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Heightened sensitivity to emotional expressions in generalised anxiety disorder, compared to social anxiety disorder, and controls

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ABSTRACT

Few studies have examined potential differences between social anxiety disorder (SAD) and generalised anxiety disorder (GAD) in the sensitivity to detect emotional expressions. The present study aims to compare the detection of emotional expressions in SAD and GAD. Participants with a primary diagnosis of GAD ($n = 46$), SAD ($n = 70$), and controls ($n = 118$) completed a morph movies task. The task presented faces expressing increasing degrees of emotional intensity, slowly changing from a neutral to a full-intensity happy, sad, or angry expressions. Participants used a slide bar to view the movie frames from left to right, and to stop at the first frame where they perceived an emotion. The frame selected thus indicated the intensity of emotion required to identify the facial expression. Participants with GAD detected the onset of facial emotions at lower intensity of emotion than participants with SAD ($p = 0.002$) and controls ($p = 0.039$). In a multiple regression analysis controlling for age, race, and depressive symptom severity, lower frame at which the emotion was detected was independently associated with GAD diagnosis ($B = -5.73$, SE = 1.74, $p < 0.01$). Our findings suggest that individuals with GAD exhibit enhanced detection of facial emotions compared to those with SAD or controls.

Social anxiety disorder (SAD) and generalised anxiety disorder (GAD) are common anxiety conditions that together affect nearly 22 million individuals in the USA in a given year (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). While much is understood about each anxiety condition, much less is known about how core aspects of their pathophysiology may differ or overlap. Relatively few studies have directly compared these two conditions across experimental tasks. Given the high comorbidity of these conditions (Kessler et al., 2005), overlap in effective treatments (e.g., antidepresants), and often incomplete response with available treatments (Ravindran & Stein, 2010), it is important to better understand which characteristics are shared and which are unique.

SAD is a distressing and impairing condition characterised by a persistent fear of embarrassment or humiliation in social or performance situations (Ruscio et al., 2008). Individuals with SAD have been consistently shown to exhibit a negative bias in the interpretation of social events (e.g., interpreting ambiguous social events in a negative fashion) (Stopa & Clark, 2000). While the pathogenesis of SAD is likely multifactorial (e.g., Hayward et al., 2008), it has been proposed that fear of social interactions might be linked in part to the way individuals with SAD perceive facial expressions. For example, individuals with clinical or subclinical SAD appear to exhibit heightened initial vigilance for threatening faces (e.g., Stevens, Rist, & Gerlach, 2009). Neuroimaging and event-related potential electroencephalographic data suggest greater activation of the amygdala (e.g., Straube, Mentzel, & Miltner, 2005) and abnormalities in the early stages of perceptual processing in
response to the presentation of threatening faces (e.g., Staugaard, 2010). A recent review found that SAD was characterized by a biased information-processing of facial expressions of emotion, including attentional bias away from negative faces, inaccurate labelling of negative emotions, enhanced recognition of negative facial displays, and increased memory for negative emotions (Machado-de-Sousa et al., 2010).

Cognitive models may explain the negative interpretation bias in individuals with SAD. Clark and Wells (1995) designed a model suggesting social anxiety persists because of the following elements: individuals are not accurately processing the social situation, are engaging in safety behaviours which prevent disconfirmation of feared outcomes, are negatively influencing other’s behaviour towards themselves, and are using their own impression of themselves as the main factor which contributes to the idea that others are negatively evaluating them. Additionally, Rapee and Heimberg (1997) suggest that the negative interpretation bias leads to stronger anxiety in social situation, which leads to increased social phobia.

