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Social anxiety disorder (SAD) is maintained by two underlying maladaptive appraisals of negative social outcomes, which are a function of maladaptive self and interpersonal judgments. Maladaptive self-judgments pertain to the belief that the individual will perform inadequately while maladaptive interpersonal judgments pertain to the belief that others will negatively evaluate the individual. This paper examines recent research done on these judgments and discusses the implications it has for the treatment of SAD in cognitive behavioural therapy (CBT). Evidence for attentional biases in SAD across behavioral and functional magnetic resonance imaging (fMRI) studies are also reviewed with a focus on the possibility for an attentional bias towards internal bodily sensations.

According to Endler (1975), different individuals have different levels of stress-susceptibility to two main stress-provoking situations. These situations include being in physical danger and being socially evaluated (Paterson & Neufeld, 1989). Social anxiety disorder (SAD) is an anxiety disorder, which falls under the latter situation, and is characterized by an excessive fear of negative social evaluation (Nakao et al., 2011). This fear interferes with the ability to participate in social interactions and may be specific to either one type of social situation (circumscribed SAD) or generalized across multiple social situations (generalized SAD). Regardless of SAD type, individuals typically experience embarrassment, show physical signs of anxiety (e.g., blushing) and show avoidance of social situations. According to cognitive models of SAD, this disorder is maintained through underlying maladaptive cognitive appraisals (Taylor & Allen, 2008). This paper will review the nature of these appraisals and investigate the implications they have for treatment through cognitive behavioural therapy (CBT). In addition, the evidence for possible attentional biases (ABs) in individuals with the disorder will also be examined.

Cognitive Models of SAD

Researchers investigating SAD have proposed a cognitive model to explain why some individuals in the general population develop social anxiety disorders and not others. According to this cognitive model, individuals with SAD tend to have a biological vulnerability for the disorder such as a neurobiological vulnerability (e.g., amygdala over activity; discussed below) or a genetic predisposition. Twin studies suggest that the heritability estimate of SAD is 0.10 (Norrholm & Ressler, 2009) and first-degree relatives of individuals with SAD tend to show more severe SAD symptoms than first-degree relatives of individuals without the disorder (Scaini et. al, 2014). In addition, individuals who have a first degree relative with SAD are at a six- to nine fold increased risk of developing SAD (Norrholm & Ressler, 2009), suggesting that genetics plays a key role in the onset of SAD.

However, a genetic predisposition alone is not sufficient for SAD onset. Rather, this genetic disposition interacts with the environment to create maladaptive cognitive appraisals that underlie the disorder. Examples of such environmental influences include traumatic childhood experiences (e.g., being bullied) and having overprotective parents. If the post-

impact of traumatic childhood experiences are severe enough, these negative social experiences may interact with a genetic predisposition or neurobiological vulnerability, leading to development of SAD (Scaini et al., 2014). Other variables such as parental behaviors may also contribute to the onset of SAD. Overprotective parents often model an overly cautious view of the world, which may reinforce avoidance behaviours and poor social skills in susceptible children. Indeed, there seems to be a positive association between having overprotective parents and the increased tendency of children with social anxiety (SA) being rejected by their peers (Scaini et al., 2014).

Cognitive Appraisals in SAD

The interaction between a biological vulnerability and negative social experiences is thought to lead to the development of two maladaptive cognitive appraisals. The first one is a tendency to overestimate the probability that negative social outcomes will occur, and the second is a tendency to overestimate the costs associated with these outcomes (Taylor & Allen, 2008). According to Paterson and Neufeld (1989), an individual's threat appraisal is influenced by the individual's assessment of the threat's likelihood of occurring as well as their assessment of the costs associated with threat occurrence. Applied to the context of SAD, individuals overestimate both of these factors, which may lead to a greater appraisal of ego threat– threat of being negatively evaluated, than what is warranted by the situation.

The tendency to overestimate the probability of a negative social outcome is a function of two maladaptive beliefs (Laurenti, Bruch & Haase, 2008). These two beliefs pertain to judgments about the self (SJ) as well as judgments about interpersonal relations (IPJ; Taylor & Allen, 2008). Maladaptive SJs in SAD relate to the individual's belief that they will perform inadequately in the social situation and

that the outcome of this performance will be negative. Maladaptive IPJs relate to the individual's belief that they will be evaluated negatively and that this outcome will be negative (Taylor & Allen, 2008). Evidence from fMRI studies supports the presence of these maladaptive beliefs in SAD by showing increased activation relative to controls in emotion-processing centers of the brain, such as the amygdala and medial pre-frontal cortices, when processing self-referential criticism (e.g. "You are an idiot"; Abraham et al., 2012).

Cognitive models of SAD propose that recovery from SAD is best achieved through the modification of these beliefs in CBT (Taylor & Allen, 2008). In particular, these models suggest that CBT's effectiveness is particularly linked through the modification of SJs (Taylor & Allen, 2008). However, recent research suggests that a greater emphasis needs to be placed on the modification of IPJs in the treatment of SAD (Laurenti et. al, 2008; Taylor & Allen, 2008).

