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An Evolutionary Perspective on the Co-Occurrence of Social Anxiety Disorder and Alcohol Use Disorder

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Abstract

Social Anxiety Disorder (SAD) commonly co-occurs with, and often precedes, Alcohol Use Disorder (AUD). In this paper, we address the relationship between SAD and AUD by considering how natural selection left socially anxious individuals vulnerable to alcohol use, and by addressing the underlying mechanisms. We review research suggesting that social anxiety has evolved for the regulation of behaviors involved in reducing the likelihood or consequences of threats to social status. The management of potential threats to social standing is important considering that these threats can result in reduced cooperation or ostracism – and therefore to reduced access to coalitional partners, resources or mates. Alcohol exerts effects upon evolutionarily conserved emotion circuits, and can down-regulate or block anxiety (or may be expected to do so). As such, the ingestion of alcohol can artificially signal the absence or successful management of social threats. In turn, alcohol use may be reinforced in socially anxious people because of this reduction in subjective malaise, and because it facilitates social behaviors – particularly in individuals for whom the persistent avoidance of social situations poses its own threat (i.e., difficulty finding mates). Although the frequent co-occurrence of SAD and AUD is associated with poorer treatment outcomes than either condition alone, a richer understanding of the biological and psychosocial drives underlying susceptibility to alcohol use among socially anxious individuals may improve the efficacy of therapeutic interventions aimed at preventing or treating this comorbidity.

Keywords: social anxiety disorder, alcohol use disorder, comorbidity, evolution

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Social Anxiety Disorder (SAD) and substance use disorders commonly co-occur in national samples (Buckner et al., 2012; Buckner et al., 2008; Grant, Stinson, Dawson, & et al., 2004; Kessler et al., 1997; Smith & Book, 2008; M. B. Stein & Stein, 2008). For instance, findings from the National Epidemiological Survey on Alcohol and Related Conditions (NESARC) – based on a representative sample of the U.S. population – suggest that the prevalence of substance use disorders among those with SAD is approximately 16% relative to only 9.35% in the general population, and that those with a substance disorder diagnosis in the past year have twice the odds of also having a past year SAD diagnosis relative to those without a substance disorder (Grant et al. 2004). This poses a significant public health concern because the conjunction of these conditions bears greater morbidity, and is associated with lower rates of treatment seeking and poorer treatment outcomes than either condition alone (Randall, Thomas, & Thevos, 2001; Schneier et al., 2010). While social anxiety disorder is known to co-occur frequently with substance use disorder sub-types, alcohol use disorder is among the most prevalent comorbid conditions (for reviews see Buckner, Heimberg, Ecker, & Vinci, 2013; Morris, Stewart, & Ham, 2005).

Specifically, epidemiological findings point to a frequent co-occurrence of SAD with alcohol use disorder (AUD), estimating that approximately 13% of those with SAD have an AUD, relative to 8.5% of the general population (Buckner et al., 2008; Grant et al., 2005; Kessler et al., 1994; Morris et al., 2005; Ross, Glaser, & Germanson, 1988; Schneier et al., 2010). To the extent that SAD precedes AUD, such comorbidity may present an opportunity for early intervention. Indeed, SAD has been found to precede AUD in as many as 80% of comorbid cases, and those with SAD at baseline have been found to have over four times the odds of developing AUD at follow-up after controlling for sociodemographic and psychiatric

confounders (including mood, personality and other anxiety disorders) (Buckner et al., 2008; Schneier et al., 2010). Though it is often impractical to disentangle the effects of other conditions occurring concurrently with SAD on alcohol use, higher co-occurrence with depression and anxiety sub-types may be associated with greater vulnerability to and severity of AUD (Hasin, Stinson, Ogburn, & Grant, 2007; Martins, Fenton, et al., 2012; Schneier et al., 2010).

In the present paper, we address proximate and ultimate explanations for the forward association between SAD and AUD. Whereas proximate explanations focus on the traits and mechanisms that make individuals with SAD susceptible to AUD and the co-development of these disorders, ultimate (distal) explanations focus on why SAD and AUD frequently co-occur by considering the evolutionary history and adaptive significance of the underlying traits and mechanisms (Nesse, 2013; D. J. Stein, 2006; Tinbergen, 1963). We now turn to the adaptive significance and proximate mechanisms underlying social anxiety, which can be viewed as the adaptive trait from which SAD deviates.

---Insert Figure 1 here---

Figure 1. A comprehensive account of a trait (circle) requires complementary proximate and ultimate explanations (hexagons) that can be further specified based on Tinbergen's (1963) four questions (squares). Here, we seek to explain the lifetime comorbidity between SAD and AUD by addressing proximate and ultimate questions about each of the underlying traits and mechanisms involved in this relationship.

