



Review

Sensitivity shift theory: A developmental model of positive affect and motivational deficits in social anxiety disorder



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HIGHLIGHTS

- Social anxiety is related to deterioration of positive emotions over time.
- We propose a developmental theory of social-motivational deficits in social anxiety disorder.
- Sensitivity shift theory predicts a developmental transition from social sensitivity to social anhedonia.
- Our theory links social anhedonia to reinforcement learning models.
- Interventions that target reward-based learning are likely to produce clinical benefit.

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ABSTRACT

Social anxiety disorder (SAD) is a common and impairing condition that emerges in early adolescence, confers significant interpersonal disability and often persists into adulthood. Prevailing interventions for socially anxious youth are largely based on cognitive-behavioral models originally developed in adult samples, but produce only modest rates of remission in adolescents. The purposes of this review are to examine plausible explanations for these modest rates of treatment response and to critically evaluate the relevance of developmental mechanisms related to reward circuitry function. In doing so, we propose Sensitivity Shift Theory (SST), an integrated theoretical model addressing the development of social anhedonia in a meaningful subset of adolescents and adults with SAD. The central prediction of SST involves a shift, or developmental transition from social sensitivity during the late childhood/early adolescent period into later-emerging social anhedonia that includes reductions in positive affect, infrequent social approach behaviors and social skills deficits. We further provide a complementary mechanistic account by which these newly identified processes may be addressed using available evidence-based treatments that influence positive affect, including mindfulness-based interventions (MBIs). Collectively, SST provides a mechanisms-focused framework for explaining relatively modest rates of response to current front-line treatments in socially anxious youth, as well as discrepant observations in SAD samples of both high- and low- levels of social motivation depending on developmental factors and learning history.

1. Introduction

Social anxiety disorder (SAD) is a common and debilitating psychiatric disorder predominantly characterized by persistent fear of one or more social or performance situations (American Psychiatric Association, 2013). In adult samples, SAD ranks among the most

prevalent of all psychiatric disorders (Kessler et al., 2005), but unlike many other anxiety disorders the average age of onset for SAD is early adolescence (Ollendick & Hirshfeld-Becker, 2002). Within this developmental period, SAD is the most common anxiety disorder with the exception of specific phobia (Merikangas et al., 2010), and in later years follows a chronic and generally unremitting course across the life span

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(Albano & Hayward, 2004). To the extent that prevention and intervention programs are likely to be maximally effective when delivered at or around the onset of the disorder (McGorry, Purcell, Goldstone, & Amminger, 2011), it therefore follows that interventions for SAD are likely to be most effective when delivered in early adolescence. To the contrary however, cognitive-behavioral therapy (CBT) has been shown to be effective for only 40–65% of youth with social anxiety (Ginsburg et al., 2011; Herbert et al., 2009; Spence, Donovan, & Brechman-Toussaint, 2000), thus ranking SAD in younger samples as one of the least responsive anxiety disorders in terms of current front-line treatments (Hudson et al., 2015). In light of generally modest rates of response and remission for SAD in the context of cognitive-behavioral treatments, alternative models of SAD genesis and treatment have emerged in recent years. These newer models are largely based on deficits in positive emotionality and reward-based learning that appear to be unique to SAD relative to other anxiety disorders. The purposes here are to propose and to critically evaluate a mechanisms-focused developmental theory that explains the emergence and consequences of social anhedonia in a meaningful subset of individuals with SAD. We further consider the context and relevance of predictions made by this model and the potential for identified mechanisms to inform intervention.

2. Phenomenology of SAD in adolescents

The prevalence of SAD in adolescents ranges between 10 and 15% in the United States (Heimberg, Stein, Hiripi, & Kessler, 2000; Merikangas et al., 2010) and Europe (Essau, Conrath, & Petermann, 1999). Age of onset data further suggest that approximately 75% of severe and persistent social anxiety symptoms onset during early adolescence, with a median age of onset of roughly 13 years (Gregory et al., 2007; Wittchen, Fuetsch, Sonntag, Muller, & Liebowitz, 1999). Phenomenologically, youth with SAD report specific fears related to negative scrutiny by others and concern that their behavior might be perceived as socially inept, leading to shame or embarrassment. Exposure to these and similar types of social or performance situations frequently provokes an immediate anxiety response that is subjectively recognized by the individual as excessive or unreasonable. Certain behavioral manifestations of SAD include gaze aversion, stooped shoulders, nail biting, and avoidance of feared situations (Ollendick, Benoit, & Grills-Taquechel, 2014). Adolescents with SAD also frequently report anxious thoughts concerning embarrassment and self-criticism that, when confronted by anxiety-provoking social situations, result in maladaptive thoughts similar to those seen in adults.

The onset and course of SAD has been linked to a variety of precipitating factors including genetic influences, temperament, attachment processes, emotion regulation difficulties, conditioning events, information processing biases, parental anxiety and parenting practices (Ollendick & Benoit, 2012; Ollendick & Hirshfeld-Becker, 2002). In recent years, several different CBT-based interventions have been developed to address many of these factors in the treatment of SAD and other anxiety disorders in youth. The majority of empirically supported interventions for adolescents with SAD are based on a series of complementary cognitive-behavioral models derived primarily from adults (Benjamin et al., 2011), featuring dysfunctional beliefs about social/evaluative situations at their core (Crozier & Alden, 2005).

The most effective forms of CBT for adolescents appear to fall in-line with theoretical accounts developed in adult samples that focus on aversive arousal, including exposure therapy, either with or without cognitive intervention (Rodebaugh, Holaway, & Heimberg, 2004). For example, the most widely disseminated treatment protocol “Coping Cat” (Kendall & Hedtke, 2006) is a manualized CBT program designed to treat a range of anxiety disorders in children including specific phobias, separation anxiety, generalized anxiety disorder as well as SAD. Disorder-specific interventions for SAD include behaviorally focused interventions (Beidel, Turner, & Morris, 2000) as well as

community-based programs featuring CBT (Baer & Garland, 2005) along with social skills training (Fisher, Masia-Warner, & Klein, 2004). Mechanistically, these interventions facilitate fear extinction and habituation processes through systematic and repeated exposure to aversive arousal, thus allowing the adolescent to re-engage in formerly feared situations. Although there is a broad range of specific techniques and exercises within the family of CBT-based interventions, these techniques are generally premised upon cognitive models that are most relevant to fear and negative affect. Empirical support for these intervention concepts has been consistent albeit modest, with response rates indicating that roughly 60% of treated youth are diagnosis free at post-treatment (Higa-McMillan, Francis, Rith-Najarian, & Chorpita, 2016; James, James, Cowdrey, Soler, & Choke, 2013). However, it has also been shown that about half of those who are diagnosis free following treatment are no longer diagnosis free at long-term follow up (Beesdo-Baum et al., 2012; Crawley, Beidas, Benjamin, Martin, & Kendall, 2008; Ginsburg et al., 2011).

Taken together, this pattern of results from CBT trials in youth and adolescents suggests that although current interventions hold promise, there is a significant gap in knowledge regarding maintaining mechanisms that are specific to youth with SAD. Moreover, attempts to explain the poor response to CBT among youth with SAD relative to those with other types of anxiety disorders have examined a host of potential variables including age-of-onset, uneven comorbidity rates (particularly depression; Hudson et al., 2015), treatment modality (group versus individual Ginsburg et al., 2011), and time since symptom emergence (Essex, Klein, Slattery, Goldsmith, & Kalin, 2010), and have systematically shown that these variables are not likely causal explanations for differential rates of treatment response. The persistence of social anxiety symptoms despite treatment cannot be explained by these factors, which suggests the existence of SAD-specific maintaining factors that remain unaddressed or are not sufficiently addressed by current interventions. One possible mechanism that may be unaddressed in CBT, but which has been receiving increased recognition in adult samples, is altered approach-related motivation and diminished positive emotion.

3. The landscape of positive affect deficits in youth and adults with SAD

Deficits in positive affect (PA) have emerged as a major factor in both the etiology and maintenance of SAD in adults (Alden, Taylor, Mellings, & Laposa, 2008; Weeks, Heimberg, Rodebaugh, Goldin, & Gross, 2012). PA broadly refers to feelings of enthusiasm, alertness, full concentration and pleasurable engagement, including emotions such as joy and curiosity (Watson, Clark, & Tellegen, 1988). Factor analytic studies have demonstrated that PA can be meaningfully represented as anticorrelated with, although not totally orthogonal to negative affect (NA; Crawford & Henry, 2004), which instead refers to a general dimension of subjective distress and displeasurable engagement including emotions such as anger, guilt sadness and fear (Watson & Clark, 1992). The behavioral readout of low PA can manifest in a wide variety of distinctive ways across diverse functional domains. For example, adults with SAD report that positive events are less likely to occur (Gilboa-Schechtman, Franklin, & Foa, 2000), indicate greater difficulty expressing positive emotions (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005), and demonstrate a reduced tendency to sustain or savor positive emotions once experienced (Eisner, Johnson, & Carver, 2009). They further report lower levels of curiosity, exploration and life satisfaction (Kashdan & Roberts, 2004), which impact day-to-day aspects of functioning. For example, state-like changes in positive subjective experiences are strongly related to the severity of daily self-reported social anxiety symptoms (Kashdan & Collins, 2010; Kashdan & Steger, 2006). Kashdan and Collins (2010) further reported data from ecological momentary assessment (EMA) methods, which revealed moment-to-moment differences in the natural lives of affected adults, who reported

less time feeling happy and relaxed, and more time feeling angry on any given day, as well as less intense positive emotions and more frequent anger episodes.

Relative to the other anxiety disorders, diminished PA appears to be unique to SAD (Brown, Chorpita, & Barlow, 1998), as it is not found in generalized anxiety disorder, separation anxiety disorder, specific phobia or panic disorder, nor can it be explained by comorbidity with depression (Kashdan, 2007). The earliest initial evidence for PA deficits in adults with social anxiety derived from a series of structural equation models (Brown et al., 1998), that distinguished SAD from other anxiety disorders on the basis of PA deficits, a finding which has been replicated in more recent years across a number of complementary quantitative models (Brown & Naragon-Gainey, 2013; Naragon-Gainey, Watson, & Markon, 2009; Rosellini & Brown, 2011). In contrast to the tripartite model of depression and anxiety (Clark & Watson, 1991), which specifically posits that anxiety disorders can be distinguished from depression on the basis of physiological hyperarousal but not diminished PA, relatively early findings from structural equation approaches consistently indicated SAD was the *only* anxiety disorder characterized by low PA. Kashdan (2007) conducted a meta-analysis of 19 studies (total $N = 2976$) evaluating the strength, consistency and construct specificity of the relationship between PA and social anxiety symptoms, and found that the constructs had a significant inverse relation ($r = -.36$; 95% CI = $-.31$ to $-.40$), an effect which remained after controlling for depressive symptoms and disorders.

