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Public Health Significance of Neuroticism

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Abstract

The personality trait of neuroticism refers to relatively stable tendencies to respond with negative emotions to threat, frustration, or loss. Individuals in the population vary markedly on this trait, ranging from frequent and intense emotional reactions to minor challenges to little emotional reaction even in the face of significant difficulties. Although not widely appreciated, there is growing evidence that neuroticism is a psychological trait of profound public health significance. Neuroticism is a robust correlate and predictor of many different mental and physical disorders, comorbidity among them, and the frequency of mental and general health service use. Indeed, neuroticism apparently is a predictor of the quality and longevity of our lives. Achieving a full understanding of the nature and origins of neuroticism, and the mechanisms through which neuroticism is linked to mental and physical disorders, should be a top priority for research. Knowing why neuroticism predicts such a wide variety of seemingly diverse outcomes should lead to improved understanding of commonalities among those outcomes and improved strategies for preventing them.

Keywords

Neuroticism; personality traits; public health; predictive utility

Personality traits quantify the marked variations in typical responding to the environment that distinguish one person from another (Mischel, 2004). Although personality traits have long been a topic of interest to psychologists, many of us are not aware of their broad and considerable importance to public health. The goal of this paper is to change that perception for one particularly important personality trait. Neuroticism refers to individual differences in negative emotional response to threat, frustration, or loss (Costa & McCrae, 1992a; Goldberg, 1993). Although the term neuroticism has its roots in Freudian theory, and the ancient philosophical and medical traditions on which psychodynamic models were based, the modern conception of neuroticism is unrelated to such theories of unconscious conflict. Rather neuroticism is usually defined today in purely descriptive psychometric terms.

Neuroticism is operationally defined by items referring to irritability, anger, sadness, anxiety, worry, hostility, self-consciousness, and vulnerability that have been found to be substantially correlated with one another in factor analyses (Costa & McCrae, 1992a; Goldberg, 1993). For individuals who are high on neuroticism, such negative emotional responses to challenges are both frequent and out of proportion to the circumstances (McCrae & Costa, 2003). In addition, persons high in neuroticism are often self-critical, sensitive to the criticism of others, and feel personally inadequate (Watson, Clark, & Harkness, 1994). The dimension of neuroticism, also

often referred to as negative emotionality or negative affectivity, is included in nearly all major models of personality traits (Matthews, Deary, & Whiteman, 2003), including three-factor (Eysenck, 1947; Tellegen, 1982) and five-factor models (Costa & McCrae, 1992b; Goldberg, 1993; Zuckerman, Kuhlman, Teta, Joireman, & Kraft, 1993).

Although the stability of neuroticism, in the sense of maintaining rank-order position relative to other persons, increases with age from adolescence through adulthood (Roberts & DelVecchio, 2000), mean neuroticism scores peak in late adolescence and decline moderately through adulthood (Costa et al., 1986; McCrae et al., 2002; Roberts & Mroczek, 2008). Mean neuroticism scores of females are slightly but significantly higher than for males (Costa, Terracciano, & McCrae, 2001) and neuroticism scores tend to be somewhat higher among individuals with lower socioeconomic status (Judge, Higgins, Thoresen, & Barrick, 1999). Therefore, it is informative that most studies of the associations between neuroticism and mental and physical health that controlled age, sex, and socioeconomic status have found that neuroticism is associated with mental and physical health independent of its correlation with these demographic factors (Kendler, Kuhn, & Prescott, 2004; Neeleman, Ormel, & Bijl, 2001; Stronks, van de Mheen, Looman, & Mackenbach, 1997).

Neuroticism can be viewed as a heterogeneous trait consisting of multiple facets that are highly correlated but partially distinct, including anger, sadness, anxiety, worry, and hostility (Weiss & Costa, 2005). Indeed, many studies of the relation between negative affectivity and adverse outcomes focus on fine-grained traits that might be considered facets of neuroticism, such as trait hostility and anger (T. W. Smith, Glazer, Ruiz, & Gallo, 2004; Suarez, Lewis, & Kuhn, 2002). The present paper focuses on the broad trait of neuroticism, but some facets of neuroticism may be more important than others for specific purposes.

It should be emphasized that there is a lack of full independence between most measures of neuroticism and measures of some mental health outcomes. In particular, the fact that some of the items that define neuroticism in most scales overlap with symptoms of depression and anxiety complicates the interpretation of correlations with these disorders (Jylha & Isometsa, 2006). In particular, in cross-sectional studies, it is possible that neuroticism and mental disorders could be correlated because persons who are currently experiencing an episode of depression or anxiety disorder might endorse more of these overlapping neuroticism items than either before or after the episode. Nonetheless, the association between neuroticism and public health outcomes is not merely an artifact of overlapping criteria. A number of longitudinal studies have controlled for shared items and concurrent depressive states and still found significant associations between the construct of neuroticism and measures of depression (Fergusson, Horwood, & Lawton, 1989; Schmutte & Ryff, 1997; Spijker, de Graaf, Oldehinkel, Nolen, & Ormel, 2007). In addition, as summarized in this paper, neuroticism is robustly linked to many mental disorders (e.g., somatoform disorders, eating disorders, schizophrenia, and substance use disorders) and to physical health problems that are not defined by symptoms that overlap with neuroticism items. Indeed, there is some evidence that neuroticism may predict a wide range of physical health problems even when depression is controlled (Bouhuys, Flentge, Oldehinkel, & van den Berg, 2004; Russo, Katon, Lin, & Von Korff, 1997). Nonetheless, greater attention to the issues of conceptual and criterion overlap in the operational definitions of neuroticism and some mental disorders is needed in the future.

This paper overviews what is known about neuroticism, attempts to explain why it has great significance for the public health, and summarizes working hypotheses to guide current research on the causal links between neuroticism and mental and physical health problems. Because funding for basic research from the National Institutes of Health is more closely tied to the public health significance of the specific research aims than ever before, I argue that

neuroticism is an important topic for policy makers and researchers in both basic and applied areas of psychology.

PUBLIC HEALTH SIGNIFICANCE OF NEUROTICISM

Neuroticism is important to public health research partly because it is robustly correlated with a wide variety of both mental and physical health problems. Neuroticism appears to be correlated with a wider range of mental and physical health problems than other personality traits (Malouff, Thorsteinsson, & Schutte, 2005; Malouff, Thorsteinsson, & Schutte, 2006; Saulsman & Page, 2004), but it certainly is not the only personality trait of public health importance. For example, there is growing evidence that conscientiousness is related to physical health through positive health practices (Hagger-Johnson & Whiteman, 2007), and sensation seeking is inversely related to injuries from traffic accidents (Heino, van der Molen, & Wilde, 1996; Schwebel, Severson, Ball, & Rizzo, 2006). Thus, although the focus of this review is on neuroticism, other personality traits also are important to public health.

