Clinical Psychology Review 69 (2019) 83–96

Contents lists available at ScienceDirect

Clinical Psychology Review

journal homepage: www.elsevier.com/locate/clinpsychrev

Review

A dual process perspective on advances in cognitive science and alcohol use disorder

Kristen P. Lindgren[a,*], Christian S. Hendershot[b,c], Jason J. Ramirez[a], Edward Bernat[d], Mauricio Rangel-Gomez[d], Kirsten P. Peterson[a], James G. Murphy[e]

[a] Department of Psychiatry & Behavioral Sciences, University of Washington, Seattle, WA, USA
[b] Centre for Addiction and Mental Health, Toronto, Ontario, Canada
[c] Department of Psychiatry, University of Toronto, Toronto, Ontario, Canada
[d] Department of Psychology, University of Maryland, College Park, MD, USA
[e] Department of Psychology, University of Memphis, Memphis, TN, USA

HIGHLIGHTS

- We review recent cognitive science advances related to alcohol use disorders.
- We focus on four domains, including implicit cognitive bias and executive function.
- Behavioral economic approaches and functional connectivity are also reviewed.
- We discuss these domains in the context of dual process models.
- We discuss implications for theory and clinical practice.

ARTICLE INFO

Keywords:
Alcohol use disorder
Dual process models
Implicit cognition
Executive function
Behavioral economics
Functional networks

ABSTRACT

There is a tremendous global and national (US) burden associated with alcohol misuse and alcohol use disorder (AUD). Further, of the mental health disorders, AUD has the widest treatment gap. Thus, there is a critical need for improved understanding of the etiology, maintenance, and treatment of AUD. The application of cognitive science to the study of AUD has a longstanding history of attempting to meet this need. In this selective review, we identified and focused on four domains of *recent* (i.e., in the last decade) applications of cognitive science to the study of AUD: implicit cognitive biases, executive function, behavioral economic approaches to alcohol decision making, and functional connectivity neuroimaging. We highlighted advances within these four domains and considered them in the context of dual process models of addiction, which focus on the contribution and interplay of two complementary neurocognitive systems (impulsive and control systems). Findings across the domains were generally consistent with dual process models. They also suggest the need for further model refinements, including integrating behavioral economic approaches and findings from functional connectivity neuroimaging studies. Research evaluating candidate interventions associated with these domains is emergent but promising, suggesting important directions for future research.

1. Introduction

There is a tremendous global (Mokdad et al., 2016) and national (U.S. Department of Health and Human Services [HHS], 2016) health burden associated with alcohol misuse and alcohol use disorder (AUD). Alcohol ranks among the top five causes of premature deaths and disability, accounting for over 88,000 deaths, costs of $250 billion in the United States annually (HHS, 2016), and 5–6% of deaths and disability globally (World Health Organization, 2015). Particularly concerning are recent findings indicating a 30% increase in high-risk drinking and a 49% increase in AUD prevalence among U.S. adults over a 10-year period (Grant et al., 2017). Critically, global and national estimates show that 78% and 90% of those with AUD, respectively, do not receive care (HHS, 2016; Kohn, Saxena, Levav, & Saraceno, 2004).

Advances in clinical and cognitive psychology have contributed substantially to the understanding and treatment of alcohol misuse and AUD, with important developments emerging from research at the intersection of these areas. For example, current psychological and

* Corresponding author at: Department of Psychiatry & Behavioral Sciences, School of Medicine, University of Washington, 1100 NE 45th Street, Suite 300, Seattle, WA 98105, USA.
E-mail address: KPL9716@uw.edu (K.P. Lindgren).

https://doi.org/10.1016/j.cpr.2018.04.002
Received 1 September 2017; Received in revised form 4 April 2018; Accepted 5 April 2018
Available online 11 April 2018

neurobiological theories of addiction etiology have strong cognitive components (e.g., Koob & Volkow, 2010), and it has long been known that alcohol has acute and long-term effects on the brain and cognition, influencing AUD course and clinical outcomes (Bates, Buckman, & Nguyen, 2013; Oscar-Berman & Marinković, 2007). In recent years, an increasingly robust body of research has implicated cognitive processes in AUD nosology, assessment, and treatment (Kwako, Momenan, Litten, Koob, & Goldman, 2016; R. W. Wiers, Gladwin, Hofmann, Salemink, & Ridderinkhof, 2013).

Our goal is to provide a targeted review of recent applications of cognitive science to the study of alcohol misuse and AUD. We seek to provide a selective but representative review of key advances, with a primary emphasis on the past decade (2008–2017). An overarching development is the refinement of *dual process* theories of addiction (e.g., Bechara, 2005; Bickel, Quisenberry, Moody, & Wilson, 2015; Koob & Volkow, 2010; R. W. Wiers et al., 2013), which contend that decisional processes underlying drug seeking, and ultimately addiction, stem from the interplay of two complementary neurocognitive systems. Note that in prioritizing this framework, we forego reviews of several long-standing and influential applications of cognitive science in alcohol research such as expectancy theory (e.g., Cooper, Frone, Russell, & Mudar, 1995; Goldman, Brown, Christiansen, & Smith, 1991) and cognitive-behavioral therapy (CBT) in favor of emergent cognitive models and interventions.

1.1. Dual process models: history and application to addictive behaviors

There are multiple dual process models, with some having stronger roots in cognitive, social, and personality psychology (e.g., Strack & Deutsch, 2004) and others having stronger roots in neuroscience (e.g., Bechara, 2005). Generally, dual process models distinguish between two classes of cognitive processes—impulsive (reflexive/automatic/spontaneous) processes and control (reflective/slower/deliberative) processes—and view both as determinants of behavior. Although dual process accounts have historically been invoked to describe multiple determinants of behavior in cognitive and social psychology (see Strack & Deutsch, 2004), the adoption and elaboration of these models to explain substance use and addiction marked a shift in emphasis for cognitive research on AUD. Further, dual process models of alcohol use and addiction have increasingly focused on the interplay between the impulsive and control systems and changes in their relative influence over the addiction cycle (see Stacy & Wiers, 2010).

In parallel, neurobiological theories of addiction have progressively moved beyond a primary emphasis on midbrain systems to emphasize the role of prefrontal cortical (PFC) regions in drug-seeking behavior, as well as the interplay between the PFC and midbrain systems (Goldstein & Volkow, 2011; Koob & Volkow, 2010). The increased emphasis on this functional interplay, and changes in the relative influence of the two systems as a function of addiction progression (e.g., Bechara, 2005; Goldstein & Volkow, 2011), is largely consistent with dual process models adapted from cognitive and social psychology. In sum, both cognitive/social psychology and neurobiological theories converged to emphasize the functional interplay between impulsive systems and control systems—and longitudinal changes in their relative influence as a function of addiction progression—as explanatory frameworks for the maintenance of drug seeking behavior.

Because the control system is viewed as functioning as a "brake" on the influence of impulsive processes, a key hypothesis under dual process models is that the impulsive system should be more influential on behavior under conditions when the control system is weak or impaired, and less influential when the control system is strong or intact (see Stacy & Wiers, 2010). Thus, both states and traits (e.g., low state or trait self-control) are hypothesized to moderate the influence of the impulsive system on alcohol consumption. Importantly, dual process models also posit a developmental shift in the balance between the impulsive and control systems as a function of addiction progression, with the impulsive system strengthening and the control system weakening, thereby perpetuating the cycle of continued drug use despite an increasing number of negative consequences.

From a neuroscience perspective, the adverse effects of chronic substance use are presumed to further undermine the efficiency of control networks, weakening the capacity for self-regulation in the face of drug cues and exacerbating this cycle (see Koob & Volkow, 2010). Dual process models also converge with incentive salience models of addiction (Robinson & Berridge, 1993), which propose that repeated exposure to drugs leads to neural sensitization wherein appetitive responses to drug cues become increasingly automatic and drug use itself becomes more compulsive. A recent meta-analysis of functional magnetic resonance imaging (fMRI) studies evaluating brain responses to drug cues indicated strong overlap in the areas activated for alcohol and other drugs, pointing to the ventral striatum, anterior cingulate cortex, and amygdala, and also suggested that cue-reactivity indexes craving (Kühn & Gallinat, 2011). Further, as addiction progresses, responsivity to alcohol cues seems to shift from the ventral to dorsal striatum, suggesting the possibility of dissociating hedonic ("liking") drinking from compulsive ("wanting") drinking (Vollstädt-Klein et al., 2010).

1.2. Dual process models: four exemplar domains

In this review, we focus on four exemplar areas of research that link to dual process models, have a robust body of research with novel, clinically relevant findings, and have the potential to yield novel treatment targets and/or interventions: *implicit cognitive biases*, *executive function*, *behavioral economic approaches to alcohol decision making*, and *functional connectivity imaging studies*. With respect to dual process models, *implicit cognitive biases* can be understood as part of the impulsive system, whereas *executive function* can be understood as part of the control system. While implicit biases and executive function are routinely considered in reviews about dual process models, we highlight recent advances related to improving their measurement, their utility as prospective predictors, and developing paradigms to target these processes clinically. *Behavioral economic approaches* to understanding alcohol-related decision making have seen particular growth in the last decade and have led to novel assessment and treatment approaches that can be integrated within an integrated within dual process framework. Finally, advances in cognitive neuroscience have been pervasive in alcohol research, and we highlight *functional connectivity imaging studies* as a means of investigating hypotheses derived from dual process models. Across these domains, we, therefore, focus on recent studies that sought to clarify when and how these classes of cognitive factors predict alcohol misuse and AUD and on novel interventions that seek to intervene at the level of these cognitive factors. Rather than attempt an exhaustive review, we highlight representative findings, discuss the implications of these findings for theory and practice, and identify critical unanswered questions. Supplemental Table 1 contains information about key measures from these four domains.

2. Advances in implicit cognitive biases

We begin with implicit cognitive biases, part of the impulsive system. Three classes of implicit cognitive biases are commonly assessed by alcohol researchers: memory (association) biases, attentional bias, and automatic action tendencies (Stacy & Wiers, 2010). We also note the emergence of interpretation bias measures (Woud, Fitzgerald, Wiers, Rinck, & Becker, 2012), which we view as a natural extension of memory biases. As discussed elsewhere (see C.E. Wiers et al., 2017), these classes of biases can be conceptualized as having a common underlying neural mechanism, tied to the release of dopamine in the mesolimbic area of the brain from alcohol (or drug) use. Repeated alcohol use results in increasing sensitization in this region, and ultimately results in alcohol cues attracting increased attention and evoking stronger approach responses. Thus, implicit cognitive biases

are commonly described as being activated automatically (i.e., spontaneously and without conscious control). While the classes of implicit cognitive biases are expected to be correlated with one another (see C. E. Wiers et al., 2017, for a rare examination of this question), they are thought to be conceptually distinct with (i) *memory biases* referring to the extent to which mental constructs become associated with alcohol (e.g., more strongly associating alcohol with good [vs. bad]); (ii) *interpretation biases* referring to the extent to which ambiguous situations are interpreted to be alcohol-related; (iii) *automatic action tendencies* referring to the extent to which there is a stronger approach (vs. avoid) response to alcohol-related cues; and (iv) *attentional bias* referring to the greater proportional allocation of attention to alcohol (vs. non-alcohol) cues. While implicit cognitive biases are typically viewed as mechanisms of drinking, we note an emergent discussion of the possibility that they may be markers of other underlying processes (see Field, Di Lemma, Christiansen, & Dickson, 2017).

The majority of measures assessing implicit biases have been in use since the mid-2000's (as compared to, for example, self-report measures of alcohol expectancies that originated in the 1980's). Measures assessing these biases fall in two categories: computer-based reaction time (RT) tasks (wherein faster responding to an alcohol-related stimulus vs. a non-alcohol-related stimulus serves as a proxy for a stronger implicit alcohol-related bias) and free association tasks (wherein generating an alcohol-related response to an ambiguous stimulus serves as a proxy for a stronger implicit alcohol-related bias). Critically, measures of implicit biases are indirect measures—that is, responses to the measures are used to *infer* the underlying cognitive/mental content (see Nosek, Hawkins, & Frazier, 2011)—versus self-report measures, which assess target behaviors relatively more directly. Measures of implicit bias, especially those relying on RT, tend to have lower internal consistency and test-retest reliability as well as smaller effect sizes when compared to self-report questionnaires that assess cognitive constructs (see Ataya et al., 2012; Reich, Below, & Goldman, 2010). Measures of implicit biases have no clinical cutoff scores and are not used for diagnostic purposes. While these are important limitations, we note the relative youth of implicit measures and their capability to predict individual differences in alcohol outcomes after controlling for established self-report measures (see Reich et al., 2010).

In the last 10 years, promising advances have included the development and validation of novel measures to assess (i) memory biases related to alcohol, drinking, and the self (i.e., implicit drinking identity; see Lindgren, Neighbors, Gasser, Ramirez, & Cvencek, 2017) and (ii) alcohol-related interpretation biases (see Salemink & Wiers, 2014; Woud et al., 2012). In addition, recent research has also seen the application of advanced statistical models, including those based on item response theory (IRT) and multinomial models (e.g., the quadruple process model; Conrey, Sherman, Gawronski, Hugenberg, & Groom, 2005). IRT has been used to improve the measurement of memory biases assessed via free response (see Shono, Grenard, Ames, & Stacy, 2014), whereas the quad model has been used to disentangle the underlying (impulsive and control) processes in RT measures of memory bias, as measures of implicit biases are not process-pure (see O'Connor, Lopez-Vergara, & Colder, 2012). fMRI has also been used to study neural correlates of RT measures of memory bias (see Ames et al., 2014), and electroencephalographic (EEG) measures have been used to evaluate neurocognitive correlates of measures of automatic action tendencies and their relation to drinking escalation (Korucuoglu, Gladwin, & Wiers, 2014, 2016).

