The Spectral Nature of Anxiety Disorders: Examining Similarities in Clinical and Subclinical Populations

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Abstract

Anxiety disorders are currently the leading mental illness in the United States. Generalized anxiety disorder (GAD) is characterized by both intense cognitive abnormalities, such as worry and intolerance of uncertainty, and physiological responses, such as a tight chest or increased heart rate. Although the diagnosis of GAD is becoming more prevalent, if the diagnostic criteria found in the Diagnostic and Statistical Manual-V (DSM-V) were relaxed, the diagnoses of GAD would approximately double. There is a large subclinical population of individuals who have anxiety symptoms that disrupt daily functioning but may not meet every diagnostic criterion, therefore never warranting a clinical diagnosis. The current paper reviews literature suggesting that individuals with clinical or subclinical levels of GAD display abnormal neural circuitry, suggesting that the two groups are more alike than they are different.

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Anxiety disorders are currently the leading mental illnesses in the United States, where 33% of the population are diagnosed with an anxiety disorder at some point during their lifetime (Bandelow & Michaelis, 2015). Anxiety is currently defined by a variety of emotional and personality traits such as worry, fear, stress, expecting negative outcomes, and rumination over perceived severity of negative outcomes (Donnellan, Moran, Moser, Schroder, & Yeung, 2013; Gu, Yu-Xia, & Yue-Jia, 2010). Anxiety disorders can be debilitating as worry can sap time and energy, and somatic symptoms detract from daily activities (Alvarez et al., 2012; American Psychiatric Association [APA], 2013; Arkin, Cornwell, Grillion, & Vytal, 2012). In the current *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5), there are 12 different diagnoses listed under the general term of anxiety disorder (APA, 2013; Banich et al., 2016). These diagnoses include social anxiety disorder, panic disorder, agoraphobia, and generalized anxiety disorder (GAD). Generalized anxiety disorder is currently one of the most prevalent anxiety disorders in the United States (Alvarez et al., 2012). Due to the prevalence of GAD, it is essential to understand the etiology and maintenance of the disorder in order to adequately treat the various symptoms that disrupt daily life.

In order to receive a diagnosis of GAD, an individual must have experienced worry over a period of at least six months, accompanied with feelings of inability to control the worry (APA, 2013). Additionally, the individual must have also experienced various somatic symptoms (i.e., increased heart rate, muscle tension) associated with the anxiety (Alvarez et al., 2012; APA, 2013). Although the diagnosis of GAD is prevalent, the criteria found in the DSM-V is especially stringent and may not capture every individual who is experiencing anxiety symptoms that disrupt his or her daily functioning (Chiu et al., 2007; Cramer, Dobos, Gass, Haller, & Lauche, 2014; Wittchen, 2002). Many individuals still experience the symptoms of GAD but do not meet the diagnostic criteria for an official diagnosis.

If the criteria for GAD found in the DSM were to relax, such as the length of time over which worry must be experienced

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before receiving a clinical diagnosis, the prevalence of GAD would approximately double (Chiu et al., 2007; Cramer et al., 2014). These individuals have anxiety at a subclinical level because their symptoms are neither severe enough nor long enough to warrant a formal clinical diagnosis, but they are still experiencing a disruption of daily functioning. It is important to separate individuals who have anxiety-like symptoms for a short amount of time due to stressful life events from people who have chronic bouts of anxiety symptoms (Abe et al., 2018; Chiu et al., 2007). However, those who have chronic bouts of continuous anxiety symptoms can either be individuals who constantly have anxiety (i.e. individuals diagnosed with GAD) or individuals who have anxiety that comes and goes for extended periods of time (i.e. individuals with subclinical generalized anxiety).

Previous literature has often focused on either healthy subjects or clinical populations, therefore ignoring the spectral nature of many psychopathologies including GAD (Besteher, Dietzek, Gaser, Langbein, & Sauer, 2017). Because anxiety disorders are spectral in nature, it has become increasingly important to understand a range of symptomatology through individuals with a clinical diagnosis and those without in order to understand the true etiology of the disorder (Besteher et al., 2017; Dannlowski et al., 2012). If research can parse out individual causes for anxious traits in psychiatrically healthy subclinical groups, understanding GAD populations may become easier as these two populations share characteristic difficulties (Chiu et al., 2007). As individuals with subclinical anxiety and individuals with diagnosed GAD may share core symptomatology, more fully understanding the subclinical populations will also lead to a better understanding of individuals diagnosed with GAD.