Association of Neuroticism with Mental Disorders

There is strong evidence that neuroticism is robustly correlated with many Axis I and II mental disorders from childhood through adulthood (Clark, Watson, & Mineka, 1994; Khan, Jacobson, Gardner, Prescott, & Kendler, 2005; Krueger & Markon, 2001; Sher & Trull, 1994; Watson et al., 1994). A meta-analysis of associations between neuroticism and diagnoses of Axis I mental disorders was conducted based on 33 population-based samples (Malouff et al., 2005). Table 1 presents effect sizes for the strength of association with the mental disorder, expressed as *d* (Cohen, 1988). A value of $d = .50$ would indicate that persons who met criteria for each mental disorder had neuroticism scores that were one-half a standard deviation higher on average than persons who did not meet criteria for the disorder. A value of $d = 1.0$ would indicate a one standard deviation difference. Conventionally, effect sizes of $d > .50$ are considered to be "medium" and effect sizes of $d > .80$ be considered to be "large" (Cohen, 1998). The meta-analysis (Malouff et al., 2005) revealed that the magnitudes of association between neuroticism and mood disorders, anxiety disorders, somatoform disorders, schizophrenia, and eating disorders were all in the large range. Furthermore, studies conducted after this meta-analysis have confirmed the robust associations between neuroticism and Axis I mental disorders (Chien, Koa, & Wub, 2007; Khan et al., 2005; Weinstock, 2006). In particular, a study of 7,588 adult twins (Khan et al., 2005) found large effect sizes for the association between neuroticism and depression, generalized anxiety disorder, and panic disorder, and medium effect sizes for phobias, alcohol and drug dependence, and antisocial personality disorder. In addition, neuroticism is robustly associated with dysthymia (Akiskal et al., 2006) and there is extensive evidence that higher neuroticism is related to the use and abuse of psychoactive substances, from nicotine and alcohol to heroin (Kornør & Nordvik, 2007; Malouff, Thorsteinsson, Rooke, & Schutte, 2007; Malouff et al., 2006; Sher & Trull, 1994).

There also is extensive evidence that neuroticism is correlated with a number of Axis II personality disorders (Clark et al., 1994; Costa & Widiger, 2002; Krueger & Markon, 2001; Saulsman & Page, 2004; Sher & Trull, 1994). As shown in Table 2, a meta-analytic review of this literature (Saulsman & Page, 2004) found medium effect sizes for associations between neuroticism and borderline, avoidant, and dependent personality disorders, and smaller effect sizes for schizotypal, paranoid, and antisocial personality disorder. It should be noted that a number of modifications of the taxonomy of personality disorders in DSM-V have been proposed that could make the definitions of personality disorders more consonant with either three or five-factor models of personality that include the trait of neuroticism (McCrae, Lockenhoff, & Costa, 2005; Widiger & Trull, 2007). Thus, neuroticism may be even more strongly linked to future diagnostic definitions of personality disorders.

Neuroticism and Comorbidity Among Common Mental Disorders

Because neuroticism is linked to increased risk for many different mental disorders, it not surprising that it also is related to greater *comorbidity* among those mental disorders (Khan et al., 2005; Middeldorp et al., 2006; Weinstock, 2006). Indeed, in one large study, variations in neuroticism explained 20–45% of the comorbidity among depression and anxiety disorders and 19–88% of the comorbidity of those disorders with alcohol dependence and drug dependence (Khan et al., 2005). This is important because persons with comorbid mental disorders have more persistent and disabling disorders, and are more likely to use high-cost mental health services, than persons who meet criteria for only one mental disorder (Kessler, Chiu, Demler, & Walters, 2005). Furthermore, higher neuroticism is associated with greater use of mental health services even when the number of comorbid mental disorders is controlled (Jylha & Isometsa, 2006; ten Have, Oldehinkel, Vollebergh, & Ormel, 2005). In addition, persons with comorbid mental disorders have significantly poorer physical health and are more likely to be high-end users of medical services (Rush et al., 2005). This means that neuroticism is centrally important to public health partly because persons with high levels of neuroticism are at high risk for having the most serious, impairing, and costly mental health problems. The combined estimated 12-month prevalence of the many Axis I and II mental disorders shown to be moderately to strongly correlated with neuroticism is upwards of 20% of the U.S. population (Narrow, Rae, Robins, & Regier, 2002). Given the enormous personal distress and impairment associated with these mental disorders and their overall impact on the economy (Merikangas et al., 2007), the association of neuroticism with mental disorders is no small matter.

Predictive Utility of Neuroticism for Mental Health Outcomes

From a public health perspective, there must be more than just concurrent correlations between a trait and adverse outcomes. The public health significance of any trait depends heavily on its ability to *predict* future adverse outcomes with an effect size large enough to be of practical importance. In the absence of such predictive utility, neuroticism could not usefully identify individuals at high risk for future adverse outcomes. Furthermore, if neuroticism did not predict future outcomes and demonstrate temporal precedence relative to the outcomes, it would be unlikely that neuroticism could be causally related to those outcomes. It is very important, then, that a number of prospective longitudinal studies have indicated that neuroticism has substantial predictive utility for a number of mental health outcomes.