To date, several studies have examined detection and interpretation of emotional facial expressions in individuals with social anxiety, with paradigms using either static facial expressions (e.g., Mullins & Duke, 2004), ambiguous facial stimuli (e.g., Jusyte & Schonenberg, 2014), or changing facial expression (Heuer, Lange, Isaac, Rinck, & Becker, 2010; Joormann & Gotlib, 2006; Montagne et al., 2006), with mixed results. Among the latter type of studies, one (Joormann & Gotlib, 2006) used a time-pressured morph movies task to assess potential differences in the intensity of facial expression needed to identify an emotion in individuals with SAD compared to those with depression and healthy controls. In this computer-based task, participants were presented with faces expressing increasing degrees of emotional intensity, slowly changing from a neutral to a full-intensity happy, sad, or angry expression. They were instructed to stop the movie when they accurately identified the emotion being expressed. While the intensity of emotion required to identify happy and sad expressions did not differ, angry facial expressions were identified “earlier” (i.e., with lower intensity expressions) for those with SAD compared to depressed and control participants. In contrast, other research using a morph movies task with no time restriction reported opposite results with individuals with SAD being less “sensitive” than controls when identifying negative facial expressions (Montagne et al., 2006). Finally, other (Heuer et al., 2010), using a similar experiments, failed to detect a significant difference between high socially anxious individuals and non-anxious controls across three types of facial expressions (angry, happy, and disgust) both with and without time pressure. Methodological differences (e.g., differences in the emotions and faces displayed) may in part explain this discrepancy. However, additional research in SAD is needed to clarify this issue.

GAD, in contrast with the social nature of SAD, involves a pattern of persistent worry and anxiety over a variety of day-to-day matters and is associated with somatic symptoms including restlessness, fatigue, concentration impairment, irritability, muscle tension, and sleep disturbance (APA, 2013). Accumulating evidence has more recently implicated emotion-regulation difficulties in the pathogenesis of GAD. For instance, individuals with GAD have been found to experience reduced tolerance of uncertainty (Dugas, Buhr, & Ladouceur, 2004), impaired ability to accept aversive psychological experiences (Roemer, Salters, Raffa, & Orsillo, 2005), and heightened experience of emotions coupled with greater difficulty modulating their emotional experience (Mennin, McLaughlin, & Flanagan, 2009).

In addition, individuals with GAD have been suggested to present both a greater perceived intensity of emotional experiences, and a difficulty identifying primary emotions including anger, sadness, or joy (e.g., Newman, Llera, Erickson, Przeworski, & Castonguay, 2013). It is thus possible that difficulty in perceiving the emotions in GAD extends to the emotion recognition of other’s expressions.

Despite distinct clinical presentations, some cross-sectional and longitudinal comorbidity data suggest that GAD and SAD might reflect alternative presentations of a single underlying psychopathology (Bruce, Machan, Dyck, & Keller, 2001). Prior research has also suggested that anxiety may be associated with specific emotion recognition alterations (Easter et al., 2005). On the other hand, individuals with trait anxiety have been suggested to recognise the facial expression of fear better than controls (Surcinelli, Codispoti, Montebonacci, Rossi, & Baldaro, 2006). Similar to SAD, individuals with GAD have finally been shown to exhibit greater vigilance for threatening faces relative to neutral faces, compared to normal controls (Bradley, Mogg, White, Groom, & de Bono, 1999).
To date, however, no study has examined performance of detection of emotional expressions in GAD or compared GAD to SAD. If both GAD and SAD shared an underlying anxiety diathesis leading to heightened emotion sensitivity, they should both be associated with detection of emotional expressions at lower intensity of expressions, compared to controls. On the other hand, if heightened sensitivity to facial emotional expression is driven by the social fears inherent in SAD, individuals with SAD should detect emotions earlier than individuals with GAD. Alternatively, if heightened sensitivity to facial emotional expression is driven by increased perceived intensity of emotional experiences reported in GAD, then GAD would be associated with enhanced emotion detection, while among those with SAD, a greater internal focus or emotional avoidance would result in reduced attention and later detection of emotions.

The present study aims to compare the detection of emotional expression between individuals with a primary diagnosis of SAD, GAD, and controls. We hypothesised that compared to controls, individuals with SAD and GAD would exhibit earlier detection of angry (threatening) faces (relative to sad and happy).

Methods

Participants

Participants were 116 individuals with a primary anxiety disorder (n = 70 with SAD, n = 46 with GAD) and 118 healthy controls, aged 18 or older, recruited through advertisement and clinical referral to the Massachusetts General Hospital. Mean (SD) age for the overall sample was 38.45 (13.32) years, and 75% (n = 128) were men (Table 1). Psychiatric diagnoses were determined by clinical interviewers certified in administering the Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 1994). Exclusions for SAD and GAD participants included lifetime psychosis, bipolar disorder, mental disorder due to a medical condition or substance, current eating disorders, and alcohol or substance use disorders within the past 6 months. Controls could have no DSM-IV Axis I disorders, with the exception of a past history of alcohol or substance use disorders in remission for at least 12 months (n = 18; 15%). Fourteen percents (n = 10) of those with primary SAD (n = 70) had a secondary GAD, while 29% (n = 13) of those with primary GAD (n = 46) had secondary SAD comorbidity. Current major depressive episode was present among 14% of participants with SAD and among 9% of those with GAD.