Adaptive significance and proximate mechanisms in social anxiety

Throughout the course of hominin evolution, humans and their ancestral predecessors encountered persistent threats to reproductive fitness. The selective pressure afforded by recurring threats has shaped the evolution of systems to mitigate their potential fitness costs (Nesse, 1990). Anxiety, which is highly conserved in mammalian species, is a keystone of the

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systems evolved to deal with fitness-related threats (Marks & Nesse, 1994). Anxiety can be defined loosely as the suite of physiological, cognitive and affective changes that produce vigilance, hyper-arousal, and attentional biases towards threat-related stimuli (Bateson, Brilot, & Nettle, 2011; Mathews, Mackintosh, & Fulcher, 1997). Recurrent threats to fitness in hominin evolution included not only physical danger from predation, violence, and pathogens, but also *social threats* to status that could result in reduced access to resources or mates, or ostracism from a social group in extreme cases (Boyer & Bergstrom, 2011; Gilbert, 2003). Increases in social group size and associated social cognitive demands have been theorized to have driven neocortex size increases in hominins (Barton & Dunbar, 1997; Byrne & Whiten, 1989; Dunbar, 2002; though see Holekamp, 2007; Reader, Hager, & Laland, 2011), and it is therefore plausible that social group complexity has increased during recent hominin evolution (Shultz, Nelson, & Dunbar, 2012). This may have resulted in strong selection pressure on mechanisms for tracking social threats, and these mechanisms may underpin social anxiety.

Anxiety is fundamentally concerned with *potential* threats to fitness, inasmuch as it prepares an organism to face a future threat that is expected to materialize (Adolphs, 2013; Boyer & Lienard, 2006; Damasio, 2010; Lang, Davis, & Ohman, 2000; Marks & Nesse, 1994; Miloyan, Bulley, & Suddendorf, 2015; Woody & Szechtman, 2011). As such, higher levels of social anxiety are associated with faster orientation towards negative social cues, and more generally to threat-related social information (Buckner, Maner, & Schmidt, 2010; Mansell & Clark, 1999; Mogg, Philippot, & Bradley, 2004; Schulze, Lobmaier, Arnold, & Renneberg, 2013). Individuals with high degrees of social anxiety also show increased gaze avoidance when confronted with threatening facial cues, although critically this appears to be most pronounced under conditions of social-evaluative threat (Buckner, DeWall, Schmidt, & Maner, 2010; Mansell, Clark, Ehlers, & Chen, 1999). Gaze avoidance away from negative emotional faces could be considered adaptive under conditions of evaluative threat (e.g., from

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a higher status individual), insofar as submission may mitigate the risk of reduced social cooperation and consequently reduced access to mates or resources (Gilbert, 2001, 2003).

Indeed, low socioeconomic status is a strong risk factor for incident SAD (Grant et al., 2005; Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996) – though this is also the case for other mental and behavioral disorders (Muntaner, Eaton, Miech, & O’Campo, 2004; Wells, Tien, Garrison, & Eaton, 1994), and low socioeconomic status may be both a cause and effect of psychopathology (Martins, Ko, et al., 2012; Miech, Caspi, Moffitt, Wright, & Silva, 1999; Yu & Williams, 1999).

Despite gaze avoidance under conditions of evaluative threat, individuals with high degrees of social anxiety tend to exhibit an attentional bias toward their own thoughts following social threat detection (Mansell, Clark, & Ehlers, 2003; Pineles & Mineka, 2005). This threat-related attentional bias, though directed inward, is associated with increased physiological arousal (McTeague et al., 2009; Mogg et al., 2004; Moscovitch, Suvak, & Hofmann, 2010; Wood, Ver Hoef, & Knight, 2014). In the context of social anxiety, a propensity for self-reflection presumably allows the individual to observe him or herself from another’s perspective, and to potentially prepare an appropriate response to a threat (Miloyan et al., 2015). Self-reflection, or attention directed toward one’s own thoughts, may be particularly active in situations where external task demands are sufficiently low (in this case, during laboratory tasks, or when an individual is not currently in the relevant social situation) (Smallwood & Schooler, 2015). In sum, clinically socially anxious individuals display many of the hallmarks of a classic anxiety profile in response to *socially* threatening stimuli, such that they more readily perceive social threat cues, are more likely to express defensive responses toward those cues, and generalize this response to situations or features of stimuli that were previously associated with socially threatening situations (Clark & Wells, 1995; Gilbert, 2001).

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Individuals with SAD tend to avoid interpersonal situations or endure them with extreme discomfort due to a pervasive fear of criticism, rejection, or humiliation. Although socially anxious individuals tend to avoid encounters with people they don't know well, or complex social situations altogether, they may relish interpersonal relations with known others, or more generally, social encounters in safe settings or environments (Martin & Quirk, 2015; Schneier, 2006; M. B. Stein & Stein, 2008). Thus, social anxiety may aptly be viewed as a continuum of sensitivity to unknown, complex, and unsafe social situations, such that individuals at one end tend to evaluate threats in such social situations more readily than those at the other.