Although evidence from adult samples has persuasively demonstrated that factors relating to low PA exert strong influence on the etiology and course of SAD, these mechanisms remain comparatively unexplored in youth samples. As one of the first systematic investigations of this relationship in youth, Chorpita, Plummer, and Moffitt (2000) replicated and extended the findings of Brown et al. (1998) of low PA in a sample of 100 youth diagnosed with mood and anxiety disorders (mean age 12.4 years). Unexpectedly, findings from structural models suggested that low PA was the *only* tripartite dimension significantly associated with social anxiety symptoms in youth, and in fact no relationship was observed between SAD and NA or physiological hyperarousal (PH), as was the case in adults. Although surprising, these findings were later supported by Kashdan's (2007) meta-analysis, which identified that mean effect sizes between PA and social anxiety symptoms varied significantly across sample type. The strongest relationship was observed in clinical samples ($r = -.41$), followed by community youth and college samples ($r = -.35$) and finally adult community or non-clinical samples ($r = -.27$), suggesting that age may play a more prominent role in the expression of PA deficits than previously thought. Further refining this association, Weisman, Rodebaugh, Brown and Mulligan (2015) directly tested the age moderation hypothesis in two large archival datasets of community participants. Results partially supported the moderating effect of age, indicating that a measure taking into account both valence and arousal did not show the expected age-driven effect, whereas a measure using only valence (not conflating arousal) supported the moderational hypothesis, revealing a stronger correlation between PA and social anxiety symptoms in youth but weaker relationship in adults. Interestingly, no moderational effect was observed between PA and symptoms of depression in youth, further underscoring the specificity of the influence of age on the expression of PA in social anxiety.

4. Sensitivity shift theory

We next present sensitivity shift theory (SST), a theoretical model that integrates recent findings in adolescent neurobiology and developmental psychopathology to mechanistically explain how neurobiological and environmental factors that peak in adolescence may potentiate the likelihood and impact of positive, approach related deficits in a meaningful subset of individuals with SAD. SST is comprised of two major components: a distinctive profile of risk variables and a pathway

primarily grounded in learning theory that, for vulnerable youth, predicts a transition from high social sensitivity into later social defeat and social-motivational deficits.

4.1. Triple vulnerability profile of risk

The basis for sensitivity shift theory originates from a series of hypotheses advanced by Schriber and Guyer (2016) who proposed that individual differences in adolescent susceptibility to social context may drive a variety of developmentally important processes such as risk-taking, prosocial versus antisocial behaviors and certain forms of psychopathology. Their model is rooted in the long-standing literature demonstrating that adolescents are not uniform in their susceptibility to influence by social context (Belsky, 1997; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Boyce & Ellis, 2005), but instead can be characterized on a continuum from strongly- to weakly-driven by contextual elements of the social environment such as peer influence and social evaluation (Guyer et al. 2012). Within this model, adolescents who are particularly sensitive to social influence are more likely to be favorably impacted by positive contexts (warm parenting, supportive peer feedback), but also more adversely impacted by negative social contexts (peer victimization, low parental warmth).

Building off this theoretical framework, we argue here that socially anxious adolescents may lie at extreme ends of these dimensions, insofar as evidence has separately illustrated both (1) high susceptibility to the influence of social context, as well as (2) disproportionate exposure to adverse environmental risk factors among adolescents with SAD (Harvey, Ehlers, & Clark, 2005; Taylor & Alden, 2005). In terms of neurobiological susceptibility, high levels of sensitivity to peer feedback is an individual difference variable in which adolescence itself is a risk factor (Guyer, McClure-Tone, Shiffrin, Pine, & Nelson, 2009; Sebastian, Viding, Williams, & Blakemore, 2010). For example, in typically developing nonanxious adolescents, fMRI studies have demonstrated that peer observation, social exclusion and merely experiencing social forms of emotion differentially modulate regions related to visceral interoception and emotional conflict monitoring including the insula and anterior cingulate, respectively (Burnett, Bird, Moll, Frith, & Blakemore, 2009; Burnett & Blakemore, 2009; Masten et al., 2009). The effects of social exclusion and social threat on these same brain regions appear to be particularly pronounced in both adolescent and adult social anxiety (Etkin & Wager, 2007; Heeren et al., 2017; Klumpp, Angstadt, & Phan, 2012), lending support to the idea that social anxiety may comprise extreme variation along a neurobiologically-based dimension of susceptibility to social context. However social anxiety symptoms are unlikely to derive from neurobiological risk alone, but also interact with environmental risk factors that similarly peak during adolescence (c.f. Paus, Keshavan, & Giedd, 2008 for comment). In support of this claim, adverse environmental conditions such as social exclusion/victimization, harsh parenting styles, parental stress and financial strain are strongly correlated with, and in some cases predictive of social anxiety symptoms in youth (Erath, Flanagan, & Bierman, 2007; Essex et al., 2010; Siegel, La Greca, & Harrison, 2009). Thus, our theoretical model posits that the period of early- to mid-adolescence may be a particularly critical time for the development of social anxiety symptoms in youth due to the intersection of risk factors that normatively and simultaneously peak during the adolescent period, as illustrated in Fig. 1.

We further contend that the joint linkage between neurobiological susceptibility to social context and environmental risk factors exists against a background of temperamental characteristics that emerge early in development and represent a unique vulnerability factor in the developmental trajectory toward social anxiety symptoms. Perhaps the most intensively studied among these is behavioral inhibition, which is defined as a relatively stable individual difference variable describing the tendency to exhibit heightened vigilance, negative affect and reactivity to novelty (Kagan, Reznick, Snidman, Gibbons, & Johnson,

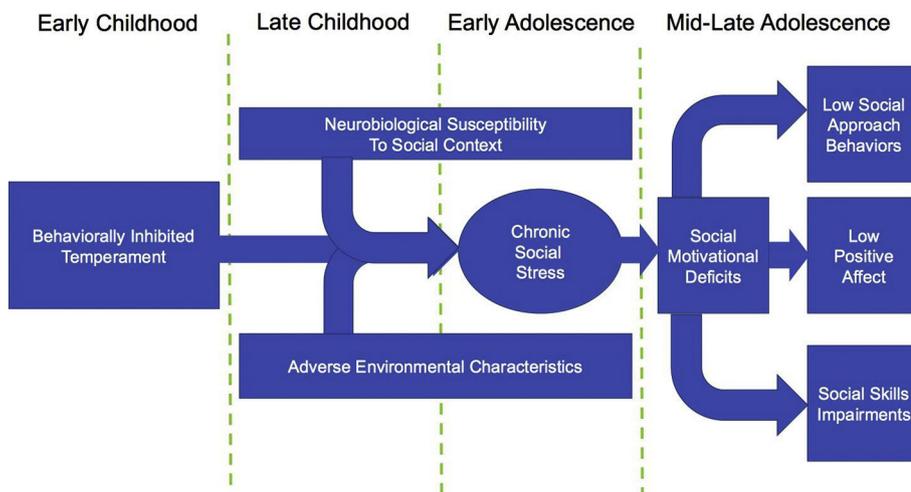


Fig. 1. Integrated theoretical model illustrating the convergence of temperamental, neurobiological and environmental vulnerability factors to produce chronic social stress during the early- to mid-adolescent period. We propose that chronic social stress eventually culminates in neurobiologically-based social motivational deficits which over time become sculpted into specific behavioral, emotional and social sequelae such as low social approach tendencies, low positive affect and social skills impairment, respectively.

1988; Ollendick et al., 2014). Behavioral inhibition is thought to emerge during the second year of life, at about the time that a child transitions from the normative period of stranger anxiety into a period of independent behavioral action to cope with threat. High levels of behavioral inhibition manifest as extreme shyness and emotional reactivity, and are strongly predictive of social anxiety symptoms in adolescence and adulthood (Schwartz, Snidman, & Kagan, 1999). Moreover, meta-analytic results have further suggested that up to 50% of behaviorally inhibited children go on to develop clinical levels of social anxiety (Clauss & Blackford, 2012), representing a sevenfold increase in risk as compared to non-inhibited children. Although early behavioral inhibition may be the single best predictor of later social anxiety symptoms, roughly half of behaviorally inhibited children do not go on to develop clinically significant levels of anxiety, which therefore leaves a significant proportion of variance yet to be explained. We assert that in combination with early temperamental characteristics, the later-emerging factors of neurobiological susceptibility to social context and repeated exposure to aversive social outcomes may explain how an early-emerging risk factor becomes sculpted into a form of social anxiety that emerges comparatively later in life. Below, we outline supportive evidence for our principal argument that the triple vulnerabilities of (1) high behavioral inhibition, (2) neurobiological susceptibility to social environment, and (3) adverse life events outline a mechanistic developmental pathway toward a form of anxiety that is uniquely social in nature, and produces long-lasting aversion to social contact and concomitant alterations in motivation-relevant neurocircuitry.

This argument originates from a growing foundation of evidence that behavioral inhibition itself represents a distinctive risk factor for altered reward-driven approach behaviors (Helfinstein, Fox, & Pine, 2012). Whereas a majority of early investigations of behavioral inhibition focused on avoidance behaviors and the functional neurobiology of fear-related learning (Fox, Henderson, Marshall, Nichols, & Ghera, 2005), more recent work has demonstrated that behaviorally inhibited children demonstrate specific and robust alterations in the dopamine (DA)-driven neurobiology of reward. The mesolimbic DA pathway or reward circuit is involved in the identification of emotionally salient stimuli in the environment (Phillips, Drevets, Rauch, & Lane, 2003), learning about outcomes related to those stimuli (Montague, Dayan, & Sejnowski, 1996) and selection of appropriate approach or avoidance behaviors (Bromberg-Martin, Matsumoto, & Hikosaka, 2010). In probing the relationship between behavioral inhibition and neural responses to monetary incentives, Guyer et al. (2006) collected fMRI data from adolescents (ages 10–15 years) during a performance based paradigm (the monetary incentive delay [MID] task; Knutson, Fong, Adams, Varner, & Hommer, 2001) in which

participants either attain a reward or avoid a loss depending on the speed of a button press. Results indicated that adolescents who had been previously characterized as behaviorally inhibited at 3 months of age demonstrated increased striatal activity as compared to non-inhibited adolescents during anticipation of both monetary gain and loss. Results further suggested that regions of interest in the striatum also tracked the magnitude of gains and losses, with greater activation corresponding to larger incentives. Striatal structures such as the nucleus accumbens (NAc), putamen and caudate are known to have roles in facilitating responses to salient incentives (Zink, Pagnoni, Martin-Skurski, Chappelow, & Berns, 2004), and the finding of magnitude- but not valence-specific enhancement of striatal responses in inhibited more so than noninhibited adolescents is suggestive of increased sensitivity to both rewards and loss. Using an experimental paradigm that systematically varied the probability of reward and requirement of action to produce desired outcomes, Bar-Haim et al. (2009) found similar effects of enhanced striatal activation during incentive anticipation in a slightly older cohort of adolescents (ages 14–18 years) who had also been characterized as behaviorally inhibited at three time points from infancy (4 months) up to 4 years of age. Results from fMRI indicated enhanced striatal responses were observed only within an experimental condition in which participants believed that the choice of an action determined the outcome and not for example when stimuli were systematically rewarded or when a simple motor response was required. The observation of agency-dependent striatal activation in behaviorally inhibited adolescents provided an important theoretical advancement by illustrating that striatal sensitivity to reward was revealed only under the belief (whether true or not) that the choice of an action determined a given outcome.