Advances in measures of attentional bias include the integration of eye-tracking technology with extant attentional bias measures (e.g., visual probe tasks). These studies have demonstrated superior internal reliability of gaze dwell times as compared to RTs in a visual probe task (Christiansen, Mansfield, Duckworth, Field, & Jones, 2015) and are providing insight into the specific processes that underlie attentional bias, including fixation time on alcohol-related images, initial orienting bias to alcohol, and their relation to alcohol consumption (Melaugh McAteer, Curran, & Hanna, 2015). Recent work focusing on within-person variability on the visual probe task has yielded preliminary, yet intriguing, findings that this variability is positively associated with hazardous drinking, suggesting that this variability may reflect a combination of attend and avoid biases among heavy drinkers (Gladwin, 2017).

A second key advance is prospective research that provides critical information about the development of implicit cognitive biases as well as their relation to the initiation and/or escalation of drinking. This is an emergent area, with relatively few independent samples and one that, to our knowledge, consists only of child, adolescent, and college student samples. Results suggest that adolescents' memory biases can predate the initiation of drinking—supporting theory (see Greenwald & Banaji, 1995) that memory biases reflect cultural and environmental factors in addition to actual (drinking) experience—and that those biases mediate the relationship between parental drinking and subsequent drinking by adolescents (Van Der Vorst et al., 2013). Relatedly, two other studies indicate that baseline memory bias in adolescents (Pieters, Burk, Van der Vorst, Engels, & Wiers, 2014) and changes in memory bias in children (Colder et al., 2014) are not associated with subsequent changes in their drinking, further suggesting that alcohol memory biases may initially serve as markers of exposure to and experience with drinking (vs. drivers of drinking). Evidence for memory biases serving as predictors of subsequent drinking has been found in high-risk (but not general community) adolescent samples (Ames, Xie, Shono, & Stacy, 2017) and adolescents who also endorsed in-home alcohol availability (Peeters, Koning, Monshouwer, Vollebergh, & Wiers, 2016). Another study with college students found that memory biases predicted increases in drinking over time (Lindgren et al., 2016) and observed bi-directional relationships between increases in memory biases and increases in risk for hazardous drinking (Lindgren, Baldwin, et al., 2017).

Prospective studies that examine attentional bias and automatic action tendencies have produced mixed findings. Studies have found (i) that automatic action tendencies, but not attentional biases, predict changes in alcohol use among adolescents who have weak negative alcohol-related expectancies (Pieters et al., 2014), (ii) that attentional biases, but not automatic action tendencies, prospectively predict adolescent drinking (Janssen, Larsen, Vollebergh, & Wiers, 2015), and (iii) that changes in automatic action tendencies over time are not associated with changes in adolescent drinking (Janssen, Wood, et al., 2015). These discrepant findings may be related to methodological differences or may suggest that the predictive relationship between implicit biases and drinking strengthens throughout development perhaps due to increasing alcohol exposure/availability and individual autonomy.

A third advance is research that tests a central tenet of the dual process model by evaluating interactions between implicit cognitive biases and the control system, with a stronger control system posited to weaken the positive relationships between implicit biases and drinking. Supporting this hypothesis, high scores on neurocognitive or self-report measures of executive function and self-control have been found to weaken the positive relationship between measures of implicit bias and drinking (for review, see R. W. Wiers et al., 2013). However, studies have also yielded null or inconsistent findings across three of the four implicit bias domains (memory bias: Ames et al., 2017; attentional bias: van Hemel-Ruiter, Wiers, Brook, & de Jong, 2016; automatic action tendencies: Christiansen, Cole, Goudie, & Field, 2012). While this research represents an important advance, the mixed findings across the implicit biases combined with the relatively narrow age range of study samples (typically, adolescents or college students) suggest the need for studies with broader samples as well as for the use of meta-analytic approaches.

The final advance we highlight is the development of candidate interventions that seek to change or modify those processes (i.e., cognitive bias modification or CBM). CBM studies have taken two forms:

clinical trials with patients (e.g., R. W. Wiers, Eberl, Rinck, Becker, & Lindenmeyer, 2011) and proof-of-concept studies with college students (e.g., R. W. Wiers, Rinck, Kordts, Houben, & Strack, 2010). Most CBM interventions focus on modifying attentional biases and automatic action tendencies. Interventions typically consist of modified versions of paradigms used to assess these biases; for example, visual probe tasks (to retrain attentional bias) modified such that visual probe cues follow neutral images (vs. alcohol-related images) with greater frequency, and approach avoidance tasks (to retrain automatic action tendencies) modified such that individuals practice avoiding alcohol-related cues with greater frequency. Recent studies have extended CBM inventions to interpretation biases in college students (e.g., Woud, Hutschemaekers, Rinck, & Becker, 2015) and show some evidence of short-term changes in the targeted bias but little evidence of long-term changes in the targeted bias, other implicit biases, or drinking.

Although initial applications of CBM were promising (R. W. Wiers et al., 2010; Wiers et al., 2011), a recent review (Christiansen, Schoenmakers, & Field, 2014) and meta-analysis (Cristea, Kok, & Cuijpers, 2016) of attentional bias and automatic action tendencies have been less enthusiastic. The meta-analysis included 17 alcohol-related studies and concluded that CBM paradigms had a small, significant effect on modifying targeted biases but that they did not lead to significant changes in substance outcomes or craving. Among the challenges of interpreting this meta-analysis is that it included both proof-of-concept studies with heavy-drinking college students and clinical trials with alcohol-dependent patients (R. W. Wiers, Boffo, & Field, 2018), and studies in which the CBM paradigms failed to shift the targeted bias, which has been argued elsewhere (Clarke, Notebaert, & MacLeod, 2014) to be an indication of an ineffective paradigm and not that CBM itself is ineffective. A key next step is to test whether CBM effects on clinical outcomes are mediated by changes in the targeted bias. We know of no published studies that do so for attention bias and two that do so for automatic action tendencies. Findings are mixed (evidence for mediation: Eberl et al., 2013; null findings: R. W. Wiers et al., 2011). Of note, Eberl et al. (2013) used CBM as an add-on to ongoing treatment (CBT), perhaps suggesting greater potential for CBM, at least with current paradigms, as an add-on [vs. stand-alone] intervention (see also R. W. Wiers et al., 2018).

In sum, the proliferation of implicit cognition research represents an important development in cognitive science research on AUD. Overall, this literature implicates implicit cognitive biases as potentially important markers of AUD risk and progression, and potential determinants of drinking behavior. Moreover, the introduction of implicit cognitive biases as potential intervention targets has generated substantial interest. Despite these advances, several caveats should be noted, including inconsistencies in the extent to which different variants of implicit cognition relate to clinically relevant outcomes, especially during adolescence. Despite some promising evidence for implicit biases as malleable intervention targets, findings are mixed regarding the efficacy of current CBM paradigms to modify the targeted bias and to alter clinical outcomes. Given the youth of these measures, the challenges inherent to developing reliable measures of implicit biases (Ataya et al., 2012), and the likelihood of small effect sizes in relating these measures to clinical outcomes, these inconsistencies might be expected. Nonetheless, critical next steps are to ascertain the reliability and generalizability of existing findings and improve existing or develop novel paradigms for bias assessment and bias modification.

3. Advances in executive function (EF)

The executive functions (EFs) comprise a group of related yet distinguishable cognitive processes undergirding higher-order cognition, self-regulation and goal-directed behavior. Viewed as central to the control system, EFs are emphasized in most cognitive and neurobiological models of addiction (e.g., Bechara, 2005; Koob & Volkow, 2010). As noted, dual process models specify that state or trait deficits in EF should promote a greater relative influence of impulsive processes on craving or consumption. These theories also predict ongoing diminishment in control systems as influencing behavior during the progression from initial use to dependence (e.g., Bechara, 2005; Bickel et al., 2015; Koob & Volkow, 2010; R. W. Wiers et al., 2013). Theories of genetic and neurodevelopmental liability for AUD (Zucker, Donovan, Masten, Mattson, & Moss, 2008), acute alcohol effects on decision-making (Field, Wiers, Christiansen, Fillmore, & Verster, 2010), and behavioral economics of addiction (Bickel et al., 2015) similarly specify a central role for EF as a mechanism that mediates dispositional and contextual risk for drinking. In short, EFs are central to most concepts emphasized in this review. We highlight advances in measurement models of EF, knowledge about acute alcohol effects on EF, studies of neurocognitive and clinical correlates, and the development and application of interventions intended to bolster or repair EFs.

EFs have been classified at various levels of resolution. An influential taxonomy (Miyake & Friedman, 2012) emphasizes three related yet separable functions: inhibition, working memory (also called updating), and set shifting. A key advance has been the use of latent variable modeling to extract common variance across these EF domains, resulting in a latent factor for “common EF” (Miyake & Friedman, 2012). Twin studies indicate that individual variation in common EF is almost entirely genetic in origin (Friedman et al., 2008). Common genetic influences on EF and substance use appear to overlap with genetic liability for externalizing traits generally, implicating lower EF abilities as an antecedent risk factor for AUD that relates to behavioral undercontrol (Gustavson et al., 2017). A cross-sectional analysis found that a latent index of common EF abilities related inversely to number of drugs used and frequency of use (but not dependence symptoms) during adolescence, and suggested that these associations were attributable to shared genetic influences (Gustavson et al., 2017).

Inhibition (also termed *response inhibition* or *inhibitory control*) refers to the ability to deliberately withhold a prepotent behavioral response, as commonly indexed by Stop Signal Task or Go/No-Go tasks. Recent years have seen a proliferation of studies examining inhibition in the context of AUD. Inhibition deficits appear to anticipate the onset of heavy drinking, the transition to AUD, and likelihood of relapse, with response inhibition deficits also following as a consequence of chronic consumption (reviewed in Field & Jones, 2017). A recent meta-analysis demonstrated relative deficits in inhibitory control in AUD populations vs. controls (Smith, Mattick, Jamadar, & Iredale, 2014).

Acute intoxication reliably impairs inhibition, representing one cognitive mechanism for impaired control over alcohol (Field et al., 2010). Functional imaging studies indicate that diminished activation in task-relevant brain regions during inhibition relates both to self-reported impaired control (Weafer et al., 2015) and the onset of heavy drinking in adolescence (Norman et al., 2011). Further, greater AUD severity relates to diminished neural response in brain regions central to inhibition and error monitoring (Claus, Feldstein Ewing, Filbey, & Hutchison, 2013). Poorer inhibition also appears related to incentive salience for alcohol, based on its association with cue-induced craving (Papachristou et al., 2013). Recent findings suggest the mere presence of alcohol cues can reduce inhibitory control, which may partly mediate the effect of cue exposure on consumption (Field & Jones, 2017).

Working memory (WM) refers to “a limited capacity system, which temporarily maintains and stores information, (and) supports human thought processes by providing an interface between perception, long-term memory and action” (Baddeley, 2003, p. 829). Most models of WM emphasize short-term retention and manipulation of information in service of goal-directed behavior, and the ability to exert top-down cognitive control in the face of task interference, as core processes (e.g., Hofmann, Schmeichel, & Baddeley, 2012). In a prospective study of community youth, lower scores on a latent index of WM predicted initial alcohol use and greater increases in use over 4 years, whereas increases in drinking did not predict change in WM (Khurana et al., 2013). Notably, associations of the WM index with drinking escalation

were mediated by impulsivity traits, including delay discounting (Khurana et al., 2013). This finding appears consistent with apparent functional overlap in cortical regions related to delay discounting and WM (Bickel, Moody, & Quisenberry, 2014). Functional imaging studies have also reported differential engagement of WM task-relevant regions as a function of AUD status (Wesley, Lile, Fillmore, & Porrino, 2017). In one study, differential activation in task-relevant regions during a WM task predicted relapse prospectively (Charlet et al., 2014).

Shifting (or set shifting) refers to the ability to alternate flexibly between tasks or response sets in accordance with changing contingencies or motivational states (Hofmann et al., 2012). Notably, whereas better WM and inhibition abilities typically index reduced impulsivity, the opposite association is observed for shifting (Miyake & Friedman, 2012). Implications of shifting abilities for self-regulation are thought to be context-dependent (Hofmann et al., 2012). Relative to inhibition and WM, comparatively few alcohol studies have focused on shifting.

A second set of advances are studies of acute alcohol effects on EFs. Acute alcohol reliably impairs inhibitory control, whereas evidence is reliable (but limited) for set shifting, and mixed for WM (Day, Kahler, Ahern, & Clark, 2015). Notably, relatively greater alcohol-induced deficits in inhibitory control predict greater laboratory consumption (Weafer & Fillmore, 2008), and lower WM performance during intoxication is also found to predict alcohol-related problems, with drinking quantity mediating this effect (Lechner, Day, Metrik, Leventhal, & Kahler, 2016). Most alcohol administration studies are limited by emphasizing a single EF. However, a recent study (Korucuoglu et al., 2017) derived a latent index of shifting ability from three tasks, showing alcohol-induced deficits (and acute tolerance) for shifting ability. Additionally, those with lower baseline shifting ability showed greater alcohol-induced impairment (Korucuoglu et al., 2017). Notably, individual differences such as personality traits and ADHD symptoms can moderate acute alcohol effects on cognitive control (Day et al., 2015). Also, perhaps consistent with dual process models, baseline WM capacity moderated the effects of intoxication on neural responses during inhibition, with lower WM predicting alcohol-related de-activation in task-relevant regions (Claus & Hendershot, 2015).

A third set of advances includes research evaluating clinical and neuroimaging correlates of EF. Generally, 50–70% of those with AUD show some degree of cognitive impairment relative to control groups (Bates et al., 2013). Neuroimaging evidence consistently supports reduced cortical volumes as a function of chronic alcohol use, with some findings suggesting greater vulnerability in frontal cortical regions (Oscar-Berman & Marinković, 2007). Importantly, structural and functional deficits tend to show recovery, particularly during early abstinence, with graded recovery over longer periods (Bates et al., 2013). A meta-analysis (Stavro, Pelletier, & Potvin, 2013) found that alcohol-dependent groups (vs. controls) showed significant deficits across cognitive domains (including those related to EF), with some evidence for smaller effect sizes after long-term abstinence, potentially reflecting ongoing recovery. While evidence supports some recovery of EFs after abstinence (Oscar-Berman & Marinković, 2007), variability in the rate and extent of recovery may be considerable (Bates et al., 2013).