In contrast to individuals with SAD who show hyperactive amygdala activity in response to socially threatening cues (Etkin & Wager, 2007; Phan, Fitzgerald, Nathan, & Tancer, 2006; M. B. Stein, Goldin, Sareen, Zorrilla, & Brown, 2002), individuals with bilateral amygdala damage demonstrate impaired detection of angry faces in a crowd, fail to account for social norms in laboratory tasks, and demonstrate poor social decision-making abilities (Bach, Hurlemann, & Dolan, 2015; Feinstein, Adolphs, Damasio, & Tranel, 2011; Kennedy, Glascher, Tyszka, & Adolphs, 2009; M. B. Stein & Stein, 2008; Tranel, Gullickson, Koch, & Adolphs, 2006). Similarly, individuals with lesions to the ventromedial prefrontal cortex (vmPFC) demonstrate impaired physiological responses to aversive social stimuli, poorly synchronize conversational rhythms with other people, and exhibit low empathy, a lack of affective theory of mind (i.e., for others' feelings but not thoughts) and abnormal social conduct, which are associated with poor real-world social and occupational outcomes in employment and relationships (Damasio, Tranel, & Damasio, 1990; Eslinger, 1998; Gordon, Tranel, & Duff, 2014; Gupta, Tranel, & Duff, 2012; Hynes, Baird, & Grafton, 2006; Shamay-Tsoory, Tibi-Elhanany, & Aharon-Peretz, 2006).

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This is not to suggest that the vmPFC and the amygdala are the sole neural substrates of social anxiety, but rather to illustrate that a lack of social anxiety can be dysfunctional because it leads to a failure to deploy appropriate social behaviors. Likewise, although SAD may not be adaptive, given that it is associated with poor functional outcomes, it may represent an extreme on a continuum of a generally adaptive trait that motivates individuals to pay appropriate credence to the navigation of social interactions in order to acquire resources, coalitional partners, and mates. Social worries such as concerns about being disliked are functional in light of the critical and detrimental implications for reproductive fitness of losing access to mates and resources in a social group (Kurzban & Leary, 2001). Indeed, it has been argued that feelings of social anxiety may lead individuals to attempt to behave in socially acceptable ways in a form of impression or reputation management, and to minimize the risk of reduced social cooperation by activating appropriate dispositional responses (e.g., inhibition, submission) in the face of socially mediated threats (Gilbert, 2001). Individuals with very high levels of social anxiety may perceive social threats and trigger such ‘social threat management’ responses very readily, which may turn out to be costly to both reproductive fitness and subjective wellbeing. Those with SAD may therefore be highly susceptible to the anxiolytic effects of alcohol, to which we now turn.

The end-directed use of alcohol

Different psychoactive drugs have overlapping though largely heterogeneous effects on the central nervous system, with varying effects on subjective state. Such compounds act on evolutionarily conserved emotion and reward/punishment systems that humans share with other mammals (Nesse & Berridge, 1997). Different emotions are associated with partially segregated neurochemical profiles, and particular classes of drugs may act differently upon these systems (Panksepp, 1998). For instance, central nervous system stimulants like cocaine act on ascending mesolimbic dopaminergic systems and serve to increase appetitive

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motivation and generate positive feelings and high levels of arousal (Panksepp, Knutson, & Burgdorf, 2002). Conversely, anxiolytic drugs such as benzodiazepines, opiates, and alcohol are thought to work, alongside their effects on other neurotransmitter systems, by facilitating the transmission of γ -Aminobutyric acid (GABA), an inhibitory neurotransmitter (for review see Ticku, 1990). Because anxiety involves a state of hyperarousal, the drug-based reduction in autonomic arousal levels can result in a perceived reduction in anxiety symptoms (Robinson & Bolton, 2013).

Many accounts of drug-taking behaviors draw on the perceived 'utility' of certain drugs in altering subjective experience and behavior as an important predictor of use (Boys & Marsden, 2003; Boys et al., 1999; Boys, Marsden, & Strang, 2001; Cappell, 1975; Müller & Schumann, 2011). In other words, people are thought to take drugs as a 'means to an end', such as changing their mood, managing a social situation, or coping with psychological distress (Boys & Marsden, 2003; Boys et al., 1999; Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010; Schafer & Brown, 1991). For instance, alcohol may be consumed in social settings both because it is expected to provide relief from aversive social anxiety states and to facilitate socialization (Cooper, Frone, Russell, & Mudar, 1995; Peele & Brodsky, 2000). In social anxiety, the motivation to drink so as to reduce the subjective 'tension' felt in social situations and to thereby facilitate social interactions is commonly evoked as a reason for alcohol consumption (Book & Randall, 2002; Cappell, 1975; Cappell & Greeley, 1987). Laboratory-based studies have demonstrated that alcohol use may lead to decreased social anxiety among both clinically and non-clinically anxious participants, and that this reduction may be based both on pharmacological anxiolytic effects and on expectancies that consumption will fulfill this role (for review see Battista, Stewart, & Ham, 2010). In one recent study on the effect of alcohol on social anxiety in real world settings amongst non-clinically anxious people, daily experience sampling revealed that subjective social anxiety

