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### Review

# Neuroticism and the brain: A quantitative meta-analysis of neuroimaging studies investigating emotion processing

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### ABSTRACT

Neuroticism is a robust personality trait that constitutes a risk factor for mood disorders. Neuroimaging findings related to neuroticism have been inconsistent across studies and hardly integrated in order to construct a model of the underlying neural correlates of neuroticism. The aim of the current meta-analysis was to provide a quantitative summary of the literature, using a parametric coordinate-based meta-analysis (PCM) approach. Data were pooled for emotion processing tasks investigating the contrasts (negative > neutral) and (positive > neutral) to identify brain regions that are consistently associated with neuroticism across studies. Significant negative and positive correlations with neuroticism were found only for the contrast (negative > neutral) after multiple comparisons correction. Differences in brain activation were found to be associated with neuroticism during fear learning, anticipation of aversive stimuli and the processing and regulation of emotion. The relationship between neuroticism and these three psychological processes and their corresponding neural correlates is discussed. Furthermore, the meta-analytic findings are incorporated into a general model of emotion processing in neuroticism.

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## 1. Introduction

Neuroticism is one of the Big Five dimensions of personality, alongside extraversion, conscientiousness, openness and agreeableness (Digman, 1990). Historically, these dimensions originated from the lexical approach in which clusters of personality descriptors were identified by performing factor analyses on personality related adjectives. The underlying assumption was that relevant personality differences become encoded in the natural language (Goldberg, 1990). Subsequent studies have consistently replicated neuroticism as a robust trait and currently, it is a fundamental part of various widely accepted taxonomies of personality (Costa and McCrae, 1989, 1992; Eysenck, 1967; Gray, 1982, 1991; McCrae and Costa, 1997; McCrae et al., 1999). High neurotic individuals express heightened emotional reactivity, especially to negative events (Canli, 2008) and experience more negative emotions, such as anxiety, depression, shame, embarrassment and guilt (Watson et al., 1994). Generally, these individuals have a negative perspective on daily life and tend to appraise events as more threatening than others. Hence, they report elevated levels of stress and regularly experience mood spillovers (Suls and Martin, 2005). In addition, high trait scorers rely on maladaptive coping strategies, such as worry and inefficient escape-avoidance strategies (Lahey, 2009; Watson et al., 1994).

Neuroticism can be defined as a general risk factor for psychopathology and it has been shown to predict a variety of disorders, specifically internalizing disorders (e.g. major depressive disorder, generalized anxiety disorder, and social phobia) but also personality disorders, schizophrenia, eating disorders, somatoform disorders and to a lesser extent, externalizing disorders and specific phobia (Kotov et al., 2010; Lahey, 2009; Ormel et al., 2004, 2013). Furthermore, neuroticism has been related to higher levels of psychiatric comorbidity, an increased risk for committing suicide and general health problems, including cardiovascular disease and disrupted immune functioning (Lahey, 2009). Indeed, Cuijpers et al. (2010) have demonstrated that the economic costs (e.g. health service and production losses) of neuroticism exceed those of common mental disorders.

Thus, neuroticism is a clinically relevant concept and it is important to identify and map its underlying neurobiological correlates. The first studies on the neural basis of neuroticism mainly used electrophysiological methods and were based on two influential neuropsychological theories of personality (see for an overview Ormel et al., 2013). First, Eysenck's theory (1967) relates individual differences in neuroticism to lower activation thresholds in the viscerocortical loop. This loop connects the cerebral cortex with the visceral brain, including the limbic system and is hypothesized to control subjective and autonomic emotional responses. Hence, it was postulated that high trait scorers are more likely to become autonomically aroused in the face of minor stressors compared to low trait scorers and because of this, experience more negative emotions (Eysenck, 1967; Matthews and Deary, 1998). Second, Gray's theory (1982, 1991) proposes five systems of which two are particularly important for personality: the behavioral inhibition

system (BIS) and behavioral activation system (BAS). The BIS system consists of frontal and limbic brain regions and is involved in the inhibition of responses, orientation of attention to potential sources of threat (e.g. punishment) and enhancement of arousal. The theory posits that this system is easily excited in individuals with an anxious personality, such as high neurotic individuals. The BAS system is related to reward and controls approach behavior (Gray, 1982, 1991; Matthews and Deary, 1998).

In later studies, attempts were made to find the neurobiological correlates of neuroticism by using neuroimaging techniques, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). Canli et al. (2001) were the first to perform an fMRI study to investigate whether neuroticism moderated brain activity to emotional stimuli. The authors found that neuroticism was associated with increased brain activation in left frontal and temporal cortical regions in response to negative emotional stimuli. Ever since, a variety of fMRI tasks have been applied to investigate the neural correlates of neuroticism, examining the following processes or phenomena: emotional face and scene processing (Britton et al., 2007; Canli et al., 2001; Chan et al., 2009; Cremers et al., 2010; Cunningham et al., 2011; Drabant et al., 2009; Haas et al., 2008; Harenski et al., 2009; Hyde et al., 2011; Jimura et al., 2009; Kehoe et al., 2012; Simmons et al., 2008), cued anticipation (Brühl et al., 2011; Coen et al., 2011; Kumari et al., 2007), the emotional Stroop-effect (Canli et al., 2004; Haas et al., 2007), emotional categorisation (Chan et al., 2008), observational fear and reward learning (Hooker et al., 2008), reward and loss processing (Fujiwara et al., 2008; Paulus et al., 2003), humor appreciation (Mobbs et al., 2005), emotional prosody (Brück et al., 2011), brand rating (Schaefer et al., 2011), theory of mind (Jimura et al., 2010), the dot-probe effect (Amin et al., 2004), the odd-ball effect (Eisenberger et al., 2005), (un)certainly processing (Feinstein et al., 2006) and working memory (Kumari et al., 2004). Furthermore, neuroticism has been associated with differences in brain structure, using cortical thickness and surface-based analysis (Bjørnebekk et al., 2013; Blankstein et al., 2009; Wright et al., 2006, 2007) and voxel-based morphometry (VBM) (Blankstein et al., 2009; Cremers et al., 2011; DeYoung et al., 2010; Hu et al., 2011; Kapogiannis et al., 2012; Omura et al., 2005; Taki et al., 2012). Most of these functional and structural neuroimaging studies focussed on limbic regions, such as the amygdala and hippocampus, as well as frontal regions, such as the anterior cingulate cortex (ACC) and medial prefrontal cortex (mPFC) (Canli, 2008). However, results regarding these areas have been largely inconsistent across studies. For example, some reports have shown that neuroticism is associated with increased activation in the amygdala (Brück et al., 2011; Chan et al., 2009; Cunningham et al., 2011; Haas et al., 2007; Harenski et al., 2009; Hooker et al., 2008), while others have revealed no such relationship (Cremers et al., 2011, 2010; Drabant et al., 2009; Haas et al., 2008; Hyde et al., 2011; Mobbs et al., 2005; Thomas et al., 2011).

In addition, neuroticism has been related to alterations in brain connectivity, using fMRI (Cremers et al., 2010), resting state fMRI (rs-fMRI) (Adelstein et al., 2011) and diffusion tensor imaging (DTI) (Bjørnebekk et al., 2013; Xu and Potenza, 2012). These connectivity

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