The utility of neuroticism in predicting first episodes of major depression was evaluated in a longitudinal study of 1,733 twins from female-female pairs and 1,862 twins from male-male pairs (Fanous, Neale, Aggen, & Kendler, 2007; Kendler, Neale, Kessler, Heath, & Eaves, 1993). Excluding participants who had already experienced an episode of depression by the time neuroticism was measured, *each* 1 standard deviation (SD) unit difference in higher neuroticism scores was associated with a 90–100% increase in the odds of developing major depression for the first time during the next 12 months in women (Kendler et al., 1993) and an 85% increase in men (Fanous et al., 2007). The strongest prospective study of neuroticism and depression to date involved 20,692 adult members of same-sextwin pairs from the population-based Swedish Twin Registry. Participants completed a measure of neuroticism in 1972–73 and were interviewed25 years later to determine if they had experienced major depression. Excluding individuals with an episode of major depression before 1974, each 1 SD difference in higher neuroticism was associated with a 31% greater risk for a first episode of major depression over the 25-year period, controlling age, sex, and extroversion (Kendler, Gatz, Gardner, & Pederse, 2006). Fewer studies of the utility of neuroticism in predicting other mental disorders have been conducted, but a prospective study of a birth cohort of 5,362 individuals found the odds of later meeting criteria for schizophrenia to be 93% greater for individuals with high neuroticism scores at age 16 (Van Os & Jones, 2001). In addition, a

longitudinal study of a New Zealand birth cohort found that risk for a suicide attempt at ages 15–21 years was 225% greater for youth in the highest quartile of neuroticism at age 14 than for youth in the lowest quartile, controlling for socioeconomic status, sensation seeking, depression and other mental disorders, and stressful life events (Fergusson, Woodward, & Horwood, 2000). Moreover, these risk factors were related to suicide attempts multiplicatively, such that the risk for a suicide attempt among youth with both high neuroticism scores and other risk factors was 60 times greater than for low risk youth (Fergusson et al., 2000). This suggests that the predictive validity of neuroticism may be particularly strong when considered in the context of negative life events and other predictors.

Association of Neuroticism with Physical Health

Persons with high scores on neuroticism scales are more likely to express medically unfounded somatic complaints (Chaturvedi, 1986; Costa & McCrae, 1987) and to have catastrophic thoughts about symptoms that lead to medical service use (Goubert, Crombez, & Van Damme, 2004). These correlates of neuroticism are very important because they involve considerable personal suffering and costly use of health services, but it appears that the link between neuroticism and physical health goes beyond unfounded medical complaints. There is both indirect and direct evidence linking neuroticism with many kinds of serious physical health problems (Brickman, Yount, Blaney, Rothberg, & De-Nour, 1996; Drossman et al., 2000; T. W. Smith & MacKenzie, 2006; Suls & Bunde, 2005).

The indirect evidence comes from studies showing strong links between physical health and mental disorders that are themselves strongly linked with neuroticism, particularly depression and anxiety disorders. A wide range of physical health problems are more common among individuals with mood and anxiety disorders, which are strongly correlated with neuroticism (Currie & Wang, 2005; Robles, Glaser, & Kiecolt-Glaser, 2005; Sareen, Cox, Clara, & Asmundson, 2005; Watkins et al., 2006). Depression and anxiety disorders are associated with disrupted immune functioning (Maier & Watkins, 1998; Pace et al., 2006; Robles et al., 2005), abnormalities in cardiac functioning (Barger & Sydeman, 2005), and increased mortality among individuals with other risk factors for cardiac disease (Penninx et al., 2001; Robles et al., 2005; Simonsick, Wallace, Blaser, & Gerkman, 1995). Again, however, the literature on anxiety and depression only provides indirect evidence regarding the association of neuroticism with physical health problems that is subject to multiple interpretations.

Fortunately, there also is growing direct evidence that neuroticism is associated with physical health problems. A number of studies suggest that neuroticism itself is associated with a wide range of physical health problems (T. W. Smith & MacKenzie, 2006), such as cardiovascular disease (Suls & Bunde, 2005), atopic eczema (Buske-Kirschbaum, Geiben, & Hellhammer, 2001), asthma (Huovinen, Kaprio, & Koskenvuo, 2001), and irritable bowel syndrome (Spiller, 2007), even when depression and other risk factors such as social support are controlled (Bouhuys et al., 2004; Russo et al., 1997). Indeed, one study found that depression did not predict poor physical health when neuroticism was controlled (Russo et al., 1997). Thus, the association between neuroticism and physical health problems may not solely reflect the result of the correlation of neuroticism with depression.

Furthermore, because neuroticism is associated with both mental and physical health problems, it is likely that neuroticism is associated with comorbidity between mental and physical health problems. This would greatly multiply the public health significance of neuroticism, as this form of comorbidity is associated with more complicated health problems, greater need for health services, and significantly poorer health outcomes (Baune, Adrian, & Jacobi, 2007; Druss et al., in press; McCaffery et al., 2006).

Predictive Utility of Neuroticism for Physical Health Outcomes

As with mental health problems, the key issue in assessing the public health significance of neuroticism for physical health problems is its predictive utility. There is growing evidence from large studies of representative samples that neuroticism significantly predicts longevity in the general population (T. W. Smith & MacKenzie, 2006). For example, a 21-year prospective study of a representative sample of 5,424 British adults found that each 1 standard deviation unit difference in greater neuroticism at baseline was associated with a statistically significant 10% greater mortality from cardiovascular disease, controlling for age, sex, socioeconomic status, smoking, alcohol consumption, physical activity, and initial health (Shipley, Weiss, Der, Taylor, & Deary, 2007). Similarly, a 6-year longitudinal study of a representative sample of 6,158 adults 65 years of age and older found that a high level of neuroticism in the first assessment predicted 33% more deaths from all causes compared to low neuroticism, controlling age, sex, race-ethnicity, education, medical conditions, and initial health (Wilson et al., 2005). In addition, a 5-year longitudinal study of 800 elderly female and male clergy found that participants with high neuroticism scores had nearly double the death rate as those with low neuroticism scores (Wilson, Leon, Bienias, Evans, & Bennett, 2004). In contrast, higher neuroticism did not predict mortality in a longitudinal study of 65- to 100-year-old participants in frail health (Weiss & Costa, 2005).

There also is important evidence that neuroticism robustly predicts morbidity and mortality in individuals with chronic diseases and cancer. For example, neuroticism strongly predicts the course of renal deterioration in type I diabetics (Brickman et al., 1996) and patients with cardiac disease (Murberg, 2004). In addition, a four-year prospective study of patients with chronic renal insufficiency found that patients with high neuroticism scores had a 38% greater mortality rate, controlling age, diabetic status, hemoglobin level, and the personality trait of conscientiousness (Christensen et al., 2002). Similarly, a 25-year Danish longitudinal study found that persons treated for cancer who were high in neuroticism had a 130% greater death rate than persons low in neuroticism (Nakaya et al., 2006). Thus, it appears that neuroticism is a robust predictor of future physical health problems and mortality.