was reduced on average by 4% with every alcoholic drink consumed (Battista et al., 2015). Additionally, a number of individual difference variables such as coping style, impulsivity, and alcohol use expectancies likely influence the relationship between social anxiety and alcohol use (Battista et al., 2010; Cooper, Russell, Skinner, Frone, & Mudar, 1992; Morris et al., 2005; Nicholls, Staiger, Williams, Richardson, & Kambouropoulos, 2014).

The artificial management of fitness threats

In what ways might psychoactive compounds act upon threat management systems involved in social anxiety? The process of threat management involves hypervigilance toward potential social threats, and is largely facilitated by anxiety and its associated attentional biases, such as prioritized attentional allocation towards threat-related stimuli (D. C. Blanchard, Griebel, Pobbe, & Blanchard, 2011; Mogg et al., 2004). As mentioned, social anxiety specifically involves faster orienting to socially threatening information as well as a difficulty disengaging attention from such stimuli (Amir, Elias, Klumpp, & Przeworski, 2003; Mathews et al., 1997; Mogg & Bradley, 1998; Mogg et al., 2004). Cues to potential threats can take many forms, and humans are also capable of voluntarily and involuntarily self-generating anxiety by evoking episodic imagery about potential threats (Miloyan et al., 2015). Nonetheless, upon the detection of perceived social threats, the individual must then determine the appropriate behavioral response option in preparation for facing those threats (Bateson et al., 2011).

As such, once a stimulus is deemed threatening, it must be appropriately managed. For instance, avoidance or ‘pre-flight’ involves increasing the distance between oneself and a potential threat, thereby reducing the risk it may pose to fitness. Social anxiety frequently results in the avoidance of social situations deemed threatening or aversive (Hofmann, Aka, & Piquer, 2014). Conversely, social submission and appeasement comprise a suite of behaviors (e.g., aversion of eye gaze) thought to be involved in the management of a perceived threat to

social status from a dominant social other that cannot be avoided or fled (Schneier, Kent, Star, & Hirsch, 2009; Trower & Gilbert, 1989). This might explain why individuals with a high degree of social anxiety demonstrate an external attentional bias away from emotional faces under conditions of social-evaluative threat (e.g., public speaking, or potential social exclusion), and simultaneous internal bias toward social threat (Buckner, DeWall, et al., 2010; Mansell et al., 2003; Mansell et al., 1999; Pineles & Mineka, 2005).

Once a particular social threat has been managed, it is appropriate for anxiety levels to decrease. Signals can be gleaned from the external environment indicating that a threat is no longer pressing – for example when one has finished a public speech, gone home, and is no longer exposed to the social threat of immediate audience evaluation. The subsequent reduction in anxiety triggered by the ‘management’ of social threats may result in a low-arousal contented positive affect, hallmarked by a reduced sensitivity to threat and punishment (Panksepp et al., 2002). However, surefire external environmental signals that a threat has been successfully and totally managed are rare (maybe impossible) for certain classes of threats (Szechtman & Woody, 2004). For instance, the management of social threats like embarrassment or humiliation may entail avoidance of threat-related social contexts with no clear end goal, especially if one expects that these things are likely to consistently re-occur (Boyer & Lienard, 2006).

Critically, social anxiety is usually attenuated once its functional role has been accomplished, and its subjective correlates become unnecessary (although it may also linger for some time after perceived management of a threat) (Nettle & Bateson, 2012). Thus, affective homeostasis usually results from the perceived successful management or perceived absence of social threats. However, it may also result from the ingestion of particular psychoactive compounds like alcohol, which can artificially down-regulate negative affective states (Battista et al., 2015). In essence, alcohol has the capacity to subdue unpleasant

subjective anxiety, thereby *artificially* signaling that an antecedent threat has been managed successfully, or that there are no pressing social threats.

