Decision Making, Risky Behavior, and Alcoholism

Chapter 13 - Section 5

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**Decision making, risky behavior and alcoholism**

A hallmark symptom of alcoholism is the continual engagement in dangerous or risky drinking behavior despite recurring social or personal difficulties such as alienation from friends and family, trouble with the law and at work, or problems with physical and emotional well-being. It has been suggested that the alcoholic’s propensity for such risky behavior is manifested by impulsive decision-making processes, wherein potential short-term appetitive outcomes of drinking (e.g. intoxication) are deemed more important than potential long-term aversive consequences of drinking (e.g., drunk driving arrests). Dual process models of alcoholism hold that separate but interrelated neurocognitive pathways to impulsive decision making exist. One reflected by weak “top-down” executive control over impulsive and compulsive urges to consume alcohol. The other reflected by a strong “bottom-up” appetitive drive in impulsive and compulsive urges to consume alcohol. The current chapter surveys evidence that functional and organizational patterns in brain executive control and appetitive drive networks characterize impulsive decision making and risky behavior in alcoholics, and that these patterns differ at different stages of alcoholism dependence and recovery.

**POOR EXECUTIVE CONTROL leads to poor decision making**

Impairments in executive cognitive ability have been consistently noted in alcoholics at various stages of alcohol dependence and recovery. Actively drinking alcoholics typically perform more poorly than non-substance abusing controls in a variety of neurocognitive domains related to attention, memory, and contingency learning (Finn and Hall, 2004, Finn et al., 2009, Bechara and Martin, 2004, Bogg and Finn, 2010). Finn et al. (2009) showed that severity of alcoholism and other related externalizing problems predicted poor short-term and working memory capacity, with the greatest impairments exhibited by those with other co-occurring externalizing behavior problems. Alcoholics’ poor cognitive capacity also has been linked with a broad constellation of personality traits related to impulsiveness, poor-harm avoidance, and antisocial behavior tendencies (Bogg and Finn, 2010). In the same vein, laboratory evidence has suggested that poor executive cognitive capacity is partially responsible for impulsive decision making in alcoholics and related externalizing syndromes.
(Finn, 2002, Endres et al., 2011, Bogg et al., 2012). Recent evidence also shows that, when combined with highly impulsive trait dispositions, poor executive cognitive capacity predicts more heavy and dangerous drinking behavior in active alcoholics (Gunn and Finn, 2013). In summary, the evidence suggests that impaired or weak executive cognitive control processes are at least partially responsible for the impulsive decision-making tendencies and risky behaviors of actively drinking alcoholics.

Alcoholics who stop drinking, and remain sober, experience recovery in some, but not all, domains of neuropsychological functioning. Studies have shown that while neuropsychological impairments in executive cognitive ability are present in recently detoxified and short-term abstinent alcoholics, such impairments may resolve with extended abstinence; nonetheless, the major recovery is observed in the first year of abstinence (Fein et al., 2006, Mann et al., 1999, Sullivan et al., 2000, Nixon and Glenn, 1995, Stavro et al., 2012). A recent meta-analysis by Stavro et al. (2012) found widespread deficits in executive cognitive ability characterized recently detoxified and short-term abstinent alcoholics; but, such impairments were dramatically attenuated in long-term abstinent alcoholics. However, Stavro et al. (2012) also noted a dearth of research on the persistence of inhibitory control/impulsive decision-making deficits in those with multi-year abstinence. A longitudinal study by Sullivan et al. (2000) found similar evidence of recovery, demonstrating alcoholics with sustained abstinence improved in short-term memory, visuospatial processing, attention, and gait and balance abilities. Likewise, Fein et al. (2006) showed that long-term abstinent (LTA) alcoholics with an average of 6.7 years abstinence did not differ from non-substance abusing controls in numerous executive cognitive domains, including cognitive abstraction/flexibility, attention, short-term/working memory, psychomotor function/reaction time, and verbal skills. For example, Fein et al. (2006) showed spatial processing deficits were present in LTAs. Thus, evidence suggests that executive cognitive impairments in attention, memory, and motor control to a large extent normalize with extended abstinence. However, as reviewed below, evidence also suggests that cognitive impairments related to the processing of appetitive and/or emotional information remain present in multi-year abstinent alcoholics.
STRONG APPETITIVE DRIVE leads to poor decision making