Procedures

All participants completed written informed consent prior to participation, and all study procedures were approved by the partners Institutional Review Board. Participants were then assessed for psychiatric diagnoses and eligibility before completing the morph movies experiment. They also completed self-report scales assessing their current affective state and psychiatric symptoms before completing the tasks. The morph movies task followed a binocular rivalry task, the results of which have been previously reported (Anderson et al., 2013). Participants received $30 financial compensation for participation.

Materials

Morph movies task

Detection of emotional expressions was assessed using a morph movies task previously described in detail (Niedenthal, Halberstadt, Margolin, & Innes-Ker, 2000). Briefly, for this task, photographs of actor/models displaying emotions were digitally morphed, with a photograph of the same actor expressing neutral emotion to create 100-frame movies in which a face initially expressing an emotion became gradually neutral. Pictures of 10 happy, sad, angry, and neutral faces of male and female actor–models were selected for this task. These images are part of a larger set of photographs pre-tested on a group of 83 participants who established the validity of the actors’ facial expressions (e.g., Niedenthal et al., 2000). Morph software (Maxwell, 1994) was used to map a set of anchor points onto an image of an actor with a neutral expression onto the same actor expressing an emotion. Digital movies composed of 100 facial composites, in which the facial expression gradually became more emotional, were then produced.

Participants were told they would view movies in which a face initially expressed neutral emotion. They were instructed to slide a bar at the bottom of the screen from left to right, playing each movie at their own speed, and to stop the movie at the first frame where they perceived the face to express an emotion. Participants were also able to slide the bar right to left to decrease emotional intensity. No verbal labels were provided about which emotion they should expect to see. Thus, the principal judgments in this
task were non-verbal. Participants clicked the mouse on a button at the bottom of the computer screen to register the frame of expression onset, and a new trial began with a neutral expression. The dependent variable was the frame at which the participant marked the initial facial expression as first visible (frame 1 = fully "neutral" expression; frame 99 = fully "emotional" expression). Each movie was presented twice and in a random order to each participant (i.e., 20 neutral-to-angry movie trials, 20 neutral-to-happy movie trials, and 20 neutral-to-sad movie trials).

Psychiatric assessment
The Structured Clinical Interview for DSM Disorders (First et al., 1994), the most widely used diagnostic instrument for assessing psychiatric disorders, was administered by doctoral-level clinicians to confirm that excluded disorders are not present and to document psychiatric and diagnostic history.

Depressive symptoms. The 10-item clinician-administered Montgomery–Asberg Depression Rating Scale (MADRS) is a psychometrically sound clinician-administered measure of depression (Montgomery & Asberg, 1979). Total scores range from 0 to 60 with higher scores reflecting greater depressive symptom severity. The MADRS has been found to be a valid and reliable interview for assessing current depressive symptom severity with high internal consistency (Cronbach’s coefficient alpha in our sample was 0.89), and excellent convergent validity with other measures of depressive symptoms. Here, the MADRS is used as a covariate.

Data analyses
A series of analyses of variance (ANOVAs) with post hoc Bonferroni tests and chi-square tests were conducted to examine group differences in socio-demographic and clinical variables. In order to examine differences in detection of emotions in SAD vs. GAD vs. control participants, we conducted a mixed-model repeated measures ANOVA with expression (angry, happy, and sad) as the repeated measure, diagnosis as the between subjects factor, and the frame at which the movie was stopped as the dependent variable. Simple effects were also examined for each emotional facial expression (angry, happy, and sad) with ANOVA with post hoc Bonferroni tests. As a follow-up analysis, in order to confirm the effect of GAD on the frame selection to detect emotion, we conducted a multiple regression analysis with the frame at which the emotion was detected as the outcome, and the GAD and SAD diagnoses as predictors, controlling for age, race, and depressive symptom severity. All analyses were performed using Stata 12.1, and the alpha level of significance was set to 0.05 (two-tailed).