One symptom present in both subclinical generalized anxiety and GAD is altered brain functioning. One example of altered brain functioning is error monitoring. Error monitoring, or the ability to detect and react to mistakes, is essential for adapting to one's surroundings in an appropriate manner (Dieterich, Riesel, & Weinberg, 2015). For example, if one is playing a computer

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game and accidentally hits the wrong button, his or her ability to detect the error would aid them in not making the same mistake in the future. Error monitoring is enhanced in individuals with generalized anxiety disorder (Aarts & Pourtois, 2010), suggesting a heightened sensitivity to errors and threats. This enhancement of error monitoring is unproductive, as time is spent worrying excessively about errors.

Although not formally diagnosed with GAD, individuals in subclinical populations exhibit differential personality traits, brain functioning, and altered error monitoring when compared to healthy controls. Therefore, individuals with subclinical anxiety may require more support than is currently offered due to the lack of duration of their symptoms, which lie outside the qualifications for a full anxiety diagnosis according to the DSM-V. This literature review will examine the research available concerning individuals with subclinical levels of anxiety in order to better understand the neural mechanisms present in this population by examining (1) trait anxiety and (2) how trait anxiety differentiates brain functioning and error monitoring.

Trait Anxiety in the Subclinical Population

Trait anxiety as currently defined is a set of personality characteristics in healthy individuals that predisposes them to anxiety disorders and other pathologies (Fu et al., 2016). Individuals high on the trait anxiety scale have a difficult time tolerating any surprising outcomes and allowing uncertainty in the future; they often ruminate on the idea of failure (Compton et al., 2010; Song & Li, 2017). Further, when faced with stressful situations, individuals with high trait anxiety are more prone to anxiety-like responses, such as hypervigilance (Fu et al., 2016; Sandi & Wegner, 2018). However, due to the spectral nature of trait anxiety, individuals high on the trait anxiety scale may never be diagnosed with anxiety or other psychopathologies (Fu et al., 2016). Trait anxiety in subclinical and clinical groups differs in duration and severity. As previously stated, in order to be diagnosed with GAD the high levels of worry must last over six months. However, those with trait

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anxiety may have periods of high anxiety but may not reach the stringent threshold of a continuous six months. This lends credence to the argument that there is a large spectrum of individuals who have subclinical anxiety but still experience anxiety symptoms for significant periods of time.

Trait anxiety can further be broken down into two subtypes: anxious apprehension and anxious arousal. In anxious apprehension, worry and introspection are frequent, but there is also an external focus in order to detect and react to threats in the environment (Banich et al., 2016; Calamia et al., 2017; Donnellan et al., 2013). The consistent worry and rumination found in anxious apprehension is thought to be one of the reasons that GAD may last over a long period of time (Donnellan et al., 2013; Heller, Miller, Nitschke, & Palmieri, 1999; Hermann, Kress, Neudert, & Stark, 2017). Individuals high on the anxious apprehension scale experience constant worry over benign future events and any ambiguity (Donnellan et al., 2013; Heller et al., 1999). In contrast, individuals with anxious arousal often experience hyper physiological symptoms, such as increased error-related negativity amplitude in the presence of threats (Donnellan et al., 2013). Although anxious arousal and anxious apprehension may be seen in the same individual, these two dimensions of anxiety have very different neurological functioning (Gu et al., 2010). The dimension of anxious apprehension or the tendency to worry excessively is found mainly in GAD and individuals with high trait anxiety. Trait anxiety in GAD has been linked to altered brain structure and functioning, as emotional centers of the brain may not be working properly in individuals who are high on the trait anxiety scale, which may cause excessive worry.

Brain Structure and Functioning

Differential brain structure and functioning has been found in a variety of psychopathologies, including GAD (Besteher et al., 2017). In many situations, it is unclear whether psychopathology leads to a change in brain functioning or if differential brain structure and functioning lends way to psychopathologies.