Taylor and Allen (2008) randomly assigned SAD patients to either an interpersonal CBT (I-CBT) treatment or a control waiting list. At pre- and post-treatment, they assessed each patients' symptom severity, biases about likelihood and costs of negative social outcomes and the amount of weight they placed interpersonal feedback on their self-worth. Compared to controls, I-CBT patients showed significant reductions in their biases about the costs and probability of negative social outcomes as well as significant reductions in the amount of weight they placed interpersonal feedback on their self-worth. Both of these reductions were significantly related to the reduction of SAD symptoms (Taylor & Allen, 2008). These patients also showed a significant reduction in maladaptive SJs, however, such reductions only had a modest relationship with symptom reduction (Taylor & Allen, 2008). Given that current treatments for SAD focus more on the modification of maladaptive SJs, which only had

a modest positive relationship with symptom reduction, these results suggest that CBT needs to start placing more focus on the modification of maladaptive IPJs when considering treatment plans for SAD.

Studies looking at the role of personality factors in the development of SAD also suggest that more emphasis needs to be placed on the modification of maladaptive IPJs in CBT. Laurenti, et al. (2008) looked at the effect of socially prescribed perfectionism (SPP; beliefs about other people's social standards) as a moderator in SA. Participants completed several measures of SA and SSP before being told of an anticipatory conversation with another person. They then completed measures on their beliefs about their anticipated performance, the standards their conversational partner might have for them and the frequency in which they engaged in positive and negative thinking about the anticipated conversation. Individuals high in SA tended to believe that they are unable to meet other people's social standards. In addition, if these individuals were also high in SPP, they showed a greater tendency to engage in negative thinking. Although only level of SA predicted the frequency of negative thoughts, an interaction between SA and SPP was observed, suggesting that SPP may worsen SA symptoms by increasing the individual's tendency to dwell on negative thoughts and feelings. Thus, in addition to modifying SJs, CBT needs to identify key maladaptive IPJs and develop techniques in which SAD patients can challenge their SPP (Laurenti et al., 2008).

Attentional Biases in SAD

Since individuals with SAD tend to place more weight on interpersonal feedback and have an extreme fear of being negatively evaluated, researchers propose that these two factors influence these individuals to interpret social ambiguities as threatening (Taylor & Allen, 2008). Interestingly, there are inconsistent

findings in the literature regarding this. Studies using fMRI indicate that SAD patients show increased activation compared to control participants in the amygdala, insula, anterior cingulate, medial prefrontal and orbitofrontal cortices (Abraham et al., 2012) when presented with threatening faces, suggesting that at the neural level there is an attentional bias (AB) for such faces. Yet at the behavioral level, there are inconsistent findings across many studies investigating the presence of such biases using the facial-dot probe task, which is a task that tracks eye movements towards dots presented on different areas of neutral and angry faces placed side-by-side (Bantin, Stevens, Gerlach & Hermann, 2015).

One possible reason for these inconsistencies is that there are vast differences in stimulus duration, stimulus reference, severity of SAD symptoms, and presence of situational anxiety across these facial-dot probe task studies (Bantin et. al, 2015). Neuroimaging studies have shown that differences in neural activations exist between individuals who show sub-clinical levels of SA and those who show clinical levels (Nakao et al., 2011). Therefore, differences in SAD severity may contribute to inconsistencies reported in the literature. More research is needed to determine whether the additional differences highlighted above also contribute to reported inconsistencies.

To determine whether ABs are present at the behavioral level, Bantin et al. (2015) performed a meta-analysis on 10 facial dot-probe task studies that reported conflicting findings. Complementing fMRI evidence found by Abraham et al. (2012), they found preliminary evidence for ABs when threatening faces were compared to neutral faces. However, no such bias was found when such faces were compared to neutral objects. Instead in these situations, they found evidence for a tendency to avoid processing threatening faces. When no such avoidance was available, individuals

processed the most salient and informative social cue, regardless of whether the cue was positive or negative. Combined with fMRI results of increased activation in emotion-processing brain centers (Abraham et al., 2012), these results suggest that SAD ABs are associated with the processing of the most emotionally salient social cue available (Bantin et al., 2015).

Although this study provides more insight into the nature of ABs in SAD, the consensus is still unclear. Only 10 studies were included so more research needs to be conducted to examine whether these preliminary results replicate. Moreover, it is still unclear whether ABs are limited to threatening facial expressions or whether it extends to threatening social cues in general. For example, it may be possible that ABs in SAD are present for negative internal body sensations (Bantin et al., 2015). The next major focus of the paper will be linking evidence from fMRI and behavioral studies that suggest a possible AB for these bodily sensations in SAD.

