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Linking “Big” Personality Traits to Anxiety, Depressive, and Substance Use Disorders: A Meta-Analysis

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We performed a quantitative review of associations between the higher order personality traits in the Big Three and Big Five models (i.e., neuroticism, extraversion, disinhibition, conscientiousness, agreeableness, and openness) and specific depressive, anxiety, and substance use disorders (SUD) in adults. This approach resulted in 66 meta-analyses. The review included 175 studies published from 1980 to 2007, which yielded 851 effect sizes. For a given analysis, the number of studies ranged from three to 63 (total sample size ranged from 1,076 to 75,229). All diagnostic groups were high on neuroticism (mean Cohen's $d = 1.65$) and low on conscientiousness (mean $d = -1.01$). Many disorders also showed low extraversion, with the largest effect sizes for dysthymic disorder ($d = -1.47$) and social phobia ($d = -1.31$). Disinhibition was linked to only a few conditions, including SUD ($d = 0.72$). Finally, agreeableness and openness were largely unrelated to the analyzed diagnoses. Two conditions showed particularly distinct profiles: SUD, which was less related to neuroticism but more elevated on disinhibition and disagreeableness, and specific phobia, which displayed weaker links to all traits. Moderator analyses indicated that epidemiologic samples produced smaller effects than patient samples and that Eysenck's inventories showed weaker associations than NEO scales. In sum, we found that common mental disorders are strongly linked to personality and have similar trait profiles. Neuroticism was the strongest correlate across the board, but several other traits showed substantial effects independent of neuroticism. Greater attention to these constructs can significantly benefit psychopathology research and clinical practice.

Keywords: Big Five, five-factor model, anxiety, depression, substance abuse

A link between personality and mental health has been hypothesized since the time of the ancient Greeks. The best known example of early theories is the doctrine of the four humors attributed to Hippocrates and Galen (Clark & Watson, 1999; Maher & Maher, 1994). It described four personality types (sanguine, phlegmatic, choleric, and melancholic) and posited that they determine vulnerability to physical and mental illness. Psychology has continued to expand this tradition since the earliest days of the discipline. For example, Freud's (1905/1953) theory of psychosexual development linked mental illness to personality types that he based on clinical observations. Pavlov (1927) and his school, on the other hand, continued to advocate the four-humor doctrine, which they reframed in terms of neuronal responses rather than humors. Interest in this topic continues to this day (Clark, 2005;

Krueger & Tackett, 2006; D. Watson & Clark, 1994). Indeed, the study of personality–psychopathology associations promises to improve our prognostic abilities and may help to elucidate the etiology of mental illness through identification of shared mechanisms. Moreover, the field now has the tools to investigate these issues with rigor and precision. Two developments in particular have made such research feasible: (a) the advent of the modern classification of mental illness and (b) the emergence of a consensus taxonomy of personality.

Modern Classification of Mental Illness

The necessity of a uniform psychiatric nomenclature has been apparent for many decades, but early classification efforts produced a variety of conflicting systems (Widiger, Frances, Pincus, Davis, & First, 1991). This confusion persisted until the introduction of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* in 1952. The first two editions of the *DSM* (American Psychiatric Association [APA], 1952, 1968) helped to bring order to the field, but they lacked a solid research base and did not define disorders precisely enough for diagnoses to be assigned reliably. The third edition of the manual (APA, 1980) sought to address both problems. The system was painstakingly overhauled with the best available data, and publication of the *DSM-III* in 1980 ushered psychiatric classification into the modern era (Wilson, 1993). The two subsequent revisions, *DSM-III-R* (APA, 1987) and *DSM-IV* (APA, 1994), made incremental improvements to the system but maintained the basic framework and focus of the

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DSM-III. The other widely used psychiatric taxonomy is the *International Classification of Diseases (ICD)*. Concerted efforts have been made in recent decades to align the *ICD* and *DSM*, and the 10th edition of the international manual (*ICD-10*; World Health Organization, 1992) corresponds closely with the *DSM-IV*. Hence, the modern approach to the diagnosis of mental illness is now established across the world. The chief achievement of this approach is the uniformity of diagnostic practices that resulted from improved reliability and widespread acceptance of the manuals (Nathan & Langenbucher, 1999).

Nevertheless, these classification systems have important limitations. One notable ongoing controversy is whether psychopathology is best represented by categories or dimensions. This question currently is unresolved, and there are many arguments in favor of the dimensional approach (for reviews, see Trull & Durrett, 2006; Widiger & Samuel, 2005). However, all versions of the *DSM* and *ICD* have followed the categorical model. Consequently, research on specific disorders typically operationalizes psychopathology as categories using one of these systems. In contrast, symptoms of mental illness typically are studied as continuous constructs. These dimensional variables differ across investigations, and a consensus dimensional classification scheme has not yet been established (D. Watson & Clark, 2006). Hence, the scope of the current study is limited to categorical diagnoses to allow for better integration of the literature, as well as clear and consistent definition of psychopathology constructs.

The validity of diagnoses represents another concern with the modern psychiatric nomenclature. Most notably, these diagnoses show high degree of co-occurrence, also known as comorbidity (Clark, Watson, & Reynolds, 1995; Krueger & Markon, 2006; D. Watson, 2009). Such an overlap suggests that existing nosologies do not classify mental illnesses optimally. Put differently, it appears that in some cases multiple diagnoses are used to describe the same basic condition. High comorbidity complicates the detection of specific correlates of mental illnesses because the majority of people with a given disorder typically qualify for a number of other diagnoses. Nevertheless, *DSM* and *ICD* systems remain the standard, and there is no widely accepted alternative that addresses these validity problems (Nathan & Langenbucher, 1999).

Despite these shortcomings, the advent of modern psychiatric classification greatly facilitated research on the epidemiology of mental illness by providing investigators with specific diagnostic criteria. Several nationally representative studies have been completed over the last two decades and established that three classes of mental disorders are especially common in the general adult population: depressive disorders (lifetime prevalence of approximately 17%; Kessler, Berglund, et al., 2005), anxiety disorders (roughly 29%; Kessler, Berglund, et al., 2005), and substance use disorders (SUD; about 35%; Compton, Conway, Stinson, Colliver, & Grant, 2005; Hasin, Hatzenbuehler, Smith, & Grant, 2005). These illnesses—collectively known as common mental disorders—have been the primary focus of many personality–psychopathology theories (Clark, 2005; Clark, Watson, & Mineka 1994; Kotov, Watson, Robles, & Schmidt, 2007; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Krueger & Tackett, 2003; D. Watson, Kotov, & Gamez, 2006), and a large number of empirical studies have examined their links with personality traits.

The present investigation is limited to these disorders to permit a detailed review of the extensive empirical and theoretical work.

Consensus Personality Taxonomy

The field of personality psychology also struggled with classification issues for much of the 20th century. A proliferation of competing taxonomies, which differed in terminology as well as the number and nature of the modeled dimensions, led many to view the field as chaotic and confusing (Clark & Watson, 1999; D. Watson, Clark, & Harkness, 1994). This lack of coherence was a serious impediment to the development of personality psychology until consensus gradually began to emerge in the 1980s. The development of a consensus was facilitated by the explicit recognition that personality is ordered hierarchically from a large number of specific traits to a much smaller number of general characteristics (Digman, 1997; Markon, Krueger, & Watson, 2005). This realization allowed personality researchers to synthesize various models that specified anywhere from two to several dozen traits into a single integrated system. The lower levels of the taxonomy have not been fully mapped out yet, but the higher order levels are already well understood. They are described by two prominent schemes, known as the “Big Five” and the “Big Three.”

The five-factor or Big Five model emerged out of a series of attempts to understand the organization of trait descriptors in the natural language (Goldberg, 1993; John & Srivastava, 1999; McCrae et al., 2000). Structural analyses of these descriptors consistently revealed five broad factors: extraversion, agreeableness, conscientiousness, neuroticism, and openness to experience (also known as openness, imagination, intellect, or culture). This structure has proven to be remarkably robust, with the same five factors observed in both self- and peer-ratings (McCrae & Costa, 1987), in analyses of both children and adults (Digman, 1997), and across a wide variety of languages and cultures (Allik, 2005; McCrae & Costa, 1997).