A final key advance is the development of interventions that seek to improve EFs. From a dual process standpoint, interventions that bolster or repair EFs can theoretically improve cognitive control and self-regulation, perhaps reducing the relative influence of impulsive processes on behavior (Bickel et al., 2015; R. W. Wiers et al., 2013). Notably, EF is proposed as a common or trans-disease intervention target across AUD and other addictive or impulse control disorders (Bickel et al., 2015). Cognitive training paradigms can broadly be divided into domain-specific (for example, CBM for implicit biases related to alcohol, as reviewed above) or domain-general interventions (Morrison & Chein, 2011). Paradigms intended to improve WM (a domain-general intervention) and inhibitory control (a domain-specific intervention) have been developed and are being tested.

There is some evidence that WM training can improve WM and potentially affect drinking outcomes. For example, adaptive (relative to non-adaptive) WM training resulted in higher WM gains and greater decreases in drinks per week among participants completing 20–25 web-based WM training sessions (Houben, Wiers, & Jansen, 2011). Notably, the mediated (indirect) effect of WM training on alcohol consumption via working memory improvements appeared stronger for participants with stronger positive implicit alcohol associations, perhaps consistent with the idea that strengthening EFs could reduce the influence of impulsive processes on craving or consumption (Houben et al., 2011). Another study found that WM training reduced delay discounting (Bickel, Yi, Landes, Hill, & Baxter, 2011). Study sample sizes are generally small, and evidence that WM training relates to changes in clinical outcomes is isolated (Houben et al., 2011), consistent with the broader WM training literature (Morrison & Chein, 2011). However, the theoretical relevance of WM training for AUD and the few studies to date suggest that further research is warranted (Bickel, Moody, & Quisenberry, 2014; R. W. Wiers et al., 2013).

Given the association of inhibition deficits with AUD severity, craving, and consumption (Field & Jones, 2017), inhibitory control training may be a promising intervention element. Notably, two meta-analyses support a significant, moderate (d = 0.40–0.45) effect of single-session inhibitory control training on immediate laboratory alcohol consumption (Allom, Mullan, & Hagger, 2016; Jones et al., 2016), supporting short-term effects of inhibitory control training on alcohol intake. Moreover, a recent study found that single-session inhibitory control training or CBM for automatic action tendencies yielded comparable reductions in laboratory alcohol consumption; however, evidence for intervention effect on targeted cognitive processes was inconclusive, and neither training type produced changes in implicit alcohol associations (Di Lemma & Field, 2017).

In sum, individual and contextual differences in EF abilities are important for alcohol misuse and AUD. In youth, lower EF ability is a risk factor for substance use, partly reflecting shared genetic influences. Theory and evidence also support declines in EFs with ongoing alcohol consumption, with recent evidence showing that even modest consumption can relate to structural brain changes (Topiwala et al., 2017). At the level of drinking episodes, situational fluctuations in EFs are considered critical for alcohol-related decision-making. Jointly, these considerations support efforts to therapeutically target EFs via cognitive training. Despite their theoretical appeal, evidence for intervention efficacy remains limited, although inhibitory control training is associated with immediate reductions in laboratory consumption among social drinkers. Among the limitations of AUD research focusing on EFs is the tendency to focus on individual constructs in isolation, despite the methodological advantages of larger measurement batteries (e.g., Miyake & Friedman, 2012). Also, relatively few studies incorporate both traditional EF tasks and implicit tasks (perhaps limiting opportunities to test dual-process hypotheses), and many findings reviewed above involve cross-sectional data.

4. Advances in behavioral economic approaches to understanding alcohol decision making

Behavioral economics is a translational research approach that integrates theoretical and methodological elements from microeconomics, psychology, and behavioral pharmacology (Bickel, Johnson, Koffarnus, MacKillop, & Murphy, 2014; Vuchinich & Tucker, 1988) to describe the cognitive, behavioral, and contextual processes that underlie decisions about how much and how often people drink. Alcohol misuse is viewed as a *reinforcer pathology* characterized by an overvaluation of alcohol as a reward relative to other available rewards (Bickel, Johnson, et al., 2014). Thus, although the pharmacological properties of alcohol are understood to be highly relevant to its appeal and abuse potential, decisions about how much and how often to drink are also assumed to be strongly influenced by contextual features such

as constraints on access to alcohol (i.e., alcohol availability and price) and other activities that might generate reward and maintain consistent patterns of behavior (e.g., employment, exercise, education, social relationships, hobbies). Choice between alcohol and alternatives is assumed to be influenced by the *temporal window* of reward receipt for the choices available in an individual's environment (Bickel, 2015). Whereas alcohol provides a reliable, immediate reward with minimal response cost, alternatives to alcohol often require sustained effort over time and are associated with delayed rewards (e.g., salutary social, vocational, and health outcomes).

A key advance in behavioral economics is research testing whether the rewarding or motivational properties of alcohol may exceed that of most available drug-free rewards among individuals who misuse alcohol, leading to increasing relative valuation of alcohol (Vuchinich & Heather, 2003). A recent study with college student heavy drinkers found that AUD symptoms were uniquely associated with diminished environmental reward availability above and beyond drinking level and depression, but that ability to experience reward was intact (Joyner et al., 2016). Other research suggests that chronic alcohol and other drug use may also lead to a diminished capacity to experience natural reward. An fMRI study found that individuals with drug dependence exhibited diminished neural activation to nondrug rewards (Lubman et al., 2009), and a study with college students found that prescription opiate misuse is associated with diminished subjective response to pleasant drug-free stimuli (Meshesha, Pickover, Teeters, & Murphy, 2017). Thus, frequent alcohol and drug use may lead to a self-perpetuating process in which neurobiological changes diminish the individual's sensitivity to substance-free natural rewards like sex, food, or exercise, which in turn increases the relative preference for alcohol (Volkow & Baler, 2015).

A second advance has been the development and validation of self-report measures to assess alcohol's *reinforcing efficacy* (also referred to as *reinforcing value*). In laboratory settings, reinforcing efficacy is quantified by the amount of behavior (e.g., lever presses, time) allocated to gain access to the reinforcer. In clinical settings, self-reports of recent patterns of activity participation and enjoyment related to alcohol use versus alcohol-free activities can assess this construct (Dennhardt, Yurasek, & Murphy, 2015). Additionally, hypothetical alcohol purchase tasks (APTs) use *demand curve* approaches that plot consumption level (and associated expenditures) as a function of drink price. APTs yield multiple indices, including peak consumption at minimum price (intensity), the price that reduces consumption to zero (breakpoint), maximum expenditure (O_{max}), the price at which demand initially decreases (P_{max}), and the aggregate slope of the demand curve (elasticity). Findings indicate that these indices are reliable and valid: They correlate with lab-based alcohol consumption and traditional measures of alcohol problem severity (e.g., AUD symptoms, craving, and drinking and driving; Amlung et al., 2016; Teeters & Murphy, 2015), comorbid substance misuse (Yurasek et al., 2011), impulsivity (Amlung et al., 2013), and symptoms of depression and PTSD (Murphy et al., 2013).

Other validation studies have focused on the link between demand and treatment outcomes and the assessment of neurocognitive correlates of demand. Regarding the former, studies have found that elevated baseline levels of alcohol demand predict poor response to brief alcohol interventions, that both behavioral and pharmacological treatments reduce demand, and that treatment-related change in demand predicts change in drinking (Bujarski, MacKillop, & Ray, 2012; Dennhardt et al., 2015; Murphy et al., 2015). Moreover, a recent translational study that examined change in demand for cocaine among rats injected with oxytocin found similar results (Bentzley, Jhou, & Aston-Jones, 2014), suggesting that demand may be a cross-species marker of addiction propensity and response to treatment. Regarding the latter, MacKillop et al. (2014) used a modified APT administered in an fMRI paradigm and found that during decisions to drink at low prices (relatively unconstrained consumption), greater activity was present in multiple distinct subunits of the prefrontal and parietal cortices. In contrast, during decisions to drink in the more elastic (price sensitive) portion of the demand curve, significantly greater activation was evident in frontostriatal regions. These results indicated that demand curves may model both the approach and restraint elements of dual process models.

A third advance is evidence that reward delay strongly influences the subjective value of the various activities available in an individual's environment, and that many of the apparently irrational decisions that often occur when one decides between competing rewards that differ in delay may be due to a unique mathematic function that characterizes the decay in reward value associated with delay (i.e., delay discounting; Bickel, Johnson, et al., 2014). Early laboratory work with human and non-human animals carefully described the hyperbolic form of the discount function: Short delays are associated with disproportionate decreases in reward value, which may account for the dynamic inconsistencies in choices and the common self-control failures that characterize AUD and addiction (e.g., preferring the larger delayed rewards associated with sobriety in the morning but shifting preference to the smaller reward of alcohol use when it is immediately available; Rachlin, Logue, Gibbon, & Frankel, 1986). The development of hypothetical money choice measures of delay discounting, which have strong test-retest reliability and are correlated with tasks that involve decisions about actual money amounts (Bickel, Johnson, et al., 2014), has facilitated translational clinical applications of behavioral economics. These tasks provide a series of choices between a larger fixed amount reward that is delayed (e.g., $100 available in 6 months) and a smaller immediate reward that is systematically varied to establish the immediate subjective equivalent of the larger delayed reward (e.g., $59 available today, $55 available today, $50 available today). Choice of the smaller, sooner reinforcer is considered “impulsive,” and choice of the larger later reinforcer is considered “self-controlled.” The choices generate estimates of the subjective immediate value of delayed monetary amounts, and these points are used to estimate a discount function (k), which reflects the degree of discounting.

A number of studies have established that although the value of all rewards decreases as their receipt is delayed, there are substantial individual differences in the degree that delayed rewards are discounted, and this discounting phenomenon may be a core feature of substance abuse (Bickel, 2015). Whereas substance use generally provides immediate reinforcement (e.g. anxiety reduction, euphoria, social facilitation), many substance-free activities (e.g. attending class, studying) do not (Murphy, Barnett, & Colby, 2006). For example, teens and young adults who sharply discount the value of delayed academic and career outcomes may be less likely to engage in the behaviors necessary for success in these domains (e.g. attending class, work productivity, learning/skill development), and more likely to engage in immediately reinforcing activities (e.g., drinking, drug use). Numerous studies have demonstrated that delay discounting is associated with continuous measures of alcohol and drug abuse severity (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017), and increases in response to laboratory stress and acute craving manipulations, withdrawal, and chronic drug use (reviewed by Bickel, Johnson, et al., 2014). Delay discounting also predicts the development of substance use (in humans and animals; Anker, Perry, Gliddon, & Carroll, 2009; Audrain-McGovern et al., 2009) and changes in substance use over time (Tucker, Roth, Vignolo, & Westfall, 2009), including response to treatment (Stanger et al., 2012).

A fourth advance in behavioral economics concerns identifying unique targets for clinical intervention. For example, an assumption of behavioral economics is that alcohol intervention approaches should also attempt to increase future orientation (reduce delay discounting) and engagement in substance-free alternatives to drinking (Koffarnus, Jarmolowicz, Mueller, & Bickel, 2013; Murphy & Dennhardt, 2016). There are evidence-based behavior therapies such as Community Reinforcement and Contingency Management that explicitly focus on developing alternatives to drinking (Carroll et al., 2012), and treatments such as behavioral activation (originally developed to treat

depression) that have been modified to address alcohol and drug misuse (Daughters et al., 2008). Similarly, the addition of a behavioral economic "Substance-Free Activity Session" that attempts to increase future orientation and engagement in goal-directed and value-consistent activity patterns was found to enhance the efficacy of a standard alcohol motivational interview (relative to control) in a heavy-drinking college student sample (Murphy et al., 2012).

In sum, behavioral economic research suggests that a comprehensive analysis of alcohol-related decision making (Kwako et al., 2016) should assess not only the valuation of alcohol but also the valuation of alternative activities, including activities associated with delayed rewards. Behavioral economic indices of alcohol reinforcing efficacy measure strength of motivation for alcohol by quantifying cost-benefit decision-making and relative alcohol-related and alcohol-free activity engagement and enjoyment. These translational indices are unique predictors of alcohol misuse severity and response to treatment. In addition, behavioral economics has led to the identification of additional treatment targets, including increased future orientation and engagement in substance-free alternatives to drinking.

5. Advances from functional connectivity imaging studies

Finally, we turn to recent advances in cognitive neuroscience focused on elucidating functional networks in the brain that are germane to the dual process model. Connectivity measures used to make inferences about brain networks can be described in terms of structural, functional, and model-based effective connectivity (Friston, 1994, 2011). Structural and functional connectivity are becoming widely reported in the current literature, providing valuable descriptions of network structure and function. This new level of description provides new opportunities to assess the nature of effective connectivity within and between these networks. Recent work suggests the presence of three broad functional brain networks that represent processes underlying a broad array of core cognitive processing (Bressler & Menon, 2010): The salience network, the control network, and the default mode network. The salience network and control network are most active during focused task performance (task-positive), relative to the default mode network, which is most active when not engaged in goal-directed behavior (task-negative) (Bressler & Menon, 2010; Park & Friston, 2013). The primary function of the salience network is to scan for the anticipation of reward, or value, and detection of potential danger. This centrally involves the anterior cingulate cortex, amygdala, and the major dopaminergic structures of the brain (Seeley et al., 2007). The salience network is functionally integrated with other brain structures that collectively constitute the control network, notably the lateral PFC, orbitofrontal PFC, and inferior frontal gyrus (Miller & Cohen, 2001; Seeley et al., 2007). In coordination with value and attention represented in the salience network, the control network becomes active to modulate and guide responding.