With the arrival of environmental contingencies that predict a status threat, such as perceived social scrutiny, anxiety may be evoked to motivate and regulate the appropriate management of the situation (e.g., fulfilling a social obligation at the expense of indulging in purely personal interests) (Marks & Nesse, 1994; Nesse, 1990). A psychoactive substance that is perceived to reduce the negative emotionality associated with this threat (e.g., alcohol) may be consumed to counteract these unpleasant feelings (Book & Randall, 2002). In offsetting this negative emotionality, relief is provided and the social anxiety subdues. Without the ingestion of such an anxiolytic compound, anxiety lingers until signals of the appropriate management of the threat are established, with slower attenuation of anxiety among individuals with more extreme manifestations of social anxiety (i.e., people with SAD) (Nettle & Bateson, 2012). Crucially, alcohol can bypass the antecedent and residual social anxiety, which may have evolved to motivate appropriate care when navigating complex social hierarchies. As a result, affective homeostasis is attainable without changes to the external environment that would otherwise only be possible by the successful navigation of a social situation and the resultant signal of appropriate threat management. In effect, the emotions and their evolved functional purpose become decoupled.

Alcohol use patterns in Social Anxiety Disorder

We now turn to the epidemiological and psychological evidence regarding alcohol use patterns in SAD. At the population level, SAD appears to be highly associated with AUD (Buckner et al., 2012; Grant et al., 2004; Hasin et al., 2007; Kessler et al., 1997; Schneier et al., 2010). As outlined above, to the extent that socially anxious individuals ingest anxiolytics in order to artificially improve affective states, they should depend on these compounds insofar as they are able to attain the desired state.

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Alcohol use among individuals with SAD is not only reinforced by improved affective state, but also by the new behavioral possibilities it entails: in cases where excessive avoidance poses its own fitness threat (i.e., difficulty finding mates), the individual may now facilitate a shift toward social approach behavior. Indeed, there is support for the notion that socially anxious individuals use alcohol as a means of avoidance coping, and alcohol consumption is associated with positive expectancies for social facilitation irrespective of social anxiety levels (Gilles, Turk, & Fresco, 2006). Furthermore, in one study participants with SAD were found to speak for longer with an interaction partner after consuming alcohol, whilst the interaction partners also exhibited more positive social behaviours towards the socially anxious participants who had consumed alcohol relative to a control beverage (Battista, MacDonald, & Stewart, 2012). In this sense, alcohol use may play an important role in attempts to reduce fitness-detrimental behaviors (i.e., sustained avoidance of social contact, repeated submission), as well as attempts to reduce subjective distress. Indeed, recent research indicates that socially anxious individuals who are high in trait approach-motivation may be particularly prone to alcohol use. This subgroup of socially anxious individuals consists of those high in reward sensitivity, rash impulsiveness and risk taking, suggesting that these individuals may be particularly prone to comorbid alcohol use due to a stronger motivation to pursue reinforcement of all kinds (Nicholls et al., 2014). Despite their apprehension toward social situations, socially anxious individuals often greatly desire interactions and relationships with others (Stein & Stein, 2008). Therefore, socially anxious individuals who are high in approach motivation may be particularly reinforced in their use of alcohol, inasmuch as consumption alters behavioral propensities in a way that facilitates their pursuit of social rewards.

Critically, alcohol has been shown to reduce performance anxiety during public speaking in individuals with SAD, to reduce perceived animosity and rejection in neutral and

angry faces, and to reduce attentional bias toward angry faces (Abrams, Kushner, Medina, & Voight, 2001; Stevens, Gerlach, & Rist, 2008; Stevens, Rist, & Gerlach, 2009). Alcohol intoxication has also been shown to significantly reduce amygdala reactivity in response to social threat-related faces (Gilman, Ramchandani, Davis, Bjork, & Hommer, 2008; Sripada, Angstadt, McNamara, King, & Phan, 2011). Furthermore, in individuals with SAD, alcohol may attenuate an implicit memory bias for social threat-related content by disrupting the consolidation of memory for socially threatening stimuli (Gerlach, Schiller, Wild, & Rist, 2006). In essence, alcohol may effectively attenuate many of the cognitive and motivational aspects of the socially anxious state, which typically prepare the individual to face a potentially threatening social situation. In individuals with clinical levels of social anxiety, this attenuation may temporarily reduce anxiety to within functional levels, and may be used to this end.

Current implications of alcohol use amongst socially anxious individuals

At the population level, anxiety disorders fall on a spectrum, and clinical levels lie on one extreme of the distribution. Clinical manifestations of social anxiety are characterized by extreme sensitivity to and avoidance of social threats (Mathews et al., 1997). Due to hypersensitivity to threat, hyperarousal, high allostatic load, and persistent avoidance of social situations, these individuals may thus be prone towards seeking out more significant readjustment towards affective homeostasis through end-directed alcohol use aimed at artificially regulating emotional and affective states. In other words, individuals lying on the extreme end of the anxiety spectrum may be particularly susceptible to the artificial signals of threat-management afforded by the ingestion of alcohol, as alcohol use may reduce subjective malaise and facilitate social behaviors (Book & Randall, 2002; Cappell, 1975; Cooper et al., 1992; Khantzian, 1997; Kushner, Sher, & Beitman, 1990; Robinson & Bolton, 2013).