The Big Three scheme includes the higher order dimensions of negative emotionality, positive emotionality, and disinhibition versus constraint (Clark & Watson, 1999; Markon et al., 2005). This model emerged from the pioneering work of Eysenck and his colleagues (H. J. Eysenck, 1947; H. J. Eysenck & Eysenck, 1976). Eysenck labeled the factors neuroticism, extraversion, and psychoticism, although the last dimension is best viewed as reflecting individual differences in disinhibition versus constraint (Clark & Watson, 1999; D. Watson & Clark, 1993). Other theorists (Gough, 1987; Tellegen, 1985; D. Watson & Clark, 1993) subsequently posited similar three-factor models. It should be noted, however, that Tellegen subsequently expanded his model into a “Big Four” scheme by subdividing positive emotionality into its “agentive” (i.e., dominant and assertive) and “communal” (i.e., sociable and affiliative) forms (see Patrick, Curtin, & Tellegen, 2002; Tellegen & Waller, 2008). Clark and Watson (1999) established that these models all converged well and defined a single common structure.

The accumulating data indicate that the Big Five and Big Three schemes overlap (Clark & Watson, 1999; Markon et al., 2005). The neuroticism and extraversion dimensions of the Big Five essentially are equivalent to the negative emotionality and positive emotionality factors of the Big Three (Clark & Watson, 1999; Markon et al., 2005; D. Watson et al., 1994). For example, in a sample of 327 students, Clark and Watson (1999) reported corre-

lations of (a) .83 between Big Five neuroticism and Big Three negative emotionality and (b) .78 between Big Five extraversion and Big Three positive emotionality. Clearly, these two dimensions are common to both structural schemes.

The situation involving the final Big Three dimension is more complex. The existing evidence indicates that disinhibition is negatively correlated with both conscientiousness and agreeableness, and includes key aspects of both of these traits. In their analysis, for instance, Clark and Watson (1999) found that Big Three disinhibition correlated $-.54$ and $-.50$ with conscientiousness and agreeableness, respectively. Importantly, approximately 50% of the variance in disinhibition was independent of these Big Five traits. Moreover, disinhibition is an important construct in its own right and has been extensively studied by psychopathology researchers, particularly in relation to SUD and antisocial behavior (Clark & Watson, 2008; Krueger et al., 2007). Finally, openness shows a moderate positive association with positive emotionality, but it is fairly independent from the Big Three (Digman, 1997; Markon et al., 2005).

In sum, these two structural schemes share some elements, but each includes distinct components. To obtain comprehensive coverage of higher order personality dimensions, we examined both models. Hence, six traits were included in the current meta-analysis: neuroticism/negative emotionality, extraversion/positive emotionality, disinhibition, conscientiousness, agreeableness, and openness. Our review is necessarily limited to these broad dimensions because lower order traits have been studied less consistently and the available data are insufficient to permit a comprehensive meta-analysis (D. Watson et al., 2006).

With the consolidation of personality psychology around a consensus framework, evidence of the taxonomy's validity began to accumulate. The Big Three and Big Five models have been replicated in many cultures across the world (Allik, 2005; Barrett, Petrides, Eysenck, & Eysenck, 1998). The appreciable longitudinal stability of personality traits has been confirmed in many long-term studies (Roberts & DelVecchio, 2000). Recent meta-analyses have established that traits contribute substantially to many important outcomes such as academic performance, occupational attainment, divorce, life satisfaction, subjective well-being, physical illness, and longevity (Heller, Watson, & Ilies, 2004; Poropat, 2009; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007; Smith & MacKenzie, 2006; Steel, Schmidt, & Shultz, 2008). Our goal in the current study was to extend this body of knowledge by evaluating links between higher order traits and common Axis I disorders.

Associations Between Traits and Psychopathology

The tripartite model of anxiety and depression (Clark & Watson, 1991) has played a prominent role in shaping work on the associations between personality and Axis I psychopathology. According to this scheme, anxiety and depression are both defined by high levels of negative affect. Furthermore, they are distinguished from each other by two specific factors: positive affect, which is low in depression, and hyperarousal, which is common in anxiety (Clark & Watson, 1991). Negative and positive affect are strongly linked to neuroticism and extraversion, respectively (D. Watson, Wiese, Vaidya, & Tellegen, 1999). Hence, Clark et al. (1994) argued that all anxiety and depressive disorders are associated with neuroticism, but depression is also negatively correlated with extraversion.

Studies have largely supported this model with one notable exception: Low extraversion was found not only in depression but also in social anxiety (Brown, Chorpita, & Barlow, 1998; Kotov et al., 2007; D. Watson, Clark, & Carey, 1988).

Further theoretical developments were spurred by evidence that common mental disorders fall into two broad spectra: internalizing (anxiety and depression) and externalizing (SUD and antisocial behavior). The internalizing spectrum can be further subdivided into a distress cluster, which includes major depressive disorder (MDD), dysthymic disorder, generalized anxiety disorder (GAD), and posttraumatic stress disorder (PTSD); and a fear cluster, which includes panic disorder, agoraphobia, social phobia, and specific phobia (see Krueger & Markon, 2006; D. Watson, 2005b). Internalizing problems have been related to neuroticism, whereas the externalizing spectrum has been linked to elevated levels of both neuroticism and disinhibition (Clark, 2005; Krueger et al., 2007; Krueger, McGue, & Iacono, 2001; D. Watson & Clark, 1993; D. Watson, Gamez, & Simms, 2005). In light of the strong association between neuroticism and distress, recent theories have argued that distress disorders are especially elevated on this trait, followed by the fear disorders and then externalizing problems (D. Watson et al., 2006).

Six major types of models have been proposed to explain the nature of these associations (see Clark, 2005; Krueger & Tackett, 2003; D. Watson & Clark, 1995). The *vulnerability* model postulates that traits contribute etiologically to development of the disorder; that is, it proposes that personality scores can predict who develops the condition among previously unaffected individuals. The *pathoplasty* model holds that traits influence the course and severity of the disorder once it develops. Thus, personality scores are hypothesized to be prognostic of clinical outcomes in people who are already ill. The *scar* model argues that psychopathology permanently changes personality, whereas the *complication* model posits that this change is temporary and lasts only while the illness is active. These models are tested by comparing personality scores obtained before onset of the disorder, during an episode of the illness, and after it fully resolves. In contrast, the *common cause* model argues that personality and psychopathology are associated because they have shared roots, such as common genetic vulnerabilities. The *spectrum* model (a version of it is known as the *precursor* model) posits that disorders and traits are best viewed as different manifestations of the same process. The clearest support for this theory would be indicated by extremely high and specific correlations between relevant traits and disorders, and by prospective evidence that personality pathology invariably precedes development of mental illness. All of these theories have received some empirical support (Bienvenu & Stein, 2003; Christensen & Kessing, 2006; Clark et al., 1994; M. H. Klein, Wonderlich, & Shea, 1993; Ormel, Oldehinkel, & Vollebergh, 2004), but the longitudinal data necessary to contrast them are too sparse to allow meaningful cumulation.

In fact, even the descriptive models described earlier are based on rather limited evidence. Only a handful of articles have attempted a comprehensive review of the links between personality traits and common Axis I disorders (Ball, 2005; Bienvenu & Stein, 2003; Clark et al., 1994; Enns & Cox, 1997). These qualitative reviews concluded that depression is associated with high neuroticism and low extraversion, with the latter effect being somewhat weaker (Clark et al., 1994; Enns & Cox, 1997). All anxiety

disorders were related to neuroticism, but specific phobia was found to have only a modest association with the trait (Bienvenu & Stein, 2003; Clark et al., 1994). In addition, social phobia and agoraphobia were reported to correlate negatively with extraversion. SUD were linked to neuroticism, disinhibition, low conscientiousness, and low agreeableness (Ball, 2005).

Importantly, these reviews focused primarily on the Big Three, and thus less is known about the role of conscientiousness, agreeableness, and openness in major Axis I disorders. Furthermore, these qualitative reviews did not provide precise estimates of effect sizes and did not directly compare trait profiles of different disorders. Hence, the magnitude of personality–psychopathology associations is not known, and even the relative standing of major disorders on these big traits is uncertain. One reason for this limited state of knowledge is the imprecision inherent in qualitative literature reviews. Another reason is the paucity of direct comparisons, as few primary studies examined the associations of personality traits with multiple disorders (those include Bienvenu et al., 2004; Gamez, Watson, & Doebbeling, 2007; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Tackett, Quilty, Sellbom, Rector, & Bagby, 2008; Trull & Sher, 1994; D. Watson et al., 1988; Weinstock & Whisman, 2006). Many more studies simply evaluated individual trait–disorder links. However, it is difficult to make inferences from comparisons across these reports because they frequently differ in sampling and measurement.

