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Review

The biological and psychological basis of neuroticism: Current status and future directions

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ABSTRACT

Neuroticism (N) is believed to reflect a stable disposition involving specific biological and psychological mechanisms that produce its robust association with psychopathology. The nature of these mechanisms remains unclear, however. Based on an extensive review of published evidence, we argue that three interesting leads are emerging. First, N may reflect individual differences in brain circuits involved in perception of and cognitive control over negative stimuli. More specifically, reduced connectivity between the left amygdala and ACC may impair extinction of the amygdala response to anxiety-eliciting stimuli. Second, the neural evidence matches the psychological findings, which associate N with a negative bias in attention, interpretation and recall of information, increased reactivity, and ineffective coping, and is consistent with findings of decreased cardiovascular flexibility. Third, current studies suggest that HPA-axis influences mood independently of N. Strong claims on N's biological basis, however, are not yet justified due to inconsistencies and lack of replication which are in part due to methodological limitations and N's heterogeneity. We discuss potential methodological improvements and substantive directions for future research.

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1. Introduction

This article reviews the evidence regarding the biological and psychological basis of the high-order personality trait Neuroticism (N). Although the term has its roots in Freudian theory, modern definitions of N are purely descriptive. Currently, N is in effect a label assigned by psychologists to a major factor that consistently emerged in factor analyses of people's response to verbal descriptors of inclinations and behaviors in daily life. This is known as the lexical paradigm of personality and is based on the assumption that language encodes what is of most importance, interest, or meaning to individuals (Goldberg et al., 1990; Matthews et al., 2003; Pervin and John, 1999). Self-report measures are the most common method of measuring N (John et al., 2008; Mathews et al., 2003; Pervin and John, 1999; Widiger et al., 1984). Objective behavioral tests have not been successful and are rarely used. Measures of N consist of items referring to negative affect, including anxiety, irritability, anger, worry, frustration, self-consciousness, sensitivity to criticism, reactivity, hostility, and vulnerability (Costa and McCrae, 1992; Eysenck and Eysenck, 1975; Ormel, 1983). Hence, N is widely defined as the tendency to experience negative affect, especially when threatened, frustrated, or facing loss.

N is the single most important factor associated with many forms of psychopathology and behavioral health, in particular the common mental disorders including anxiety, depressive, and substance use disorders (see for reviews Kotov et al., 2010; Lahey, 2009). The prospective associations between N and psychopathology have prompted many in the field to consider N a robust independent predictor of psychopathology (e.g., Fanous et al., 2007; Kendler and Prescott, 2006; Khan et al., 2005; Krueger et al., 1996; Lahey, 2009; Ormel and Wohlfarth, 1991; Ormel et al., 2001; van Os et al., 2001; Vink et al., 2009). However, some authors have raised concerns about the etiological significance of the association (Claridge and Davis, 2001; Duncan-Jones et al., 1990; Ormel et al., 2004b). First, measures of N and psychopathology, in particular anxiety and depressive disorders, overlap to a large extent and may thus partially reflect the same phenotype rather than a causal relationship. Second, it is unclear to what extent the prospective studies controlled fully for earlier episodes of mental disorder and (subclinical) psychiatric symptoms present at the time of the assessment of N. This is important because N is considerably increased during episodes of (subthreshold) psychopathology, and part of this state effect may persist after full remission of the episode (Kendler et al., 1993; Ormel et al., 2004a; Rohde et al., 1994). Finally, although N has been conceptualized as a stable personality characteristic, it has been shown that test–retest correlations steadily decrease with increasing time intervals (Ormel and Rijsdijk, 2000; Roberts and DelVecchio, 2000; Watson and Clark, 1984). Furthermore, its genetic and environmental sources overlap to a large extent with those of the common mental disorder, suggesting that N and psychopathology may both be outcomes with overlapping etiologies (Carey and DiLalla, 1994; Fergusson and Horwood, 2001; Hettema et al., 2006; Ormel et al., 2012; van Os and Jones, 1999). Thus, for N to become etiologically informative, we must clarify its basis.

The analysis of the biological and psychological bases of N described in this paper may help to clarify the construct, elucidate its relationship with psychopathology, and support development of specific etiological hypotheses for both N and CMDs. We identified relevant studies using Web of Science and Pubmed and included human studies on correlates of N in biological domains such as the central nervous system (CNS) autonomic nervous system (ANS), and hypothalamic–pituitary–adrenal (HPA) axis, and in psychological domains such as cognition, coping, and emotional processes. We used multiple keywords for each domain in our literature search that covered the past 40 years. We not only included studies on N; but also studies on 'negative affectivity'; which is a dimension of subjective dysphoria and unpleasurable engagement (Rothbart et al., 2000; Watson and Clark, 1984). These two concepts are used interchangeably in the literature because of consensus that individuals scoring high on N measures also exhibit negative affectivity (Shankman and Klein, 2003).

2. The biological basis of neuroticism

2.1. Methodological shifts in the studies on the biological basis

Studies on the basis of N have shown a gradual but persistent shift in both research focus and methodology. 'Early' studies focused on the question whether N is linked to physiological overresponsiveness using *global* measures of peripheral physiological indicators and CNS arousability. Gradually, the availability of neuroimaging techniques with better spatial resolution shifted the focus to N's link with emotional reactivity and emotion regulation, by opening up the possibility of studying *specific* brain regions and their interactions.

The first studies were strongly inspired by Eysenck's and Gray's influential theories, which postulate that N reflects excessive physiological responsiveness (or arousability) of certain brain systems, which predisposes individuals to psychopathology (Eysenck, 1967; Eysenck and Eysenck, 1985; Gray and McNaughton, 2000). Eysenck's theory links N to lower activation thresholds in the sympathetic nervous and limbic systems. The limbic system, which consists of the hippocampus, amygdala, septum, and hypothalamus, regulates emotional states such as fear, anxiety, and aggression. Higher activation levels and lower thresholds within the limbic system would explain why high-N individuals are more easily upset in the face of minor stressors, whereas low-N people

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