Extending these findings into clinically diagnosed adolescents, Guyer et al. (2012) again used the MID task to test the hypothesis that incentive magnitude would modulate striatal responses in a sample of adolescents diagnosed with SAD as compared to a matched sample of adolescents with generalized anxiety disorder (GAD) and a control group matched on age, sex, IQ, and pubertal status. Confirming earlier work focused on behavioral inhibition, results of this study indicated that adolescents with a current diagnosis of SAD also demonstrated enhancement in the caudate and putamen according to incentive magnitude but not valence. As no such effects were observed in youth with GAD or matched controls, this further underscores the specificity of reward-related dysfunction in socially anxious youth relative to other forms of anxiety. The consistent finding of enhanced striatal responses to both negatively and positively incentivized non-social stimuli in adolescence may reveal theoretically important clues about the effects of context and the means by which aberrant reward responses become socially-specific as development progresses.

4.2. Mechanisms of transition from social sensitivity into social anhedonia

Next, we review work supporting a mechanistic pathway originating in this vulnerability profile and progressing into chronic social stress and eventually reward-circuitry deficits in affected adults. Although a precise and overarching definition of chronic stress has been elusive, we define this as a condition of unremitting environmental demands that exceed the adaptive capacity of the organism, resulting in nonspecific psychological and biological consequences (cf. Cohen, Janicki-Deverts, & Miller, 2007 for comment). A central premise of sensitivity shift theory is that over time, the convergence of these neurobiological and environmental risk factors will eventually produce a narrowly-defined form of chronic stress that disproportionately impacts the social domain. This is based on the proposition that for behaviorally inhibited children entering into the adolescent period, their generally enhanced sensitivity to both negative and positively valenced events would be expected to eventually coincide with normatively increasing sensitivity to social context. In fact, behavioral inhibition may itself be a risk factor for increased environmental sensitivity (Pluess, 2015), reinforcing the idea that genetic and temperamental factors interactively relate to social susceptibility in adolescents by enhancing environmental sensitivity to normally motivating cues such as peer feedback (Guyer et al., 2014). This proposition has been directly supported by findings that reveal enhanced function in key structures in adolescent midbrain DA circuitry during social evaluation and loss as well as social exclusion (Casey, Jones, & Hare, 2008). Thus, behaviorally inhibited children transitioning into the adolescent period appear to be more profoundly impacted by motivationally salient outcomes that are *social in nature*.

In considering the pathway from social susceptibility into social stress, Schriber and Guyer (2016) propose a learning-based mechanism of canalization toward social stress for vulnerable adolescents. Based on the work of Pluess (2015), as well as prior work illustrating sensitivity to incentive-predicting cues in adolescents with a history of behavioral inhibition (Henderson, Pine, & Fox, 2015), they suggest that this trait enhances detection, processing and responding to environmental cues that predict the most motivationally relevant outcomes. Caouette and Guyer (2014) further propose that behaviorally inhibited adolescents demonstrate heightened vigilance specifically toward the consequences of action-contingent performance, thus accelerating the learning produced by both aversive and appetitive outcomes. Although on the one hand, this enables rapid adaptation to transient environmental conditions, on the other hand conditions of chronic social adversity can entrench long-term consequences. Specifically, we add the proposition here that repeated encounters with peers or caregivers that are disproportionately characterized by unfavorable outcomes would be expected to modify the associative strength between social cues in the environment and their outcomes in a predictable direction. In particular, seminal learning theories suggest that distress-producing cues increase in their salience due to repeated cue-outcome pairing and elicit a conditioned avoidance response due to their learned predictive value (Rescorla & Wagner, 1972). Related theories propose that the impact of aversive outcomes or losses are weighted more heavily than gains, wins or otherwise favorable outcomes (Kahneman & Tversky, 1979), further predisposing sensitive individuals to disproportionate impact of aversive consequences.

In the context of this learning framework, we argue that future expectancies about social outcomes are dramatically modified by heightened sensitivity toward environmentally salient social cues. The logic behind this proposition is as follows. As early as Pavlov (1927), initially neutral stimuli (S) have been shown to eventually acquire affective properties after repeatedly preceding some biologically significant event. The response-independent learning described by Pavlov combines well with instrumental learning theories which specify that a cue-driven response (R) and unpleasant outcome (O) disincentivizes future responses by transitioning such cues from emotionally-neutral to distress-producing (Jean-Richard-Dit-Bressel, Killcross, & McNally,

2018). For individuals with a dispositional tendency to prioritize certain stimuli for subsequent associative learning, as well as a tendency to experience aversive outcomes based on instrumental responses to those cues it therefore follows that this combination of factors would culminate in at least the following two outcomes. First, emotionally neutral (albeit environmentally salient) stimuli will eventually acquire aversive motivational qualities. Second, aversive outcomes will eventually produce behavioral avoidance, or a suppressive effect on the behaviors that caused them. This associative learning model directly connects to certain principles within the broader SAD phenotype, to the extent that this form of fear is cue-driven by social context, and prioritizes distress-producing cues for behavioral control through preemptive avoidance. Although it is presently unclear what nervous system mechanisms specifically mediate this relationship, the pathway outlined above requires repeated associative events, and thus requires adverse developmental context, consistent with prior theoretical accounts and behavioral data suggesting markedly increased frequency of such events in socially anxious youth.

A major prediction specified by sensitivity shift theory is that over time, prolonged social stress will produce deficits in social motivation by affecting key brain structures related to the translation of motivation into action. Support for the chronic social-stress → social anhedonia connection comes from multiple levels of analysis including both pre-clinical animal models (Berton et al., 2006; Golden, Covington, Berton, & Russo, 2011), as well as extensive human data indicating that repeated experiences of social aggression leads to social defeat stress and lasting changes in neural systems that profoundly alter motivation for social interaction (Huhman, 2006). Although the functional neurobiology of motivationally-relevant DAergic circuitry is complex and does not operate in isolation (cf. Haber & Knutson, 2010), social stress selectively alters this circuit in rodents by remodeling DA-mediated signaling in the ventral striatum or nucleus accumbens (NAc), a key structure involved in linking motivation to action control (Harsay et al., 2011). In turn, this produces a characteristic behavioral syndrome that includes hedonic changes, weight loss, circadian abnormalities and social withdrawal (Krishnan et al., 2007; Savignac et al., 2011). These alterations are normalized by chronic administration of antidepressant medication in rodent models (Cao et al., 2010), which has also been the case in human trials examining pharmacology for SAD as well (Blanco et al., 2003). Experimental evidence from rodent models suggests that chronic stress produces both anhedonia and motivational deficits but only when stressors are inescapable or uncontrollable (Mangiavacchi et al., 2001), similar in principle to the long-standing literature on learned-helplessness (Abramson, Seligman, & Teasdale, 1978; Maier & Watkins, 2005).

Animal models further confirm that vulnerability versus resistance to social defeat lies within remarkably specific neurobiological variation in key structures of the mesolimbic DA circuit (Berton et al., 2006; Razzoli, Andreoli, Michielin, Quarta, & Sokal, 2011). Krishnan et al. (2007) provided evidence that phenotypic variability in social defeat for genetically similar (inbred) mice depends on levels of brain-derived neurotrophic factor (BDNF) in the NAc. BDNF promotes growth and differentiation of new synaptic connections as well as neuronal plasticity and remodeling (Binder & Scharfman, 2004). In the case of vulnerable mice, Krishnan et al. (2007) reported that significantly increased BDNF concentrations in the NAc were further associated with long-lasting upregulation of firing in ventral tegmental area (VTA) DAergic neurons that terminate in the NAc, which could be reversed by genetic knockdown of the BDNF gene in the VTA but not NAc. This suggests that the susceptibility to social defeat is mediated by genetic dysregulation of events within the VTA that enhance neuroplasticity in the NAc after social stress. Interestingly, human post-mortem brain tissue from depressed patients was also examined by Krishnan et al. (2007), revealing a remarkably similar effect. Although anhedonia measures were not reported, a 40% increase in BDNF protein was observed in the NAc of depressed patients as compared to controls, an

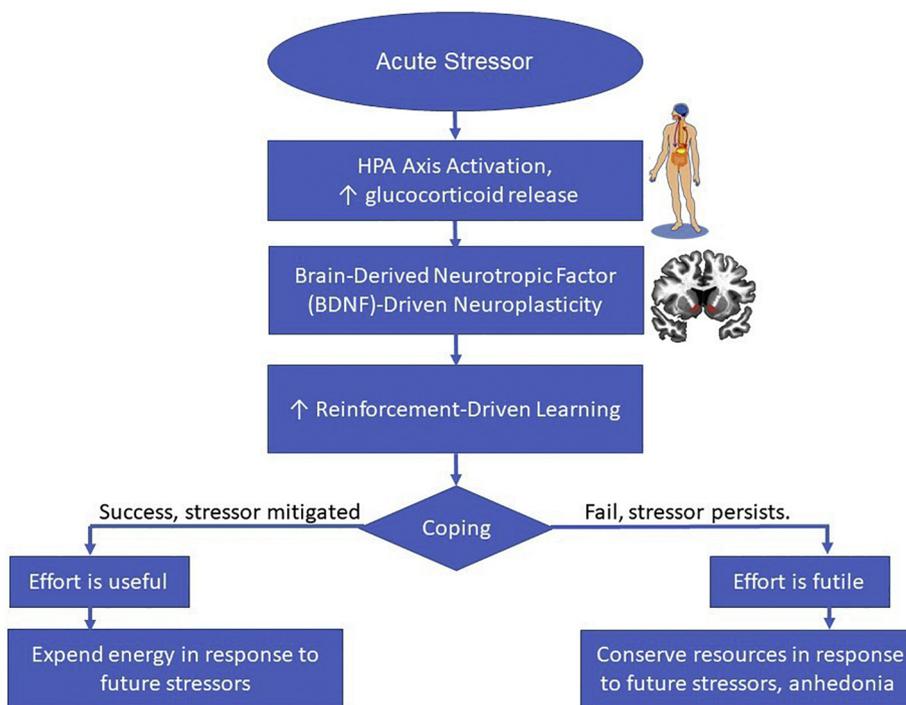


Fig. 2. Proposed biological and behavioral mechanisms involved in the transition from social stress to social anhedonia. Transient stressors enhance learning by promoting BDNF-driven neuroplasticity in the ventral striatum, amplifying the impact of coping outcomes. Behavioral responses to future stressors are likely to reflect learning history, such that prior success in coping leads to increases in future effort expenditure when facing acute stress. Conversely, persistent failure in limiting the impact of stressors on the organism leads to a decrease in active coping and conservation of resources as is seen in anhedonia.

effect that could not be explained by chronic antidepressant treatment.