The default mode network corresponds to stimulus-independent thought that is detached from the external environment, including mental explorations based on personal introspection, autobiographical memories, and thoughts of the future (Buckner, Andrews-Hanna, & Schacter, 2008; Spreng, Mar, & Kim, 2009). Areas making up the default mode network include the medial prefrontal regions and posterior parietal regions, particularly the posterior cingulate and precuneus (Fransson & Marrelec, 2008; Raichle & Snyder, 2007). There is an ongoing interplay between the task-positive salience network and control network and task-negative default mode network underlying behavior. For example, increased default mode network activity has been shown to precede lapses in attention and focus during task performance (Buckner et al., 2008), and such shifts are also associated with error commission (Eichele et al., 2008). The shifts between networks and how they are controlled is a highly active current area of investigation (see for example, Menon & Uddin, 2010). Current research focuses on both within and between network activity, and the degree to which within and between network activity is modulated between task-positive and task-negative processing.

Key advances include theoretical work relating functional networks and dual process models of AUD and resulting empirical tests. Of note, the default mode network has not been linked to the dual process model in the same way as the salience network and control network have, but multiple studies now present compelling evidence that the default mode network structure (X. Ma et al., 2015) and function (N. Ma et al., 2011; Wang et al., 2015) are disrupted in individuals with substance use disorders, including AUD. Regarding the linkage between the salience network and control network to the dual process model, the impulsive and control systems have been mapped conceptually onto the salience network and control network, respectively (Gladwin, Figner, Crone, & Wiers, 2011; Hutchison, 2010). Empirical work has begun to validate this perspective. For example, a recent study found that abnormalities in connectivity between subcortical areas that process reward (e.g., nucleus accumbens) and cortical areas that govern cognitive-behavioral control (e.g. dorsolateral PFC; dlPFC) were associated with substance use disorders more broadly (Motzkin, Baskin-Sommers, Newman, Kiehl, & Koenigs, 2014). Another study reported a more complex relationship between AUDs and resting state dlPFC activity, a key region in the control network, finding that those with AUDs evidenced increased left dlPFC activity, related to reward/approach tendencies, but decreased right dlPFC activity, related to regulatory/inhibitory processing (Krmpotich et al., 2013). Other work suggests a central role for the salience network in modulating other network behavior (e.g. control network, default mode network), and that this regulatory process may be disrupted for substance use disorders (Zhu, Sundby, Bjork, & Momenan, 2016). Interestingly, there is also evidence that such salience network effects may have a genetic basis (Hong et al., 2010).

A third advance is findings that alcohol appears to change the behavior of these networks. For example, the salience network, responsible for the allocation of neural resources to relevant actions or events, is altered by alcohol consumption (Grodin, Cortes, Spagnolo, & Momenan, 2017). Further evidence suggests that salience network changes are markers of the progression of the AUD: Acute alcohol use appears to dampen the functional connectivity between the anterior insula and the dorsal anterior cingulate cortex (Gorka, Phan, & Childs, 2017)—structures identified as being important components of the salience network (Bressler & Menon, 2010). Further, there is evidence that the progression of AUDs may disrupt how the salience network can modulate the control network and default mode network (Zhu et al., 2016). Another study focused on alcohol dependent individuals found that all three key functional networks (default mode network, salience network, and control network) show within- and between-network connectivity changes (Müller-Oehring, Jung, Pfefferbaum, Sullivan, & Schulte, 2015), which are mediated by sex, age, and history of use (Müller-Oehring et al., 2017). Evidence also suggests that such changes may continue during alcohol abstinence, where, for example, alterations in the control network continue (Kim, Im, Lee, & Lee, 2017).

With regard to treatment, assessing effects on brain networks is in the beginning stages. However, the utility of assessing networks is already becoming clear. For example, it has been shown that interventions targeting neurotransmission of addictive disorders (dopaminergic agents) modulate the connection between the control network and default mode network, improving cognitive control of individuals living with AUD (Schmaal et al., 2013). The importance of the network interconnections is further supported by the fact that treatments targeting only one network, like the control network, do not lead to functioning levels comparable to healthy controls (Wilcox et al., 2015). Thus, broadly, the conceptual link between the dual process model and salience and control networks is widely considered, and empirical evidence is beginning to demonstrate the utility of this idea.

In sum, functional networks are emerging as a central new framework from which to understand broad swaths of human affect and

cognition, including goal-directed behavior and inward reflective processing. This framework has rapidly infused current work on understanding brain function associated with alcohol misuse and AUD, and is likely to play an increasing role in inferences about AUD from neuroimaging data in the coming years.

6. Implications for theory

The dual process framework provides the theoretical backdrop to our review, and advances described herein are generally consistent with this framework. While we have largely treated dual process models as if they are static, they are evolving. Reformulations of dual process models have been proposed generally (see Cunningham, Zelazo, Packer, & Van Bavel, 2007) as well as for addiction, specifically (see Gladwin et al., 2011). These models are informed by emerging neuroscience findings, including the absence of evidence for two distinct systems (Keren & Schul, 2009, as cited in Gladwin et al., 2011). Among the key elements of this reformulation is a shift away from conceptualizing cognitive processes as two distinct systems and toward conceptualizing them as two interactive systems that vary in terms of activation speed (see Cunningham et al., 2007). Cognitive processing is argued to be iterative and hierarchical, with "lower" brain regions (subcortical, limbic regions) coming online first and leading to rapid evaluations based on preexisting associations, and with "higher" brain regions (those mediated by the PFC) coming online later, allowing more reflective cognitive operations that can modulate or reinterpret earlier evaluations (Cunningham et al., 2007). Key uses of the iterative processing model include Gladwin et al.'s (2011) discussion of adolescence (a critical period with contrasting paces of brain development for systems implicated in impulsive processing [faster development] vs. control processing [slower development]) and adolescent substance use.

Important future research directions include whether—across stages of development, or AUD progression—there are individual differences in the speed in which the impulsive and control systems come online, what factors influence whether and how (often) initial evaluations are updated (i.e., [re]processing), and the effects of acute and chronic alcohol use on cognitive (re)processing. We also note a key practical constraint to the potential to fully evaluate this reformulation: Current behavioral measures of impulsive and control systems take 5–10 min to complete and essentially treat the underlying processes as static, suggesting the need for novel measures that can capture the rapid dynamics of iterative (re)processing.

As reformulations of dual processing models continue, one key question is how to integrate emerging cognitive neuroscience findings of *three* primary functional brain networks (i.e., salience network, control network, & default mode network) with *dual* process perspectives that are now established in multiple domains. There is a particular need to integrate the default mode network, as early theoretical work attempting this integration with dual-process models focused only on the salience network (impulsive system) and control network (control system). This need is underlined by more general findings from neuroscience studies—namely, that the default mode network is increasingly understood in terms of a dynamic interplay with the salience network and control network (Sutherland, McHugh, Pariyadath, & Stein, 2012; Zhu et al., 2016)—and alcohol-related studies—namely, that disruptions in the default mode network have generally been linked to AUD (Correas et al., 2016). Future models will need to be more comprehensive, possibly leading to the abandonment of the dual systems approach in favor of a unitary model that focuses on the interplay of component processes. Truly integrating cognitive neuroscience findings creates a need to capture dynamic shifts in functional brain networks, which calls for measures with faster time scales. EEG offers inherently higher time-resolution than fMRI, and there is thus potential for EEG (as well as event-related potential measures) to offer important, novel information about the shifts (e.g., Aviyente, Tootell, & Bernat, 2017; Cavanagh, Cohen, & Allen, 2009).

We also note the importance of integrating motivational accounts of alcohol misuse and AUD into dual process approaches (see R. W. Wiers & Gladwin, 2017, for an initial model). There are emergent findings that motivation to restrain drinking can further modulate two-way interactions between impulsive and control processes on drinking (O'Connor & Colder, 2015). Further, behavioral economics findings indicate the unique influence of valuation of the future (delay discounting), sensitivity of drinking decisions to cost (alcohol demand), and of alternative rewards on alcohol motivation (Bickel, Johnson, et al., 2014). There are also long-standing and robust theories that focus on drinking motives (Cooper et al., 1995) and alcohol expectancies (Goldman et al., 1991) which are important to integrate fully into dual process models.

7. Clinical implications

Advances in these exemplar domains have implications for clinical assessment and treatment, which we discuss in turn. First, despite the clear relevance of implicit cognitive biases, EF, behavioral economics, and functional networks for AUD clinical course, treatment engagement, and relapse, their use (and that of standardized cognitive assessments, generally) in addiction treatment settings is rare. Thus, information on rates and correlates of cognitive recovery during treatment, as well as the potential for cognitive assessments/tasks to inform treatment matching, is limited. The literature on EF is relatively more developed than the other domains and might serve as a model for future research. Recently, neuroscience-informed assessment initiatives, including behavioral and neuroimaging measures/tasks, have been advanced, emphasizing EF and incentive salience (including attentional bias) as priority assessment domains (Kwako et al., 2016). While we are enthusiastic about these initiatives in principle—data from clinical populations is essential—we note that many suggested measures (as well as measures from our reviewed exemplar domains) are not reliable enough to act as standalone assessments for screening or diagnostic purposes, and/or present a substantial assessment burden (e.g., neuroimaging). Similarly, we note the emerging understanding of patterns of functional network behavior as indictors of dysfunction (Menon, 2011). Ensuing models could potentially serve as a means of validation for novel diagnostic approaches, or perhaps disrupted or aberrant network behavior may eventually become a diagnostic indicator. Ultimately, all of these efforts could inform AUD nosology, but whether they will show clinical utility by informing or resulting in actionable clinical decisions is unclear.

We discuss treatment implications for each domain individually. Regarding implicit cognitive biases, CBM research represents a burgeoning subfield of alcohol research. Across various implicit biases and CBM paradigms, a general conclusion is that these interventions, while theoretically justified, are not ready for primetime. While we note the promise of CBM, particularly CBM for automatic action tendencies as an add-on treatment in clinical populations, much needs to be learned in terms of *how* and *for whom* CBM works. Initial hopes that CBM could serve as a stand-alone intervention or be effective for individuals ambivalent about or not intending to change their drinking have given way to more modest hopes that it might serve as an adjunct to evidence-based treatments for treatment-seeking clinical populations. We note the potential for game-like elements to increase engagement in CBM, motivation for change, and, ultimately, the efficacy of CBM (see Boendermaker, Prins, & Wiers, 2015), as well as their potential to reduce efficacy (see Boendermaker, Sanchez Maceiras, Boffo, & Wiers, 2016).

We hope that CBM paradigms can be refined (or novel paradigms developed) to the point that their effects on drinking, and on theoretical mediators, are easily and widely replicated. Mindfulness training has also shown some promise in weakening the relationships between implicit cognitive biases and drinking (see Ostafin, Bauer, & Myxter, 2012), suggesting a more general need for research testing whether

other established or in-development psychotherapies or medications for alcohol misuse or AUD have an effect on implicit cognitive biases. Finally, the notion that baseline implicit cognitive biases might predict response to treatment is also consistent with dual process models. However, evidence of their ability to predict treatment response is scant but promising (e.g., Wolff, von Hippel, Brener, & von Hippel, 2015).

Findings from clinical interventions targeting EF also indicate a need for improvement. While WM training is an appealing adjunctive treatment for AUD (Bickel, Moody, & Quisenberry, 2014), the evidence base is insufficient to infer efficacy. While the research base on inhibitory control training suggests that single-session inhibitory control training can lead to reductions in laboratory alcohol consumption (Allom et al., 2016; Jones et al., 2016), these results are based on a handful of trials, have not been extended to longer-term drinking outcomes, and are generally limited to young social drinkers. Objectives for future work include establishing efficacy of cognitive training interventions in clinical populations (e.g., can they reduce heavy drinking and prevent relapse) and studying practical barriers (e.g., can multi-session training protocols be implemented with sufficient adherence in outpatient settings). Additional areas for cognitive training research include evaluating scalability (e.g., delivery via internet and via mobile devices) and examining behavioral, pharmacological, or neurostimulation methods as a means of augmenting intervention effects.

Behavioral economic approaches suggest unique targets for interventions, but research is scant in several domains. Interventions have been developed or adapted to focus on alcohol-free activities, but individuals with more severe alcohol problems or AUD may require interventions that provide tangible rewards (e.g., contingency management) and/or enhance the ability to experience reward. An 8-session Mindfulness-Oriented Recovery Enhancement intervention that uses experiential and mindfulness exercises to enhance the ability to notice and experience drug-free rewards has been developed for individuals in opiate treatment and has promising preliminary findings (Garland, Howard, Zubieta, & Froeliger, 2017). Next steps include extension to the alcohol domain. Increasing the salience of the delayed outcomes of behavior and learning to view behaviors as patterns (vs. discrete acts) can reduce impulsive responses (Loewenstein & Prelec, 1993); one could imagine interventions that seek to target them. Additionally, focused thinking/writing about potential positive future events (Episodic Future Thinking) has been found to reduce delay discounting and alcohol demand in laboratory studies (Bickel, 2015), but this has not been tested as an intervention for alcohol misuse and AUD.

Finally, findings from functional connectivity imaging studies have important clinical implications. As more information emerges about the nature of network disruptions related to AUDs, interventions may be evaluated by their ability to change network behavior, or interventions may eventually directly target such disruptions as a treatment goal. Alternatively, although pharmacological approaches have evolved dramatically in the last years, most treatments are oriented to single targets, such as hormones or neurotransmitter systems. Only a few medications (e.g., modafinil and naltrexone) have managed to make network level changes. Psychopharmacology in the treatment of AUD will benefit from integration of a network perspective, aiming for network targets and accounting for side effects.