Meta-analysis can address these shortcomings of qualitative reviews. It can account for design differences between studies and derive quantitative estimates of effect sizes from all available information. Meta-analysis, of course, has its own limitations (Hunter & Schmidt, 2004). First, it is limited by the state of the literature. For instance, if existing studies are all cross-sectional, the meta-analysis would not be able to examine temporal relations. Second, if the published literature is biased toward a particular conclusion (i.e., a file-drawer effect), this distortion will influence results of the quantitative review unless the unpublished studies are also obtained. Third, meta-analysis often integrates information from studies that vary dramatically in their design and methodological quality, which can dilute true effects. This problem can be addressed by correcting for unreliability of measures and stratifying analyses by relevant characteristics of primary studies. Importantly, most of these concerns also apply to qualitative literature reviews; meta-analysis offers greater rigor in synthesizing the literature and has tools for addressing these limitations.

Three meta-analyses of personality–psychopathology associations have been published to date. Malouff, Thorsteinsson, and Schutte (2005) analyzed data from 33 samples to evaluate links between the Big Five traits and mental disorder. They concluded that mental illness in general is associated with high neuroticism (Cohen's $d = 0.92$), low conscientiousness ($d = -0.66$), low extraversion ($d = -0.41$), and low agreeableness ($d = -0.38$) but not openness ($d = 0.05$). They also investigated the impact of design characteristics on results and found that (a) studies that use the NEO family of personality measures (Costa & McCrae, 1992) report stronger effects and (b) investigations that include a control group produce somewhat smaller estimates. This meta-analysis described the general personality profile associated with mental illness but did not examine individual *DSM* disorders, which may differ substantially on these dimensions. In fact, the study found appreciable trait differences between the major diagnostic classes

(e.g., mood disorders vs. somatoform disorders). Unfortunately, the corresponding groups were too small (two to seven studies) to establish disorder-specific profiles.

Malouff, Thorsteinsson, Rooke, and Schutte (2007) examined the links of the Big Five traits to alcohol involvement, a broad concept that ranges from ever having had alcohol to a diagnosis of alcohol dependence. However, this literature was also reviewed by Ruiz, Pincus, and Schinka (2008), who evaluated associations of these traits with SUD and antisocial personality disorder. We focused on the latter study, as it was more specific to mental illness and included the relevant literature reviewed by Malouff et al. The SUD analysis of Ruiz et al. (2008) was based on 22 samples and showed that these disorders are moderately associated with neuroticism ($r = .26$, which corresponds to $d = 0.54$), low conscientiousness ($r = -.32$ or $d = -0.68$), and low agreeableness ($r = -.20$ or $d = -0.41$) but not extraversion ($r = -.06$ or $d = -0.12$). The correlation with openness was not reported. The antisocial personality disorder analysis was based on 35 samples and revealed a pattern of results similar to that of SUD. Specifically, the disorder was associated with average levels of extraversion ($r = .06$ or $d = 0.12$), low conscientiousness ($r = -.30$ or $d = -0.63$), and low agreeableness ($r = -.38$ or $d = -0.82$). Neuroticism showed essentially no effect ($r = .10$ or $d = 0.20$), however. These parallels between SUD and antisocial personality disorder are consistent with classifying them together in the externalizing category. The present meta-analysis focused on Axis I conditions, however, and thus only SUD were considered in the current study. Ruiz et al. also found that effect sizes are greater in clinical than in community samples.

Unfortunately, the Ruiz et al. (2008) and Malouff et al. (2005) studies, as well as the Malouff et al. (2007) review, were limited to samples that had data on all Big Five traits, which is a small subset of the available literature. Moreover, they included studies of symptoms as well as full *DSM* diagnoses. Both articles found that symptom studies report smaller effect sizes, thus reducing overall estimates of trait–disorder associations.

The Current Study

Our primary aim was to describe patterns of personality–psychopathology associations and estimate their strength as precisely as possible. We sought to extend prior research by examining a broad range of specific mental disorders and including all relevant personality data on these diagnoses. We evaluated the links between the depressive, anxiety, and substance use disorders and the higher order traits (i.e., the Big Five plus disinhibition) because common diagnoses and broad personality dimensions have been the primary focus of this literature. The inclusion of multiple disorders allowed us to examine their trait profiles side by side and make inferences about the differential role of specific personality characteristics in various mental illnesses. We were unable to evaluate causality, as longitudinal data still are too limited, but we hoped to identify candidate traits for research on the etiology of common disorders. Our main objective was to describe the interface of personality and psychopathology. We also evaluated several potential moderators of these associations: population sampled, personality measure used, various characteristics of psychopathology assessment (diagnostic system, diagnosis time frame, and ascertainment method), and whether a control group

was included in the study. All of these design characteristics may affect results of primary investigations and lead to inconsistencies among them. In sum, the present study is the most comprehensive quantitative review of links between traits and common mental disorders to date.

A key analytic decision for our study was whether to cumulate effect sizes as Pearson's r or Cohen's d . Indeed, the Ruiz et al. (2008) meta-analysis chose the former approach, whereas Malouff et al. (2005) used the latter. There are important differences between the two statistics (Hunter & Schmidt, 2004, p. 280). Pearson's r is reduced when comparison groups (disorder and control in this case) are not of equal size, and this reduction is proportionate to the inequality. Cohen's d , on the other hand, is independent of the relative group size. In other words, d describes the difference between diagnostic and control groups in standard units and can be used to construct a personality profile of a disorder. In contrast, r reflects the amount of variance in the trait attributable to the disorder. The former focus is closer to the aims of the present study, as we were interested in trait profiles of psychiatric conditions, and thus data were cumulated with d . Importantly, r is the right statistic for cumulating associations between two continuous variables (with reasonably normal distributions) and is appropriate for meta-analyses that operationalize mental illnesses continuously. However, as mentioned earlier, we chose to focus on dichotomous diagnoses to ensure clear and consistent definition of psychopathology constructs.

Prior research suggested several hypotheses as described in the previous section. First, we expected neuroticism to correlate with all disorders, but to show the strongest links to the distress disorders (i.e., MDD, dysthymic disorder, GAD, and PTSD), followed by the fear disorders (i.e., panic disorder, agoraphobia, social phobia, and specific phobia), and then SUD. Second, we predicted that MDD, dysthymic disorder, social phobia, and agoraphobia would have particularly low extraversion scores. Third, we hypothesized that the personality profile of SUD would be defined by high disinhibition, low conscientiousness, and low agreeableness. Fourth, we anticipated that openness would display relatively weak associations with all disorders considered.

With regard to moderators, we expected to see larger effects in patient populations, based on the data reported by Ruiz et al. (2008). The findings presented by Malouff et al. (2005) led us to predict stronger effects for the NEO family of measures and weaker effects in studies that included a control group. We also hypothesized that current diagnoses would be associated with more extreme trait profiles than lifetime diagnoses, because active mental illness can potentially bias personality assessment toward greater pathology and inflate effect sizes, as described by the complication model (see Clark et al., 1994; D. Watson et al., 2006; Widiger & Smith, 2008). The existing literature did not justify strong hypotheses with regard to the diagnostic system and ascertainment method moderators, but we examined their effects in an exploratory manner.

Method

Inclusion and Exclusion Criteria

Psychiatric classification underwent a dramatic revision with publication of the *DSM-III* in 1980, which overhauled diagnostic

practices and significantly enhanced their reliability. Given our focus on modern personality and psychopathology constructs, the review was limited to the 28-year period starting on January 1, 1980, and ending on December 31, 2007. To ensure precise definition of these constructs, we specified two inclusion criteria. First, the study had to employ a standardized and validated personality measure that mapped clearly onto our target higher order dimensions. Scales tapping only a specific component of the general trait were not accepted. Second, ascertainment of diagnoses had to be done by a trained rater according to one of the modern classification systems, namely the *DSM-III*, *DSM-III-R*, *DSM-IV*, *ICD-9*, *ICD-10*, or Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1975). Self-report diagnostic instruments were not included. Also, we restricted our review to analyses of adult personality measures, because trait scores have markedly different properties in childhood, including substantially lower temporal stability (Roberts & DelVecchio, 2000). We confirmed this by reviewing age ranges of all included articles. Furthermore, only English-language reports were considered. Studies were excluded if we could not obtain information necessary for the computation of effect sizes either from the article or from the authors. We were able to compute effect sizes from any of the following statistics: Cohen's d , Pearson's r , t or F statistic, and means and standard deviations of personality scales in diagnostic groups. The latter was by far the most common type of information available. Finally, we excluded samples that had fewer than 15 people with a target disorder, as we judged the informational value of such data to be too limited to warrant analysis.