Psychometric evidence has suggested that impulsive decision-making in alcoholism and related externalizing syndromes comprises three independent domains: myopia to the future, unwillingness to delay gratification, and difficulties with passive avoidance (Dom et al., 2007). Myopic decision-making reflects insensitivity to the long-term aversive consequences of appetitive goal-directed behavior and has been assessed with simulated gambling tasks such as the Iowa Gambling Task (Bechara et al., 1994). Preference for immediate gratification reflects a tendency to choose smaller immediate appetitive outcomes over larger delayed appetitive outcomes and has been assessed with intertemporal choice paradigms such as the delay discounting task (DDT) (Rachlin et al., 1991). Poor passive avoidance refers to difficulty stopping or withholding proponent or appetitive behavior so as to avoid sub-optimal or aversive outcomes and has been assessed with measures of inhibitory control, such as the stop-signal (SST) (Logan et al., 1997), go/no-go (GNG) (Newman et al., 1985), or balloon analog risk task (BART) (Lejuez et al., 2002).

Acute alcohol use has disinhibiting effects on behavior control, impairing rational decision making and fueling such risky behavior as gambling, getting a tattoo, or having unprotected sex (Lin et al., 2005, Purdie et al., 2011, Tzambazis and Stough, 2000). Separate studies have shown active alcoholics typically make more impulsive or disinhibited decisions than non-substance abusing controls, with those presenting other co-occurring externalizing symptoms exhibiting the poorest performances on the Iowa Gambling Task, DDT, and GNG (Cantrell et al., 2008, Finn et al., 2002, Mazas et al., 2000, Endres et al., 2011, Bobova et al., 2009). Evidence also suggests that impaired processing of aversive feedback leads to impulsive decision-making in alcoholism and related externalizing syndromes, and this information processing impairment is associated with factors that predate the onset of clinical problems, such as a family history of alcoholism (Finn et al., 1994, Fein and Chang, 2008).

Impulsive decision-making tendencies also have been observed in abstinent alcoholics, and unlike impairments in executive cognitive ability, evidence has suggested that impulsive decision-making tendencies persist for weeks, months, and even years of abstinence. Research
has shown abstinent alcoholics to be less willing to delay gratification in the DDT relative to non-substance abusing controls, and this effect was still present after controlling for impulsive personality traits (Mitchell et al., 2005). Evidence also has suggested recently detoxified alcoholics make more myopic decisions in the Iowa Gambling Task than controls with little to no improvement in this capacity at 1 month follow (Dom et al., 2007). Similar myopic decision-making tendencies have also been found in short-term and long-term abstinent alcoholics (Fein et al., 2010b), with short-term and long-term abstainers showing comparable impulsive decision-making during the Iowa Gambling Task relative to non-substance abusing controls (Ando et al., 2012). Long-term abstinent alcoholics also are known to have persistent difficulties with emotion perception and affective decision-making (Fein et al., 2010b, Foisy et al., 2007, Philippot et al., 1999, Kornreich et al., 2001, Endres and Fein, 2012). Endres and Fein (2012) recently showed that LTA’s persistent difficulties with passive avoidance in an affective GNG task were largely due to a history of externalizing problems and independent of more general difficulties in lexical decision-making. However, research has suggested a disassociation between antisocial symptoms and traits in LTA, suggesting that antisocial behaviors return to normal levels in the face of elevated antisocial and reward-seeking trait dispositions (Fein and Fein, 2012). In light of the fact that alcoholics can remain sober in spite of elevated levels of impulsive traits and decision-making tendencies, researchers have suggested that other compensatory mechanisms come into play, such as heightened executive control and conscientiousness aid in abstinence maintenance (Ando et al., 2012, Camchong et al., 2013a). When together, research has shown impulsive decision-making tendencies are present in alcoholics throughout the recovery process, but such impulsive personality and decision-making tendencies do not appear to militate against the ability to maintain abstinence.