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In any case, it is important to understand brain structure and functioning and how it differs in psychopathologies and in subclinical populations with anxiety. The brain structures most closely associated with anxiety are the amygdala as an emotional processing center and the prefrontal cortex as center of control for higher emotional processing (Barr et al., 2011; Binder, Martin, Nemeroff, & Ressler, 2009; Dannlowski et al., 2012). These two brain regions play a role in the processing of threats and worry in both individuals with GAD and subclinical anxiety.

Amygdala

The amygdala is a small structure in the brain that is thought to be a general emotional processing center and more specifically involved with the negative emotions of fear and anxiety (Barr et al., 2011; Dannlowski et al., 2012). The amygdala is part of the larger limbic system that includes multiple structures, such as the prefrontal cortex. Much like the larger limbic system, the prefrontal cortex also has essential roles in emotional monitoring. A characteristic trait of GAD is differential, and possibly enhanced, emotional processing as seen by abnormal amygdala activity (Beesdo-Baum, Hilbert, & Leuken, 2014; Binder et al., 2009; Calamia et al., 2017; Davidson et al., 2009). In people high on the trait-anxiety scale, higher levels of worry and rumination show reduced amygdala gray matter but increased amygdala activation (Barr et al., 2011; Sandi & Wegner, 2018). Overall, higher levels of worry and rumination may be associated with increased amygdala activity in spite of reduced volume. This also suggests that hyperactive emotional processing centers may lead to excessive worry and hyper attentiveness to threats in even subclinical populations.

Although the amygdala by itself has been implicated in both GAD and subclinical populations, there are also connectivity differences between the amygdala and prefrontal cortex in individuals with high trait anxiety, whether formally diagnosed with anxiety or not (Beesdo-Baum et al., 2014; Dannlowski et

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al., 2012). Individuals with high trait anxiety showed greater connectivity with the amygdala and prefrontal cortex when compared to individuals with low trait anxiety (Barr et al., 2011), suggesting that when the amygdala is overactive, the prefrontal cortex is also overactive due to the hyper connectivity between the two areas.

The differences in the amygdala's structure, functioning, and connectivity may play a key role in the appearance and maintenance of anxiety symptoms in subclinical populations. Again, it is difficult to infer the direction of causation in this situation, but just understanding the differences can aid in a more holistic view of the neural mechanisms of subclinical anxiety.

Prefrontal Cortex

The prefrontal cortex on its own is another brain region implicated in both clinical and subclinical anxiety. It is thought to be involved in executive functioning, including social and moral behaviors and higher emotional processing (Anderson, Bechara, Damasio, Damasio, & Tranel, 1999). The prefrontal cortex has also been found to show elevated activity in individuals diagnosed with GAD, although this is suggested to be a response to anxiety symptoms instead of an underlying cause (Binder et al., 2009). As previously mentioned, greater connectivity between the amygdala and prefrontal cortex has been observed in a subclinical anxiety population. Additionally, greater prefrontal cortex activation has been seen in high trait anxiety individuals with a stronger response for words that carried negative emotional valence (Dannlowski et al., 2012). Abnormal prefrontal activity has also been linked with hyperactivity of the limbic regions, which may suggest that the prefrontal cortex is not inhibiting the emotional centers of the brain as well as it should be (Dannlowski et al., 2012). Abnormal prefrontal cortex activity and hyperactivity of the limbic regions may suggest that the prefrontal cortex is a hyperactive brain region that may contribute to the states of worry and distress over possible negative outcomes.

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Error Monitoring in GAD

Error monitoring is a process through which individuals detect errors, and subsequently correct behavior, in order to improve in the future (Donnellan et al., 2013). Individuals with GAD are hypervigilant in their error monitoring (Hajcak, McDonald & Simons, 2003; Hajcak, Olvet, & Weinberg, 2010), therefore manifesting as a heightened awareness and sensitivity to errors. Even at subclinical levels, anxiety symptoms are associated with enhanced error monitoring (Aarts & Pourtois, 2010; Chan, Ng, & Schlaghecken, 2012). Especially for individuals with high trait anxiety, errors are particularly aversive and great lengths are taken to avoid committing errors (Inzlicht, Mennin, & Proudfit, 2013).