Literature Search

We obtained studies using five search strategies. First, we searched three online databases—PsycINFO, Medline, and Dissertation Abstracts—using a combination of trait and disorder names. Trait terms included names of common models, various labels for the six traits, and names and acronyms of standard personality inventories, specifically *Big Five*, *Five Factor Model*, *Big Three*, *Three Factor Model*, *NEO-FFI*, *NEO-PI*, *NEO-PI-R*, *Big Five Inventory*, *BFI*, *Goldberg*, *Eysenck Personality Questionnaire*, *EPQ*, *Multidimensional Personality Questionnaire*, *MPQ*, *Schedule for Nonadaptive and Adaptive Personality*, *SNAP*, *General Temperament Survey*, *GTS*, *California Psychological Inventory*, *CPI*, *neuroticism*, *extraversion*, *openness*, *agreeableness*, *conscientiousness*, *negative emotionality*, *negative temperament*, *positive emotionality*, *positive temperament*, *psychoticism*, and *disinhibition*. Disorder terms included labels commonly applied to the target disorders, specifically *mood disorder*, *anxiety disorder*, *depression*, *dysthymia*, *dysthymic disorder*, *generalized anxiety disorder*, *GAD*, *posttraumatic stress disorder*, *post-traumatic stress disorder*, *PTSD*, *social phobia*, *panic disorder*, *agoraphobia*, *specific phobia*, *simple phobia*, *obsessive-compulsive disorder*, *OCD*, *externalizing*, *alcohol abuse*, *substance abuse*, *alcohol dependence*, *drug dependence*, and *substance dependence*. We combined these two sets of terms and limited the results to the English language and 1980–2007 period, which yielded 7,156

abstracts.¹ Some of these were redundant selections made by different search engines, however.

We reviewed each abstract, eliminating studies that clearly did not collect diagnostic or personality data, and screened the remaining articles. We similarly examined all articles included in the previous two meta-analyses (Malouff et al., 2005; Ruiz et al., 2008). We also searched reference sections of all selected articles and identified 416 additional potentially relevant reports. Selected studies were most commonly published in the *Journal of Abnormal Psychology*, *Journal of Affective Disorders*, *Personality and Individual Differences*, and *Psychiatry Research*. Hence, we also reviewed all issues of these four journals published since January 2000. Moreover, we posted requests for information on three listservs: *Psychiatry Research*, *Society for a Science of Clinical Psychology*, and *Society for Personality and Social Psychology*. Finally, we contacted 54 research teams requesting unpublished data or information missing from their published reports. This produced another 17 potentially relevant studies. Thus, 7,589 abstracts were reviewed in total (see Figure 1 for derivation of the analysis sample).

This broad screening identified 426 potentially eligible studies. A number of them were eliminated after review of the full text of the articles: Eighty-two did not meet our criteria, 106 were redundant with another article already included in the database, and necessary data could not be obtained for 63 even after contacting the authors. The remaining 175 studies were included in the meta-analysis. However, 86 of them did not have a healthy control group and thus lacked a reference necessary for computation of effect sizes.

To be as inclusive as possible, we searched for control groups and used them to compute missing effect sizes. These samples came from three sources. First, we examined articles already included in the meta-analysis for a matching control group. Studies reporting on the disorder in question were excluded from the search, and thus no control group was used more than once in a given analysis. This approach produced reference data for 27 samples. Second, we obtained normative data from manuals of relevant measures, which yielded 15 additional control groups representative of the general population. Third, we searched the

literature for large representative community studies using names of relevant measures as keywords. This approach yielded 95 reports, 44 of which provided control data. A control group was matched to a given study according to the following procedure. First, we identified control samples that were drawn from the same population (e.g., general population, students, medical patients, or veterans) and completed the same personality measure administered in the same language. Then, if more than one study was available, we matched further on basic gender and age distributions. If multiple samples fit these criteria, we selected the largest one.

Data Coding

Some studies compared diagnostic groups on multiple personality measures. To maintain independence of observations, we included only one effect size per trait–disorder comparison. Specifically, we selected the measure that assessed the most traits. For instance, if patients with MDD were compared with a control group on the Revised NEO Personality Inventory (NEO-PI-R) and on another measure of neuroticism, we chose the former, as it taps five relevant traits. We adopted this approach to reduce heterogeneity of estimates resulting from methodological differences. A number of articles reported effect sizes for both concurrent and delayed associations (i.e., the correlation of Time 1 diagnostic status with both Time 1 and Time 2 personality scores). In such situations, we recorded only concurrent associations, as all other studies were concurrent and inclusion of delayed effects would introduce additional heterogeneity. Prospective relations can be especially informative and thus warrant a separate analysis. Unfortunately, only five of the included studies reported such data, and the length of delay varied considerably, which made meaningful cumulation of these effect sizes impossible. Finally, several articles reported trait–disorder associations in multiple samples. If a separate control group was available in each sample, these comparisons were considered independent observations. Otherwise, the data were aggregated with formulas that follow directly from the analysis-of-variance approach to partitioning variance (Kirk, 1995):

$$M_a = \frac{N_1 * M_1 + N_2 * M_2}{N_1 + N_2}$$

$$SD_a = \sqrt{\frac{(N_1 - 1)SD_1^2 + (N_2 - 1)SD_2^2}{(N_1 - 1) + (N_2 - 1)} + \frac{(N_1 - 1)(M_1 - M_a)^2 + (N_2 - 1)(M_2 - M_a)^2}{(N_1 - 1) + (N_2 - 1)}}$$

In these formulas, N is size of a diagnostic group, M is the mean, and SD is the standard deviation of a personality scale in that group. Subscripts 1, 2, and a indicate Sample 1, Sample 2, and their aggregate, respectively. Next, the aggregated data were com-

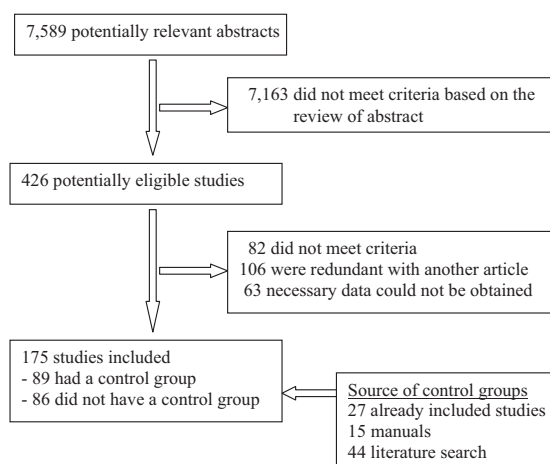


Figure 1. Derivation of the analysis sample.

¹ An abstract search with the same criteria indicated that relevant literature increased by 22% over the January 2008–April 2010 period. These studies could not be included because of the time lag inherent in research and in the publication process, but this increase is unlikely to have resulted in material changes to the present findings.

pared with the reference data (from the internal or assigned control group), resulting in one effect size per trait.

Initially, we planned to analyze data for substance dependence and substance abuse separately. However, we found only two articles that specifically examined abuse and were eligible for inclusion. Hence, we analyzed all data on SUD under one rubric. We also considered a general category of “anxiety disorder” for studies that reported on a diagnostic group composed of multiple anxiety disorders and did not segregate them further. However, only two such articles were found, which were too few to analyze. In contrast, the number of depression studies was sufficient to examine the general diagnostic group of *unipolar depression*—for studies that did not distinguish between different depressive disorders—as well as specific dysthymic disorder and MDD diagnoses (number of studies $K = 18, 15,$ and $65,$ respectively). Hence, we analyzed each of these three categories.