Results
Participants’ characteristics
Participants’ characteristics are reported in Table 1. There was an approximately equal balance of sex across the three groups; however, the three groups differed significantly on age (p = 0.002) and race (p = 0.007).
Group differences in detection of emotional expression

Overall, the frame at which participants stopped the movie was normally distributed, although there were a number of trials in which participants stopped the movie on the first frame (indicating that they saw an emotional expression in the “neutral” face; less than 0.05% of trials) or final frame (maximum expression; 2.86% of trials). Although there was a statistically significant group difference in the number of trials stopped at the first or final frame (2.9% in controls, 3.76% in participants with SAD, and 1.56% in participants with GAD, \( \chi^2(2) = 28.78, p < 0.001 \)), the same overall pattern of findings held when analyses excluded trials that were stopped on the first or last frame. Thus, the data reported here include all trials. Participants detected affective expressions differently depending on their diagnosis (Table 1 and Figure 1).

We conducted a mixed-model repeated measures ANOVA, with expression (angry, happy, and sad) as the repeated measure, diagnosis as the between subjects factor, and the frame at which the movie was stopped as the dependent variable. Because sphericity assumptions were not met (Mauchly’s \( W = 0.658; p < 0.001 \)), Greenhouse–Geisser correction was applied. After inclusion of age, race, and depressive symptom severity as covariates, both the main effect of diagnosis, \( F(2, 402) = 3.0, p < 0.05 \), and the main effect of expression in the movie, \( F(2, 402) = 623.5, p < 0.001 \) were significant, while the interaction between the two factors was not, \( F(4, 402) = 1.65, p = 0.18 \). Bonferroni post hoc tests revealed that all groups required the smaller number of frames to detect happy expressions, followed by angry expressions, and the greatest number of frames to detect for sad expressions (Table 1). The GAD group also detected faces at a lower frame than SAD (\( p = 0.002 \)) and controls (\( p = 0.039 \)) (Table 1 and Figure 1).

Association of GAD diagnoses with detection of emotional expression

A multiple regression analysis (\( F(5, 606) = 4.99, p < 0.001 \)) revealed that lower frame at which the emotion was detected was only independently associated with age (\( B = -0.11, SE = 0.04, p < 0.05 \)) and GAD diagnosis (\( B = -5.73, SE = 1.74, p < 0.01 \)).

Discussion

This study is the first to compare performance in the detection of emotional expression across two anxiety disorders. Furthermore, to our knowledge, no prior studies have examined performance in the detection of emotional expression among individuals with GAD. Participants with a primary diagnosis of GAD required lower intensity of emotion to detect the onset of facial expressions compared to participants with SAD or controls. These group differences were not accounted for by depression symptom severity.

While some have proposed that GAD and SAD might reflect a common underlying pathology (Bruce et al., 2001; Pine, Cohen, Gurley, Brook, & Ma, 1998), our results suggest that distinct underlying pathophysiological processes regarding emotion detection may be present. Our findings of enhanced detection of emotion in GAD compared to SAD are consistent with recent research showing differences in emotion dysregulation between the disorders. For instance, individuals with GAD appear to experience their emotions more intensely than those with SAD (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Similarly, Blair et al. (2008) found in a functional neuroimaging study different patterns of cerebral activation in response to neutral, fearful, and angry facial expressions between individuals with GAD and SAD. However, their results showed that participants with GAD had a lower level of neural activation in response to the presentation of negative faces (relative to neutral faces) compared to those with SAD. Although seemingly inconsistent, these findings may be explained by contemporary models of GAD which suggest that individuals with GAD both experience heightened negative affect and employ affect-suppressing strategies (i.e., worry), which may explain...
their increased sensitivity to emotionally salient facial expressions but reduced neural activation (Mennin, Heimberg, Turk, & Fresco, 2002; Turk et al., 2005). Future research should examine how different contexts and stimuli impact emotion-regulation strategies used by those with GAD.