It is still not completely clear whether poor decision making and risky behavior contribute to the vulnerability of becoming an alcoholic or whether they are a consequence of chronic alcohol use. There is evidence supporting the idea that poor decision making exists before alcohol dependence. A longitudinal study on young adults in college found that poor decision making (as assessed with the Iowa Gambling Task) at second year of college is associated with heavy drinking two years later (Goudriaan et al., 2009). Poor decision making
(assessed with the Iowa Gambling and Stroop tasks) has also been identified in individuals at risk for alcoholism (with family history of alcoholism) when compared to healthy controls (Lovallo et al., 2006). Heavy drinking adolescents have shown poor decision making and higher impulsivity than light drinking adolescents (Field et al., 2007).

There is also evidence suggesting that chronic alcohol consumption during adolescence, a period in which decision making brain regions are still developing, has a long-term effect on adult decision making. A longitudinal study using a rodent model of chronic alcohol use found that rats that were exposed to chronic alcohol use during adolescence had poor decision making (did not properly weigh benefits and costs) and preferred more risky options (chose large but uncertain rewards over small but certain rewards) when performing a probability-discounting instrumental response task in adulthood versus rats that were exposed to a placebo (Nasrallah et al., 2009). Further research needs to be conducted to better determine whether poor decision making is present before or is a consequence of alcoholism.

Other aspects of behavior related to poor decision making in alcoholics

Even though poor decision making has been identified in alcoholics and individuals at risk of becoming alcoholics, this behavioral deficit is not specific to alcoholism. Poor decision making in alcoholism has been associated with other aspects of behavior such as antisocial tendencies, stress, number of detoxifications, and onset of alcohol dependence.

An individual with antisocial personality disorder does not adhere to accepted societal norms and often engages in criminal and other impulsive behavior (APA, 1994). Antisocial Personality Disorder is more prevalent in alcoholics than in healthy controls (Compton et al., 2005, Goldstein et al., 2007). Previous research has proposed that poor decision making in alcohol dependent individuals is increased in those with a comorbid Antisocial Personality Disorder. A study by Miranda et al (2009) compared risky decision making (Iowa Gambling Task performance) between men with alcohol dependence (abstinent >30 days) with and without ASPD comorbidity compared with non-substance abusing control men. Both alcoholic groups made significantly more risky decisions (manifested as poorer Iowa Gambling Task performance) than the control group. However, while alcoholics without ASPD comorbidity
showed a slow and steady improvement in advantageous decision making during the task, alcoholics with ASPD comorbidity showed an initial improvement but were unable to sustain it. Moreover, authors reported evidence that impulsive behavioral traits (e.g., propensity for rash impulsive behavior) mediated disadvantageous decision making in alcoholics with ASPD comorbidity. These findings suggest that poor decision making in alcoholics is closely related to impulsive antisocial tendencies. Because there is evidence that alcoholics’ antisocial behavior resolves with extended periods of abstinence, while antisocial disposition does not (Fein and Fein, 2012), findings of poor decision making in long-term abstinent alcoholics might be related to a persistent disposition to appetitive drive tendencies.

There is evidence that decision making improves when stress is induced in actively heavy drinkers. A study that compared decision making in heavy and light drinkers reported that heavy drinkers made significantly more disadvantageous decisions than light drinkers (with more attention to gains and less attention to losses). When anticipatory stress was induced (by asking them to give a speech), decision making was improved in both heavy and light drinkers (by increasing attention to losses) (Gullo and Stieger, 2011). Evidence from this study suggests that stress is needed to better focus attention on negative consequences and hence better decision making. It should be noted, however, that the effect of stress on decision making may be different in individuals that have been diagnosed with alcohol dependence or substance dependence (Zhang et al., 2011). In addition, the level of stress induced by requesting participants to give a speech may not be severe, chronic, or realistic enough to negatively affect drinkers’ decision making.