From each article we coded data necessary for computation of effect sizes, sizes of diagnostic groups, and six study characteristics that we expected to moderate associations: personality measure, sample type, diagnostic system used, method of diagnosis, diagnosis time frame, and control group status (i.e., whether the article included a reference group or we had to locate control data elsewhere). We observed a large variety of designs among the studies and grouped them into a smaller number of conceptual categories to obtain a sufficient number of effect sizes in each category across the analyses. Hence, personality measure was coded as NEO family (NEO-PI [Costa & McCrae, 1985], NEO-PI-R [Costa & McCrae, 1992], or NEO Five-Factor Inventory [NEO-FFI; Costa & McCrae, 1992]), Eysenck’s inventories (Maudsley Personality Inventory [H. J. Eysenck, 1959], Eysenck Personality Inventory [H. J. Eysenck & Eysenck, 1964], Eysenck Personality Questionnaire [H. J. Eysenck & Eysenck, 1975], or Eysenck Personality Questionnaire–Revised [H. J. Eysenck & Eysenck, 1991]), or other. Sample was coded as patient, epidemiologic, or other. Diagnostic system was coded as *DSM-IV/ICD-10* or earlier. Method of diagnosis was coded as the Structured Clinical Interview for *DSM* (SCID; First, Spitzer, Gibbon, & Williams, 1995), completely structured interview (e.g., Diagnostic Interview Schedule [Robins, Helzer, Croughan, & Ratcliff, 1981], Composite International Diagnostic Interview [Kessler & Üstün, 2004], Schedules for Clinical Assessment in Neuropsychiatry [Wing et al., 1990], and Diagnostic Interview Schedule for Children [Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000]), informal-clinical diagnosis, or other. Time frame was coded as current or broader (e.g., 12-month, lifetime). For SUD, we also coded the substance of choice for the sample as primarily alcohol, primarily drugs, or mixed. Finally, we recorded the reliability of personality scales (Cronbach’s α) in the study sample whenever it was available. We also attempted to record data on the reliability of diagnoses, but such information was reported in only five studies, and therefore we could not use it.

Study information was coded by advanced undergraduate research assistants and reviewed by one of the authors. All inconsistencies were discussed until consensus was achieved. To evaluate the reliability of the resulting ratings, we blindly recoded 25 articles. The agreement was perfect for effect sizes, means, standard deviations, group sizes, and reliability estimates. Interrater reliability also was very high for the moderator variables, with a kappa of 1.00 for measure, .90 for sample, .86 for diagnostic

system, .86 for method, 1.00 for time frame, and 1.00 for control group status. Reliability of diagnostic group assignment was just as high ($\kappa = .89$), and all disagreements concerned depressive disorders. Specifically, two studies initially were assigned to unipolar depression analysis, but recoding revealed that they were better classified under MDD. Data on individual studies are reported in Table 1. Distribution of reliability estimates is presented in Table 2.

Statistical Analyses

We converted all effect size information to d s prior to analysis using standard formulas (Hunter & Schmidt, 2004). Conventionally, d of less than $|0.50|$ is considered a small effect size; d in the $|0.50| - |0.79|$ range is viewed as medium, whereas d of $|0.80|$ or greater is large (J. Cohen, 1988). In the computation of d , group membership was coded so that 1 indicated a diagnostic group and 0 indicated a healthy control group. Hence, a positive effect size implies that the diagnostic group is elevated on the trait in question. If a scale was keyed in direction opposite to that of the trait (e.g., the Constraint scale of the Multidimensional Personality Questionnaire [Tellegen, 1982] taps the low end of disinhibition), the sign of the corresponding effect size was reversed.

The meta-analyses followed Hunter and Schmidt (2004) procedures. We used a random-effects model, which is a recommended meta-analytic approach because it takes into account true differences among studies as well as differences among participants (National Research Council, 1992; Schmidt, Oh, & Hayes, 2009). In other words, the random-effects model allows the true effect size to differ across studies. Hence, each analysis produced an average effect size and an 80% credibility interval (CrI). This CrI defines the range within which the true effect sizes of 80% of the studies fall. Thus, we were able to describe the distribution of true associations, rather than just the average estimate and its precision. To determine whether true differences are appreciable, we estimated their contribution to the observed variability among effect sizes. If this contribution is less than 25% of the total variance, the population of studies can be considered essentially homogenous (Hunter & Schmidt, 2004), and moderators would not be hypothesized. We also sought to adjust effect sizes for unreliability but could not correct them individually, as reliability information was often unavailable. Consequently, we corrected overall estimates using artifact distribution (i.e., the distribution of all available α estimates) for a given trait, which is a standard procedure for such cases (Hunter & Schmidt, 2004, pp. 137–188).

First, we conducted 66 meta-analyses to describe the associations between the six traits and the 11 disorders (we also conducted supplemental analyses of the SUD subgroups). Second, we adjusted the resulting effect sizes on extraversion, disinhibition, conscientiousness, agreeableness, and openness for the potentially confounding effects of neuroticism. Specifically, we converted Cohen’s d s into Pearson’s r s using formulas that account for uneven sizes of the disorder and control groups (Hunter & Schmidt, 2004, p. 284). Next, we computed partial correlations using estimates of trait intercorrelations from Markon, Krueger, and Watson’s (2005) meta-analysis. We then converted these partial correlations back to d s. Third, we performed moderator analyses of 30 unadjusted estimates that were based on a sufficiently large number of studies. Specifically, we stratified effect sizes by each moderator in turn and carried out a meta-analysis

Table 1
Summary of Effect Size Estimates and Study Characteristics

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time		
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>						frame	Control	Drug
Major depressive disorder														
Aben et al. (2002) ^a	41			68				1	3	1	1	1	1	0
N		33.50	7.30		28.30	6.40	0.77							
E		37.60	6.50		40.10	4.80	-0.45							
O		31.20	6.30		33.60	6.10	-0.39							
A		44.50	5.70		43.50	5.00	0.19							
C		44.80	5.90		45.50	5.30	-0.13							
Abou-Saleh & Coppen (1984); Damas-Mora et al. (1982)	55			57				2	1	2	3	1	1	
N		12.06	1.99		7.78	5.06	1.11							
E		9.69	1.51		13.19	4.83	-0.97							
D		2.02	0.68		3.69	4.30	-0.54							
Aggen et al. (2005)	211			1,870				2	2	2	1	2	2	0
N		7.50	3.20		5.50	3.20	0.63							
E		5.30	2.50		5.20	2.40	0.04							
Akiskal et al. (1995); Hirschfeld et al. (1989)	447			370				3	1	2	4	1	1	0
N		32.50	11.30		11.50	9.60	1.99							
E		14.30	7.20		19.50	6.00	-0.78							
K. W. Anderson & McLean (1997); Piedmont (1993)	63			36				1	1	2	1	1	1	1
C		40.80	11.90		47.30	10.30	-0.57							
Andrews & Slade (2002) ^a	409			9,538				2	2	1	2	2	2	0
N		6.80	2.45		2.27	3.22	1.42							
Angst (1998) ^b	151			283				3	2	2	4	2	2	0
N		17.80	6.40		13.50	5.50	0.74							
E		18.30	7.60		20.20	7.90	-0.24							
A		18.30	7.40		15.90	6.90	-0.34							
Bagby et al. (1996); Brummett et al. (2003) ^a	100			99				1	1	2	4	1	1	1
N		120.11	25.50		61.72	19.29	2.58							
E		89.74	25.38		109.16	17.68	-0.89							
O		121.32	19.59		112.49	16.49	0.49							
A		115.83	17.21		132.67	14.54	-1.06							
C		101.34	24.02		125.90	17.90	-1.16							
Bagby & Rector (1998) ^a ; Costa & McCrae (1985)	146			632				1	1	2	4	1	1	1
N		123.40	23.50		77.73	20.68	2.15							
E		85.70	22.10		101.24	17.20	-0.85							
O		115.30	19.00		109.46	17.22	0.33							
A		46.80	8.40		48.97	9.16	-0.24							
C		41.40	10.80		52.91	9.49	-1.18							
Bech et al. (1986) ^a ; Mortensen (2006)	73			450				2	1	2	3	1	1	1
N		13.80	6.03		5.93	4.72	1.60							
E		9.40	4.57		14.58	4.50	-1.15							
D		3.73	2.40		3.40	2.03	0.16							
Berlanga et al. (1999); Fullana et al. (2004) ^a	42			40				2	1	1	3	1	1	1
N		15.02	3.66		8.97	5.47	1.31							
E		7.62	4.29		14.67	2.59	-1.98							
D		5.24	3.47		1.43	1.30	1.44							
Bienvenu et al. (2004) ^a	133			297				1	2	2	2	2	2	0
N		91.83	20.87		73.52	18.47	0.95							
E		104.08	18.20		108.57	15.25	-0.28							
O		105.83	15.71		103.03	15.79	0.18							
A		124.87	15.11		123.30	15.35	0.10							
C		113.08	18.17		119.09	16.92	-0.35							