Another point to be considered regarding our current findings is that the results may have been impacted by the lack of time pressure in the task. Since the task was not time-pressured and participants could go back and forth through the movie, responses could have stemmed not from early perceptual processes but from late cognitive/response processes. These late processes could be influenced by various factors that may drive GAD participants to select a lower intensity of emotion including more intense feeling of emotions. Individuals with GAD have also been suggested to exhibit a greater perceived intensity of emotional experiences (e.g., Newman et al., 2013). It is possible, for example, that participants with GAD selected a frame with less emotion intensity as a strategy to avoid heightened feeling of emotions. The increased amount of time allowed for decision making exposed participants to an increasingly ambiguous situation without feedback. Prior research suggests that individuals with GAD may exhibit low tolerance of uncertainty (Dugas et al., 2004). Although germane to all anxiety disorders (McEvoy & Mahoney, 2012), intolerance of uncertainty is particularly pronounced among individuals with GAD relative to those with SAD (Boswell, Thompson-Hollands, Farchione, & Barlow, 2013). Thus, while the nature of worry and anxiety inherent in GAD may have led to detection of emotional faces at lower frame than for SAD or controls, an alternate explanation is that the untimed nature of the task, which allowed individuals to scroll back and forth through the movie, enabled individuals with GAD to be more careful in their assessments and/or greater concern about missed detections led them to report detection with earlier frames. Further studies identifying the specific factors and processes that may explain the unique response strategies used by participants with GAD are thus warranted.

Contrary to our hypotheses, the detection of emotional expression also did not differ between individuals with SAD and controls. These results are consistent with the findings of Heuer et al. (2010) and Justye and Schonenberg (2014), but at odds with another study (Joormann & Gotlib, 2006) that found "earlier" detection of angry faces among SAD compared to controls in a time-pressured morph movie task. One possibility is that individuals with SAD are more sensitive to detecting angry facial expressions only under time pressure. As previously suggested (Staugaard, 2010), longer exposure durations to the emotionally salient faces may allow for more detailed processing of the stimuli, thereby diminishing differences between individuals with SAD and controls. In other words, because the affective reaction elicited by the presentation of emotional faces may be short-lived, conscious processing overrides automatic processes (Staugaard, 2010). On the other hand, consistent with our findings, Heuer et al. (2010) also failed to observe any differences in emotion detection regardless of time pressure. Additional studies are needed to clarify the role of time pressure in processing emotional expressions.

It is worth noting that happy expressions were detected earlier than angry and sad across all groups. Prior literature has also reported that individuals required a lower intensity of emotion to identify happy expressions compared to other expressions including sadness (Joormann & Gotlib, 2006; Schonenberg et al., 2014). As suggested previously (Barrett & Niedenthal, 2004), such findings may reflect the differences in the intensity of the end-point emotional expressions displayed across emotion type. Because movie frames are morphed composites of a neutral and an end-point expression each happy, angry, and sad morphed movie contains differing amounts of emotional information at a given frame. An alternative explanation may be that the happy expression was easier to detect since it was the only positive emotion. The other two (angry and sad) were negative and similar to each other. It is possible that participants did not respond to the detection of a change but to the detection of a change that enabled them to differentiate between these two emotions.

Some limitations need to be acknowledged. First, anxiety comorbidities were allowed in the current study and may have limited the study’s internal validity for cross-disorder comparisons. Although the presence of some secondary GAD in the SAD group and vice versa would be more likely to diminish rather than enhance group differences. In addition, regression analyses confirmed the association of GAD with emotion detection at a lower frame. In addition, our design did not allow differentiation of sensitivity to emotion or more simple differences in facial information, and we cannot rule out that participants may have rated visual changes in general (i.e.,
merely detected facial contrasts). Similarly, as indicated above, the design also did not allow for differentiation between perceptual processes and later processes related to criterion (expectancy, interpretation). Finally, we did not assess intelligence quotient.

Despite these limitations, our findings provide preliminary evidence that GAD might be associated with lower threshold for detection of facial emotions. Although these results warrant replication, they provide evidence in favour of distinct pathophysiological processes between individuals with GAD and SAD. Our findings thus point to the importance of incorporating disorder-specific treatment strategies in the treatment of GAD including emotion-regulation training and decreased reliance on detrimental strategies (e.g., worry, avoidance). Future research aiming to identify the specific factors and processes explaining the unique response strategies used by participants with GAD are warranted.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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