Although the molecular mechanisms by which social stress selectively promotes neuroplasticity in the NAc is still a topic of active investigation, some prior work has focused on hormonal and neurotransmitter products emanating from the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis is activated by a wide variety of psychological stressors, and resultant increases in serum concentrations of glucocorticoids serve an immediate role in facilitating adaptive behavioral responses such as fight-flight-freeze (Porges, 2003). Numerous studies have demonstrated that stress via HPA function has variable consequences on BDNF levels, decreasing concentrations in certain areas of the brain including the hippocampus and prefrontal cortex (Duman & Monteggia, 2006) and upregulating it in striatum as noted above. Several studies have demonstrated HPA axis dysregulation in anhedonic depressed patients (Plotsky, Owens, & Nemeroff, 1998), and further that these patients benefit from pharmacological modification of glucocorticoid receptors (Wolkowitz et al., 1999). The precise signaling pathway mediating stress-induced neuroplastic changes remains unclear, however evidence suggests that the relationship between BDNF and neurotransmitter release may be linked through interactions between its receptor (tyrosine kinase receptor; TrkB) and the cortical glucocorticoid receptor (GR). Numakawa et al. (2009) illustrated in cultured cortical cells that chronic administration of a synthetic glucocorticoid causes GR downregulation and reduced BDNF-activated signaling, which can be recovered by GR overexpression, restoring BDNF-mediated neurotransmitter release. Interactions between glucocorticoids released by the HPA axis and these receptors appears to be critical in mediating the effect of stress specifically on neuroplasticity via BDNF. Thus, the stress-induced crosstalk between the HPA axis and the DA-centric reward system may in fact be mediated by more basic molecular mechanisms influencing neuroplasticity. Given the increasingly accepted role for the NAc as a final common pathway in translating expected value into action, upregulated sensitization to plasticity in this region, as suggested by increases in BDNF levels (which are in turn produced by chronic glucocorticoid release) would be expected to sensitize reinforcement-based learning about the expected gain or loss to be realized under conditions of stress.

Human neuroimaging work has corroborated the idea that chronic stress sensitizes responses to novel stressors by affecting dopaminergic

pathways in the brain. Pizzagalli and colleagues have shown across a number of studies that anhedonia in depressed patients is associated with altered responses to normally rewarding incentives. In a study using the MID task in a sample of unmedicated depressed adults and matched control subjects, Pizzagalli et al. (2009) demonstrated significantly weaker striatal responses to monetary gains in depressed patients, as well as a significant correlation between anhedonic symptoms and caudate volume. Similarly, Smoski et al. (2009) reported hyporesponsivity in striatal regions of depressed adults during reward selection anticipation and feedback during a monetized decision making task. Forbes et al. (2009) reported a similar general pattern in depressed adolescents, and further that the degree of hyporesponsivity during anticipation of monetary reward predicts real-world experiences of positive affect. Pizzagalli (2014) summarized this work by proposing that anhedonia is preceded by dysfunctional interactions between stress and reward systems. Recent work has elaborated on this seminal idea, specifically illustrating that striatal DA responses are critically dependent on the perceived controllability of stressors.

The ability of the organism to cope, or manage the demands of stress through behavioral control is critically important in coding the effect of stress on the DA system. Coping is a complex construct that can be broadly defined as a behavior or cognitive strategy that mediates the impact of stress on an organism (Folkman & Lazarus, 1988). As discussed, enhanced striatal DA release has been observed in response to stressors that are avoidable or controllable, whereas inhibition of DA release is associated with inescapable stressors (Cabib & Puglisi-Allegra, 2012). This supports the idea that neural responses to stressors are dependent on learning history. In particular, learning from the consequence of prior actions that either mitigate or prolong the impact of stress promotes adaptive functioning by replicating behaviors that promote survival and suppressing behaviors that produce no result. For example, DA has a role in conditioned avoidance and physical escape from foot shock in mice (Oleson, Gentry, Chioma, & Cheer, 2012), as well as suppression of effort when shock is inescapable (Anisman, Glazier, & Sklar, 1981). It has been suggested elsewhere that this is because if active coping is sustained in the face of unavoidable or uncontrollable stressors, exhaustion of energy resources will inevitably result, thus leaving the organism unable to exploit changes in the environment if they occur (Cabib & Puglisi-Allegra, 2012).

We propose that social anhedonia follows a similar model, as illustrated in Fig. 2. Repeated coping failures in the face of chronic uncontrollable social stress, combined with enhancements in neurobiologically-driven learning would be expected to reduce reward responsiveness due to the learned futility of coping efforts. The paradoxical finding of sensitization in DA-driven learning pathways in anhedonia ultimately leading to blunted hedonic behaviors can thus be understood in terms of reinforcement learning processes as they relate to successful versus unsuccessful coping with stress. For a sensitized learner, feedback from successful as well as failed coping attempts provides equally important information. Success in coping assigns increased utility to actions that mitigated acute stress, thus increasing the likelihood of repeating them again to manage future demands. On the other hand, consistent failed attempts to mitigate acute stress should reduce energy expenditure and increase conservation of resources because the organism has learned that actions stored within its existing repertoire are incapable of producing any useful result. In either case, by rapidly updating expectations about the likelihood of reducing aversive stimulation through behavioral action, the organism can sculpt a suite of behavioral responses that are stored and maintained within individual synapses, neurons and neuronal circuits to guide behavior through relatively automated motivational systems including affect.

This culminates in a final major prediction specified by SST: that the end stage of this social anhedonia phenotype is characterized by (1) reductions in social approach behavior, (2) low positive affect, and (3) impairments in social skills. These narrowly defined phenotypes are downstream traits each with their own unique connection to suppression of normally rewarded behavior, as delineated below.

1. Reductions in social approach behavior, for example, can be understood in simple behavioral terms as infrequent social interactions. We argue that such behaviors are not purely avoidant to the extent that they are not exclusively due to expectancies about aversive stimulation, but also because the costs conferred by unpleasant stimulation are not exceeded by the anticipated gain to be realized. This would shift the balance of decision-making in favor of behavioral avoidance not necessarily due to threat expectancies such as fear of negative evaluation, but because the low anticipated payoff combines to produce a net negative expected value. Thus, the decision not to engage in repeated social behavior is potentially explainable in terms of the relative imbalance between approach and avoidance tendencies.
2. Positive affect, as noted earlier is an approach-related motivational system characterized by pleasurable emotions. Approach-driven motivational experience steers behaviors toward primary rewards such as food, sex and drugs of addiction as well as formerly rewarded stimuli. For socially anxious individuals, diminished pleasure in the context of social interaction can be explained in terms of the mechanistic pathway outlined above, in which adversity, stress and learning about coping failures converge to knock down expectations of future reward. Our model specifically theorizes that a leaking effect of PA over time is the result of the decoupling between social interaction and the concomitant experience of pleasure. In other words, social anhedonia establishes a risky learning context as affected individuals learn to connect social interaction experience to its affective consequences. For affected individuals who experience a lack of pleasurable engagement in the midst of social interaction, it follows logically that the positive anticipatory quality of motivating affect embodied by PA would be adversely affected.
3. Social skills impairments are a widely studied phenotype within the broader social anxiety category, particularly in adolescents. We argue here that social skills impairment is a consequence of infrequent interaction opportunities during the developmental period in which these skills are typically forged. In turn, the reduced frequency of practice opportunities can be explained by low approach motivation to engage in social interaction. In light of the

significance and weight awarded to peer relationships during this developmental window, this may have secondary effects in which socially anxious adolescents are less motivated to engage in behaviors that are valued by peers, leading to erosion of social support networks. Consistent with this idea, adolescents with SAD report fewer friends (La Greca & Lopez, 1998), lower quality friendships (Festa & Ginsburg, 2011) and greater social skills difficulties (Greco & Morris, 2005). In addition, our allegation that social skills deficits mark a relatively later stage in the ontogeny of the disorder may relate to the clinical treatment literature, which has illustrated generally inconsistent rates of success when treating social skills impairments in socially anxious adults (Fedoroff & Taylor, 2001; Ponniah & Hollon, 2009). This may be due to the relatively late and distal position of this behavioral phenotype and its role as a pathognomonic feature rather than a direct causal attribute.

4.3. Summary of theoretical model

In sum, we propose that social susceptibility factors that naturally peak during adolescence are potentiated by the presence of behavioral inhibition, and predispose affected individuals to disproportionate influence by adverse social context such as unfavorable parent/caregiver/peer interactions. Although it is not clear yet whether adverse social conditions are caused by behavioral inhibition, this combination of factors in turn produces a narrowly defined form of chronic stress that is especially relevant to social contexts, and is similar in principle to the DSM-defined SAD diagnosis. Over time, repeated aversive social interactions produce a social defeat phenotype that is characterized by low social approach behaviors, deficits in positive affect and ultimately social skills impairments due to less frequent social interaction and practice opportunities during the developmental period in which such skills are typically established. The developmental shift from social sensitivity in childhood and early adolescence toward social defeat in mid-late adolescence and adulthood may explain paradoxical findings in neuroimaging work that has separately illustrated both an early increase, and as well as a later decrease in striatal responses to normally rewarding incentives in socially anxious youth and adults respectively (Guyer, Choate, Detloff, et al., 2012; Richey et al., 2014; Richey et al., 2017). Whereas children and youth may display enhanced processing biases toward motivationally relevant cues in the environment, the influence of social stress may be observable in these same brain regions after prolonged unsuccessful coping efforts, eventually producing a social defeat syndrome marked by motivational deficits. Plus, the developmental timing of these events may also explain the unique age-of-onset pattern of SAD, which is characterized by a steep increase in prevalence beginning at about age 10 (Beesdo et al., 2007), 50% of new cases diagnosed by age of 13 and 90% of cases emerging by age 23 (Beesdo, Pine, Lieb, & Wittchen, 2010; Stein, 2006). Whereas, if temperamental characteristics alone were causally sufficient for the development for social anxiety symptoms, then one would predict a relatively flat distribution of new cases after the early childhood period.