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
Booij & Van der Does (2007) ^a ; Sanderman et al. (1995)	39			475				2	1	1	1	2	1	
N		6.45	3.44		4.40	3.20	0.64							
E		6.18	3.27		6.70	3.00	-0.17							
D		2.30	1.34		2.00	1.40	0.22							
Bos et al. (2006); Sanderman et al. (1995)	100			849				2	1	1	2	2	1	
N		6.50	3.30		4.10	3.30	0.73							
E		6.90	3.80		7.20	3.20	-0.09							
Brieger et al. (2003) ^a ; Körner et al. (2002)	107			1,908				1	1	1	1	1	1	
N		41.43	8.13		31.44	7.44	1.34							
E		32.72	7.00		38.40	6.00	-0.94							
O		38.68	5.36		36.60	5.52	0.38							
A		42.27	4.55		42.48	5.64	-0.04							
C		42.90	6.24		44.52	6.60	-0.25							
Brown (2007) ^a ; J. Gomez (1984)	160			20				2	1	1	4	1	1	
N		18.33	3.84		10.60	3.40	2.04							
Buckley et al. (1999)	20			15				2	1	2	3	1	0	
N		19.10	2.90		6.70	6.00	2.76							
E		8.10	5.10		12.60	6.10	-0.81							
D		3.40	2.60		3.10	2.70	0.11							
Chapman et al. (2007) ^a	19			343				1	3	1	1	1	0	
N		59.21	9.61		43.72	9.25	1.67							
E		40.53	11.07		52.19	9.49	-1.22							
O		51.58	10.87		49.88	9.43	0.18							
A		48.05	11.48		56.23	9.28	-0.87							
C		41.42	11.58		50.18	9.62	-0.90							
Clark et al. (2003); Clark et al. (1996) ^a	148			74				3	1	1	1	1	1	
N		20.50	5.40		11.70	7.18	1.45							
E		10.40	5.60		18.16	6.42	-1.32							
D		8.80	5.20		10.39	5.45	-0.30							
Cutrona et al. (2005) ^a	47			702				3	2	1	2	2	0	
N		7.02	4.07		4.22	3.52	0.79							
E		9.70	2.77		10.72	2.59	-0.39							
D		3.77	2.28		2.85	2.06	0.44							
Davidson et al. (1985) ^a ; Brodaty et al. (2004)	39			61				2	1	2	3	1	1	
N		12.30	5.00		3.70	2.20	2.41							
De Fruyt et al. (2006) ^{a,b} ; Rolland & Mogenet (2001)	599			1,958				3	1	1	3	1	1	
N		29.44	8.95		39.26	10.36	0.98							
E		43.95	9.71		41.85	9.51	-0.22							
O		41.60	9.30		47.59	7.12	-0.78							
A		45.57	8.03		48.72	7.64	-0.41							
C		47.82	9.84		50.03	9.53	-0.23							
Du et al. (2002)	53			53				1	1	1	3	1	0	
N		34.30	6.53		13.20	7.25	3.06							
E		18.70	8.68		30.10	7.16	-1.43							
O		27.30	7.18		30.80	5.96	-0.53							
A		28.90	6.11		34.60	6.08	-0.94							
C		27.80	8.01		35.00	6.48	-0.99							
Duberstein et al. (2001); Savla et al. (2007)	77			234				1	1	2	1	1	1	
N		108.80	25.30		85.68	16.17	1.23							
E		93.20	21.70		106.56	13.87	-0.83							
O		99.30	17.00		104.62	11.99	-0.40							
A		125.00	17.60		122.06	13.40	0.20							
C		112.30	21.10		116.73	14.68	-0.27							
Duggan et al. (2003) ^a ; R. J. King et al. (1988)	263			43				2	1	2	3	1	1	
N		15.50	5.00		6.80	4.50	1.76							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Ellenbogen & Hodgins (2004) ^a	21			132											
N		86.76	26.88		71.48	18.74	0.76		1	1	2	1	1	0	
E		103.00	16.78		111.02	13.67	-0.57								
O		108.33	12.96		111.92	14.69	-0.25								
A		125.67	15.20		126.81	12.63	-0.09								
C		119.43	21.94		125.06	12.83	-0.39								
Enns et al. (2000)	94			112					1	1	1	1	1	0	
N		36.54	7.07		20.73	8.04	2.08								
E		16.66	7.58		30.26	6.57	-1.93								
O		26.52	6.70		29.80	6.85	-0.48								
A		29.09	6.80		31.82	6.14	-0.42								
C		25.80	7.85		34.50	6.54	-1.21								
Enns & Cox (2005); Egan et al. (2000)	171			1,025					1	1	1	3	1	1	
N		37.66	6.02		19.50	8.60	2.19								
Fountoulakis et al. (2007) ^a	40			120					2	1	1	2	1	0	
N		14.05	5.63		8.75	5.45	0.96								
E		11.91	4.83		14.70	3.93	-0.67								
D		3.82	2.15		3.75	3.58	0.02								
Freire et al. (2007)	45			30					3	1	1	1	1	0	
N		34.70	9.40		23.90	10.70	1.09								
E		24.70	8.60		30.20	7.80	-0.66								
Friedman-Wheeler (2006)	25			46					1	3	2	1	2	0	
N		37.81	9.03		27.62	8.20	1.20								
Gamez et al. (2007)	52			402					3	3	1	1	1	0	
N		62.65	8.39		46.61	9.10	1.78								
E		39.32	11.29		49.18	9.64	-1.00								
D		51.60	9.57		48.05	8.36	0.42								
Grace & O'Brien (2003)	63			40					2	3	1	3	1	0	
N		15.03	5.45		6.10	4.01	1.81								
E		7.88	5.26		12.40	4.97	-0.88								
D		2.81	2.57		2.00	4.09	0.25								
Hecht et al. (1998) ^c	48			48					3	1	2	1	1	0	
N							0.92								
E							-0.30								
Heerlein et al. (1996)	27			21					3	1	1		1	0	
N		13.40	7.10		7.10	5.90	0.95								
E		14.80	6.10		15.60	6.20	-0.13								
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	885			3,468					1	1	1	4	2	1	
N		126.65	21.78		82.92	24.12	1.85								
E		84.67	24.11		118.50	20.00	-1.62								
O		102.10	23.95		117.97	19.48	-0.78								
A		131.72	19.08		121.22	15.56	0.64								
C		98.08	25.04		115.27	19.09	-0.84								
Ignjatovic & Svrakic (2003) ^a	30			30					1	1	1	1	1	0	
N		103.20	17.14		83.10	14.76	1.26								
E		100.83	15.59		110.30	14.65	-0.63								
O		106.30	16.69		111.00	12.86	-0.32								
Jain et al. (1999); Balch & Scott (2007)	24			33					1	1	2	1	1	1	
N		60.30	12.20		44.02	9.80	1.50								
E		41.90	12.10		61.33	8.68	-1.89								
O		62.30	8.50		48.94	11.37	1.30								
A		45.40	11.50		46.48	10.91	-0.10								
C		41.00	11.20		50.48	11.04	-0.85								
Kendler et al. (2007) ^a	4,400			14,876					2	2	1	2	2	0	
N		3.66	2.43		2.44	2.15	0.55								
D. N. Klein et al. (1988); A. C. King et al. (2003)	35			11					2	1	2	4	1	1	
E		11.90	5.40		14.60	3.70	-0.54								