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In the case of individuals with SAD, whose evolved systems for the perception and management of social threats are very readily engaged (perhaps exacerbated by the scale of interpersonal interactions with unknown others brought about by modern sociality), this ‘bypassing’ may be welcome, and more readily sought-out. Indeed, the results of a longitudinal study suggest that most individuals with SAD had not recovered after 10 years of follow-up, and those with sub-threshold social anxiety symptoms also report experiencing increased distress and functional impairment relative to asymptomatic controls (Fehm, Beesdo, Jacobi, & Fiedler, 2008; Keller, 2005).

Previous studies have found a shared genetic factor underlying psychopathology (Pettersson, Larsson, & Lichtenstein, 2015; Pickens, Sviki, McGue, & LaBuda, 1995). Associations between familial factors (i.e., history of psychopathology) and alcoholism have also long been observed (Winokur, Reich, & Rimmer, 1970). These factors may account to some extent for the onset of both SAD and AUD. Nevertheless, the onset of SAD precedes that of AUD in most cases (Grant et al., 2005; Hofmann et al., 2014). For example, it has been estimated that 50% of cases of SAD occur before age 11, and approximately 80% occur before age 20 (Stein & Stein, 2008). This suggests that social anxiety may negatively impact wellbeing from a young age (Kashdan & Herbert, 2001), and alcohol use for coping reasons may be initiated as a consequence (Buckner et al., 2012; Buckner et al., 2008). Indeed, approximately 80% of all AUD cases occur prior to 40 years of age (Eaton et al., 2012). Alcohol use among socially anxious individuals is usually much harder to control than in individuals reporting fewer social anxiety symptoms, and early drinking in order to cope is known to predict later alcohol *misuse* in adulthood (Blumenthal, Leen-Feldner, Frala, Badour, & Ham, 2010; Patrick, Schulenberg, O'Malley, Johnston, & Bachman, 2011; Villarosa, Madson, Zeigler-Hill, Noble, & Mohn, 2014).

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Expectancies that a drug will assist with coping may form before first use of that drug via familial or cultural learning, and are associated with an earlier onset of problem drinking (Jester et al., 2015). Indeed, positive expectancies that alcohol will aid in the management of anxious affect or in the facilitation of social behaviors may be an important moderating factor in the relationship between social anxiety and alcohol use (Burke & Stephens, 1999). After learning about the effects of a drug, many of the cognitive and motivational aspects associated with anxiety state may come to be attenuated merely by the belief that one has consumed alcohol, such as a reduction in subjective, physiological and behavioral anxiety in response to a social stressor after consuming placebo beverages (Himle et al., 1999; Wilson & Abrams, 1977; Wilson, Abrams, & Lipscomb, 1980). The effect of this expectancy-mediated placebo effect and of actually consuming alcohol may be additive, such that actual alcohol consumption more potently reduces social anxiety than the placebo alone (Abrams et al., 2001).

With continued use, psychoactive substances may come to bypass the functional affective mechanisms that have been shaped by natural selection for the regulation of social behavior. It is well established that continued drug taking can negatively affect declarative memory, emotion, motivation, learning and other cognitive processes, through changes to brain structure and function (Gould, 2010). In the context of moderate or normal-range social anxiety, substance use may preclude adaptive anxiety-related processes to take root, potentially barring effective management of manifest and potential social threats. Thus, in the case of individuals within the normal healthy range of social anxiety, alcohol use may result in riskier social behavior, including conflicts with other people: indeed, alcohol use is commonly involved in problem social behaviors (Collins & Messerschmidt, 1993).

Despite perceived facilitation of subjectively more pleasant states in the short-run, excessive drug use can exacerbate physical and mental health in the longer-term in some

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cases. As the effects of a substance wear off, the short-term positive fitness signal wanes, sometimes resulting in withdrawal symptoms after chronic use that can mimic pre-drug levels of anxiety or worse (Kushner, Abrams, & Borchardt, 2000). Disruption of neurobiological circuits underlying emotion regulation may be an important factor in this deterioration. Considering that drugs may be taken to regulate emotional state, the fact that continued use may exacerbate dysregulation of emotion is of particular note. Indeed, difficulties in emotion regulation are associated with increased drug use (Berking et al., 2011; Fucito, Juliano, & Toll, 2010), and individuals with substance use disorders often exhibit impaired negative emotion regulation capacities, with structural and functional changes to the prefrontal cortex proposed to underlie such difficulties (Kober, 2013).