Major predictions specified by this theory include:

1. A developmental shift in striatal responses from early sensitivity during the late childhood through early adolescent period toward later insensitivity during the mid- to late-adolescence and early adulthood periods, coinciding with the transition from social sensitivity to social defeat.
2. Developmental narrowing of context away from domain-general motivational deficits in late childhood/ early adolescence, toward social specific deficiencies in detecting and learning from positive social gestures or behaviors.
3. The progression from social susceptibility to chronic social stress is based upon associative learning mechanisms that edit future expectations about the likely outcomes of actions.
4. Social skill impairment will correlate with the degree of

motivational deficits, as well as the age at which the transition from social sensitivity to social anhedonia occurs.

5. Insensitivity to social reward and concomitant deficits in positive affect mark the end-stage of a social defeat syndrome produced by prolonged social stress.

5. Transdiagnostic and nomological network considerations

The principal aim of this review and theoretical account has been to contextualize social anhedonia within social anxiety symptoms and SAD. However, this is not to imply that social anhedonia is specific to SAD to the exclusion of all other DSM-5 diagnoses. To the contrary, substantial prior work has demonstrated that social anhedonia is observed across a range of psychiatric conditions including depression (Enneking et al., 2019), schizophrenia (Blanchard, Collins, Aghevli, Leung, & Cohen, 2011; Ritsner, Ratner, Mendyk, & Gooding, 2018), autism spectrum disorder (Gadow & Garman, 2018) and eating disorders (Tchanturia et al., 2012). Within each of these conditions, social anhedonia appears to have distinct risk and comorbidity patterns, which may offer insight into transdiagnostic mechanisms and consequences. We consider these effects here, as well as the inter-relationships between sensitivity shift theory and other models of SAD in the broader nomological network.

5.1. Transdiagnostic relevance and patterns of comorbidity

Although still a relatively new area of inquiry, systematic evaluation of social anhedonia age-of-onset and risk factors collectively suggest the presence of manifold contributors and pathways toward this outcome. In a recent international population-based survey of social anhedonia in more than 19,000 individuals, Dodell-Feder and Germaine (2018) reported that among other things, social anhedonia prevalence is highly variable across the lifespan, more common in males than females, and is associated with significant social and economic disadvantage. These authors further report that in the general population, rates of social anhedonia steadily increase beginning around age 9, reach an initial plateau at roughly 15 years of age, and then increase at a relatively reduced rate until 43 years of age, followed by a steady decline into old age. Elaborating on these population-based patterns, empirical examinations of age-of-onset in disordered samples have identified correlations between social anhedonia and age of symptom onset in schizophrenia. Ritsner et al. (2018) reported that earlier onset of illness was significantly associated with lower social/interpersonal pleasure in patients with either schizophrenia or schizoaffective disorder. Longitudinal data reported by Blanchard, Horan, and Brown (2001) further indicate that after initial onset, social anhedonia is an enduring trait in schizophrenia, but only transiently related to clinical status in depression, an effect which could not be accounted for by symptom severity. In autism spectrum disorder (ASD) social motivational deficits and social anhedonia tend to be observed relatively early in development (Chevallier, Kohls, Troiani, Brodtkin, & Schultz, 2012; Gadow & Garman, 2018), and are thought to be stable into adulthood (Carre et al., 2015).

Caution is warranted in summary interpretation of these age-of-onset findings given the nascent character of systematic work in this area. However, these patterns suggest that on the one hand, elevated social anhedonia may reflect an early-emerging and temporally enduring trait in neurodevelopmental disorders such as schizophrenia and autism, but on the other hand may be reflective of current symptomatology (or as an episode indicator) in affective disorders (Blanchard et al., 2001). The emergent research on risk factors for social anhedonia across these broad domains further supports the existence of highly distinct mechanisms, with anhedonia in schizophrenia perhaps related to diminished encoding and retrieval processes that influence reports of noncurrent feelings (Strauss & Gold, 2012), whereas anhedonic symptoms in depression may instead stem from altered incentive processing

and reinforcement learning mechanisms (Barch, Pagliaccio, & Luking, 2016). These developmental timing and risk effects also indicate that social anhedonia is unlikely to be specific either to SAD or even to affective disorders more broadly. It may instead be a transdiagnostic outcome toward which manifold risk-chains coalesce. As such, the developmental pathway illustrated by our theory may represent only one of many possible pathways.

In keeping with this idea that multiple nonredundant developmental paths may eventually culminate in a similar syndrome of social anhedonia, consideration of these effects may offer insight into both risk and comorbidity patterns of trans-diagnostic relevance. Anhedonia is considered a core diagnostic feature of depression, which itself is among the most common comorbidities among adolescents and young adults with SAD (Chavira, Stein, Bailey, & Stein, 2004; Stein et al., 2001). The risk-chain described by sensitivity shift theory may potentially explain certain patterns of comorbidity in adults with SAD, although this remains an area for future study. For example, prior work has explored the relationship between components of the tripartite model to types of social anxiety (e.g., performance anxiety, social interaction anxiety; Hughes et al., 2006), revealing that low PA is more specifically related to social interaction anxiety in the context of SAD. Conversely, the relationship between types of social anxiety and types of depression remains an emergent area of study. A testable hypothesis derived from our theory is that anhedonic depression should be observed at a higher frequency than sad mood or dysphoria in the context of SAD. Furthermore, in SAD with comorbid depression (of secondary or later onset), social anhedonia in particular should be observed at a disproportionately higher rate relative to physical anhedonia which includes reduced capacity to experience physical pleasures such as tastes and touch. Such a finding would support the idea that social anhedonia is itself a risk factor for transition into depression for SAD patients, and would also be consistent with work in non-clinical populations suggesting that social defeat may be a particular risk factor presaging anhedonic depression (Brewer & Olive, 2014; Cheung, Gilbert, & Irons, 2004; Gilbert, Allan, Brough, Melley, & Miles, 2002).

Deficits in positive affect and reward-seeking are central to our theoretical model, however disruptions in these positively-valenced systems co-exist with other core features of the disorder including negatively-valenced processes such as fear and avoidance (Heimberg, Brozovich, & Rapee, 2010). Interactions between these systems have been considered within the Research Domain Criteria (RDoC) framework (Cuthbert & Insel, 2013), which has promoted empirical study of core domains of functioning rather than DSM-5 diagnostic thresholds and boundaries. Fear, for example, figures prominently in the RDoC matrix, although it has not been thoroughly explicated in terms of its relationship specifically with social anhedonia. In the context of anhedonia more broadly however, the impact of negatively-valenced systems have been examined using the lens of RDoC (c.f. Dillon et al., 2014), collectively suggesting that threat and fear can mechanistically affect specific parameters of reward processing. For instance, acute stress appears to negatively impact reward responsiveness in healthy adults (Bogdan & Pizzagalli, 2006), an effect that appears to be related to alterations in sensitivity to reinforcement contingencies. Within the RDoC ecosystem, substantial additional work will be required in order to understand the consequences of social threat on reinforcement learning specifically as they relate to social anhedonia.

Results of this future work will have important implications for treatment. Currently, medication as well as most front-line psychotherapeutic approaches do not sufficiently address motivational and reward processing deficits, although evidence appears to suggest that these processes have a direct and specific effect on reward-based learning. Auerbach, Pagliaccio, and Pizzagalli (2019) recently commented on the importance of moving beyond a conceptualization of anhedonia as a monolithic entity, and instead moving toward an understanding that involves dissociable components such as anticipatory, consummatory and reward-learning processes. In a similar sense, we

recommend a granular approach to translating fundamental research into treatment of social anhedonia. For example, work guided by RDoC has suggested that fear and stress acutely alter expectations about cue-reward and action-outcome contingencies, which then alters behaviors based on the expectation of value. An important step will be to evaluate the influence of social threat on parameters of reinforcement based learning, such as sensitivity to social reward, the ability to modulate behavior based on social reinforcement, and the neurobiological factors that subserve these processes. This nuanced view could provide an important context for exploring more targeted treatments and preventative interventions by building upon prior work that breaks down the complexity of positively-valenced systems into mechanistic relationships between stress, threat and reward processing.

Although prior work has established that a significant proportion of socially anxious adults present with symptoms of social anhedonia (Brown, Silvia, Myin-Germeys, & Kwapil, 2007; Cicero, Krieg, Becker, & Kerns, 2016), there is considerable heterogeneity in social anxiety symptomatology and trajectories of risk in adolescents (Nelemans et al., 2014; Weems, 2008). In light of this pervasive heterogeneity, social anhedonia is not proposed here as a definitional characteristic of all SAD cases, but may instead be a specific developmental path that is characteristic of some but not all individuals with SAD. Additional future work will be necessary in order to identify underlying brain-behavior profiles associated with this trajectory, which will have eventual implications for personalization of treatment based specifically on these processes. For example, assessment of social anhedonia characteristics among individuals presenting for treatment will inform whether clinicians focus on exposure-based treatment, treatment aimed at modulating positive affect or some combination thereof.

5.2. Connections to existing theories of motivational deficits in SAD

It is also important to consider how sensitivity shift theory compares and contrasts with other extant theories of positive motivational deficits in SAD. For example, Kashdan, Weeks, and Savostyanova (2011) provided a major theoretical contribution to the understanding of positive motivational deficits in SAD with their self-control resource depletion model. Although complex, the essence of the self-control resource depletion model suggests that individuals with SAD expend disproportionate cognitive resources in pursuit of heterogeneous self-control acts during social interaction. As a result, a concomitant decrease in goal-directed behavior is theorized to occur, due specifically to the depletion of finite cognitive resources that would otherwise be available to coordinate them. The pattern borne out by this self-regulatory depletion mechanism is believed to dampen positive interpersonal outcomes and positive affect by degrading social relationships which are a primary source of positive experiences and events (Baumeister & Leary, 1995). Excessive self-control efforts during social interaction may further represent a specific maintaining factor for SAD symptoms in adults by lowering the quality of interpersonal relationships and eroding social/interpersonal support networks. Inasmuch as social relationships are themselves a major source of positive experiences (Baumeister & Leary, 1995), the maladaptive cycle of resource depletion and missed opportunities to build interpersonal connections may entrench long-term interpersonal consequences that are difficult to reverse.