Another aspect that has been found to affect decision making in individuals with alcohol dependence is the number of detoxifications an individual has been through. A study that compared a group with fewer than 2 detoxifications (low-detox group) versus a group with >2 detoxifications (high-detox group) reported that the low-detox group had a significantly greater rate of improvement in choices throughout the Iowa Gambling Task than the high-detox group (Loeber et al., 2009). This evidence suggests that individuals with repeated withdrawal episodes lack the ability to make proper decisions and to adapt behavior to newly learned rules.
More research needs to be conducted to find out whether poor decision making leads to repeated relapse episodes or whether the negative effects of repeated withdrawals affect decision making.

Higher levels of impulsive decision making have been associated with early-onset alcoholism (Dom et al., 2006). Early-onset alcoholics have shown higher levels of impulsive decision making during a delayed discounting task than late-onset alcoholics and controls (Dom et al., 2006). This claim, however, needs to be further examined considering other factors related to poor decision making in early-onset alcoholism such as fewer years of education, potentially higher genetic load, or longer lifetime alcohol use.

**Neural correlates of decision making and risky behavior in alcoholism**

Behavioral aspects of alcoholism such as poor decision making and risky behavior have been associated with corresponding brain functional differences from non-substance abusing individuals. The executive control (principally mediated by cortical regions particularly in frontal cortex) and appetitive drive (principally mediated by striatal regions) brain networks have prominent roles in decision making and risky behavior in alcoholism. During its early stages, alcohol consumption is a goal-directed behavior, initiated and executed by regions within the executive control network (such as dorsolateral prefrontal cortex and anterior cingulate cortex) with its rewarding effects processed by appetitive drive regions (such as nucleus accumbens). After alcohol is repeatedly consumed, evidence for or consistent with poor decision making, consumption becomes more automatic (with more involvement of appetitive drive regions such as caudate and putamen) and less voluntary (with less involvement of executive control regions) (Everitt and Robbins, 2005). Alcohol consumption shifts to a more habitual mode, particularly as a response to avoid withdrawal symptoms. An individual with alcohol dependence seeks alcohol compulsively, a behavior that has been associated with increased activity of appetitive drive regions when presented with an alcohol cue, and a lack of engagement of prefrontal regions when required to stop a prepotent, maladaptive behavior such as alcohol consumption. The quality of interaction between and within these functional networks (frontal executive and striatal appetitive networks) is
becoming an increasingly relevant topic in alcoholism and addiction in general. Both the attenuation of the executive control and the enhancement of the appetitive drive networks are closely related to behavioral aspects of alcoholism such as poor decision making and subsequently risky behavior.

To examine functional brain activity and synchrony associated with poor decision making in alcoholics, participants are required to perform tasks while functional magnetic resonance imaging (fMRI) data are collected. Numerous neuroimaging studies have used specific tasks to tap into functional brain differences associated with poor decision making and risky behavior during alcohol use, abuse and dependence.

Brain function associated with decision making in binge-drinking

A study by Xiao et al (2012) compared brain functional activity of 14 binge-drinking adolescents while they made decisions during the Iowa Gambling Task compared with 14 adolescents who had never consumed alcohol. Binge drinkers had both behavioral and brain functional differences manifested as inflexible poor decision making (kept making disadvantageous decision) together with higher activity than non-drinkers in limbic brain regions known to play a role in the emotional aspects of decision making (amygdala and insula). Because they also found that adolescents that had higher limbic activity while performing the decision making task had more self-reported drinking problems, Xiao et al. (2012) proposed that a hyperactive bottom-up affective network may be a good candidate for a biological marker that can be used to identify individuals at risk of alcohol dependence. A previous behavioral study used the original Iowa Gambling Task and a variant of the Iowa Gambling Task to dissect whether poor decision-making found in binge-drinkers is attributable to insensitivity to long-term consequences (lack of executive control, planning) or to hypersensitivity to reward (enhanced appetitive drive) (Johnson et al., 2008). After comparing Iowa Gambling Task performance between adolescent binge-drinkers to adolescent never-drinkers, Johnson et al (2008) found that poor decision making in binge-drinkers is associated to hypersensitivity to reward. Results from these studies provide evidence that at early stages of alcohol abuse there is enhanced behavior ruled by appetitive drive together with a hyperactive bottom-up network.
Brain function associated with decision making in active drinkers with alcohol dependence