The ability to regulate one's negative emotions in times of stress or anxiety is critical for effective daily functioning. That alcohol, despite its initial subjective benefits, may in some cases come to impair this ability through neurobiological changes to the PFC leads to a possible feedback loop in which a decreased capacity for emotion regulation results in increased drug use (to fulfill this role), with this drug use causing further difficulties in regulating emotions through damage to the PFC (Bechara et al., 2001). In turn, the individual may become ever more reliant on drugs to artificially simulate states associated with the absence or management of social threats, thereby contributing to a pattern of addiction with limited or transient improvement to overall anxiety symptomology. An interaction between short-term anxiety *reduction* during alcohol use with long-term anxiety *escalation* from chronic use and withdrawals may thereby result in a feed-forward cycle wherein anxiety symptoms and alcohol use exacerbate one another (for review see Kushner et al., 2000). Importantly, while the preceding discussion has focused on alcohol, the same temporal sequence may occur with other substances that have anxiolytic properties (Buckner, Bonn-Miller, Zvolensky, & Schmidt, 2007; Buckner, Schmidt, Bobadilla, & Taylor, 2006).

More broadly, the perspectives outlined in this paper have some relevant clinical implications. To recapitulate, humans have evolved to strive towards the minimization of potential harm from perceived social threats. The advent of readily accessible alcoholic beverages - particularly in social settings (Beck & Treiman, 1996; Single & Wortley, 1993) - provides a mechanism for reducing perceived threat via the consumption of alcohol. This suggests that the underlying drives associated with minimizing perceived social threats (which have evolved over millennia) are presently co-opted by a relatively novel feature of the environment. Understanding this, clinicians and practitioners could conceptualize strategies that enable patients not solely to cope with their social anxiety, but also to reduce the degree to which they perceive and attend to social threat, and in turn reduce the alcohol use that it may have engendered (for example see Koster, Fox, & MacLeod, 2009).

Phylogenetic and Comparative Considerations

Animal models of the relationship between social status and alcohol consumption have revealed a number of interesting parallels to alcohol use patterns observed in humans. For instance, socially dominant laboratory rats tend to drink less than subordinate conspecifics (R. J. Blanchard, Hori, Tom, & Blanchard, 1987; Wolffgramm & Heyne, 1991), and fight stress increases the consumption of alcohol more so in submissive than in alpha mice (Hilakivi-Clarke & Lister, 1992). Furthermore, low dominance standing predicts the development of problematic alcohol consumption in colony-housed rats (Ellison & Potthoff, 1984). Thus, it seems likely that the basic cognitive and motivational systems underlying susceptibility to alcohol use among socially anxious individuals as outlined above may not be unique to humans. Further research with non-human great apes could prove particularly informative given recent evidence suggesting that in at least one wild chimpanzee group alcohol consumption is occasionally but widely practiced (Hockings et al., 2015). Observational field

studies might seek to explore how this usage varies between individuals as a function of social status in such alcohol-using groups.

Some authors have argued that humans have evolved cognitive specializations for the use of psychoactive substances to aid in functional behaviours (e.g. Müller & Schumann, 2011). However, whether or not humans have evolved mechanisms leading them to use alcohol in ‘strategic’ ways is auxiliary to our argument, as our argument here does not address a potential *adaptationist* account of the interaction between SAD and AUD. Specifically, we are *not* postulating that humans have, on account of many generations of interaction with alcohol, evolved domain-specific cognitive and motivational systems to mediate its use for reasons relating to reproductive success; nor are we suggesting that socially anxious people have evolved specific regulatory mechanisms leading them to seek out alcohol in order to facilitate their reproductive success. Instead, we have drawn upon separate evolutionary accounts of social anxiety, and of substance use, in order to shed light on which cognitive and motivational aspects of social anxiety leave individuals with SAD in particular at higher risk of developing AUD. Namely, we have suggested that *because* social anxiety has evolved over successive generations as a tool for the effective perception and regulation of social threats in social groups, and *because* alcohol can affect and attenuate the systems involved in this social threat-perception, it is not surprising that high levels of social anxiety (i.e. SAD) are associated with risky alcohol use (i.e. AUD) in contemporary environments as an artificial means to down-regulate threat perception and facilitate social interactivity.