Although the mechanisms and overall nature of the depletion effect is an ongoing area of investigation (Inzlicht, Schmeichel, & Macrae, 2014), recent research has generally supported the predictions made by Kashdan et al. (2011) in adult samples. For example, results from a daily diary study from O'Toole, Zachariae, and Mennin (2017) indicated higher rates of experiential avoidance among college students high in social anxiety symptoms, particularly at higher levels of negative intensity. Similarly, Weisman, Rodebaugh, Lim, and Fernandez (2015) found that adults with SAD reported significantly higher scores on scales reflecting maladaptive emotion regulation strategies, including difficulty focusing on other things when upset and nonacceptance of

emotional responses.

Sensitivity shift theory provides a developmental context that combines well with the predictions made by Kashdan et al. (2011). In particular, a point of connection between these theories concerns the initially higher expectations for reward that prompt the additional effort that is essential to self-control resource depletion theory. Indeed, heightened effort during social interaction (prior to transition into social defeat) would be not only predicted by our theory, but also required to produce the concomitant decrease in resources that interfere with engagement in potentially rewarding situations. Thus, the self-control acts that are central to self-control resource depletion theory may themselves be the result of initially heightened allocation of choice behaviors toward social reward seeking, which later suffer from the rate-suppressing effect of social defeat. The empirical work suggesting that these patterns are observed in adults with SAD may also be specifically reflective of the timeline that is central to sensitivity shift theory. For example in their sample of middle-aged adults, Weisman, Rodebaugh, Lim, and Fernandez (2015) did not find evidence for their central hypothesis that emotion regulation and personality trait variables varied according to SAD diagnosis. Whereas, O'Toole et al., (2017), in a sample of college students, did find corroborative evidence that this comparatively younger sample did indeed report more frequent use of experiential avoidance strategies in a group higher in SAD symptoms, particularly at higher levels negative emotional intensity. Thus, these discrepant age-effects could potentially be explainable in terms of the developmental timeline of reward sensitivity and concomitant effort expenditure. Additional work will however be required in order to more fully understand the role of developmental factors in precipitating excessive acts of self-control.

In a conceptually related theoretical model, Kimbrel (2008) provides an account of SAD genesis and maintenance couched in terms of Gray's (1970, 1982, 1991) Reinforcement Sensitivity Theory (RST). RST describes a biologically-based framework of personality subsystems including the Fight-Flight-Freeze System (FFFS), the Behavioral Approach System (BAS) and the Behavioral Inhibition System (BIS). Kimbrel (2008) points out that the BAS, in particular, is relevant to motivational deficits seen in SAD, inasmuch as its principal role is to motivate approach-related behavior to both conditioned and unconditioned stimuli (Corr, 2004). Within this approach, low BAS sensitivity is construed as a risk factor for SAD by indirectly facilitating FFFS activity. This idea was later supported in an analogue sample of young adults with high social anxiety symptoms (Kimbrel, Mitchell, & Nelson-Gray, 2010), indicating that low BAS sensitivity but not low PA was associated with social interaction anxiety. In contrast to our theoretical account here, Kimbrel (2008) further notes that high BAS sensitivity may alone be sufficiently protective against the development of SAD specifically because it suppresses FFFS activity. We postulate that in light of prior evidence for higher levels of reward sensitivity in behaviorally inhibited youth (Lahat, Benson, Pine, Fox, & Ernst, 2018), that high BAS sensitivity may be a risk factor for SAD perhaps only when combined with an adverse learning history which together would be expected to accelerate the learned futility of social effort expenditure. This is also consistent with more recent observations of low self-reported BAS in adults with social anxiety symptoms, as demonstrated by Kimbrel et al. (2010).

5.3. Relevance to theoretical models of social skills deficits

Our prediction that social skills deficits are a consequence of diminished social motivation must be considered in light of previous work illustrating that adults with SAD do not necessarily demonstrate poor social skills (Angelico, De Sousa Crippa, & Loureiro, 2013), as well as evidence that treating social skills deficits may enhance treatment outcomes in SAD (Beidel et al., 2014; Herbert et al., 2005). Moreover, some conceptualizations of SAD also argue that these individuals have adequate social skills, but excessive anxiety during social interaction

consumes limited attentional resources, leaving comparatively less cognitive capacity left over to devote to skillful social interaction (Hopko, McNeil, Zvolensky, & Eifert, 2001). Thus, some have argued that social skills deficits in this population may be better conceptualized as a performance deficit due to situationally-specific anxiety.

Support for social skills training (SST) interventions in SAD remains mixed, with some reports of little to no evidence for their efficacy when delivered alone (i.e. not combined with CBT or exposure-based treatment; Ponniah & Hollon, 2008). Other investigations have however indicated that SST enhances outcomes when combined with CBT (Herbert et al., 2005), or exposure therapy (Beidel et al., 2014), although at least one other study has not found a similar effect when combining SST with an interpersonal treatment approach (Stravynski et al., 2000). It has also been demonstrated that a distinction emerges when comparing actual versus perceived skills deficits. For example, Cartwright-Hatton, Tschernitz, and Gomersall (2005) demonstrated that in a sample of children aged 10–11 who participated in a conversation with an unfamiliar adult, independent raters could not distinguish between the high and low anxiety group. Yet, socially anxious children rated themselves as appearing significantly less skilled. In a similar study, Miers, Blote, and Westenberg (2010) found that peers perceived poorer social skills in a group of socially anxious adolescents (13–17 years old) as compared to a group of nonanxious adolescents during a videotaped speech. Using latent class growth modeling (LCGM), this group later found that observer-reported social skills deficits in youth between 9 and 17 years of age was specifically related to a latent class of adolescents characterized by initially high and subsequently variable scores of social anxiety symptoms (Miers, Blote, de Rooij, Bokhorst, & Westenberg, 2013). However, observer-rated social skills problems were not related to other latent classes defined by moderate or low social anxiety symptoms.

Taken together, conceptualizations of the genesis, maintaining roles and treatment relevance of real and perceived social skills deficits in this population are not without controversy. In the context of this dispute, sensitivity shift theory may offer guidance to future work that seeks to clarify how youth with perceived and/or actual social skills deficits develop. A central question awaiting future testing is whether motivational deficits mediate the relationship between social anxiety symptoms and social skills deficits, either real or perceived. It will similarly be important to examine whether age moderates the association between social anxiety symptoms and motivational deficits. These models can be combined to form a testable model of moderated mediation (Type I conditional indirect effects), in which age would be predicted to influence the magnitude of the association between social anxiety and motivational deficits, the latter of which should mediate the relationship between social anxiety and social skills. If some combination of these predictions are correct, then experimental therapeutic efforts can be adapted to test presumed mechanisms such as diminished practice effects as would be predicted by our theory, versus anxiety-driven performance deficits as predicted by others (Hopko et al., 2001). These distinct causal pathways may also be meaningfully related to the effects of age. For example, in light of evidence for significant variation in both self- and observer ratings of social skills deficits in children and adolescents, it is plausible that motivational deficits may lead to a form of later-emerging social skills impairments, whereas anxiety-driven interference with skillful interaction would be observed earlier and would further be expected to be unrelated to motivational impairment.

5.4. Relevance to theoretical models of safety behaviors

Although social skills deficits are posited to be one of the behavioral sequelae of social anhedonia, there is also evidence to suggest that social skills deficits in SAD may instead be the manifestation of safety behaviors (Alden & Bieling, 1998). Safety behaviors are observed across a range of anxiety disorders (Schmidt et al., 2012), and are defined as

covert and overt avoidance behaviors aimed at preventing feared outcomes (Kim, 2005; Wells et al., 1995). While safety behaviors may initially provide relief from fear-inducing stimuli in the short term, in the long term they are believed to exacerbate and maintain anxiety disorders by preventing habituation experiences and interfering with disconfirmation of fear-related beliefs (McManus, Sacadura, & Clark, 2008). Safety behaviors are known to take a variety of forms in SAD, including talking only briefly, avoiding self-disclosure or avoiding discussion of oneself entirely (Clark & Wells, 1995).

In two experimental studies of adults with social anxiety reported by Taylor and Alden (2010), participants who were instructed to eliminate their self-identified safety behaviors during a laboratory-based social interaction task reported improvements in self-judgments and future social predictions. Experimental evidence has further suggested that reductions in safety behaviors in adults with SAD leads to beneficial changes in motivational processes that promote positive social interactions (Plasencia, Taylor, & Alden, 2016). Similarly, Taylor and Alden (2011) reported that reductions in safety behaviors in adults with SAD led to increases in social approach behaviors during a laboratory-based social interaction task as well more positive reactions on the part of an interaction partner. Results additionally indicated that adults with SAD who were instructed to reduce safety behaviors both perceived and actually elicited more positive interpersonal responses than a control group who were not instructed to reduce safety behaviors. Together, these findings raise the possibility that safety behaviors (1) are themselves an obstacle to skillful social interaction, and (2) may also imply heightened social motivation to engage in social interaction, insofar as they facilitate social proximity while simultaneously guarding against feared outcomes. The latter point is somewhat paradoxical to a central claim in sensitivity shift theory, that social skills deficits are caused by infrequent practice opportunities, which in turn is caused by diminished social motivation.

To elaborate on the role of motivation in safety behaviors, sensitivity shift theory offers several specific predictions that are couched in prior evidence that not all safety behaviors serve the same function. Clark and Wells (1995) first posited that SAD-related safety behaviors could be grouped into avoidance and impression-management strategies. Avoidance strategies are thought to reduce engagement in social interaction (i.e. limited eye-contact, excessive brevity), whereas impression-management strategies are intended to favorably influence one's impression on others by controlling and regulating information during social interaction. Impression-management strategies imply an interpersonally-driven motivation to create a favorable impression in the minds of others due to highly valued social outcomes (Leary & Kowalski, 1990). Consistent with this idea, Plasencia, Alden, and Taylor (2011) demonstrated that individuals with SAD who used avoidance-type safety behaviors during a laboratory-based social interaction reported higher state-anxiety during interaction, and less motivation to engage in a future interaction with a conversation partner. Similarly, Hirsch, Meynen, and Clark (2004) found that avoidance-motivated safety behaviors were more associated with negative perceptions by others, whereas impression-management motivated safety behaviors were not.