The neural correlates of decision making have been examined in active drinkers with a diagnosis of alcohol dependence (Amlung et al., 2012). Amlung et al (2012) compared brain activity during delayed reward discounting between active drinkers with an alcohol dependence diagnosis (AUD+; n = 13) versus active drinkers without an alcohol dependence diagnosis (AUD-; n = 12). AUD+ showed more impulsive decision making and higher activity in executive control (dorsolateral prefrontal cortex) and attention (precuneus) regions than AUD- during delayed reward decisions. Although the study has a small sample size, was limited to men, but did compare drinkers with a control (non-drinking) group, this study provides important evidence of greater brain activity in executive control regions when alcoholics are required to delay gratifications, a finding that may reflect alcoholics’ increased demand of executive control when required to make decisions on behavior ruled by appetitive drive. These neural differences need to be explored further in larger samples that include a comparison to control groups.

Brain function associated with decision making in short-term abstinent alcoholics

Although it is important to understand the neural network differences in active drinkers, findings from these studies may be affected by the acute effects of alcohol and caution needs to be taken when comparing such results with those in abstinent treated samples. Active drinking samples may comprise a different population from treated samples - they may not have an alcohol use disorder (AUD) or have less severe AUDs than treatment samples (Fein and Landman, 2005, Di Sclafani et al., 2008, Fein et al., 2010a). The neural substrates of decision making have been extensively investigated in short-term abstinent alcoholics (STAA).

Park et al (2010) compared brain activity during reward-guided decision making between 20 STAA with at least 7 days of abstinence (mean = 16.9 days of abstinence) and 16 healthy controls. Subjects performed a task that required them to make a choice between two abstract stimuli to be allocated a probabilistically pre-determined reward. This task measured learning rates and outcome because subjects needed to update the reward values associated with the stimuli to guide their future responses. While STAA did not differ from controls in
striatal activity associated with predicting errors during task performance, they did show significantly lower fronto-striatal synchrony (i.e., ventral striatum and right dorsolateral prefrontal cortex) when presented with feedback of wins versus losses. Park et al (2010) suggest that while STAA do not have impaired brain activity associated with the representation of prediction error in the ventral striatum (nucleus accumbens), they do not seem to relay this information to brain regions that mediate higher executive control processes (dorsolateral prefrontal cortex - DLPFC) and are not able to integrate reward-related information for proper control of behavior following a reward (Park et al., 2010). While this study was limited to only men and to the examination of functional synchrony of the ventral striatum, it provides valuable evidence pointing to differences in functional synchrony between brain regions that mediate appetitive drive and regions that guide decision making in STAA versus healthy controls. The lack of interaction between these executive control and appetitive drive regions may underlie alcoholics’ difficulty in guiding future reward-related choices and goal-directed behavior. Furthermore, results from this study suggest that these neural network differences are evident after short periods of abstinence.

A study by Li et al (2009) investigated differences in brain activity specifically related to impulsive control and risk-taking between 24 STAA and 24 healthy controls using the stop-signal task (SST). By dissecting the component processes of SST performance (response inhibition, error processing, post-error slowing, risk taking) they could examine neural differences specific to discrete aspects of impulse control and risky behavior. Authors reported that while groups did not differ in overall SST performance, STAA compared to healthy controls showed significantly (1) lower left DLPFC activity when inhibiting a response, (2) lower right DLPFC activity during post-error slowing (behavioral adjustment after an error), (3) lower amygdala, striatal, and posterior cingulate cortex activity during post-trial speeding (representing a risk-taking decision), and (4) higher activity in visual cortex and anterior cingulate cortex (ACC) when failing to stop a response. Results from this study showed that even though overall performance did not differ between groups, STAA showed differences in brain activity during individual trials that reflect specific instances of poor decision making such as post-trial speeding and failure to stop a response.
Evidence from studies mentioned above suggest that poor decision making and risky behavior in STAA are associated with brain functional differences in regions within the executive control (lower activity) and appetitive drive (higher activity) networks known to be involved in cognitive and emotional control. Whether these brain functional differences predispose to alcohol problems or are a consequence of unhealthy alcohol use is not clear.