Neuroticism and Quality of Life

Any discussion of adverse outcomes associated with neuroticism is incomplete without considering the positive outcomes that are inversely associated with it. The extensive literature on associations between personality traits and aspects of subjective well being has been reviewed recently and will not be repeated here (Ozer & Benet-Martinez, 2006; Steel, Schmidt, & Shultz, 2008). It is important to note, however, that neuroticism is an important trait partly because it is inversely related to marital satisfaction (Gattis, Berns, Simpson, & Christensen, 2004), occupational success (Ozer & Benet-Martinez, 2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007), and overall quality of life (Arrindell, Heesink, & Feij, 1999; Lynn & Steel, 2006; Ozer & Benet-Martinez, 2006). Interestingly, there is recent evidence based on a large and representative twin sample indicating that much of the genetic influences on subjective well being are shared with neuroticism (Weiss, Bates, & Luciano, 2008).

Thus, overall, there is strong evidence from prospective studies that greater neuroticism is related to adverse outcomes. Indeed, it predicts shorter, less happy, less healthy, and less successful lives to a meaningful extent.

CAUSAL INFLUENCES ON NEUROTICISM

Given the robust utility of neuroticism in predicting many significant human outcomes, it is essential that the causal mechanisms linking neuroticism to those outcomes be discovered. In order to lay a foundation for working hypotheses regarding these causal mechanisms, however,

it is first necessary to review current evidence on the genetic and environmental causal influences on neuroticism itself. This is because some of the causal influences on neuroticism may also influence many of the mental and physical health outcomes and partly explain their associations with neuroticism.

Genetic Influences on Neuroticism

Neuroticism is substantially heritable, with estimates of its heritability varying somewhat by sex and age. The heritability of neuroticism peaks in early adolescence and early adulthood, with no sex differences in heritability at those ages (Gillespie, Evans, Wright, & Martin, 2004; Lake, Eaves, Maes, Heath, & Martin, 2000; Rettew et al., 2006; Viken, Rose, Kaprio, & Koskenvuo, 1994). In this age range, 50–60% of the variance in neuroticism scores is estimated to be attributable to genetic factors in both sexes. After early adulthood, the heritability of neuroticism declines gradually with age in both sexes, but slightly more so in males, resulting in somewhat greater heritability in females during later adulthood (Eaves, Eysenck, & Martin, 1989; Lake et al., 2000). Thus, it is possible that there are sex differences in some of the genes that influence neuroticism, or sex differences in their expression, at older ages that result in these small sex differences in heritability in later adulthood (Macaskill, Hopper, White, & Hill, 1994; Rettew et al., 2006).

Most molecular genetic studies of neuroticism have focused on polymorphisms of genes thought to be good candidates for neuroticism because they are associated with neural systems related to emotion. For example, the serotonergic system is known to be involved in emotion processing (Leonardo & Hen, 2006). The association between a polymorphism (5-HTTLPR) of the serotonin transporter gene and neuroticism has been investigated many times. This is because the serotonin transporter is involved in the reuptake of serotonin from the synapse and variations in transporter functioning can influence serotonergic transmission. The 5-HTTLPR polymorphism is of particular interest because (a) it is a functional polymorphism that influences gene products that influence activity of the serotonin transporter on neurons (Haddley et al., 2008), and (b) because the serotonin transporter is the target of some medications that are effective in treating anxiety and depression (Leonardo & Hen, 2006).

Three separate meta-analyses of published studies concluded that there is a small but significant difference in neuroticism scores between persons with at least one short 5-HTTLPR allele and persons with only long alleles (Schinka, 2005; Schinka, Busch, & Robichaux-Keene, 2004; Sen, Burmeister, & Ghosh, 2004, 2005). A fourth meta-analysis initially did not find a significant association between 5-HTTLPR and neuroticism (Munafo et al., 2003), but a revised and updated report of this meta-analysis confirmed the significant association between 5-HTTLPR and neuroticism (Munafo, Clark, & Flint, 2005). This conclusion is consistent with findings that variations in the serotonin transporter gene in mice are related to behaviors suggestive of emotionality that may be analogous to neuroticism (Leonardo & Hen, 2006; Murphy & Lesch, 2008). In addition, functional magnetic resonance imaging has revealed that variations in alleles of 5-HTTLPR are related to variations in the activation of the amygdala and related brain structures in response to threatening stimuli (Brown & Hariri, 2006; Hariri et al., 2002; Passamonti et al., 2008). Because anatomic and functional variations in the same brain structures have been linked to variations in neuroticism (Canli, 2004; Haas, Omura, Constable, & Canli, 2007; Omura, Constable, & Canli, 2005), this research also supports the hypothesis that polymorphisms of 5-HTTLPR are linked to neuroticism.

In addition to the serotonin transporter, there is evidence from candidate gene studies that polymorphisms that encode serotonin receptors also are related to neuroticism. This comes from studies of both humans (including genomic imaging studies) and genetically modified mice and includes the 5-HT1A and 5-HTR2A receptors (Golimbet et al., 2002; Lesch & Canli, 2006). Similarly, the G72 gene, which influences glutamate receptor function, has been found

in multiple samples to be associated with neuroticism (Rietschel et al., in press). Molecular studies of other promising candidate genes that may be associated with neuroticism have yielded less consistent findings to date (Hettema, An et al., 2006; Lang et al., 2005; Sen et al., 2003; Willis-Owen et al., 2005).

Several genetic studies of neuroticism not involving the advance specification of candidate genes also have been conducted. Genome-wide linkage studies, which use large numbers of markers selected from across the genome to examine patterns of inheritance within families, have not yielded consistent findings (Fullerton et al., 2003; Nash et al., 2004), perhaps partly because of limitations in the statistical power of linkage studies (Risch & Merikangas, 1996). A more powerful recent genome-wide association study of unrelated individuals failed to find significant associations between single nucleotide polymorphisms (SNPs) and neuroticism (Shifman et al., 2008), but another recent study found associations of neuroticism and SNPs in MAMDC1 (van den Oord et al., 2008).