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
Krueger (1999) ^b	157			543				3	2	2	2	2	0	
N		0.34	1.07		-0.29	0.87	0.68							
E		-0.17	1.00		0.09	0.99	-0.27							
D		-0.09	1.08		0.09	0.97	0.19							
Krueger et al. (2001) ^b	823			1,007				3	2	2	1	2	0	
N		83.79	13.94		78.16	12.53	0.43							
E		118.90	13.76		121.65	13.07	-0.20							
D		147.18	15.28		148.57	14.61	0.09							
Lehman et al. (1997)	144			51				3	1	2	4	1	0	
N		138.40	14.03		116.78	12.33	1.59							
E		142.14	14.26		155.84	10.42	-1.03							
D		165.98	14.55		159.52	13.85	0.45							
Lyness et al. (1998) ^a	18			181				1	3	2	1	1	0	
N		27.78	4.82		15.59	7.98	1.57							
E		21.67	7.39		27.88	5.85	-1.03							
O		24.11	5.45		25.56	5.55	-0.26							
A		31.39	7.11		34.71	5.45	-0.59							
C		28.50	8.33		34.51	5.97	-0.97							
McBride et al. (2005) ^a ; Siegler & Brummett (2000)	959			2,379				1	1	1	1	1	1	
N		114.60	26.17		76.77	22.05	1.62							
E		90.86	22.66		110.69	19.14	-0.98							
O		113.82	20.64		112.50	19.90	0.07							
A		121.70	18.67		123.19	15.80	-0.09							
C		103.32	23.56		125.92	18.30	-1.13							
McGlashan et al. (2000) ^a ; Clark et al. (2009)	559			561				3	1	2	1	2	1	
D		11.19	5.71		8.80	5.80	0.42							
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	559			1,638				1	1	2	1	2	1	
N		119.28	24.01		74.55	20.25	2.10							
E		92.30	22.44		110.12	18.60	-0.91							
O		117.12	21.86		114.30	18.81	0.14							
A		115.10	19.46		124.50	15.35	-0.57							
C		100.72	24.81		123.30	17.81	-1.14							
McWilliams et al. (2003) ^a ; Murray et al. (2003)	298			527				1	1	1	3	1	1	
N		36.13	7.08		17.80	8.20	2.35							
E		18.02	7.38		28.30	6.60	-1.49							
O		26.12	7.10		28.70	6.60	-0.38							
A		29.82	6.78		32.60	5.60	-0.46							
C		25.93	8.79		34.10	6.50	-1.10							
Middeldorp et al. (2006) ^a	191			1,057				3	2	1	2	2	0	
N		70.70	25.90		46.10	24.20	1.01							
E		55.80	16.50		60.70	15.60	-0.31							
Miller et al. (2004) ^{a,b}	34			332				3	3			1	0	
N		50.21	7.00		42.39	9.27	0.86							
E		46.96	9.54		56.29	8.33	-1.10							
D		50.66	9.56		52.34	10.06	0.17							
Mongrain & Leather (2006) ^a ; D. Watson et al. (2004)	166			580				3	3	1	1	2	1	
N		26.24	6.24		22.22	7.00	0.59							
E		25.36	7.28		28.12	6.46	-0.41							
O		40.20	6.00		38.87	6.00	0.22							
A		33.12	6.12		35.36	5.31	-0.41							
C		34.29	6.21		34.17	5.89	0.02							
Mulder & Joyce (2002); Dunbar & Lishman (1984)	48			30				2	1	2	1	1	1	
N		17.00	4.80		9.50	4.40	1.61							
E		8.20	5.10		11.40	4.70	-0.65							
D		5.50	4.10		2.90	2.60	0.72							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Nowakowska et al. (2005)	25			47											
N		97.40	19.70		66.00	18.70	1.65	1	1	1	1	1	1	0	
E		105.40	22.80		116.10	16.40	-0.57								
O		127.20	23.80		124.20	19.00	0.14								
A		115.20	15.60		123.00	12.90	-0.56								
C		105.70	18.40		122.90	16.00	-1.02								
Painuly et al. (2007)	40			20				3	1	1	4	1	1	0	
N		0.78	1.16		-0.60	1.17	1.18								
E		-0.44	1.36		-0.46	1.24	0.02								
Petersen et al. (2001); Tokar et al. (1999)	76			485				1	1	2	1	1	1	1	
N		64.55	11.49		50.09	10.92	1.31								
E		35.00	11.75		52.34	11.98	-1.45								
O		49.90	12.84		51.51	11.80	-0.13								
A		46.89	14.64		49.84	12.56	-0.23								
C		37.47	15.17		50.46	11.46	-1.08								
Pickering et al. (2003)	108			105				2	1	1	2	1	1	0	
N		19.57	3.64		8.72	5.02	2.48								
E		8.86	5.82		14.98	4.92	-1.13								
D		5.56	3.85		4.24	2.97	0.38								
Roy (1998)	97			56				2	1	2	3	1	1	0	
N		15.66	5.68		5.40	4.20	1.98								
E		11.04	5.42		13.60	4.40	-0.51								
D		4.09	3.35		3.00	2.10	0.37								
Rytsälä et al. (2006) ^a	264			437				2	1	1	2	1	1	0	
N		17.37	3.96		9.35	5.20	1.68								
Sauer et al. (1997); Maier et al. (1995)	90			228				3	1	2	1	1	1	1	
N		11.40	5.90		7.30	5.40	0.74								
E		9.90	6.50		9.80	5.10	0.02								
Scheibe et al. (2003); Sen et al. (2004)	289			340				1	1	2	1	1	1	1	
N		124.84	22.75		85.15	20.47	1.84								
E		86.54	22.50		108.07	17.52	-1.08								
O		116.24	20.03		103.63	17.15	0.68								
A		45.11	8.97		48.25	8.30	-0.36								
C		40.32	10.82		47.16	6.45	-0.78								
Scott et al. (1995); Lamey et al. (2006)	20			18				2	1	2	3	1	1	1	
N		14.00	4.50		7.55	5.22	1.33								
Stanković et al. (2006)	35			20				1	1	1	3	2	2	0	
N		120.77	25.19		80.75	22.27	1.66								
E		77.91	18.27		108.85	12.92	-1.87								
O		93.09	20.66		113.65	17.15	-1.06								
A		123.80	18.98		127.10	17.47	-0.18								
C		109.20	19.09		130.75	17.83	-1.16								
Strong (2003)	25			47				1	1	1	1	1	1	0	
N		97.36	19.69		65.98	18.70	1.65								
E		105.40	22.82		116.06	16.35	-0.57								
O		127.20	23.81		124.23	19.03	0.14								
Trull & Sher (1994) ^a	38			280				1	3	2	2	2	2	0	
N		25.24	8.66		16.76	6.72	1.21								
E		25.61	8.83		31.57	5.88	-0.95								
O		32.18	6.23		28.17	6.10	0.66								
A		30.50	6.57		32.99	5.72	-0.43								
C		25.71	6.75		32.35	6.28	-1.05								
D. Watson (2005a)	324			3,854				1	2	2	2	1	1	0	
N		25.57	6.06		18.45	4.98	1.40								
E		26.72	5.38		28.97	4.79	-0.47								
O		28.38	4.61		28.43	4.37	-0.01								

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Schrader (1994); Hirschfeld et al. (1989)	52			370											
N		34.70	10.40		11.50	9.60	2.39	2	1	2	1	2	1		
Schreindorfer (2002); Phillips et al. (2006)	15			112											
N		71.28	10.95		52.16	10.47	1.82	1	3	1	1	1	1		
E		32.31	13.43		54.39	11.23	-1.92								
O		58.79	10.50		54.74	11.73	0.35								
A		39.96	11.77		47.95	12.35	-0.65								
C		33.78	15.00		49.88	11.12	-1.38								
Spijkerman et al. (2005) ^a	119			349											
N		6.20	3.40		2.50	2.70	1.28	2	3	1	2	1	0		
E		6.00	2.80		6.40	207.0	0.00								
Williams et al. (2007)	22			74											
N		9.20	2.90		5.90	3.30	1.03	2	3	1	3	2	0		
E		4.20	3.70		7.90	3.70	-1.00								
D		2.00	1.30		1.80	1.60	0.13								
Wise et al. (1995); Tokar et al. (1999) ^a	50			485											
N		77.50	25.70		50.09	10.92	2.10	1	1	2	4	1	1		
E		32.20	33.20		52.34	11.98	-1.32								
O		55.70	30.10		51.51	11.80	0.29								
A		46.40	35.30		49.84	12.56	-0.21								
C		21.50	26.50		50.46	11.46	-2.13								
Dysthymic disorder															
Andrews & Slade (2002) ^a	110			9,538											
N		7.80	2.57		2.27	3.22	1.72	2	2	1	2	2	0		
Angst (1998)	36			283											
N		23.90	6.20		13.50	5.50	1.86	3	2	2	4	2	0		
E		13.80	6.30		20.20	7.90	-0.83								
A		21.20	7.40		15.90	6.90	0.76								
Bienvenu et al. (2004) ^a	18			297											
N		94.50	15.64		73.52	18.47	1.14	1	2	2	2	2	0		
E		90.83	14.52		108.57	15.25	-1.17								
O		103.72	13.06		103.03	15.79	0.04								
A		127.78	10.70		123.30	15.35	0.30								
C		107.44	12.69		119.09	16.92	-0.70								
Bijl et al. (1998) ^a	194			5,492											
N		12.35	6.34		3.00	3.39	2.65	3	2	2	2	2	0		
Brown (2007) ^a ; Emery et al. (1996)	56			3,084											
N		17.86	3.54		9.40	5.20	1.63	2	1	1	4	1	1		
Hayden & Klein (2001) ^a ; H. J. Eysenck & Eysenck (1975)	83			5,574											
N		17.81	3.76		11.53	5.39	1.17	2	1	2	1	1	1		
E		8.76	5.23		12.84	4.87	-0.84								
D		6.82	3.65		3.11	2.75	1.34								
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	358			3,468											
N		125.24	22.43		82.92	24.12	1.77	1	1	1	4	2	1		
E		80.13	21.81		118.50	20.00	-1.90								
O		101.63	22.29		117.97	19.48	-0.83								
A		130.44	19.06		121.22	15.56	0.58								
C		94.22	24.49		115.27	19.09	-1.07								
Katon et al. (2002); Egan et al. (2000)	282			1,025											
N		25.44	7.80		19.50	8.60	0.70	1	3	2	4	1	1		
D. N. Klein et al. (1988); S. B. G. Eysenck et al. (1980)	32			654											
E		8.00	5.50		13.51	4.78	-1.14	2	1	2	4	1	1		
McGlashan et al. (2000) ^a ; Clark et al. (2009)	119			561											
D		11.29	5.39		8.80	5.80	0.43	1	1	2	1	2	1		

