subtypes of alcoholics. *Journal of Studies on Alcohol*, 48(6), 523-527.
- Weissenborn, R., & Duka, T. (2003). Acute alcohol effects on cognitive function in social drinkers: their relationship to drinking habits. *Psychopharmacology*, 165(3), 306-312. doi:10.1007/s00213-002-1281-1
- Whiteside, S. P., & Lynam, D. R. (2003). Understanding the role of impulsivity and externalizing psychopathology in alcohol abuse: application of the UPPS impulsive behavior scale. *Experimental and Clinical Psychopharmacology*, 11(3), 210-217.
- Zuckerman, M., & Neeb, M. (1979). Sensation seeking and psychopathology. *Psychiatry Research*, 1(3), 255-264.



Psychology - Selected Papers

Edited by Dr. Gina Rossi

ISBN 978-953-51-0587-9

Hard cover, 330 pages

Publisher InTech

Published online 02, May, 2012

Published in print edition May, 2012

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How to reference

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Xavier Noël (2012). Alcoholism: An Impulsive/Disinhibition Disorder?, Psychology - Selected Papers, Dr. Gina Rossi (Ed.), ISBN: 978-953-51-0587-9, InTech, Available from: <http://www.intechopen.com/books/psychology-selected-papers/alcoholism-an-impulsive-disinhibition-disorder>

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