8. Future research directions

Significant advances from the four domains have occurred over the past decade and led to major reformulations of cognitive theories of alcohol misuse. These advances have facilitated novel assessment and treatment approaches. Nonetheless, much remains to be learned about these exemplar domains, and we close with suggestions for future research.

First, there is a clear need for research about ways individual differences and demographic characteristics interact with constructs from the exemplar domains. Our knowledge is generally limited due to the relative abundance of studies with adolescents and college students and the scarcity of studies with clinical samples (including individuals with AUD who are not treatment-seeking) as well as with older adults, and evidence within the reviewed domains is scant. Yet there are important preliminary data that these cognitive processes may differ as a function of these characteristics (e.g., implicit biases: age, race and gender differences have been observed, see Werntz, Steinman, Glenn, Nock, & Teachman, 2016; behavioral economics: low income is a risk factor for steep delay discounting, see Bickel, Johnson, et al., 2014; functional connectivity networks: age and gender differences play key roles, see Gong et al., 2009). Further, some have noted the potential for these characteristics to be associated with differences in motivation for treatment (EF: potential cultural differences in motivation for cognitive training, see Au et al., 2015). Thus, we recommend both a general expansion of populations studied as well as targeted research aimed at evaluating demographic and individual differences in the exemplar domains, and how those characteristics might interact with one another and/or other factors to predict response to or preferences for treatment. Finally, research that investigates potential underlying factors or mechanisms for observed differences is essential.

Second, further research is needed to systematically integrate behavioral economic and dual process indices of alcohol motivation and associated risk factors. While a few studies have begun doing so (e.g., Luehring-Jones et al., 2016; Ramirez, Dennhardt, Baldwin, Murphy, & Lindgren, 2016), research is scant. One obvious point of integration is to develop implicit measures of the relative strength of motivation for alcohol relative to important and personally relevant substance-free stimuli, and the relative valuation of immediate versus future rewards (the latter construct has been measured almost exclusively by self-report hypothetical monetary choice tasks). It would also be interesting to investigate the efficacy of behavioral economic approaches to increasing future orientation and valuation of substance-free rewards for modifying implicit cognitive biases. As described above, behavioral economic intervention approaches have shown promise in reducing delay discounting, and more research is needed to evaluate their potential efficacy as elements of more general EF interventions. Behavioral economic demand curve and delay discounting measures may also have utility as mechanisms of behavior change for EF intervention approaches that should presumably increase valuation of the future and the extent to which decisions to drink are sensitive to drink price.

Third, large-sample, prospective studies that include batteries of measures of implicit biases and EF tasks, and incorporate latent variable modeling and prospective assessments could help reconcile findings within the implicit cognitive bias and EF literatures, respectively. Ideally, prospective cohort studies would also characterize changes in implicit cognition and EF domains as both predictors and consequences of consumption. We note also that prospective studies incorporating measures of EF *and* implicit biases are needed to evaluate key developmental predictions of dual process theories. Including behavioral economic measures would also further theory development and integration.

We also recommend (updated) meta-analyses to clarify findings from numerous published studies that vary in subject population and measurement. For example, we note the mixed findings evaluating interactions between implicit cognitive biases and control processes. Results from an unpublished meta-analysis evaluating this interaction (see Ames, Xie, Aragon, & Stacy, 2015) generally found support for control processes weakening the relationship between implicit cognitive biases and drinking, with stronger relationships observed in adults (vs. adolescents) in substance use. Greater use of meta-analytic techniques will be helpful to clarify *which* measures of EF domains are more likely to moderate *which* measures of implicit cognitive biases in *which* populations with *which* patterns or histories of drinking. Greater use of meta-analysis can also be helpful within the exemplar domains.

Finally, we note the need for continued improvement in measures. As noted throughout this review, many current measures are too

unreliable for screening and diagnostic purposes, which greatly reduces their utility in the context of cognitive neuroscience-informed assessment batteries for AUD (see Kwako et al., 2016). While the application of emerging technologies (e.g., the use of eye-tracking to evaluate attentional bias) and complex statistical models (e.g., latent variable approaches, the quad model, IRT) is novel and promising, there are inherent, practical limitations to their adoption. Further, the extent to which these applications improve the prediction of alcohol misuse and AUD is not yet clear. Relatedly, improvements to the platforms to implement these measures are also critical. Many are costly, require programming expertise, and cannot be easily integrated with commercial online survey programs. Options that are open-source, free, and less cumbersome will be critical (see Carpenter et al., 2017, for one promising possibility). Finally, a key goal of improved measurement will be to provide the best match possible between the phenomenon of interest and the context in which it is actually measured (including attention to population, setting, and individuals' cognitive and motivational states; see Sayette, 2016).

9. Conclusion

Recent advances in cognitive science have improved our understanding of the etiology, maintenance, and treatment of alcohol misuse and AUD. Four exemplar domains—implicit cognitive biases, EF, behavioral economic approaches, and functional connectivity imaging studies—were highlighted in this review. These domains and their findings can be integrated into and are generally consistent with the dual process model framework. Most methods used to assess these domains are emergent, and though there are demonstrations of their predictive validity, they are presently not appropriate for diagnostic purposes. Treatment efforts, including CBM and cognitive training, have some promise but are not ready for wide-spread dissemination. Dual process model frameworks continue to evolve and future refinements are needed. Finally, there is a need for basic and clinical research on individual and demographic differences and with broader samples that seeks to improve measurement of these domains, that implements prospective studies with large-scale samples, and that makes greater use of meta-analytic techniques.

Role of funding sources

Manuscript support was provided by grants from the National Institute on Alcohol Abuse and Alcoholism (R01AA024732, PI: Lindgren; R01AA021763, PI Lindgren; R01AA024930, Multiple PIs: MacKillop and Murphy; R01AA020829, PI: Murphy; R01AA022087-03S1, PI: Lee); and the Canadian Institutes of Health Research and Canada Research Chairs Program (no grant #, PI: Hendershot). NIAAA and the Canadian Institutes of Health Research and Canada Research Chairs Program had no role in the writing the manuscript, or the decision to submit this paper for publication.

Contributors

All authors (Lindgren, Hendershot, Ramirez, Bernat, Rangel-Gomez, Peterson, & Murphy) contributed to and approved the final manuscript. All took part in planning meetings for this review and helped shape its content and organization. The lead authors of the sections were as follows: introduction (Lindgren & Hendershot), implicit cognitive biases (Lindgren & Ramirez), executive function (Hendershot), functional connectivity imaging (Bernat & Rangel-Gomez), behavioral economics (Murphy), implications for theory (Lindgren). Peterson was lead for literature searches. All authors contributed to the clinical implications and future directions.

Conflict of interest

All authors declare that they have no conflicts of interest.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cpr.2018.04.002.

Acknowledgements

We are grateful to Micheal Sayette and Reinout Wiers for sharing their thoughts on key advances in cognitive science and AUD.

References

Allom, V., Mullan, B., & Hagger, M. (2016). Does inhibitory control training improve health behaviour? A meta-analysis. *Health Psychology Review, 10*(2), 168–186. http://dx.doi.org/10.1080/17437199.2015.1051078.

Ames, S. L., Grenard, J. L., He, Q., Stacy, A. W., Wong, S. W., Xiao, L., ... Bechara, A. (2014). Functional imaging of an alcohol-Implicit Association Test (IAT). *Addiction Biology, 19*(3), 467–481. http://dx.doi.org/10.1111/adb.12071.

Ames, S. L., Xie, B., Aragon, J., & Stacy, A. W. (2015). *Moderating effects of control functions on implicit cognitive processes in health behavior: A meta-analysis.* (Unpublished manuscript).

Ames, S. L., Xie, B., Shono, Y., & Stacy, A. W. (2017). Adolescents at risk for drug abuse: A 3-year dual-process analysis. *Addiction, 112*(5), 852–863. http://dx.doi.org/10.1111/add.13742.

Amlung, M., Few, L. R., Howland, J., Rohsenow, D. J., Metrik, J., & MacKillop, J. (2013). Impulsivity and alcohol demand in relation to combined alcohol and caffeine use. *Experimental and Clinical Psychopharmacology, 21*(6), 467–474. http://dx.doi.org/10.1037/a0034214.

Amlung, M., Morris, D. H., Hatz, L. E., Teeters, J. B., Murphy, J. G., & McCarthy, D. M. (2016). Drinking-and-driving-related cognitions mediate the relationship between alcohol demand and alcohol-impaired driving. *Journal of Studies on Alcohol and Drugs, 77*(4), 656–660. http://dx.doi.org/10.15288/jsad.2016.77.656.

Amlung, M., Vedelago, L., Acker, J., Balodis, I., & MacKillop, J. (2017). Steep delay discounting and addictive behavior: A meta-analysis of continuous associations. *Addiction, 112*(1), 51–62. http://dx.doi.org/10.1111/add.13535.

Anker, J. J., Perry, J. L., Gliddon, L. A., & Carroll, M. E. (2009). Impulsivity predicts the escalation of cocaine self-administration in rats. *Pharmacology, Biochemistry and Behavior, 93*(3), 343–348. http://dx.doi.org/10.1016/j.pbb.2009.05.013.

Ataya, A. F., Adams, S., Mullings, E., Cooper, R. M., Attwood, A. S., & Munafò, M. R. (2012). Internal reliability of measures of substance-related cognitive bias. *Drug and Alcohol Dependence, 121*(1–2), 148–151. http://dx.doi.org/10.1016/j.drugalcdep.2011.08.023.

Au, J., Sheehan, E., Tsai, N., Duncan, G. J., Buschkuehl, M., & Jaeggi, S. M. (2015). Improving fluid intelligence with training on working memory: A meta-analysis. *Psychonomic Bulletin & Review, 22*(2), 366–377. http://dx.doi.org/10.3758/s13423-014-0699-x.

Audrain-McGovern, J., Rodriguez, D., Epstein, L. H., Cuevas, J., Rodgers, K., & Wileyto, E. P. (2009). Does delay discounting play an etiological role in smoking or is it a consequence of smoking? *Drug and Alcohol Dependence, 103*(3), 99–106. http://dx.doi.org/10.1016/j.drugalcdep.2008.12.019.

Aviyente, S., Tootell, A., & Bernat, E. M. (2017). Time-frequency phase-synchrony approaches with ERPs. *International Journal of Psychophysiology, 111*, 88–97. http://dx.doi.org/10.1016/j.ijpsycho.2016.11.006.

Baddeley, A. (2003). Working memory: Looking back and looking forward. *Nature Reviews Neuroscience, 4*(10), 829–839. http://dx.doi.org/10.1038/nrn1201.

Bates, M. E., Buckman, J. F., & Nguyen, T. T. (2013). A role for cognitive rehabilitation in increasing the effectiveness of treatment for alcohol use disorders. *Neuropsychology Review, 23*(1), 27–47. http://dx.doi.org/10.1007/s11065-013-9228-3.

Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience, 8*(11), 1458–1463. http://dx.doi.org/10.1038/nn1584.

Bentzley, B. S., Jhou, T. C., & Aston-Jones, G. (2014). Economic demand predicts addiction-like behavior and therapeutic efficacy of oxytocin in the rat. *Proceedings of the National Academy of Sciences of the United States of America, 111*(32), 11822–11827. http://dx.doi.org/10.1073/pnas.1406324111.

Bickel, W. K. (2015). Discounting of delayed rewards as an endophenotype. *Biological Psychiatry, 77*(10), 846–847. http://dx.doi.org/10.1016/j.biopsych.2015.03.003.

Bickel, W. K., Johnson, M. W., Koffarnus, M. N., MacKillop, J., & Murphy, J. G. (2014). The behavioral economics of substance use disorders: Reinforcement pathologies and their repair. *Annual Review of Clinical Psychology, 10*, 641–677. http://dx.doi.org/10.1146/annurev-clinpsy-032813-153724.

Bickel, W. K., Moody, L., & Quisenberry, A. (2014). Computerized working-memory training as a candidate adjunctive treatment for addiction. *Alcohol Research: Current Reviews, 36*(1), 123–126. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4432851/.

Bickel, W. K., Quisenberry, A. J., Moody, L., & Wilson, A. G. (2015). Therapeutic opportunities for self-control repair in addiction and related disorders: Change and the limits of change in trans-disease processes. *Clinical Psychological Science: A Journal of the Association for Psychological Science, 3*(1), 140–153. http://dx.doi.org/10.1177/2167702614541260.

Bickel, W. K., Yi, R., Landes, R. D., Hill, P. F., & Baxter, C. (2011). Remember the future:

Working memory training decreases delay discounting among stimulant addicts. *Biological Psychiatry, 69*(3), 260–265. http://dx.doi.org/10.1016/j.biopsych.2010.08.017.

Boendermaker, W. J., Prins, P. J. M., & Wiers, R. W. (2015). Cognitive Bias Modification for adolescents with substance use problems – Can serious games help? *Journal of Behavior Therapy and Experimental Psychiatry, 49*, 13–20. http://dx.doi.org/10.1016/j.jbtep.2015.03.008.

Boendermaker, W. J., Sanchez Maceiras, S., Boffo, M., & Wiers, R. W. (2016). Attentional bias modification with serious game elements: Evaluating the shots game. *JMIR Serious Games, 4*(2), http://dx.doi.org/10.2196/games.6464.

Bressler, S. L., & Menon, V. (2010). Large-scale brain networks in cognition: Emerging methods and principles. *Trends in Cognitive Sciences, 14*(6), 277–290. http://dx.doi.org/10.1016/j.tics.2010.04.004.

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network. *Annals of the New York Academy of Sciences, 1124*(1), 1–38. http://dx.doi.org/10.1196/annals.1440.011.

Bujarski, S., MacKillop, J., & Ray, L. A. (2012). Understanding naltrexone mechanism of action and pharmacogenetics in Asian Americans via behavioral economics: A preliminary study. *Experimental and Clinical Psychopharmacology, 20*(3), 181–190. http://dx.doi.org/10.1037/a0027379.