Environmental Influences on Neuroticism

It is clear that there are both genetic and environmental causal influences on neuroticism (Rettew et al., 2006; Viken et al., 1994). In discussing this topic, it useful to distinguish two kinds of environments. Shared environments are common to members of a family and make them more similar on a trait. In contrast, non-shared environments are not experienced by all members of a family (e.g., an automobile accident involving only one sibling) and make members of a family less similar. Estimates of the magnitude of shared environmental influences on neuroticism from several large twin studies are essentially zero, but there appear to be substantial non-shared environmental influences on neuroticism (Fullerton, 2006; Lake et al., 2000). It would be a mistake to conclude that only nonshared environments influence neuroticism, however (Rutter, 2007a). It is possible that environments that siblings share, such as the stress of living in poverty, strongly influence neuroticism, but their causal influences do not appear in estimates of shared environmental influences in twin studies (Plomin, DeFries, & Loehlin, 1977). This can happen when the shared environment operates through gene-environment interactions (Jinks & Fulker, 1970; Johnson, 2007; Robinson, 2004). That is, if the impact of a shared environment is *moderated* by genes, it would not contribute to the estimate of shared environmental influences. This is because gene-environment interactions with shared environmental factors tend to make identical (monozygotic) twins, who share essentially 100% of their polymorphic genes, more similar to one another. In contrast, because fraternal (dizygotic) twins only share 50% of their polymorphic genes on average, interactions between shared environments and genes would only make fraternal twins more similar when they happen to share the same allele(s) of the relevant polymorphism(s). Otherwise, gene-environment interactions make dizygotic twins and other siblings less similar and contribute to the estimate of nonshared environmental influences in twin studies. Thus, it is possible that shared aspects of the environmental influences exert causal influences on neuroticism, but do so through gene-environment interactions.

At this point in time, very little is known about the *specific* shared and nonshared environmental experiences that influence neuroticism. Several studies have found modest correlations between neuroticism scores in adults and their retrospective recall of intrusive parenting (Reti et al., 2002), having been abused during childhood (Allen & Lauterbach, 2007; Roy, 2002), lack of religious upbringing (Willemsen & Boomsma, 2007), and low parental involvement (McCrae & Costa, 1988) when they were children. A search of the literature did not locate any published studies that prospectively linked parenting in childhood to the offspring's later neuroticism, however. Retrospective studies are useful for generating hypotheses about potential environmental influences on personality, but they are highly subject to potentially serious recall biases. Thus, there is a clear and pressing need for longitudinal studies that can

test hypotheses prospectively regarding the causal influence of specific experiences on later neuroticism (Rutter, 2007b). These would need to determine that such early experiences influence future neuroticism when early measures of neuroticism are controlled and rule out relevant confounds, such as shared genetic influences between the experience and neuroticism. It is essential that future studies of causal environmental influences on neuroticism not be conducted in isolation, however, but take the interplay of genes and environments fully into consideration (Johnson, 2007; Robinson, 2004).

CAUSAL LINKS BETWEEN NEUROTICISM AND ADVERSE OUTCOMES

Causal Links Between Neuroticism and Mental Disorders

Three working hypotheses have been proposed regarding the causal links between neuroticism and mental disorders. It seems likely that these hypotheses are not mutually exclusive, but refer to mechanisms that work together to foster mental disorders. First, there is substantial evidence that the same genes that influence neuroticism also influence the many different mental disorders that are strongly correlated with neuroticism. Second, persons higher in neuroticism appear to be more likely to experience stressful events and have less social support. Third, it is possible that neuroticism is associated with more pronounced and less well-regulated emotional responses to stressful events.

1. Overlapping genetic influences—There is growing evidence that neuroticism and mental disorders are correlated partly because they are partly influenced by the same genes (Hettema, Neale, Myers, Prescott, & Kendler, 2006; Silberg, Rutter, Neale, & Eaves, 2001; Stein & Stein, 2008). Indeed, twin studies indicate that one-third to two-thirds of the genetic variance in broad range of mental disorders is shared with neuroticism (Carey & DiLalla, 1994; Fanous, Gardner, Prescott, Cancro, & Kendler, 2002; Hettema, Neale et al., 2006). It is not surprising, then, that the genetic influences on neuroticism also increase risk for *comorbidity* among the many forms of mental disorders correlated with neuroticism (Khan et al., 2005).

There is mounting evidence that the 5-HTTLPR polymorphism may be one of the specific genetic variants that influence both neuroticism and the mental disorders correlated with it. For example, persons with at least one copy of the short allele of 5-HTTLPR drink more alcohol than persons with two long alleles (Munafo, Lingford-Hughes, Johnstone, & Walton, 2005). This is potentially important because neuroticism is associated with this polymorphism and higher neuroticism scores are associated with alcohol use disorders (Khan et al., 2005; Malouff et al., 2005). There also is consistent evidence that the short allele of 5-HTTLPR is associated with increased risk for depression among persons who have experienced stressful life events (Caspi et al., 2003; Lotrich & Pollock, 2004; Uher & McGuffin, 2008). Indeed, 17 studies have replicated this finding with only 3 failures to replicate (Rutter, 2008).

Furthermore, the interaction of 5-HTTLPR and stressful life events has been substantiated in genomic imaging studies. For example, activation of the amygdala and hippocampus in response to faces varies dramatically according to the number of stressful life events experienced by the individual, but particularly in persons with at least one short allele of 5-HTTLPR (Canli et al., 2006). Furthermore, two studies found that neuroticism entirely mediates the association between the 5-HTTLPR gene and depression (Jacobs et al., 2006; Munafo, Clark, Roberts, & Johnstone, 2006). In addition, the results of a number of cognate studies of non-human animals are consistent with the hypothesis that variants of the serotonin transporter gene interact with stress levels to influence negative emotionality (Caspi & Moffitt, 2006). There also is growing evidence that the G72 gene, which is associated with neuroticism, is also associated with major depression and schizophrenia (Rietschel et al., in press; Shi, Badner, Gershon, & Liu, 2008), suggesting that G72 could prove to be another gene that

increases risk for both high neuroticism and some of the mental disorders that are correlated with it.

2. Neuroticism, stressful events, and social support—The second hypothesized causal link between neuroticism and mental disorders is an indirect link through negative life events and social support. There is evidence from a number of longitudinal studies that persons high in neuroticism live their lives in ways that increase the likelihood of many kinds of negative life events. For example, individuals with high neuroticism scores are more likely to experience daily hassles, have conflicted and unstable relationships with friends, experience stressful physical health problems, and lose employment (Bolger & Zuckerman, 1995; Gunthert, Cohen, & Armeli, 1999; Hankin, Fraley, & Abela, 2005; Kendler, Gardner, & Prescott, 2003; Magnus, Diener, Fujita, & Pavot, 1993; Suls & Martin, 2005; van Os, Park, & Jones, 2001). This is the case both when the life events are subjectively judged to be stressful by the individual and when they are objectively rated by experimenters blind to neuroticism scores (Bolger & Zuckerman, 1995; Gunthert et al., 1999; Hankin et al., 2005; Kendler et al., 2003; Magnus et al., 1993; Suls & Martin, 2005; van Os et al., 2001). The higher frequency of stressful events in the lives of persons with high levels of neuroticism is important because it is likely that stressful life events precipitate a range of mental disorders (Ehring, Ehlers, & Glucksman, 2006; Kendler et al., 2004; Parslow, Jorm, & Christensen, 2006).