We suggest here that individual differences in social motivation as predicted by sensitivity shift theory should be related to the type of safety behaviors selected, which in turn should be associated with the magnitude of social skills deficits. These specific predictions could be stated as follows: impression-management strategies, to the extent they are a marker of increased social motivation should therefore be associated with some combination of (1) heightened neurobiological sensitivity to social reward during the early to mid-adolescent period (2) heightened motivation to engage in future social interactions, and (3) normative or strong social skills. On the other hand, individuals who have transitioned into social anhedonia should report higher use of avoidance strategies, which should further be associated with a combination of (1) blunted neurobiological responses to social rewards

during the late-adolescent to early adulthood period (2) reduced motivation to engage in future social behaviors and (3) social skills deficits. Future work will be required in order to systematically examine these predictions. Outcomes from such work will likely be important for refining treatments aimed at eliminating safety behaviors, which in turn will have implications for overall quality of life and wellbeing for persons with SAD. For example, Alden and Bieling (1998) hypothesized that insofar as friendships and intimate personal relationships are thought to deepen specifically due to a process of mutual self-disclosure (Altman & Taylor, 1973; Jourard, 1971), it therefore follows that safety behaviors which were initially meant to facilitate social interaction will ironically lead to negative reactions from others. Thus, safety behaviors in SAD may eventually culminate in a self-fulfilling prophecy of social rejection.

6. Implications for intervention

The growing evidence base for the importance of motivational deficits in socially anxious adults (and their developmental precursors in adolescence and youth) highlights the need for experimental manipulations that directly modulate newly identified neural and behavioral targets. Yet, most gold-standard treatment concepts for SAD in youth are derived from overarching cognitive theories of social anxiety genesis and maintenance developed and tested in adult samples. While it is true that these cognitive models are well-supported in adults, and there is also good evidence to believe in their efficacy for socially anxious youth, a key point that emerges from the evidentiary framework for sensitivity shift theory is that *developmentally specific factors may play a major role* in the genesis of social anhedonia. Accordingly, we argue that in light of the evidence reviewed above, mechanistically-informed treatments and systematic examinations of their effects are needed to target the processes that lead from social stress to social motivational deficits.

Currently there are no intervention concepts that comprehensively target all factors in the developmental risk chain outlined by sensitivity shift theory as illustrated in Fig. 1. There are, however, a number of empirically-supported interventions developed in adult samples that do target facets of these processes and may therefore offer a roadmap for developing mechanistically-informed interventions for children and youth at risk for social anhedonia. Although not an exhaustive list (cf. Chakhssi, Kraiss, Sommers-Spijkerman, & Bohlmeijer, 2018 for a systematic review), we describe a number of particularly relevant interventions that target positive valence systems as well as a brief review of their evidentiary bases.

6.1. Positive affect treatment (PAT)

Drawing from affective neuroscience and experimental psychopathology research, Craske, Meuret, Ritz, Treanor, and Dour (2016) presented Positive Affect Treatment (PAT), a modular, transdiagnostic intervention approach that targets deficits in the appetitive reward system. PAT is delivered across 15 weeks through individual sessions of face-to-face therapist contact. Three conceptually inter-related modules target core processes in anhedonia, including anticipation/wanting, consumption/liking and reward-based learning. Exercises within these modules were selected to directly target positive affect and learning-related mechanisms in the appetitive reward system. For example, module one is rooted in pleasant events scheduling, in which patients plan for and engage in daily activities that are inherently pleasurable, and then spend time in-session recounting the pleasant emotions and experiences generated by the exercises. In module two, cognitive training exercises aim to identify positive features of the self, imagine positive outcomes, and attribute these positive outcomes to the self to promote a sense of mastery. Module three involves compassion training, lovingkindness meditation, gratitude contemplation, generosity and appreciative joy. In a recent randomized controlled trial in 96

adults with clinically severe symptoms of depression or anxiety (Craske et al., 2019), PAT was compared to a negative affect training (NAT) that is premised upon principles of cognitive behavioral therapy. Relative to this comparator condition, PAT resulted in significantly greater improvements in positive affect at post-treatment and 6-month followup, as well as reductions in the probability of suicidal ideation at 6-month followup.

Although PAT is a relatively new innovation, it appears that the exercises within this approach are a strong fit with the mechanistic risk factors described by sensitivity shift theory. In particular, PAT is likely to target the reinforcement based learning systems that are thought to be at the heart of the transition from social sensitivity to social defeat (Fig. 2). Therefore, delivering PAT during late childhood or early adolescence may prevent or even reverse progression into anhedonia for socially anxious youth. Although PAT may not lessen the impact of adverse social events, it is plausible to hypothesize that targeting this mechanism would modify the pathway to social anhedonia by restoring and sustaining social reward-seeking behavior. However, the application of PAT to youth and adolescent samples awaits future testing.

6.2. Positive activity intervention (PAI)

Taylor, Lyubomirsky, and Stein (2017) presented the Positive Activity Intervention (PAI), along with supportive data from a pilot study of 29 adults seeking treatment for anxiety and/or depression. Participants were randomly assigned to either PAI (N = 16) or wait-list control condition (N = 13). The PAI condition involved 10 1-hour weekly psychosocial treatment sessions based on a modular, manualized approach. Each session focused on a highly thematic lesson, such as noticing and amplifying positive events, gratitude and counting one's blessings and acts of kindness, among others. Results of this small randomized trial indicated that after 10 weeks, the PAI group reported significantly larger improvements in positive affect and psychological wellbeing as compared to the wait-list. Follow-up assessments further revealed that initial gains were maintained up to 6 months subsequent to the conclusion of treatment.

The sample reported in Taylor et al. (2017) was comprised of participants with depression and anxiety. Although social anxiety was not specifically targeted, the finding that a psychosocial intervention such as PAI may alter positive affect is highly consistent with prior experimental manipulations of conceptually related processes within social anxiety. For example, Taylor and Amir (2012) demonstrated in a sample of socially anxious adults, a computerized task (approach-avoidance training; AAT) that focuses on modifying approach biases influenced a relationship-building task with a trained confederate. In another socially anxious sample, Taylor, Bomyea, and Amir (2011) demonstrated that a strategically targeted computerized training method designed to train attention toward positive emotional cues influenced subsequent emotional reactivity to a laboratory-based social stressor. As such, the processes and themes within PAI appear to fit well with motivational deficits in social anxiety. Given the nature of the themes in PAI, it is further plausible that the exercises facilitate approach-related motivation toward social interaction, although this remains an untested idea. However if this is the case, then PAI offers high potential to directly target these systems in adolescents prior to transition into social anhedonia, which will have implications for building positive emotions and well-being. Specifically, sustaining approach-related motivation toward social sources of reward would be particularly important in preventing the transition into social defeat for at-risk youth.

6.3. Acts of kindness (AK)

Alden and Trew (2013) demonstrated that a structured psychosocial intervention focused on training in (and promotion of) acts of kindness resulted in significant increases in positive affect in a sample of adults

with high levels of social anxiety. Participants (N = 143) were randomly assigned to either the acts of kindness (AK) condition, or to a condition aimed at reducing negative affect (NA), or a neutral comparator condition (activity monitoring). Kind acts were defined as behaviors that benefit others, typically at some cost to oneself. The AK condition involved planning and enacting three kind acts a day on 2 days each week over a 4-week period. Results indicated that engaging in kind acts influenced change in positive affect from pre- to post-treatment as well as measures of weekly mood. However, neither the NA-focused condition nor the daily monitoring control condition increased PA. Kind acts also increased satisfaction with social relationships, and lessened concerns over protecting oneself from negative social outcomes.

As one of the few empirical examinations of hedonic enhancement in a socially anxious sample, Alden and Trew (2013) provide important context for understanding the effects of behavioral exercises specifically intended to increase PA. The AK intervention may be a particularly profitable area of future research in youth and adolescent samples for a number of reasons. For example, applications of the AK intervention in youth may have the direct benefit of modifying the pathway toward social anhedonia by addressing the hypothesized effect of adverse social context, which is central to sensitivity shift theory. Prior work has shown that self-compassion and interpersonal cohesion can buffer the effects of peer victimization on non-suicidal self injury (Jiang et al., 2016). It is possible in a similar sense that acts of kindness could modify the effect of peer victimization on anhedonia by promoting adaptive strategies to cope with negative events (Allen & Leary, 2010), and creating sense of connectedness with others (Kerr, O'Donovan, & Pepping, 2015). From a practical perspective, acts of kindness could also be more acceptable in this sensitive population for whom exposure exercises may be prohibitively aversive.

6.4. Mindfulness-based interventions (MBI)

Mindfulness can be defined as the awareness that arises when paying attention in the present moment, on purpose and non-judgmentally (Kabat-Zinn and University of Massachusetts Medical Center/Worcester. Stress Reduction Clinic, 1990). When someone is "being mindful," the attitudinal quality of not judging and allowing experience to unfold with curiosity rather than trying to control it may reduce the impact of positive and negative affective states when triggered (Brewer, Van Dam, & Davis, 2015). This is particularly relevant in the case of social anxiety, in which periods of transient stress can reactivate negative emotional patterns and thinking—a hallmark of cognitive and emotional reactivity (Scher, Ingram, & Segal, 2005). Broadly consistent with this proposition, there is now growing evidence showing that mindfulness training can be efficacious for adults with anxiety conditions, broadly construed. Meta-analyses of mindfulness-based interventions delivered in person, such as Mindfulness-Based Stress Reduction (MBSR), and Mindfulness-Based Cognitive Therapy (MBCT) have yielded effect sizes ranging from .30 to 1.0 for anxiety (Goyal et al., 2014; Hofmann, Sawyer, Witt, & Oh, 2010; Khoury et al., 2013).

Connections between MBIs and sensitivity shift theory are revealed when considering the interface between mindfulness practice and reinforcement-based learning, the latter of which is theorized to be a central mechanism involved in the transition from social stress to social anhedonia. A key point in our theoretical model is that social anxiety development is intimately tied to DA-linked mechanisms of learning, and that vulnerable individuals demonstrate particular sensitivity to the consequences of social action. Our theory specifically posits that persistent unfavorable social outcomes culminate in anhedonia due to the learned futility of social effort expenditure. Taking advantage of the same learning mechanisms, mindfulness may reconnect social interaction with its potential for reward by enhancing detection, registration and processing of pleasurable engagement with the environment when

and if it occurs. In line with this idea, Geschwind, Peeters, Drukker, van Os, and Wichers (2011) reported data from a randomized controlled trial of Mindfulness-Based Cognitive Therapy (MBCT) in adults vulnerable to depression. Results indicated that MBCT was associated with increased experiences of momentary positive emotions, as well as greater appreciation of and responsiveness to pleasant daily life activities.