Future directions

More work is needed to understand how alcohol use and social anxiety co-occur across the lifespan, and the underlying mechanisms that lead to increased alcohol use among socially anxious individuals. The following research questions may lead to an improved understanding

of the frequent co-occurrence of SAD and the use of psychoactive substances including alcohol:

1. SAD is a chronic condition, with symptoms persisting to some degree for many years (Wittchen & Fehm, 2003). Many cases of SAD are estimated to occur during adolescence, and most cases are estimated to have first onset within the first 20 years of life (Kessler et al., 2005; M. B. Stein & Stein, 2008). The peak incidence of substance use disorders in the general population also appears to hover around 20 years of age, with about half of all incident cases occurring by that point (Eaton et al., 2012; Kessler et al., 2005). What is the longitudinal relationship between social anxiety and substance abuse beginning in adolescence and early adulthood, when the emergence of these disorders appears to be most common? As young adults enter new situations marked by social change and uncertainty, in which evolved mechanisms for detecting and managing social threats to fitness become heavily engaged (Boyer & Bergstrom, 2011), are those who experience higher levels of social anxiety thereby more likely to turn to alcohol, or more likely to develop problematic usage patterns? For example, clinical SAD represents a risk factor for AUD (Buckner et al. 2008; Schenier et al. 2010), but so too does sub-clinical social anxiety (Crum & Pratt, 2014). Adolescents and young adults with high levels of social anxiety may be particularly prone to alcohol use on account of the availability and high prevalence of this behavior in social contexts and settings in Western countries (Beck & Treiman, 1996; Single & Wortley, 1993), with implications for finding a partner. More research is needed to understand the temporal relationship between SAD and the onset of AUD.
2. Higher degrees of social anxiety are associated with fixation to external cues of social threat, submissive behaviors (which entail avoidance of threat cues), and heightened inward attention to threat (Buckner, Maner, et al., 2010; Mansell & Clark, 1999; Mogg

et al., 2004; Schulze et al., 2013). Alcohol has been found to reduce social anxiety and attentional bias to angry faces (Abrams et al., 2001; Stevens et al., 2008; Stevens et al., 2009). However, it remains to be seen whether reduced anxiety and attentional bias toward threatening faces is also associated with reduced inward attention (i.e. during self reflection) toward threat among socially anxious individuals under the influence of alcohol. Heightened inward attention toward threatening situations in one's imagination of future scenarios may represent a significant source of distress, but may also have adaptive benefits because it precipitates the avoidance of future threats (Miloyan, Bulley & Suddendorf, 2015). Alcohol might change behavioural propensities by altering expectations for the likelihood of social threat in such anticipated future scenarios (Miloyan, Pachana & Suddendorf, 2014).

3. Laboratory rats and mice tend to self-administer anxiolytic drugs when in anxiety-like states (Manzo, Donaire, Sabariego, Papini, & Torres, 2014; Olsson & Sherwin, 2006), and there are established models of social stress and social anxiety in rats such as the 'resident intruder paradigm' (Vidal, Buwalda, & Koolhaas, 2011). More research is needed to determine how social anxiety in various animals affects a propensity to self-administer alcohol (or analogues). In rats there is evidence that alcohol has an anxiolytic effect in situations of anticipated social stress (Tornatzky & Miczek, 1995). Further cross-species comparative research may therefore provide a more detailed understanding of the evolutionary history of mechanisms underlying susceptibility to alcohol (or anxiolytic drug) use in socially anxious humans. For instance, does the dominance hierarchy structure of a given species affect the tendency of individuals to self-administer alcohol in response to social stressors, contingent on social rank?
4. What are the current social consequences of alcohol use among socially anxious individuals? Do socially anxious individuals who use alcohol and other anxiolytics

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report having larger social networks than those who do not use substances, because these substances facilitate social approach behavior? Likewise, are socially anxious individuals who use certain anxiolytic drugs and alcohol more likely to be married or have children (reproductive success) than those who do not use such substances?

Conclusion

Despite its costs to wellbeing at high levels, some degree of social anxiety is frequently adaptive; it promotes the detection and management of socially mediated threats to fitness. However, extreme manifestations of social anxiety appear to confer little benefit to the individual in the present environment, due to chronic and aversive subjective states, pronounced avoidance of social encounters, and the frequent engagement of inappropriate social behaviors. Due to the continuous deployment of metabolic resources aimed at monitoring and managing social situations, and the sometimes persistent avoidance of certain social contexts, socially anxious individuals may be particularly prone to artificially alter their sustained negative emotional states with alcohol that signals the management or absence of social threats. Furthermore, although end-directed alcohol use may also capacitate the individual to approach and more effectively cope with previously avoided situations, prolonged (mis)use may eventually result in negative long-term outcomes (Kushner et al., 2000). In this way, some cases of SAD may come to be expressed in tandem with an AUD, and these disorders may come to compound upon one another (Schneier et al., 2010). On the other hand, the early detection of SAD may result in effective prevention of additional morbidity and the emergence of an AUD. Regardless, individuals with high social anxiety and heightened approach motivation may be strongly driven to obtain social rewards despite the anxiety this entails, and may therefore seek out a “quick” way of facilitating this through artificial means (e.g. alcohol use). Interventions that do not effectively address such social and

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biological drives may be less effective. On the bright side, clinicians may be able to harness these drives in order to sustain compliance and more effectively deliver psychosocial treatments.

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