Evidence for an AB for Negative Bodily Sensations in SAD

Individuals diagnosed with SAD tend to overestimate the visibility of their anxiety in social situations than what is observed during video recordings. One possible reason may be that these individuals have a tendency to dwell on negative thoughts and feelings (Laurenti et al., 2008). This tendency to dwell on such negativity may encourage behaviors that negatively impact social performance. Relative to controls, SAD patients tend to engage in more fidgeting, less smiling reciprocity, more self-talk, and more reassurance-seeking behaviour in social interactions, leading to all individuals in the interaction to rate the conversation as less smooth and engaging (Heerey & Kring, 2007; Lundy & Drouin, 2016).

Moreover, rumination in SAD has been found to mediate the positive relationship between stress and SA symptoms (Valenas &

Szentagotai-Tatar, 2015). This suggests that this tendency to overestimate anxiety visibility may create a state of arousal that is detrimental to performance. According to the Yerkes-Dodson effect, once arousal increases beyond an optimal level, performance deteriorates (Anderson, 1994). Many fMRI studies indicate that compared to controls, individuals with high SA experience higher levels of anxiety and have decreased activation in the left cerebellum, left precuneus and bilateral posterior cingulate cortex (PCC) while performing social tasks. The PCC is suggested to play a role in self-focused attention, while the precuneus is suggested to play a role in rating one's own traits relative to that of other individuals' (Nakao et. al, 2011). Supplementing Laurenti et al.'s (2008) findings, increased self-focused attention from these brain activations may increase the tendency to ruminate on negative thoughts and affect, leading to poorer performance in social situations (Nakao et al., 2011; Lundy & Drouin, 2016).

Looking specifically at the impact of stress on cognitive performance in SAD individuals, Koric et al. (2012) found that SAD individuals maintained the same level of activation in the right ventrolateral prefrontal cortex (VLPFC), an area associated with anxiety, across high stress and low stress tasks. In comparison, healthy controls had decreased activation in this area during more stressful tasks compared to less stressful tasks. Individuals with SAD also exhibited decreased activation in the dorsolateral prefrontal cortex (DLPFC), an area associated with cognitive control, in the more stressful condition compared to controls (Koric et al., 2012). These findings suggest that SAD individuals may have poorer cognitive control and higher levels of anxiety while under stress. Therefore, stress during social situations may impair social performance in SAD by interfering with cognitive functions, such as attentional allocation.

Combining findings from the two studies discussed above, stress elicited by rumination in SAD may impair social performance in three ways. One way is by influencing the content of cognition. Individuals with SAD tend to worry about ego threat and constantly assess the costs associated with being negatively socially evaluated. Another way is through decreased capacity deployment. Instead of allocating attention to the conversation, SAD individuals may allocate attention to their own anxiety as suggested by the reported deceased activation in DLPFC (Koric et al., 2012) and the observed increased levels self-talk and reassurance-seeking behaviors during social interactions relative to controls (Heerey & Kring, 2007). Lastly, stress may induce intrusive associations such as being distracted by anxiety visibility as well as their fear of not being able to meet other people's standards (SPP).

Although CBT is considered to be the most effective treatment for SAD (Taylor & Allen, 2008), it currently places more focus on the modification of maladaptive SJs than IPJs, which when successfully modified is only modestly associated with symptom reduction (Taylor & Allen, 2008). Converging evidence from the studies reviewed in this paper suggest that treatment for SAD should also consider therapeutic techniques targeted towards the modification of maladaptive IPJs. Given that individuals with SAD tend to overestimate the visibility of their anxiety, it is possible that an attentional bias towards negative bodily sensations may reinforce maladaptive beliefs about their social competence and how others will evaluate their performance. One technique that may prove particularly useful for the treatment of SAD is behavioural exposure therapy. Having patients receive feedback from conversational partners on the actual visibility of their symptoms may be effective in successfully modifying maladaptive judgments on how others will perceive them in social interactions

(Laurenti et al., 2008). These modifications may subsequently increase the amount of attention allocated to the actual conversation and lead to improvements in their social performance, which may reinforce positive beliefs about their social competence.

However, while the reviewed studies suggest a tentative attentional bias towards negative bodily sensations, it is important to note that there are limitations to these studies that make it difficult to make concrete conclusions about the underlying cognitive and attentional biases in SAD. As discussed above, there are differences across studies in many aspects of the methodology used to examine SAD, which may contribute to inconsistent findings in the literature. Using differences in symptom severity as an example, there have been reported differences in the activation patterns seen in individuals with clinical and subclinical SAD (Nakao et al., 2011), which may lead to differences in how these individuals perform in social situations under stress. In addition, only some individuals with SA have high SPP, leading to these particular individuals to dwell on negative thoughts more frequently than SA individuals who have low SPP (Laurenti et al., 2008). Another important limitation to note is that many studies investigating SAD typically use tasks involving anticipated conversations. Consequently, it is unclear how these findings would translate to actual social interactions. Therefore, in order to make more concrete conclusions about the underlying mechanisms of SAD, future research should carefully consider the influence of SAD symptom severity, personality traits (e.g., SPP), the type of experimental task used (e.g., anticipated vs. actual conversations) and the presence of situational anxiety or stress when designing studies.

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