Along these same lines, mindful awareness of the consequences of social action may promote rediscovery that social effort expenditure (for example initiating a conversation, attending a social gathering) has at least a non-zero likelihood of producing a gratifying outcome. Conversely, distraction or disengagement from the emotional experience of the present moment is unlikely to modify former action-outcome associations. Because no violation of prior beliefs or predictions occurs, disengagement or distraction from emotional experience will, by definition, obviate the possibility of new learning. Thus, sensitivity shift theory exposes a direct treatment target that is potentially articulated by mindfulness practice, wherein restoration of the positively motivating quality of social interaction involves *recoupling* the formerly broken association between social effort expenditure and its potential for reward. This target and hypothesized mechanism are illustrated as Fig. 3, which overlays theoretically relevant constructs onto the processes of social anhedonia genesis, maintenance and reversal. Fig. 3 further illustrates a major connection between our account of social anhedonia and a potential mechanism of action in mindfulness practice. By reinstating the formerly eroded action-outcome association between social effort expenditure and its potential for naturally rewarding consequences (e.g., social support, friendship building, romantic relationships), mindfulness practice may interrupt the maintenance cycle that is promoted by distraction and aversion. Ultimately, by enhancing sensitivity to pleasurable experience as it naturally occurs, mindfulness training may reestablish the motivational properties of social context.

The impact of mindfulness practice is not necessarily limited to reinforcement learning-based modification of behavior based on positive experiences, but may also impact learning about negative outcomes as well. For example, when avoidance behaviors become a strategy for triggers such as fear of negative evaluation, the reduction in the fear can lead to a process of negative reinforcement (Skinner, 1963). Over time, this behavior becomes habitual in nature, operating largely out of conscious control (Suhler & Churchland, 2009). What previously was learned as a coping mechanism for fear self-perpetuates over time and prevents new learning about distress-producing cues in the environment. In fact, mindfulness training may be unique in its ability to tap into reinforcement learning itself. Reinforcement learning is dependent upon experiencing the outcomes of actions, however habitual avoidance or habitual negative emotional responses may block awareness of present-moment reward value when action-outcomes yield a positive result.

Research specifically linking MBIs to behavioral and neurobiological mechanisms of reward remains in its infancy (Brewer, 2019). However, some systematic work on the effects of mindfulness on the neural systems of reward and valuation has been conducted by Kirk, Gu, Harvey, Fonagy, and Montague (2014) and Kirk and Montague (2015). In a neuroimaging investigation using a simple conditioning task, experienced (lifelong Zen) meditators demonstrated attenuated reward prediction error signals in the striatum, representing a reduction in the difference between expected versus actual outcomes during reinforcement learning (Kirk & Montague, 2015). This effect was later observed in naïve (never meditating) subjects randomized to a mindfulness training condition (Kirk, Pagnoni, Hetu, & Montague, 2019). Results of this work suggest that value signals in the brain may be causally influenced by mindfulness practice, ultimately leading to a systematic change in expectancies about reward outcomes. Although the processes by which this occurs remains an active topic of inquiry, Kirk et al., (2019) hypothesized that mindfulness training may modify Bayesian priors of future value expectancies such that a single

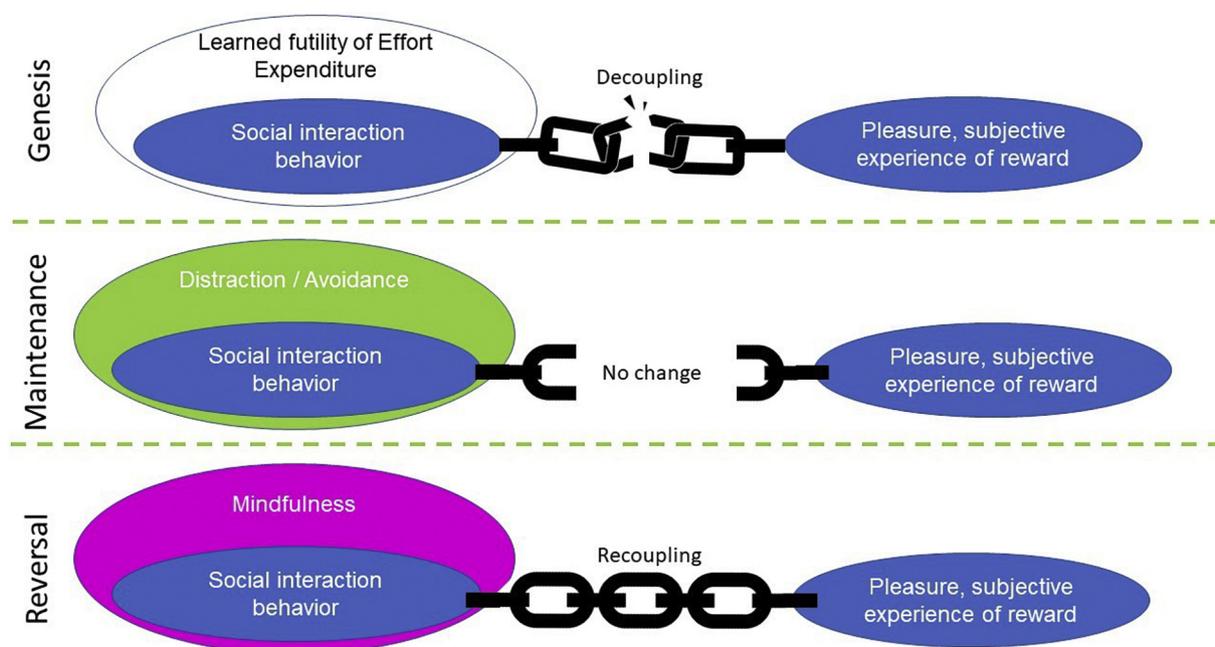


Fig. 3. Conceptual representation of hypothesized mechanism of action in mindfulness practice as it relates to social anhedonia. The strength of action-outcome associations between social interaction behavior and the subjective experience of reward or pleasure is illustrated in the context of learned futility of effort, avoidance and mindfulness practice, which relate to the genesis, maintenance and reversal of social anhedonia, respectively. As specified by our theory, mindfulness practice may directly treat social anhedonia by modifying the formerly broken association between social interaction and subjective experience of pleasure.

hypothesized outcome is less favored as the learner moves toward a stance where no specific hypothesis is favored for prediction. They further noted that this particular effect of mindfulness training may shift the learner toward a stance of “equipotent poise,” that is still capable of producing learning through the same mechanisms but is less disturbed by experience, a perspective that could be compared to the Buddhist concept of equanimity (Desbordes et al., 2014).

Systematic work evaluating the impact and mechanisms of MBIs in SAD similarly remains in the early stages. Prior evidence from randomized controlled trials in adults with SAD has consistently indicated comparable, although not superior treatment outcomes in MBIs as compared to CBT in adults (Goldin et al., 2016; Kocovski, Fleming, Hawley, Huta, & Antony, 2013; Koszycki, Bengler, Shlik, & Bradwejn, 2007). In the only known trial to utilize a sample of youths with SAD, Ebrahimejad, Poursharifi, Bakshshour Roodsari, Zeinodini, and Noorbakhsh (2016) conducted a small (N = 30) RCT in female adolescents who met diagnostic criteria for SAD. Participants were randomly assigned to either a treatment condition utilizing an MBCT protocol or a no treatment control condition. Individuals in the MBCT group showed reduced self-reported social anxiety symptoms and improvement in self-esteem. While studies utilizing MBIs among adolescents have not addressed mechanisms of change, a recent pilot study from Strege, Swain, Bochicchio, Valdespino, and Richey (2018), evaluating the impact of MBCT on adults with SAD suggested that improvement in SAD symptoms may specifically relate to change in positive, approach related emotion. Thus, we do not argue here that MBIs are likely to be universally ideal for all SAD cases, but may instead be a particularly strong fit for certain combinations of symptom characteristics related to social anhedonia. Indeed, future work will be required in order to determine whether targeting cases who are specifically low in PA may result in added benefit from MBIs. Further benefit could be realized in adolescent samples when considering the high potential and precedent for implementation of mindfulness practices via telehealth or remote delivery via mobile technology (Fish, Brimson, & Lynch, 2016).

7. Conclusions

This review had four principal aims. First we sought to describe and critically evaluate current behavioral and neurobiological work in the area of positive, approach related deficits in adolescent SAD. Second, we used this as an evidentiary framework for presenting sensitivity shift theory, an integrative theoretical model explaining the developmental pathway from temperamental and environmental factors into social stress and resultant reward-related deficits particularly in the social domain. Third, we considered the transdiagnostic and transtheoretical relevance of sensitivity shift, with specific predictions that add to and in some cases contrast with extant theory. Finally, we sought to provide a complementary mechanistic account by which certain interventions might directly address mechanisms of change exposed by our model.

Conclusions presented here should also be considered in light of the limitation that this was not a systematic review of the literature. The broad scope of SST, covering clinical outcome studies, neuroimaging work, animal models, learning theory and developmental neurobiology of SAD means that a fully systematic review of all components would exceed the scope of a single scholarly contribution. Rather, we took a targeted approach, searching multiple publicly available databases (PubMed, Scopus and Web of Science) with terms including but not limited to the major content domains of SST (“social anxiety/social phobia”, “positive affect, motivation, reward”, “fMRI, dopamine, learning”) across major developmental periods of interest (“adolescent, youth, child”). Future work examining subtopics from a systematic perspective are likely to be of scientific value, and would motivate future refinements of the theory.

In summary, adolescence appears to be a particularly critical period for the development of SAD symptomatology due to the convergence of temperamental and environmental factors that peak during this developmental window. The theoretical approach presented here provides a number of testable predictions, and considering the generally modest rates of remission in trials of CBT for socially anxious youth, future work in this area will benefit from systematic manipulation of neural systems involved in learning and appetitive motivation as they relate to SAD symptomatology. Our overall model brings together multiple

levels of analysis, including molecular and systems neuroscience perspectives, preclinical animal models as well as human neuroimaging and clinical trial methodologies. These connections may inform targeted intervention efforts and encourage a more mechanistic delineation of the causal relationships between social anxiety and positive, approach-related deficits.

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Contributors

All authors materially participated in the preparation of this manuscript. J. Richey supervised the team, coordinated writing tasks and wrote the majority of the manuscript. J. Brewer wrote portions of the manuscript related to mechanisms of action in mindfulness-based interventions. H. Sullivan-Toole and M. Stregge wrote the portion of the manuscript describing clinical trials of mindfulness based interventions in adolescents. J. Kim-Spoon wrote portions of the manuscript related to adolescent and developmental factors. S. White wrote portions of the manuscript related to technology-enabled interventions. T. Ollendick wrote portions of the manuscript related to adolescent social anxiety phenomenology and current status of treatment.

Conflict of interests

Dr. Brewer owns stock in Claritas Mindsciences, Claritas Mindsciences had no role in any activities related to the present manuscript. All other authors declare that they have no conflicts of interest, financial or otherwise.

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