To consider just one specific example of neuroticism influencing risk for negative life events, a number of longitudinal studies have shown that the neuroticism scores of both partners measured before marriage predict future separation or divorce (Donnellan, Conger, & Bryant, 2004; Karney & Bradbury, 1997; Kelly & Conley, 1987; Roberts et al., 2007; Rogge, Bradbury, Hahlweg, Engl, & Thurmaier, 2006; Tucker, Kressin, Spiro, & Ruscio, 1998). Because other longitudinal studies have shown that divorce predicts mental health problems, substance abuse, and mortality (Hemström, 1996; Ikeda et al., 2007;C. Lee & Gramotnev, 2007; S. Lee et al., 2005; Overbeek et al., 2006; Perreira & Sloan, 2001), it is reasonable to hypothesize that neuroticism indirectly promotes adverse outcomes partly by increasing the likelihood of divorce.

Similarly, there is evidence that neuroticism predicts lower levels of social support (Kendler, Gardner, & Prescott, 2002; Kendler, Gardner, & Prescott, 2006). This could mean that individual differences in neuroticism are one factor influencing the initiation and maintenance of supportive social relationships. This could be important because social support is linked with depression and partially mediates the association between neuroticism and depression (Finch & Graziano, 2001; Kendler et al., 2002; Kendler, Gardner et al., 2006). Furthermore, greater social support predicts shorter episodes of depression in prospective studies (Lara, Leader, & Klein, 1997; Neeleman, Oldehinkel, & Ormel, 2003).

3. Neuroticism and emotional reactivity to stressful life events—The construct of neuroticism would have little meaning if persons high in neuroticism did not respond with negative emotions more frequently and intensely when they experience stressful life events. There is mounting evidence in support of the construct, however. For example, participants with high neuroticism scores responded to a standard negative mood induction task with more negative affect than participants lower in neuroticism (Larsen & Ketelaar, 1991). Similarly, a 30-day daily diary study of persons with rheumatoid arthritis found that persons with higher neuroticism scores experienced greater negative affect on days when negative events occurred than persons with lower neuroticism scores did (Zautra, Affleck, Tennen, Reich, & Davis, 2005). Moreoever, there is replicated evidence from longitudinal studies that persons with high neuroticism scores are more likely than other persons to develop anxiety and depression following negative life events (Fanous et al., 2002; Hutchinson & Williams, 2007; Jacobs et al., 2006; Kendler et al., 2004; Ormel & Wohlfarth, 1991; Parslow et al., 2006).

Coping style may be an important component of the greater emotional reactivity of persons high in neuroticism (Matthews et al., 2003). There is evidence that persons high in neuroticism use fewer problem-focused and more emotion-focused strategies to cope with stress (Watson & Hubbard, 1996). In addition, there is evidence that persons high in neuroticism are more likely to use inefficient escape-avoidance strategies to cope with stress (Bolger, 1990). Thus, it appears that persons high in neuroticism have both an increased likelihood of experiencing negative life events and an increased magnitude of emotional reactivity to those events, partly because of how they cope with stress.

Causal Links Between Neuroticism and Physical Health Problems

Some of the causal links between neuroticism and physical health problems may be the same as for mental health problems. Like mental health problems, it is possible that neuroticism may be linked to physical health problems partly because of shared genetic influences. In addition, like mental health problems, it is likely that neuroticism is linked to physical health problems partly because neuroticism leads to experiencing more stressors and less social support (Contrada, Cather, & O'Leary, 1999;T. W. Smith & MacKenzie, 2006) or benefitting less from social support (Holt-Lunstad, Smith, & Uchino, 2008). This is important because lower social support is associated with increased risk for health problems (Uchino, 2006) and stress increases risk for health problems, particularly when it is unremitting and the individual is older or in poor health (Schneiderman, Ironson, & Siegel, 2005).

In addition, two other specific causal mechanisms have been hypothesized through which neuroticism may be linked to physical health problems that may not be involved in mental health problems. First, it is possible that higher neuroticism is associated with greater sympathetic and hypothalamic-pituitary-adrenal (HPA) reactivity and with greater alterations of the immune system in response to stressful life events (Futterman, Kemeny, Shapiro, & Fahey, 1994; Gillespie et al., 2004). In turn, these enhanced physiological responses to stress may contribute to health problems (Contrada et al., 1999; Friedman, 2000;T. W. Smith & MacKenzie, 2006). Second, it is likely that persons high in neuroticism are more likely to engage in behaviors that increase the risk of health problems (Contrada et al., 1999).

Neuroticism and Physiological Stress Reactivity—There is growing evidence that neuroticism moderates the magnitude of physiological responses to stressors, but this evidence is not fully consistent. Several studies suggest that individuals high in neuroticism have larger and more prolonged sympathetic responses to stressors (Norris, Larsen, & Cacioppo, 2007; Riese et al., 2007; Vogeltanz & Hecker, 1999), greater cardiovascular reactivity (Muth, Koch, & Stern, 2000), and higher morning levels of cortisol (Portella, Harmer, Flint, Cowen, & Goodwin, 2005), but have blunted cortisol response to stress (Oswald et al., 2006; Phillips, Carroll, Burns, & Drayson, 2005). Hormonal challenge studies suggest that this blunted cortisol stress response in individuals with high neuroticism scores may reflect down-regulation of the HPA system due to prolonged cortisol elevation (McCleery & Goodwin, 2001; Zobel et al., 2004). Neuroticism also is related to disruption of circadian rhythms (Murray, Allen, Trinder, & Burgess, 2002) and is correlated with abnormalities of the immune system (Bouhuys et al., 2004). Higher neuroticism has been found to be associated with more prolonged suppression of secretory immunoglobulin following a stressor (Hennig, Possel, & Netter, 1996), atypical response of natural killer cells to stress (Borella et al., 1999), diminished antibody response to vaccination (Phillips et al., 2005), and higher leukocyte counts (Daruna, 1996). Leukocyte count is important because it is indicator of inflammation and chronic infection and is a strong predictor of mortality from all causes, particularly from cardiovascular disease (Margolis et al., 2005).