Carpenter, T., Pogacar, R., Pullig, C., Kouril, M., LaBouff, J., Aguilar, S. J., ... Chakroff, A. (2017, August 11). Building and analyzing Implicit Association Tests for online surveys: A tutorial and open-source tool. Retrieved from https://psyarxiv.com/6xdyj.

Carroll, K. M., Nich, C., LaPaglia, D. M., Peters, E. N., Easton, C. J., & Petry, N. M. (2012). Combining cognitive behavioral therapy and contingency management to enhance their effects in treating cannabis dependence: Less can be more, more or less. *Addiction, 107*, 1650–1659. http://dx.doi.org/10.1111/j.1360- 0443.2012.03877.x.

Cavanagh, J. F., Cohen, M. X., & Allen, J. B. (2009). Prelude to and resolution of an error: EEG phase synchrony reveals cognitive control dynamics during action monitoring. *The Journal of Neuroscience, 29*(1), 98–105. http://dx.doi.org/10.1523/JNEUROSCI.4137-08.2009.

Charlet, K., Beck, A., Jorde, A., Wimmer, L., Vollstadt-Klein, S., Gallinat, J., ... Heinz, A. (2014). Increased neural activity during high working memory load predicts low relapse risk in alcohol dependence. *Addiction Biology, 19*(3), 402–414. http://dx.doi.org/10.1111/adb.12103.

Christiansen, P., Cole, J. C., Goudie, A. J., & Field, M. (2012). Components of behavioural impulsivity and automatic cue approach predict unique variance in hazardous drinking. *Psychopharmacology, 219*(2), 501–510. http://dx.doi.org/10.1007/s00213-011-2396-z.

Christiansen, P., Mansfield, R., Duckworth, J., Field, M., & Jones, A. (2015). Internal reliability of the alcohol-related visual probe task is increased by utilising personalised stimuli and eye-tracking. *Drug and Alcohol Dependence, 155*, 170–174. http://dx.doi.org/10.1016/j.drugalcdep.2015.07.672.

Christiansen, P., Schoenmakers, T. M., & Field, M. (2014). Less than meets the eye: Reappraising the clinical relevance of attentional bias in addiction. *Addictive Behaviors, 44*, 43–50. http://dx.doi.org/10.1016/j.addbeh.2014.10.005.

Clarke, P. J. F., Notebaert, L., & MacLeod, C. (2014). Absence of evidence or evidence of absence: Reflecting on therapeutic implementations of attentional bias modification. *BMC Psychiatry, 14*, 8. http://dx.doi.org/10.1186/1471-244X-14-8.

Claus, E. D., Feldstein Ewing, S. W., Filbey, F. M., & Hutchison, K. E. (2013). Behavioral control in alcohol use disorders: Relationships with severity. *Journal of Studies on Alcohol and Drugs, 74*(1), 141–151. http://dx.doi.org/10.15288/jsad.2013.74.141.

Claus, E. D., & Hendershot, C. S. (2015). Moderating effect of working memory capacity on acute alcohol effects on BOLD response during inhibition and error monitoring in male heavy drinkers. *Psychopharmacology, 232*(4), 765–776. http://dx.doi.org/10.1007/s00213-014-3711-2.

Colder, C. R., O'Connor, R. M., Read, J. P., Eiden, R. D., Lengua, L. J., Hawk, L. W., & Wieczorek, W. F. (2014). Growth trajectories of alcohol information processing and associations with escalation of drinking in early adolescence. *Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors, 28*(3), 659–670. http://dx.doi.org/10.1037/a0035271.

Conrey, F. R., Sherman, J. W., Gawronski, B., Hugenberg, K., & Groom, C. J. (2005). Separating multiple processes in implicit social cognition: The quad model of implicit task performance. *Journal of Personality and Social Psychology, 89*(4), 469–487. http://dx.doi.org/10.1037/0022-3514.89.4.469.

Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *Journal of Personality and Social Psychology, 69*, 990–1005. http://dx.doi.org/10.1037/0022-3514.69.5.990.

Correas, A., Cuesta, P., López-Caneda, E., Rodríguez Holguín, S., García-Moreno, L. M., Pineda-Pardo, J. A., ... Maestú, F. (2016). Functional and structural brain connectivity of young binge drinkers: A follow-up study. *Scientific Reports, 6*, 1–8. http://dx.doi.org/10.1038/srep31293.

Cristea, I. A., Kok, R. N., & Cuijpers, P. (2016). The effectiveness of cognitive bias modification interventions for substance addictions: A meta-analysis. *PLoS One, 11*(9), e0162226. http://dx.doi.org/10.1371/journal.pone.0162226.

Cunningham, W. A., Zelazo, P. D., Packer, D. J., & Van Bavel, J. J. (2007). The iterative reprocessing model: A multilevel framework for attitudes and evaluation. *Social Cognition, 25*(5), 736–760. http://dx.doi.org/10.1521/soco.2007.25.5.736.

Daughters, S. B., Braun, A. R., Sargeant, M. N., Reynolds, E. K., Hopko, D. R., Blanco, C., & Lejuez, C. W. (2008). Effectiveness of a brief behavioral treatment for inner-city illicit drug users with elevated depressive symptoms: The Life Enhancement Treatment for Substance Use (LETS Act!). *The Journal of Clinical Psychiatry, 69*(1), 122–129. http://dx.doi.org/10.4088/JCP.v69n0116.

Day, A. M., Kahler, C. W., Ahern, D. C., & Clark, U. S. (2015). Executive functioning in alcohol use studies: A brief review of findings and challenges in assessment. *Current Drug Abuse Reviews, 8*(1), 26–40. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4638323/.

Dennhardt, A. D., Yurasek, A. A., & Murphy, J. G. (2015). Change in delay discounting and substance reward value following a brief alcohol and drug use intervention. *Journal of the Experimental Analysis of Behavior, 103*(1), 125–140. http://dx.doi.org/10.1002/jeab.121.

Di Lemma, L. C. G., & Field, M. (2017). Cue avoidance training and inhibitory control training for the reduction of alcohol consumption: A comparison of effectiveness and investigation of their mechanisms of action. *Psychopharmacology, 234*(16), 2489–2498. http://dx.doi.org/10.1007/s00213-017-4639-0.

Eberl, C., Wiers, R. W., Pawelczack, S., Rinck, M., Becker, E. S., & Lindenmeyer, J. (2013). Approach bias modification in alcohol dependence: Do clinical effects replicate and for whom does it work best? *Developmental Cognitive Neuroscience, 4*, 38–51. http://dx.doi.org/10.1016/j.dcn.2012.11.002.

Eichele, T., Debener, S., Calhoun, V. D., Specht, K., Engel, A. K., Hugdahl, K., ... Ullsperger, M. (2008). Prediction of human errors by maladaptive changes in event-related brain networks. *Proceedings of the National Academy of Sciences of the United States of America, 105*(16), 6173–6178. http://dx.doi.org/10.1073/pnas.0708965105.

Field, M., Di Lemma, L., Christiansen, P., & Dickson, J. (2017). Automatic avoidance tendencies for alcohol cues predict drinking after detoxification treatment in alcohol dependence. *Psychology of Addictive Behaviors, 31*(2), 171–179. http://dx.doi.org/10.1037/adb0000232.

Field, M., & Jones, A. (2017). Elevated alcohol consumption following alcohol cue exposure is partially mediated by reduced inhibitory control and increased craving. *Psychopharmacology, 234*(19), 2979–2988. http://dx.doi.org/10.1007/s00213-017-4694-6.

Field, M., Wiers, R. W., Christiansen, P., Fillmore, M. T., & Verster, J. C. (2010). Acute alcohol effects on inhibitory control and implicit cognition: Implications for loss of control over drinking. *Alcoholism: Clinical and Experimental Research, 34*(8), 1346–1352. http://dx.doi.org/10.1111/j.1530-0277.2010.01218.x.

Fransson, P., & Marrelec, G. (2008). The precuneus/posterior cingulate cortex plays a pivotal role in the default mode network: Evidence from a partial correlation network analysis. *NeuroImage, 42*(3), 1178–1184. http://dx.doi.org/10.1016/j.neuroimage.2008.05.059.

Friedman, N. P., Miyake, A., Young, S. E., DeFries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual differences in executive functions are almost entirely genetic in origin. *Journal of Experimental Psychology: General, 137*(2), 201–225. http://dx.doi.org/10.1037/0096-3445.137.2.201.

Friston, K. J. (1994). Functional and effective connectivity in neuroimaging: A synthesis. *Human Brain Mapping, 2*(1–2), 56–78. http://dx.doi.org/10.1002/hbm.460020107.

Friston, K. J. (2011). Functional and effective connectivity: A review. *Brain Connectivity, 1*(1), 13–36. http://dx.doi.org/10.1089/brain.2011.0008.

Garland, E. L., Howard, M. O., Zubieta, J., & Froeliger, B. (2017). Restructuring hedonic dysregulation in chronic pain and prescription opioid misuse: Effects of mindfulness-oriented recovery enhancement on responsiveness to drug cues and natural rewards. *Psychotherapy and Psychosomatics, 86*(2), 111–112. http://dx.doi.org/10.1159/000453400.

Gladwin, T. E. (2017). Attentional bias variability and cued attentional bias for alcohol stimuli. *Addiction Research and Theory, 25*(1), 32–38. http://dx.doi.org/10.1080/16066359.2016.1196674.

Gladwin, T. E., Figner, B., Crone, E. A., & Wiers, R. W. (2011). Addiction, adolescence, and the integration of control and motivation. *Developmental Cognitive Neuroscience, 1*(4), 364–376. http://dx.doi.org/10.1016/j.dcn.2011.06.008.

Goldman, M. S., Brown, S. A., Christiansen, B. A., & Smith, G. T. (1991). Alcoholism and memory: Broadening the scope of alcohol-expectancy research. *Psychological Bulletin, 110*(1), 137–146. http://dx.doi.org/10.1037/0033-2909.110.1.137.

Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews Neuroscience, 12*(11), 652–669. http://dx.doi.org/10.1038/nrn3119.

Gong, G., Rosa-Neto, P., Carbonell, F., Chen, Z. J., He, Y., & Evans, A. C. (2009). Age- and gender-related differences in the cortical anatomical network. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 29*(50), 15684–15693. http://dx.doi.org/10.1523/JNEUROSCI.2308-09.2009.

Gorka, S. M., Phan, K. L., & Childs, E. (2017). Acute calming effects of alcohol are associated with disruption of the salience network. *Addiction Biology*. http://dx.doi.org/10.1111/adb.12537.

Grant, B. F., Chou, S. P., Saha, T. D., Pickering, R. P., Kerridge, B. T., Ruan, W. J., ... Hasin, S, D. (2017). Prevalence of 12-month alcohol use, high-risk drinking, and DSM-IV alcohol use disorder in the United States, 2001–2002 to 2012-2013: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *JAMA Psychiatry*. http://dx.doi.org/10.1001/jamapsychiatry.2017.2161.

Greenwald, A. G., & Banaji, M. R. (1995). Implicit social cognition: Attitudes, self-esteem, and stereotypes. *Psychological Review, 102*(1), 4–27. http://dx.doi.org/10.1037/0033-295X.102.1.4.

Grodin, E. N., Cortes, C. R., Spagnolo, P. A., & Momenan, R. (2017). Structural deficits in salience network regions are associated with increased impulsivity and compulsivity in alcohol dependence. *Drug and Alcohol Dependence, 179*, 100–108. http://dx.doi.org/10.1016/j.drugalcdep.2017.06.014.

Gustavson, D. E., Stallings, M. C., Corley, R. P., Miyake, A., Hewitt, J. K., & Friedman, N. P. (2017). Executive functions and substance use: Relations in late adolescence and early adulthood. *Journal of Abnormal Psychology, 126*(2), 257–270. http://dx.doi.org/10.1037/abn0000250.

van Hemel-Ruiter, M. E., Wiers, R. W., Brook, F. G., & de Jong, P. J. (2016). Attentional bias and executive control in treatment-seeking substance-dependent adolescents: A cross-sectional and follow-up study. *Drug and Alcohol Dependence, 159*, 133–141.