As error monitoring may be hyperactive in individuals with GAD when compared to psychiatrically healthy controls, understanding the neural mechanisms of error monitoring in GAD becomes of interest. The error-related negativity (ERN) is an event-related brain potential derived from electroencephalogram (EEG) data (Clawson, Clayson, & Larson, 2014) that is often used to quantify neural responses to errors. More specifically, the ERN is a negative deflection in the amplitude of recorded brain potentials thought to be the neural marker of error detection and correction (Bernstein, Coles, & Scheffers, 1995; Clawson et al., 2014). The ERN is thought to represent error detection as the negative peak is consistently higher (i.e. more negative) when an individual gives an erroneous response versus when a correct response is made during various computerized tasks (Clawson et al., 2014). The ERN is thought to originate from the anterior cingulate cortex (ACC) (Clawson et al., 2014), a brain region that has been suggested to deal with higher emotional behavior and error detection (Hajcak et al., 2014). Enhanced error monitoring, as indexed by the ERN, has been seen in both subclinical populations and populations diagnosed with GAD (Hajcak et al., 2014), suggesting a mutual enhancement of error monitoring neural processes.

In a subclinical population, the ERN is related to characteristic worry and enhanced error monitoring, and therefore high trait anxiety individuals have a more negative (or increased) ERN when

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compared to controls (Bennett, Glazer, Moran, Moser, & Schroder, 2017; Collins, Luu, & Tucker, 2000; Hajcak et al., 2014; Hajcak & Olvet, 2008; Lin, Moran, Moser, & Schroder, 2015). The ERN in both clinical and subclinical generalized anxiety is thought to be specific to the anxious apprehension subtype of anxiety when examined in low and high trait anxiety individuals (Donnellan et al., 2013). This specificity of the ERN only to anxious apprehension suggests that the consistent worry found in subclinical populations may be related to an increase in worry over future errors.

Although error monitoring seems to be enhanced in individuals high on the trait anxiety scale, performance on cognitive tasks is not impaired in these individuals (Donnellan et al., 2013). Anxiety as a disorder does not impair effectiveness but instead may impair the efficiency of cognitive processing (Basten, Fiebach, & Stelzel, 2011; Calvo, Derakshan, Eysenck, & Santos, 2007). Therefore, individuals high on the trait anxiety scale may have to employ more cognitive resources in order to achieve the same performance as their peers (Inzlicht et al., 2013; Lin et al., 2015). Accordingly, as a task becomes increasingly demanding on cognitive functioning, it becomes more difficult for individuals with high trait anxiety to compensate effectively (Calvo et al., 2007; Arkin et al., 2012). It is after this significant increase in task difficulty that performance, as measured by accuracy, begins to decline in individuals with anxiety symptoms. This phenomenon is thought to be due to the distracting nature of worry and rumination as these thoughts take up cognitive resources that could be used instead to monitor errors (Ansar & Derakshan, 2011; Basten et al., 2011; Bishop, 2009). The increased need for cognitive control in order to perform as well as peers is cognitively demanding and therefore often takes a large toll on the individual.

Whether at clinical or subclinical levels, anxiety may affect cognitive functioning, including error monitoring (Beutel et al., 2018; Chan et al., 2012). Although subclinical individuals are not diagnosed with GAD, this enhanced error monitoring may lead to a constant state of worry over errors or negative outcomes.

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Conclusion

Individuals with subclinical anxiety experience daily disruption and dysfunction from their anxiety-specific symptoms. However, these individuals are often overlooked because they do not reach the clinical threshold for GAD. Better understanding the neural mechanisms related to subclinical populations and how those neural mechanisms relate to daily functioning will aid a population that for so long has been forgotten.

Future research should investigate additional similarities between subclinical anxiety and GAD to inform future versions of the diagnostic criteria and treatment options. Other avenues of future research could determine the neural correlates of personality traits, such as worry, that may relate to both subclinical and clinical GAD. Overall, understanding subclinical populations of anxiety will aid in understanding a variety of psychopathologies and could potentially improve the lives of many individuals, both those who are diagnosed and those who are not.

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