**Brain function associated with decision making in individuals at risk for alcoholism**

Neuroimaging studies on adolescents at risk for alcoholism have identified brain functional differences associated with poor decision making in such samples. A study by Acheson et al (2009) compared brain function between 15 adolescents with a family history of alcoholism (FHP) and 19 with a negative family history of alcoholism (FHN) when performing the Iowa Gambling Task. While groups did not differ in behavioral performance, the FHP group showed more activity in left dorsal anterior cingulate cortex (ACC) and left caudate nucleus than the FHN group. Because activity in dorsal ACC has been associated with risk prediction and activity in caudate nucleus with risky responses, Acheson et al (2009) suggest that FHP need to recruit additional brain regions for proper decision making involving risk-taking when compared to FHN. A study by Cservenka et al (2012) provided a different perspective on predisposing biological markers of alcoholism. They investigated whether there were differences in functional activity associated with risk-taking when FHP versus FHN adolescents performed the “Wheel of Fortune” decision making task (WOF). While there were no behavioral differences in risk-taking, FHP showed significantly lower activity in right dorsolateral prefrontal cortex (DLPFC) and right cerebellar tonsil than FHN in the risky versus safe decision making contrast. Authors suggest that attenuated DLPFC response to risk-taking in FHP may be associated with poorer cognitive control and hence poorer behavioral regulation in FHP, making them vulnerable to failure in the decision to avoid risk-taking and ultimately contributing to out of control alcohol consumption.

A study by Norman et al (2011) investigated the neural correlates of another aspect that contributes to risky behavior in adolescents, the lack of response inhibition. They examined brain function in adolescents while they performed a go/no-go task that measured response
inhibition and response selection. Based on follow-up interviews assessing alcohol use information (after a mean of 4.2 years), the sample was classified as either heavy alcohol users (n=21) or healthy controls (n=17). While there were no behavioral differences in impulse control during the go/no-go task at baseline, adolescents that later became heavy alcohol users showed significantly lower activity during no-go trials than healthy controls in prefrontal regions (inferior frontal gyrus, dorsolateral and medial frontal gyri), striatum (putamen) and inferior parietal lobules. The authors suggest that when required to exert control over a prepotent response, individuals that are at risk of becoming alcoholics show less responsive frontal regions even before the onset of drinking problems. Although these findings are important, they still do not provide decisive evidence for a predetermined predisposition because these may indicate that future problem drinkers have either (1) a pre-existing disadvantage (maybe genetic) due to poor engagement of frontal regions needed to properly exert executive control on impulsive and risky behaviors or (2) a delayed cortical maturity, in which activation in the executive control network is still not fully specialized yet, but never reaches its full potential because its development is later stunted by alcohol use. More studies need to be conducted to fully address these issues.

Brain function associated with decision making in long-term abstinent alcoholics

To investigate whether brain functional differences identified in actively drinking or recently abstinent alcoholics are permanent or can be overcome with long periods of abstinence, it is essential to examine long-term abstinent alcoholics. The examination of executive and appetitive drive networks during rest in long-term abstinent alcoholics (LTAA) has yielded results consistent with an ongoing compensatory mechanism in LTAA. A study by Camchong et al (2013a) that compared resting state synchrony of executive and appetitive drive networks between 23 LTAA (with more than 18 months of abstinence from alcohol use) and 23 healthy controls found that LTAA have (1) lower synchrony of appetitive drive regions and (2) greater synchrony of executive control regions than healthy controls. Moreover, these resting state synchrony differences were positively correlated with performance in a task that measured cognitive flexibility in decision making (e.g., higher resting state synchrony of
executive control regions significantly correlated with better performance). Results from this study reflect adaptive mechanisms in brain ongoing functional organization that may support proper executive control of behavior in long-term abstinent alcoholics and successfully stop behavior that may lead to relapse. A follow-up study by Camchong et al. (2013b) that studied this effect in STAA (6–15 weeks of abstinence) found ordered effects from STAA to LTAA within both the executive control and appetitive drive networks: higher resting state synchrony of the executive control network (Figure 1) and lower resting state synchrony of the reward processing network (Figure 2) than healthy controls. These results provided further evidence that this compensatory mechanism follows an adaptive progression from short- to long-term abstinence. Longitudinal studies of brain functional organization need to be conducted to explore the hypothesis suggesting that synchrony within the executive control network progressively increases and synchrony within the appetitive drive network progressively decreases with length of abstinence.

