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Review article

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.

Contents

1. Introduction

Social Anxiety Disorder (SAD) and substance use disorders commonly co-occur in national samples (Buckner et al., 2012,

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2008; Grant et al., 2004; Kessler et al., 1997; Smith and Book, 2008; Stein and 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 use disorder diagnosis in the past year have twice the odds of also having a past year SAD diagnosis relative to those without a substance use 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 et al., 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 et al. (2013) and Morris et al. (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 et al., 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 et al., 2007; Martins et al., 2012a; 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; Stein, 2006; Tinbergen, 1963) (Fig. 1). 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.

2. 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 systems evolved to deal with fitness-related threats (Marks and 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 et al., 2011; Mathews et al., 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 and 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 and Dunbar, 1997; Byrne and Whiten, 1989; Dunbar, 2002; though see Holekamp (2007) and Reader et al. (2011)), and it is therefore plausible that social group complexity has increased during recent hominin evolution (Shultz et al., 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 and Lienard, 2006; Damasio, 2010; Lang et al., 2000; Marks and Nesse, 1994; Miloyan et al., 2015; Woody and 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 et al., 2010b; Mansell and Clark, 1999; Mogg et al., 2004; Schulze et al., 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-

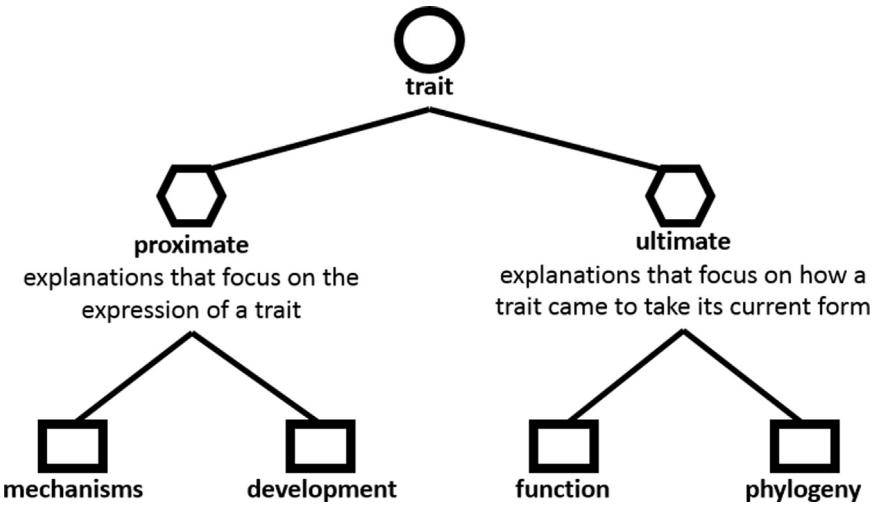

Fig. 1. A comprehensive account of a trait (circle) requires complementary proximate and ultimate explanations (hexagons) that can be further specified based on Tinbergen's (1963) four questions (squares). Here, we seek to explain the lifetime comorbidity between SAD and AUD by addressing proximate and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