In contrast, some studies have not found neuroticism to be related to differences in cortisol reactivity to a stressor (Hennig et al., 1996; Schommer, Kudielka, Hellhammer, & Kirschbaum, 1999) and one study found that higher neuroticism was associated with lower heart rate and lower levels of serum norepinephrine during an aversive challenge (LeBlanc, Ducharme, & Thompson, 2004). Such discrepancies may be the result of the generally small samples used in this area of research, nonlinear associations between neuroticism and stress reactivity, methodological differences across studies, and demographic differences in samples. For example, there is some evidence that the greater physiological responsiveness associated with higher neuroticism is considerably more robust in females than in males (Hennig et al., 1996; Oswald et al., 2006). It will be very important to consider such factors when attempting to resolve these discrepancies and test the important hypothesis that neuroticism moderates health-related physiological responsiveness to stressors.

There is emerging evidence from twin studies that the hypothesized moderation of physiological reactivity by neuroticism may be partly due to some genes influencing both neuroticism and physiological reactivity (Riese et al., 2007). At the molecular level, persons with at least one copy of the short allele of 5-HTTLPR have been found to have higher resting cortisol levels than persons with two long alleles (Jabbi et al., 2007). Similarly, the association between neuroticism and some health problems may reflect common genetic influences. For example, irritable bowel syndrome is associated with neuroticism (Spiller, 2007) and the short allele of 5-HTTLPR has been found to be associated with irritable bowel syndrome in humans (Yeo et al., 2004) and with analogous bowel dysfunctions in mice (Murphy & Lesch, 2008).

Neuroticism and Health-Risk Behaviors—A second hypothesized link between neuroticism and physical health is through behaviors that increase risk for health problems (Contrada et al., 1999;T. W. Smith & MacKenzie, 2006). Persons high in neuroticism are markedly more likely to smoke (Breslau, Novak, & Kessler, 2004; Malouff et al., 2006; Morissette, Tull, Gulliver, Kamholz, & Zimering, 2007; Terracciano & Costa, 2004) and to become dependent on alcohol and other drugs (Larkins & Sher, 2006). These behaviors robustly increase risk for cancer, asthma, emphysema, cardiovascular disease, and other diseases. In addition, persons high in neuroticism are much more likely to engage in unprotected sex, perhaps as a way to cope with aversive mood states or due to emotional impulsiveness or lack of assertiveness (Cooper, Agocha, & Sheldon, 2000; Trobst et al., 2000). Indeed, the odds of engaging in unprotected sex by individuals who are at least 1 standard deviation above the mean of neuroticism are 8–10 times greater than for individuals who are at least 1 standard deviation below the mean (Hoyle, Fejfar, & Miller, 2000). In turn, risky sexual behavior increases the odds for HIV/AIDS, cervical cancer, and other diseases.

COULD NEUROTICISM PLAY A ROLE IN THE PREVENTION OF ADVERSE OUTCOMES?

Ultimately, the public health significance of neuroticism hinges on whether knowledge that neuroticism is a potent risk factor for many adverse outcomes can be translated into the prevention of those outcomes. Perhaps the most likely way in which this is could happen is through improved understanding of the basic nature of each of the health and mental health problems associated with neuroticism. That is, achieving a full understanding of why each disorder is related to neuroticism is almost certain to advance our understanding of both neuroticism and each disorder. Indeed, understanding why high neuroticism places persons at increased risk for such seemingly diverse outcomes as depression, schizophrenia, diabetes, asthma, irritable bowel syndrome, and heart disease could change how we conceptualize each of these disorders in fundamentally important ways. Because improved understanding often leads to improved prevention and treatment, discovering how neuroticism is related to each

outcome should eventually lead to improved, and potentially innovative, ways of preventing and alleviating each of the many health and mental health problems linked to neuroticism.

In addition, it is possible, but by no means certain, that innovative approaches to prevention could be developed based on the predictive relation between neuroticism and adverse outcomes. For example, it has been suggested that mental health clinicians routinely administer a five-factor personality scale to determine if more extensive assessments of personality disorders related to neuroticism and other traits should be conducted (Widiger & Trull, 2007). Analogously, it might be possible to inexpensively screen large numbers of individuals in the community for high neuroticism scores (e.g., over the internet). For these reasons, it is useful to speculate about ways in which screening for neuroticism could possibly play a role in future prevention strategies.

First, persons with high scores on the screen could be advised to obtain further individual physical and mental health “check ups” that would include tests specific to each of the mental or physical disorders associated with neuroticism (e.g., tests for cognitive and emotional vulnerability to suicide or tests for high cholesterol). Individuals found to be at risk for any specific adverse outcome using those tests, could then be provided with interventions tailored to their needs (e.g., cognitive-behavior therapy and antidepressants to reduce risk of suicide or statins to reduce cholesterol). If much higher proportions of screen positives than screen negatives were found to be at high risk for one or more of the specific adverse outcomes, such a strategy might prove to be an effective method of identifying at-risk persons. That would only be the case, however, if substantial numbers of persons were found to be at risk for a condition that had not been identified in other ways, such as during routine medical visits. For example, it is possible that few persons would be discovered by screening on neuroticism who did not already know that they were at risk for cardiovascular disease. Even so, a potential benefit of wide-spread screening on neuroticism might be the inexpensive early detection of at-risk persons at an early stage when preventive interventions would be most helpful.

The potential benefits of such large scale screening on neuroticism also would need to be balanced against the numbers of false positives, however. That is, if even if the threshold for the screen was carefully set, any foreseeable screen will identify some persons who screen high on neuroticism but are not actually at high risk for any adverse outcome. This is an issue of concern because persons who are incorrectly identified as being at high risk for serious physical or medical disorders using measures of neuroticism could experience unnecessary worry and subject an already over-burdened health care system to unnecessary expense. In addition, the use of neuroticism scales as a screening measure could tend to stigmatize diversity in this dimension of normal personality.

There has been a trend in the United States toward lowering thresholds for physical health indices such as blood pressure and cholesterol for the purpose of identifying more people in need of preventive treatment. Recent analyses suggest that this *may* have yielded little increase in health benefits, but may have caused increases in adverse side effects because more individuals are being treated, adverse consequences of labeling, and the diversion of funds from potentially more effective health programs (Kaplan & Ong, 2007). Much remains to be learned about this topic, but the same clearly could happen with neuroticism if the specificity of the screen was low and/or the screening threshold was set too low. Great caution should be exercised as we consider the potential utility and wisdom of screening on neuroticism to identify people in need of interventions. Nonetheless, because neuroticism is robustly related to so many different adverse outcomes, this approach to prevention should not be prematurely passed over just because it is challenging.