http://dx.doi.org/10.1016/j.drugalcdep.2015.12.005.
Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences, 16*(3), 174–180. http://dx.doi.org/10.1016/j.tics.2012.01.006.
Hong, L. E., Hodgkinson, C. A., Yang, Y., Sampath, H., Ross, T. J., Buchholz, B., ... Stein, E. A. (2010). A genetically modulated, intrinsic cingulate circuit supports human nicotine addiction. *Proceedings of the National Academy of Sciences, 107*(30), 13509–13514. http://dx.doi.org/10.1073/pnas.1004745107.
Houben, K., Wiers, R. W., & Jansen, A. (2011). Getting a grip on drinking behavior: Training working memory to reduce alcohol abuse. *Psychological Science, 22*(7), 968–975. http://dx.doi.org/10.1177/0956797611412392.
Hutchison, K. E. (2010). Substance use disorders: Realizing the promise of pharmacogenomics and personalized medicine. *Annual Review of Clinical Psychology, 6*, 577–589. http://dx.doi.org/10.1146/annurev.clinpsy.121208.131441.
Janssen, T., Larsen, H., Vollebergh, W. A. M., & Wiers, R. W. (2015). Longitudinal relations between cognitive bias and adolescent alcohol use. *Addictive Behaviors, 44*, 51–57. http://dx.doi.org/10.1016/j.addbeh.2014.11.018.
Janssen, T., Wood, M. D., Larsen, H., Peeters, M., Vollebergh, W. A., & Wiers, R. W. (2015). Investigating the joint development of approach bias and adolescent alcohol use. *Alcoholism: Clinical and Experimental Research, 39*(12), 2447–2454. http://dx.doi.org/10.1111/acer.12899.
Jones, A., Di Lemma, L. C., Robinson, E., Christiansen, P., Nolan, S., Tudur-Smith, C., & Field, M. (2016). Inhibitory control training for appetitive behaviour change: A meta-analytic investigation of mechanisms of action and moderators of effectiveness. *Appetite, 97*, 16–28. http://dx.doi.org/10.1016/j.appet.2015.11.013.
Joyner, J. K., Pickover, A. M., Soltis, K. E., Dennhardt, A. A., Martens, M. P., & Murphy, J. G. (2016). Deficits in access to reward are associated with college student alcohol use disorder. *Alcoholism: Clinical and Experimental Research, 40*(12), 2685–2691. http://dx.doi.org/10.1111/acer.13255.
Keren, G., & Schul, Y. (2009). Two is not always better than one: A critical evaluation of two-system theories. *Perspect. Psychol. Sci. 4*, 533–550.
Khurana, A., Romer, D., Betancourt, L. M., Brodsky, N. L., Giannetta, J. M., & Hurt, H. (2013). Working memory ability predicts trajectories of early alcohol use in adolescents: The mediational role of impulsivity. *Addiction, 108*(3), 506–515. http://dx.doi.org/10.1111/add.12001.
Kim, S., Im, S., Lee, J., & Lee, S.-G. (2017). Disrupted control network connectivity in abstinent patients with alcohol dependence. *Psychiatry Investigation, 14*(3), 325–332. http://dx.doi.org/10.4306/pi.2017.14.3.325.
Koffarnus, M. N., Jarmolowicz, D. P., Mueller, E. T., & Bickel, W. K. (2013). Changing delay discounting in the light of the competing neurobehavioral decision systems theory: A review. *Journal of the Experimental Analysis of Behavior, 99*(1), 32–57. http://dx.doi.org/10.1002/jeab.2.
Kohn, R., Saxena, S., Levav, I., & Saraceno, B. (2004). The treatment gap in mental health care. *Bulletin of the World Health Organization, 82*(11), 858–866. http://dx.doi.org/10.1590/S0042-96862004001100011.
Koob, G. F., & Volkow, N. D. (2010). Neurocircuitry of addiction. *Neuropsychopharmacology, 35*(1), 217–238. http://dx.doi.org/10.1038/npp.2009.110.
Korucuoglu, O., Gladwin, T. E., & Wiers, R. W. (2014). Preparing to approach or avoid alcohol: EEG correlates, and acute alcohol effects. *Neuroscience Letters, 559*(Supplement C), 199–204. http://dx.doi.org/10.1016/j.neulet.2013.12.003.
Korucuoglu, O., Gladwin, T. E., & Wiers, R. W. (2016). The effect of acute alcohol on motor-related EEG asymmetries during preparation of approach or avoid alcohol responses. *Biological Psychology, 114*, 81–92. http://dx.doi.org/10.1016/j.biopsycho.2015.12.012.
Korucuoglu, O., Sher, K. J., Wood, P. K., Saults, J. S., Altamirano, L., Miyake, A., & Bartholow, B. D. (2017). Acute alcohol effects on set-shifting and its moderation by baseline individual differences: A latent variable analysis. *Addiction, 112*(3), 442–453. http://dx.doi.org/10.1111/add.13684.
Krmpotich, T. D., Tregellas, J. R., Thompson, L. L., Banich, M. T., Klenk, A. M., & Tanabe, J. L. (2013). Resting-state activity in the left executive control network is associated with behavioral approach and is increased in substance dependence. *Drug and Alcohol Dependence, 129*(1–2), 1–7. http://dx.doi.org/10.1016/j.drugalcdep.2013.01.021.
Kühn, S., & Gallinat, J. (2011). Common biology of craving across legal and illegal drugs–a quantitative meta-analysis of cue-reactivity brain response. *European Journal of Neuroscience, 33*(7), 1318–1326. http://dx.doi.org/10.1111/j.1460-9568.2010.07590.x.
Kwako, L. E., Momenan, R., Litten, R. Z., Koob, G. F., & Goldman, D. (2016). Addictions neuroclinical assessment: A neuroscience-based framework for addictive disorders. *Biological Psychiatry, 80*(3), 179–189. http://dx.doi.org/10.1016/j.biopsych.2015.10.024.
Lechner, W. V., Day, A. M., Metrik, J., Leventhal, A. M., & Kahler, C. W. (2016). Effects of alcohol-induced working memory decline on alcohol consumption and adverse consequences of use. *Psychopharmacology, 233*(1), 83–88. http://dx.doi.org/10.1007/s00213-015-4090-z.
Lindgren, Kristen P., Baldwin, Scott A., Olin, Cecilia C., Wiers, Reinout W., Teachman, Bethany A., Norris, Jeanette, ... Neighbors, Clayton (2017). Evaluating within-person change in implicit measures of alcohol associations: Increases in alcohol associations predict increases in drinking risk and vice versa. *Alcohol and Alcoholism*. http://dx.doi.org/10.1093/alcalc/agy012.
Lindgren, K. P., Neighbors, C., Gasser, M. L., Ramirez, J. J., & Cvencek, D. (2017). A review of implicit and explicit substance self-concept as a predictor of alcohol and tobacco use and misuse. *The American Journal of Drug and Alcohol Abuse, 43*(3), 237–246. http://dx.doi.org/10.1080/00952990.2016.1229324.
Lindgren, K. P., Neighbors, C., Teachman, B. A., Baldwin, S. A., Norris, J., Kaysen, D., ... Wiers, R. W. (2016). Implicit alcohol associations, especially drinking identity, predict drinking over time. *Health Psychology, 35*(8), 908–918. http://dx.doi.org/10.1037/hea0000396.
Loewenstein, G. F., & Prelec, D. (1993). Preferences for sequences of outcomes. *Psychological Review, 100*(1), 91–108. http://dx.doi.org/10.1037/0033-295X.100.1.91.
Lubman, D. I., Yücel, M., Kettle, J. W. L., Scaffidi, A., MacKenzie, T., Simmons, J. G., & Allen, N. B. (2009). Responsiveness to drug cues and natural rewards in opiate addiction: Associations with later heroin use. *Archives of General Psychiatry, 66*(2), 205–213. http://dx.doi.org/10.1001/archgenpsychiatry.2008.522.
Luehring-Jones, P., Dennis-Tiwary, T. A., Murphy, J. G., Dennhardt, A., Lindgren, K. P., Yarmush, D. E., & Erblich, J. (2016). Favorable associations with alcohol and impaired self-regulation: A behavioral economic analysis. *Drug and Alcohol Dependence, 163*, 172–178. http://dx.doi.org/10.1016/j.drugalcdep.2016.04.017.
Ma, N., Liu, Y., Fu, X.-M., Li, N., Wang, C.-X., Zhang, H., & Zhang, D.-R. (2011). Abnormal brain default-mode network functional connectivity in drug addicts. *PLoS One, 6*(1), e16560. http://dx.doi.org/10.1371/journal.pone.0016560.
Ma, X., Qiu, Y., Tian, J., Wang, J., Li, S., Zhan, W., & Xu, Y. (2015). Aberrant default-mode functional and structural connectivity in heroin-dependent individuals. *PLoS One, 10*(4), e0120861. http://dx.doi.org/10.1371/journal.pone.0120861.
MacKillop, J., Amlung, M. T., Acker, J., Gray, J. C., Brown, C. L., Murphy, J. G., ... Sweet, L. H. (2014). The neuroeconomics of alcohol demand: An initial investigation of the neural correlates of alcohol cost-benefit decision making in heavy drinking men. *Neuropsychopharmacology, 39*(8), 1988–1995. http://dx.doi.org/10.1038/npp.2014.47.
Melaugh McAteer, A., Curran, D., & Hanna, D. (2015). Alcohol attention bias in adolescent social drinkers: An eye tracking study. *Psychopharmacology, 232*(17), 3183–3191. http://dx.doi.org/10.1007/s00213-015-3969-z.
Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Sciences, 15*(10), 483–506. http://dx.doi.org/10.1016/j.tics.2011.08.003.
Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: A network model of insula function. *Brain Structure and Function, 214*(5–6), 655–667. http://dx.doi.org/10.1007/s00429-010-0262-0.
Meshesha, L. Z., Pickover, A. M., Teeters, J. B., & Murphy, J. G. (2017). A longitudinal behavioral economic analysis of non-medical prescription opioid use among college students. *The Psychological Record, 67*(2), 241–251. http://dx.doi.org/10.1007/s40732-017-0235-2.
Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*(1), 167–202. http://dx.doi.org/10.1146/annurev.neuro.24.1.167.
Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions: Four general conclusions. *Current Directions in Psychological Science, 21*(1), 8–14. http://dx.doi.org/10.1177/0963721411429458.
Mokdad, A. H., Forouzanfar, M. H., Daoud, F., Mokdad, A. A., El Bcheraoui, C., Moradi-Lakeh, M., ... Murray, C. J. L. (2016). Global burden of diseases, injuries, and risk factors for young people's health during 1990–2013: A systematic analysis for the Global Burden of Disease Study 2013. *The Lancet, 387*(10036), 2383–2401. http://dx.doi.org/10.1016/S0140-6736(16)00648-6.
Morrison, A. B., & Chein, J. M. (2011). Does working memory training work? The promise and challenges of enhancing cognition by training working memory. *Psychonomic Bulletin and Review, 18*(1), 46–60. http://dx.doi.org/10.3758/s13423-010-0034-0.
Motzkin, J. C., Baskin-Sommers, A., Newman, J. P., Kiehl, K. A., & Koenigs, M. (2014). Neural correlates of substance abuse: Reduced functional connectivity between areas underlying reward and cognitive control. *Human Brain Mapping, 35*(9), 4282–4292. http://dx.doi.org/10.1002/hbm.22474.
Müller-Oehring, E. M., Jung, Y.-C., Pfefferbaum, A., Sullivan, E. V., & Schulte, T. (2015). The resting brain of alcoholics. *Cerebral Cortex, 25*(11), 4155–4168. http://dx.doi.org/10.1093/cercor/bhu134.
Müller-Oehring, E. M., Kwon, D., Nagel, B. J., Sullivan, E. V., Chu, W., Rohlfing, T., ... Pohl, K. M. (2017). Influences of age, sex, and moderate alcohol drinking on the intrinsic functional architecture of adolescent brains. *Cerebral Cortex,* 1–15. http://dx.doi.org/10.1093/cercor/bhx014.
Murphy, J. G., Barnett, N. P., & Colby, S. M. (2006). Alcohol-related and alcohol-free activity participation and enjoyment among college students: A behavioral theories of choice analysis. *Experimental and Clinical Psychopharmacology, 14*(3), 339–349. http://dx.doi.org/10.1037/1064-1297.14.3.339.
Murphy, J. G., & Dennhardt, A. A. (2016). The behavioral economics of young adult substance abuse. *Preventive Medicine, 92*, 24–30. http://dx.doi.org/10.1016/j.ypmed.2016.04.022.
Murphy, J. G., Dennhardt, A. A., Skidmore, J. R., Borsari, B., Barnett, N. P., Colby, S. M., & Martens, M. P. (2012). A randomized controlled trial of a behavioral economic supplement to brief motivational interventions for college drinking. *Journal of Consulting and Clinical Psychology, 80*, 876–886. http://dx.doi.org/10.1037/a0028763.
Murphy, J. G., Dennhardt, A. D., Yurasek, A. M., Skidmore, J. R., Martens, M. P., MacKillop, J., & McDevitt-Murphy, M. E. (2015). Behavioral economic predictors of brief alcohol intervention outcomes. *Journal of Consulting and Clinical Psychology, 83*(6), 1033–1043. http://dx.doi.org/10.1037/ccp0000032.
Murphy, J. G., Yurasek, A. M., Dennhardt, A. A., Skidmore, J. R., McDevitt-Murphy, M. E., MacKillop, J., & Martens, M. P. (2013). Symptoms of depression and PTSD are associated with elevated alcohol demand. *Drug and Alcohol Dependence, 127*(1–3), 129–136. http://dx.doi.org/10.1016/j.drugalcdep.2012.06.022.
Norman, A. L., Pulido, C., Squeglia, L. M., Spadoni, A. D., Paulus, M. P., & Tapert, S. F. (2011). Neural activation during inhibition predicts initiation of substance use in adolescence. *Drug and Alcohol Dependence, 119*(3), 216–223. http://dx.doi.org/10.1016/j.drugalcdep.2011.06.019.

Nosek, B. A., Hawkins, C. B., & Frazier, R. S. (2011). Implicit social cognition: From measures to mechanisms. *Trends in Cognitive Sciences, 15*(4), 152–159. http://dx.doi.org/10.1016/j.tics.2011.01.005.

O'Connor, R. M., & Colder, C. R. (2015). The prospective joint effects of self-regulation and impulsive processes on early adolescence alcohol use. *Journal of Studies on Alcohol and Drugs, 76*(6), 884–894. http://dx.doi.org/10.15288/jsad.2015.76.884.

O'Connor, R. M., Lopez-Vergara, H. I., & Colder, C. R. (2012). Implicit cognition and substance use: The role of controlled and automatic processes in children. *Journal of Studies on Alcohol and Drugs, 73*(1), 134–143. http://dx.doi.org/10.15288/jsad.2012.73.134.

Oscar-Berman, M., & Marinković, K. (2007). Alcohol: Effects on neurobehavioral functions and the brain. *Neuropsychology Review, 17*(3), 239–257. http://dx.doi.org/10.1007/s11065-007-9038-6.

Ostafin, B. D., Bauer, C., & Myxter, P. (2012). Mindfulness decouples the relation between automatic alcohol motivation and heavy drinking. *Journal of Social and Clinical Psychology, 31*(7), 729–745. http://dx.doi.org/10.1521/jscp.2012.31.7.729.

Papachristou, H., Nederkoorn, C., Havermans, R., Bongers, P., Beunen, S., & Jansen, A. (2013). Higher levels of trait impulsiveness and a less effective response inhibition are linked to more intense cue-elicited craving for alcohol in alcohol-dependent patients. *Psychopharmacology, 228*(4), 641–649. http://dx.doi.org/10.1007/s00213-013-3063-3.