Differences in brain function and organization in prefrontal executive control regions are not specific to decision making and risky behavior in alcoholics. Alcoholics have shown similar differences as described above in neural activity in executive control regions (dorsolateral prefrontal cortex and anterior cingulate cortex) during tasks that hone in on other aspects of executive function such as spatial working memory (Pfefferbaum et al., 2001) or verbal working memory (Desmond et al., 2003, Cservenka and Nagel, 2012). Moreover, brain activity and functional organization differences associated with executive control differences have been identified in other samples with addiction to other substances such as cocaine and nicotine (Camchong et al., 2011, Gu et al., 2010, Janes et al., 2010).

Although neuroimaging studies have identified brain function and organization differences in executive and appetitive drive networks in alcoholics, these differences vary depending on whether the alcoholic is actively drinking or has been abstinent for short or extended periods of time. A number of studies reviewed in the sections above do not show performance differences between alcoholic and comparison groups, but instead find differences between groups in brain activation patterns associated with comparable
performance. Such findings are often interpreted as indicating lack of efficiency in alcoholics vs. controls of specific brain regions in performing some function. However, it is important to remember that such a statement is a hypothesis, not a finding. If an impairment in some function is present (less efficient processing by a brain region is an impairment), there should be experimental challenges that will result in impaired performance on some task. A recent paper by Chanraud et al (2013) found that while controls and recovering alcoholics had similar performance levels during a spatial working memory task, they recruited different brain networks. While controls recruited prefrontal-cerebellar regions known to mediate working memory, recovering alcoholics recruited two additional fronto-cerebellar networks, presumably to compensate and achieve normal working memory performance (Chanraud et al., 2013). It is also important to emphasize that the phenomena discussed above are not necessarily reflected in all aspects of behavior. For example, Fein et al. (2004) found impaired performance on the Iowa Gambling Task in long-term abstinent alcoholics – they were able to achieve multi-year abstinence despite the persistent decision making impairments on the Iowa Gambling Task. In contrast, Fein et. al. (2006) found normal Iowa Gambling Task performance in actively drinking treatment naïve alcoholics. These individuals drank harmfully (a poor decision) despite having normal Iowa Gambling Task performance. These examples point out that propensities and inherited vulnerabilities do not fully control behavior. The research reviewed above suggests some of the adaptive changes that may take place in the development of alcoholism and in the struggle to achieve long-term abstinence.
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still present in alcoholics after mid- to long-term abstinence. *Journal of Studies on Alcohol, 62*, 533-42.


Figure 1. (A) Three-dimensional MNI brain in neurological orientation with slices cut at $z = 21$ and $z = 43$ with region (in blue) that showed a linear trend (NSAC < STAA < LTA) in strength of resting-state synchrony in the executive control network. (B) Bar graph illustrating significant linear trend between groups in strength of RSS between subgenual anterior cingulate (sgACC) and right dorsolateral prefrontal cortex (DLPFC) in STAA (green bar), LTA (beige bar) and NSAC (blue bar). Red lines represent significant post-hoc differences between groups. MNI, Montreal Neurological Institute; LTA, long-term abstinent alcoholics; STAA, short-term abstinent alcoholics; NSAC, non-substance abusing controls.
Figure 2. (A) Three-dimensional MNI brain in neurological orientation with slice cut at z = 14 with region (in orange/red) that showed a linear trend (NSAC>STAA>LTA) in strength of resting-state synchrony in the appetitive drive network. (B) Bar graph illustrating significant linear trends between groups in strength of RSS between nucleus accumbens (NAcc) and appetitive drive regions in STAA (green bars), LTA (beige bars) and NSAC (blue bars). Red lines represent significant post-hoc differences between groups. MNI, Montreal Neurological Institute; LTA, long-term abstinent alcoholics; STAA, short-term abstinent alcoholics; NSAC, non-substance abusing controls.