A second possible innovative approach based on screening for neuroticism might be to attempt to reduce high levels of neuroticism in order to indirectly reduce risks for all of the many mental and physical health problems associated with it. Even if the indirect reduction in the prevalence of each individual adverse outcome were modest, it is possible that such a strategy could be cost-effective because of the sheer number of adverse outcomes associated with neuroticism. To date, no interventions for reducing neuroticism have been identified, but such interventions seem feasible.

The potential utility of this approach to prevention would depend both on the safety, cost, and effectiveness of reducing neuroticism and on the extent to which reducing neuroticism actually resulted in reductions in the adverse health and mental health outcomes associated with neuroticism, all of which are currently unknown. The utility of such large-scale preventive interventions would depend partly on whether reducing neuroticism requires interventions more like supplementing drinking water with fluoride to prevent dental carries than like long-term individual psychotherapy. Assuming for the sake of argument that they were effective in reducing neuroticism, encouraging people with high neuroticism scores to use inexpensive interventions with few adverse side effects such as participation in moderate physical exercise (Koukouvou et al., 2004) or taking omega-3 dietary supplements (Conklin et al., 2007) might be cost-effective, even if they yielded only modest reductions in each of the many adverse outcomes associated with neuroticism. More expensive interventions would need to produce larger decreases in physical and mental disorders to be cost-effective. Although much remains to be learned, prescription medication might provide a way to reduce neuroticism (Knutson et al., 1998), but only if the benefits outweighed the side effects and costs. Similarly, adaptations of cognitive and behavioral interventions like those developed for stress management (Antoni et al., 2000; Cruess et al., 2000; Hampel, Meier, & Kummel, 2008), to prevent anxiety disorders and depression (Bienvenu & Ginsburg, 2007; Stice, Rohde, Seeley, & Gau, 2008), or to treat borderline personality disorder (Woodberry & Popenoe, 2008) could possibly prove to be cost-effective for reducing the broad dimension of neuroticism if they were implemented in group settings and yielded significant reductions in the incidence of physical and mental disorders. This is not because these interventions are inexpensive but because the mental and physical disorders associated with neuroticism are extremely expensive in human and monetary terms. Little empirical evidence can be brought to bear on these potential prevention strategies at present, but they are very worthy of consideration.

Any serious discussion of preventive intervention targeting neuroticism also must consider other possible negative iatrogenic effects. Because wide-spread preventive interventions to reduce neuroticism might even have the unintended effect of reducing adaptive levels of fearfulness and wariness to unsafe levels in some persons, care would need to be taken. This concern might be minimized by intervening only with persons with high levels of neuroticism who requested the intervention, but in some dangerous environments in which cues signaling danger are subtle, such as some urban environments, even relatively high levels of neuroticism might be adaptive in some cases (Matthews et al., 2003).

A third possible way to use of neuroticism in prevention could be to routinely assess neuroticism only in persons with the existing medical conditions that appear to deteriorate more quickly in persons who have high neuroticism scores, such as diabetes and cardiovascular disease. At a minimum, physicians could provide more intensive monitoring or interventions to prevent decline in these individuals. This could be particularly beneficial if a better understanding of the role of neuroticism in such diseases leads to improved treatments for patients high in neuroticism. In addition, is possible that interventions to reduce neuroticism could mitigate against disease progression. Again, much remains to be learned about such important topics.

FUTURE DIRECTIONS FOR RESEARCH

It is clear that the public is an important stakeholder in research on neuroticism. Because of its enormous significance to the public health, an intensive research effort to fully understand relations among neuroticism, mental health, physical health, and quality of life would be well justified. A number of detailed causal models of the multiple mechanisms linking neuroticism and physical health outcomes have been proposed (Contrada et al., 1999;T. W. Smith, 2006). These need further testing and similar causal models for mental health outcomes need testing. It is likely that the best-supported and most useful constructs from each alternative model could be combined to create more comprehensive and useful models. Furthermore, it could be very useful and revealing to develop truly integrated models that would simultaneously explain causal relations between neuroticism and both mental and physical health outcomes in the same model. These adverse outcomes share too much in common to be treated entirely separately in such models. Moreover, including both physical and mental health outcomes in the same models will reveal much about their previously underestimated commonalities.

In addition, the time is right to invest in a better understanding of the genetic and environmental factors that influence neuroticism and play a role in linking it to adverse outcomes. Current evidence strongly suggests that many of the genes influencing neuroticism function as pleiotropic general risk genes that also influence risk for many different mental disorders (Hettema, Neale et al., 2006; Jang & Livesley, 1999) and many different physical health problems (Johnson & Krueger, 2005; McCaffery et al., 2006). Therefore, identifying the genes associated with neuroticism and their interplay with environmental factors should be a top priority for both mental and physical health research. A plausible working model for future studies is that that the etiology of each mental and physical health disorder results from the combined interplay of general-risk genes and environments and disorder-specific genes and environments (Gillespie et al., 2004; Khan et al., 2005; Lahey & Waldman, 2003). To fully understand the etiology of each disorder, it will be necessary to identify each of these factors and how they work together. Understanding the causal pathways linking neuroticism to the many adverse outcomes associated with it will likely reveal a great deal about commonalities in the mechanisms underlying many seemingly distinct mental health and health problems. This should facilitate the development of both integrated causal models and innovative interventions for preventing and treating the many adverse outcomes associated with neuroticism.

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Table 1

Cohen's *d* effect sizes for associations between neuroticism and Axis I mental disorders from the meta-analysis by Malouff et al. (2005).

Mood Disorders	Anxiety Disorders	Somatoform Disorders	Alcohol and Drug Disorders	Schizophrenia	Eating Disorders
1.54	1.04	1.20	.54	1.08	1.29

Note: For effect sizes expressed as Cohen's *d*. $d > .50$ = "moderate effect"; $d > 1.0$ = "large effect." All associations are significant at $p < .0001$.

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Table 2

Cohen's *d* effect sizes for magnitudes of associations between neuroticism and Axis II personality disorders from the meta-analysis conducted by Saulsman and Page (2004)

Borderline	Avoidant	Dependent	Schizotypal	Paranoid	Antisocial	Narcissistic
.55	.54	.50	.41	.32	.10	.02

Note: Cohen's *d* > .50 = "moderate effect", *d* > 1.0 = "large effect." All associations p < .0001, except for narcissitic personality disorder which was not significant at p < .05.