Park, H.-J., & Friston, K. (2013). Structural and functional brain networks: From connections to cognition. *Science, 342*(6158), 1238411. http://dx.doi.org/10.1126/science.1238411.

Peeters, M., Koning, I., Monshouwer, K., Vollebergh, W. A. M., & Wiers, R. W. (2016). Context effects of alcohol availability at home: Implicit alcohol associations and the prediction of adolescents' drinking behavior. *Journal of Studies on Alcohol and Drugs, 77*(5), 749–756. http://dx.doi.org/10.15288/jsad.2016.77.749.

Pieters, S., Burk, W. J., Van der Vorst, H., Engels, R. C., & Wiers, R. W. (2014). Impulsive and reflective processes related to alcohol use in young adolescents. *Frontiers in Psychiatry, 5*, 1–8. http://dx.doi.org/10.3389/fpsyt.2014.00056.

Rachlin, H., Logue, A. W., Gibbon, J., & Frankel, M. (1986). Cognition and behavior in studies of choice. *Psychological Review, 93*(1), 33–45. http://dx.doi.org/10.1037/0033-295X.93.1.33.

Raichle, M. E., & Snyder, A. Z. (2007). A default mode of brain function: A brief history of an evolving idea. *NeuroImage, 37*(4), 1083–1090. http://dx.doi.org/10.1016/j.neuroimage.2007.02.041.

Ramirez, J. J., Dennhardt, A. A., Baldwin, S. A., Murphy, J. G., & Lindgren, K. P. (2016). Alcohol-approach inclinations and drinking identity as predictors of behavioral economic demand for alcohol. *Experimental and Clinical Psychopharmacology, 24*(5), 356–366. http://dx.doi.org/10.1037/pha0000086.

Reich, R. R., Below, M. C., & Goldman, M. S. (2010). Explicit and implicit measures of expectancy and related alcohol cognitions: A meta-analytic comparison. *Psychology of Addictive Behaviors, 24*(1), 13–25. http://dx.doi.org/10.1037/a0016556.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews, 18*(3), 247–291. http://dx.doi.org/10.1016/0165-0173(93)90013-P.

Salemink, E., & Wiers, R. W. (2014). Alcohol-related memory associations in positive and negative affect situations: Drinking motives, working memory capacity, and prospective drinking. *Psychology of Addictive Behaviors, 28*, 105–113. http://dx.doi.org/10.1037/a0032806.

Sayette, M. A. (2016). The role of craving in substance use disorders: Theoretical and methodological issues. *Annual Review of Clinical Psychology, 12*, 407–433. http://dx.doi.org/10.1146/annurev-clinpsy-021815-093351.

Schmaal, L., Goudriaan, A. E., Joos, L., Krüse, A. M., Dom, G., van den Brink, W., & Veltman, D. J. (2013). Modafinil modulates resting-state functional network connectivity and cognitive control in alcohol-dependent patients. *Biological Psychiatry, 73*(8), 789–795. http://dx.doi.org/10.1016/j.biopsych.2012.12.025.

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., & Greicius, M. D. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *Journal of Neuroscience, 27*(9), 2349–2356. http://dx.doi.org/10.1523/JNEUROSCI.5587-06.2007.

Shono, Y., Grenard, J. L., Ames, S. L., & Stacy, A. W. (2014). Application of item response theory to tests of substance-related associative memory. *Psychology of Addictive Behaviors, 28*(3), 852–862. http://dx.doi.org/10.1037/a0035877.

Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in behavioural inhibition in substance abuse and addiction: A meta-analysis. *Drug and Alcohol Dependence, 145*, 1–33. http://dx.doi.org/10.1016/j.drugalcdep.2014.08.009.

Spreng, R. N., Mar, R. A., & Kim, A. S. N. (2009). The common neural basis of autobiographical memory, prospection, navigation, theory of mind, and the default mode: A quantitative meta-analysis. *Journal of Cognitive Neuroscience, 21*(3), 489–510. http://dx.doi.org/10.1162/jocn.2008.21029.

Stacy, A. W., & Wiers, R. W. (2010). Implicit cognition and addiction: A tool for explaining paradoxical behavior. *Annual Review of Clinical Psychology, 6*, 551–575. http://dx.doi.org/10.1146/annurev.clinpsy.121208.131444.

Stanger, C., Ryan, S. R., Fu, H., Landes, R. D., Jones, B. A., Bickel, W. K., & Budney, A. J. (2012). Delay discounting predicts adolescent substance abuse treatment outcome. *Experimental and Clinical Psychopharmacology, 20*(3), 205–212. http://dx.doi.org/10.1037/a0026543.

Stavro, K., Pelletier, J., & Potvin, S. (2013). Widespread and sustained cognitive deficits in alcoholism: A meta-analysis. *Addiction Biology, 18*(2), 203–213. http://dx.doi.org/10.1111/j.1369-1600.2011.00418.x.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and Social Psychology Review, 8*(3), 220–247. http://dx.doi.org/10.1207/s15327957pspr0803_1.

Sutherland, M. T., McHugh, M. J., Pariyadath, V., & Stein, E. A. (2012). Resting state functional connectivity in addiction: Lessons learned and a road ahead. *NeuroImage, 62*(4), 2281–2295. http://dx.doi.org/10.1016/j.neuroimage.2012.01.117.

Teeters, J. B., & Murphy, J. G. (2015). The behavioral economics of driving after drinking among college drinkers. *Alcoholism: Clinical and Experimental Research, 39*, 896–904. http://dx.doi.org/10.1111/acer.12695.

Topiwala, A., Allan, C., Valkanova, V., Zsoldos, E., Filippini, N., Sexton, C., ... Ebmeier, K. (2017). Moderate alcohol consumption as risk factor for adverse brain outcomes and cognitive decline: Longitudinal cohort study. *BMJ, 357*, J2353. http://dx.doi.org/10.1136/bmj.j2353.

Tucker, J. A., Roth, D. L., Vignolo, M. J., & Westfall, A. O. (2009). A behavioral economic reward index predicts drinking resolutions: Moderation revisited and compared with other outcomes. *Journal of Consulting and Clinical Psychology, 77*(2), 219–228. http://dx.doi.org/10.1037/a0014968.

U.S. Department of Health and Human Services (HHS), Office of the Surgeon General (2016). Facing addiction in America: The Surgeon General's report on alcohol, drugs, and health. Retrieved from https://addiction.surgeongeneral.gov/surgeon-generals-report.pdf.

Van Der Vorst, H., Krank, M., Engels, R. C. M. E., Pieters, S., Burk, W. J., & Mares, S. H. W. (2013). The mediating role of alcohol-related memory associations on the relation between perceived parental drinking and the onset of adolescents' alcohol use. *Addiction, 108*(3), 526–533. http://dx.doi.org/10.1111/add.12042.

Volkow, N. D., & Baler, R. D. (2015). NOW vs LATER brain circuits: Implications for obesity and addiction. *Trends in Neurosciences, 38*(6), 345–352. http://dx.doi.org/10.1016/j.tins.2015.04.002.

Vollstädt-Klein, S., Wichert, S., Rabinstein, J., Bühler, M., Klein, O., Ende, G., & Mann, K. (2010). Initial, habitual and compulsive alcohol use is characterized by a shift of cue processing from ventral to dorsal striatum. *Addiction, 105*(10), 1741–1749. http://dx.doi.org/10.1111/j.1360-0443.2010.03022.x.

Vuchinich, R. E., & Heather, N. (2003). Introduction: Overview of behavioural economic perspectives on substance use and addiction. In R. E. Vuchinich, & N. Heather (Eds.). *Choice, behavioural economics and addiction* (pp. 1–31). . [e-book] https://doi.org/10.1016/B978-008044056-9/50042-7.

Vuchinich, R. E., & Tucker, J. A. (1988). Contributions from behavioral theories of choice to an analysis of alcohol abuse. *Journal of Abnormal Psychology, 97*(2), 181–195. http://dx.doi.org/10.1037/0021-843X.97.2.181.

Wang, Z., Suh, J., Li, Z., Li, Y., Franklin, T., O'Brien, C., & Childress, A. R. (2015). A hyper-connected but less efficient small-world network in the substance-dependent brain. *Drug and Alcohol Dependence, 152*, 102–108. http://dx.doi.org/10.1016/j.drugalcdep.2015.04.015.

Weafer, J., Dzemidzic, M., Eiler, W., Oberlin, B. G., Wang, Y., & Kareken, D. A. (2015). Associations between regional brain physiology and trait impulsivity, motor inhibition, and impaired control over drinking. *Psychiatry Research: Neuroimaging, 233*(2), 81–87. http://dx.doi.org/10.1016/j.pscychresns.2015.04.010.

Weafer, J., & Fillmore, M. T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology, 201*(3), 315–324. http://dx.doi.org/10.1007/s00213-008-1284-7.

Werntz, A. J., Steinman, S. A., Glenn, J. J., Nock, M. K., & Teachman, B. A. (2016). Characterizing implicit mental health associations across clinical domains. *Journal of Behavior Therapy and Experimental Psychiatry, 52*, 17–28. http://dx.doi.org/10.1016/j.jbtep.2016.02.004.

Wesley, M. J., Lile, J. A., Fillmore, M. T., & Porrino, L. J. (2017). Neurophysiological capacity in a working memory task differentiates dependent from nondependent heavy drinkers and controls. *Drug and Alcohol Dependence, 175*, 24–35. http://dx.doi.org/10.1016/j.drugalcdep.2017.01.029.

Wiers, C. E., Gladwin, T. E., Ludwig, V. U., Gröpper, S., Stuke, H., Gawron, C. K., ... Bermpohl, F. (2017). Comparing three cognitive biases for alcohol cues in alcohol dependence. *Alcohol and Alcoholism, 52*(2), 242–248. http://dx.doi.org/10.1093/alcalc/agw063.

Wiers, R. W., Boffo, M., & Field, M. (2018). What's in a trial? On the importance of distinguishing between experimental lab-studies and randomized controlled trials; the case of cognitive bias modification and alcohol use disorders. *Journal of Studies on Alcohol and Drugs*. (in press) https://livrepository.liverpool.ac.uk/3010437/ (ISSN 0096-882X) .

Wiers, R. W., Eberl, C., Rinck, M., Becker, E., & Lindenmeyer, J. (2011). Re-training automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychological Science, 22*(4), 490–497. http://dx.doi.org/10.1177/0956797611400615.

Wiers, R. W., & Gladwin, T. E. (2017). Reflective and impulsive processes in addiction and the role of motivation. In R. Deutsch, B. Gawronski, & W. Hofmann (Eds.). *Reflective and impulsive determinants of human behavior* (pp. 173–188). New York: Psychology Press.

Wiers, R. W., Gladwin, T. E., Hofmann, W., Salemink, E., & Ridderinkhof, K. R. (2013). Cognitive bias modification and cognitive control training in addiction and related psychopathology: Mechanisms, clinical perspectives, and ways forward. *Clinical Psychological Science: A Journal of the Association for Psychological Science, 1*(2), 192–212. http://dx.doi.org/10.1177/2167702612466547.

Wiers, R. W., Rinck, M., Kordts, R., Houben, K., & Strack, F. (2010). Retraining automatic action-tendencies to approach alcohol in hazardous drinkers. *Addiction, 105*(2), 279–287. http://dx.doi.org/10.1111/j.1360-0443.2009.02775.x.

Wilcox, C. E., Mayer, A. R., Bogenschutz, M. P., Ling, J., Dekonenko, C., & Cumbo, H. (2015). Cognitive control network function in alcohol use disorder before and during treatment with lorazepam. *Substance Use & Misuse, 50*(1), 40–52. http://dx.doi.org/10.3109/10826084.2014.957771.

Wolff, N., von Hippel, C., Brener, L., & von Hippel, W. (2015). Implicit identification with drug and alcohol use predicts retention in residential rehabilitation programs. *Psychology of Addictive Behaviors, 29*(1), 136–141. http://dx.doi.org/10.1037/

adb0000004.
World Health Organization (2015). Alcohol. Retrieved from http://www.who.int/mediacentre/factsheets/fs349/en/.
Woud, M. L., Fitzgerald, D. A., Wiers, R. W., Rinck, M., & Becker, E. S. (2012). 'Getting into the spirit': Alcohol-related interpretation bias in heavy-drinking students. *Psychology of Addictive Behaviors, 26*, 627–632. http://dx.doi.org/10.1037/a0029025.
Woud, M. L., Hutschemaekers, M. H., Rinck, M., & Becker, E. S. (2015). The manipulation of alcohol-related interpretation biases by means of Cognitive Bias Modification–Interpretation (CBM-I). *Journal of Behavior Therapy and Experimental Psychiatry, 49*, 61–68. http://dx.doi.org/10.1016/j.jbtep.2015.03.001.
Yurasek, A. M., Murphy, J. G., Dennhardt, A. A., Skidmore, J. R., Buscemi, J., McCausland, C., & Martens, M. P. (2011). Drinking motives mediate the relationship between reinforcing efficacy and alcohol consumption and problems. *Journal of Studies on Alcohol and Drugs, 72*(6), 991–999. http://dx.doi.org/10.15288/jsad.2011.72.991.
Zhu, X., Sundby, K., Bjork, J. M., & Momenan, R. (2016). Alcohol dependence and altered engagement of brain networks in risky decisions. *Frontiers in Human Neuroscience, 10*. http://dx.doi.org/10.3389/fnhum.2016.00142.
Zucker, R. A., Donovan, J. E., Masten, A. S., Mattson, M. E., & Moss, H. B. (2008). Early developmental processes and the continuity of risk for underage drinking and problem drinking. *Pediatrics, 121*(Suppl. 4), S252–272. http://dx.doi.org/10.1542/peds.2007-2243B.