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	119			1,638				1	1	2	1	2	1	
N		127.50	18.95		74.55	20.25	2.63							
E		85.68	19.36		110.12	18.60	-1.31							
O		113.95	20.95		114.30	18.81	-0.02							
A		110.03	18.75		124.50	15.35	-0.93							
C		98.82	23.71		123.30	17.81	-1.34							
Middeldorp et al. (2006) ^a	28			1,220				3	2	1	2	2	0	
N		84.50	25.00		49.10	25.50	1.39							
E		46.90	16.90		60.30	15.70	-0.85							
Oxman et al. (2001) ^a ; Murray et al. (2003)	169			527				1	3	1	4	1	1	
N		25.42	7.98		17.80	8.20	0.94							
Roy et al. (1985)	11			11				2	1	2	3	1	0	
N		15.30	4.60		4.30	3.40	2.72							
E		11.40	4.90		14.60	3.70	-0.74							
D		3.20	2.00		2.50	1.60	0.39							
D. Watson (2005a)	114			3,854				1	2	2	2	1	0	
N		26.05	6.40		18.45	4.98	1.51							
E		25.75	5.33		28.97	4.79	-0.67							
O		28.24	4.78		28.43	4.37	-0.04							
				Generalized anxiety disorder										
Andrews & Slade (2002) ^a	335			9,538				2	2	1	2	2	0	
N		7.44	2.84		2.27	3.22	1.61							
Bienvenu et al. (2004) ^a	32			297				1	2	2	2	2	0	
N		94.06	21.56		73.52	18.47	1.09							
E		104.38	22.06		108.57	15.25	-0.26							
O		105.91	15.97		103.03	15.79	0.18							
A		128.16	16.92		123.30	15.35	0.31							
C		108.47	18.01		119.09	16.92	-0.62							
Bijl et al. (1998) ^a	81			5,492				3	2	2	2	2	0	
N		10.87	7.30		3.00	3.39	2.26							
Brown (2007) ^a ; Cramer (1993)	132			1,455				2	1	1	4	1	1	
N		17.75	3.37		8.06	5.06	1.96							
Gamez et al. (2007)	39			402				3	3	1	1	1	0	
N		62.31	8.21		46.61	9.10	1.74							
E		39.10	11.70		49.18	9.64	-1.02							
D		51.24	8.77		48.05	8.36	0.38							
R. Gomez & Francis (2003)	40			40				2	3	1	3	1	0	
N		11.17	2.42		2.42	1.77	4.13							
E		3.48	2.34		8.18	2.30	-2.03							
Hoehn-Saric et al. (1993); R. J. King et al. (1988)	103			43				2	3	2	1	1	1	
N		17.42	4.08		6.80	4.50	2.52							
Hummelen et al. (2007) ^a	323			3,468				1	1	1	4	2	0	
N		129.48	20.88		82.92	24.12	1.95							
E		86.55	22.62		118.50	20.00	-1.58							
O		102.83	22.35		117.97	19.48	-0.77							
A		130.10	19.18		121.22	15.56	0.56							
C		98.28	25.58		115.27	19.09	-0.86							
Kendler et al. (2007) ^a	223			14,876				2	2	1	2	2	0	
N		4.75	2.51		2.44	2.15	1.07							
Krueger (1999)	18			543				3	2	2	2	2	0	
N		0.96	0.94		-0.29	0.87	1.42							
E		0.05	0.93		0.09	0.99	-0.05							
D		0.05	0.80		0.09	0.97	-0.04							
Krueger et al. (2001) ^{a,b}	20			1,007				3	2	2	1	2	0	
N		93.46	11.75		78.16	12.53	1.22							
E		124.36	15.02		121.65	13.07	0.21							
D		149.63	16.19		148.57	14.61	-0.07							
McGlashan et al. (2000) ^a ; Clark et al. (2009)	143			561				1	1	2	1	2	1	
D		11.54	6.01		8.80	5.80	0.47							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
McGlashan et al. (2000) ^a ; Siegler & Brummett (2000)	143			2,379				1	1	2	1	2	1	
N		128.34	21.34		76.77	22.05	2.34							
E		90.15	23.55		110.69	19.14	-1.06							
O		115.17	20.97		112.50	19.90	0.13							
A		111.36	20.51		123.19	15.80	-0.73							
C		99.14	24.77		125.92	18.30	-1.43							
Middeldorp et al. (2006) ^a	72			1,176				3	2	1	2	2	0	
N		80.40	22.30		47.90	24.90	1.31							
E		52.50	16.90		60.50	15.60	-0.51							
D. Watson (2005a)	113			3,854				1	2	2	2	1	0	
N		26.67	5.75		18.45	4.98	1.64							
E		27.14	5.60		28.97	4.79	-0.38							
O		28.97	4.69		28.43	4.37	0.12							
				Posttraumatic stress disorder										
Andrews & Slade (2002) ^a	105			9,538				2	2	1	2	2	0	
N		6.78	3.03		2.27	3.22	1.40							
Brodsky et al. (2004)	39			61				2	3	1	3	1	0	
N		6.6	1.4		3.7	2.2	1.50							
Davidson et al. (1988); Cramer (1993)	15			1,455				2	1	2	3	1	1	
N		16.80	4.10		8.06	5.06	1.73							
Davidson et al. (1987)	30			16				2	1	2	3	1	0	
N		17.7	2.5		9.4	5.6	2.14							
Fauerbach et al. (2000); Piedmont (1993)	18			36				1	3	2	1	1	1	
N		56.72	8.9		52.2	8.9	0.51							
E		53.37	9.4		54.6	8.3	-0.14							
O		48.42	7.9		53.9	10.7	-0.56							
A		39.53	8.5		51.0	8.0	-1.40							
C		46.84	10.2		47.3	10.3	-0.04							
Gamez et al. (2007)	41			402				3	3	1	1	1	0	
N		63.3	8.99		46.61	9.10	1.84							
E		42.66	12.30		49.18	9.64	-0.66							
D		51.74	9.02		48.05	8.36	0.44							
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	166			3,468				1	1	1	4	2	1	
N		125.9	24.0		82.9	24.1	1.78							
E		86.9	24.5		118.5	20.0	-1.56							
O		101.1	23.0		118.0	19.5	-0.86							
A		133.2	18.7		121.2	15.6	0.76							
C		96.7	24.6		115.3	19.1	-0.96							
Kamen (2002)	18			42				3	3	1	4	1	0	
N		58.39	8.36		45.76	9.99	1.33							
E		35.83	12.8		47.12	9.01	-1.10							
D		44.33	9.95		42.43	7.69	0.23							
McFarlane (1988)	11			34				2	3	2	3	1	0	
N		11.1	4.3		6.3	4.2	1.14							
McGlashan et al. (2000) ^a ; Clark et al. (2009)	218			561				1	1	2	1	2	1	
D		11.46	5.71		8.8	5.8	0.46							
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	218			1,638				1	1	2	1	2	1	
N		125.19	20.01		74.55	20.25	2.50							
E		91.59	22.16		110.12	18.60	-0.97							
O		116.98	21.83		114.30	18.81	0.14							
A		113.16	18.47		124.50	15.35	-0.72							
C		100.59	23.14		123.30	17.81	-1.23							
Miller et al. (2004) ^a	603			332				3	1	2	1	1	0	
N		70.18	11.61		42.39	9.27	2.56							
E		56.86	14.15		56.29	8.33	0.05							
D		49.06	10.16		52.34	10.06	-0.32							

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Miller & Resick (2007); Clark et al. (1996) ^a	143			74				3	3	1	4	1	1		
N		59.39	7.93		51.53	9.97	0.91								
E		41.29	12.27		49.04	11.46	-0.65								
D		46.38	9.63		52.74	9.40	-0.67								
Talbert et al. (1993); Costa & McCrae (1985)	100			632				1	3	2	4		1		
N		85	8.7		50.0	10.0	3.56								
E		44	8.7		50.0	10.0	-0.61								
O		42	10.4		50.0	10.0	-0.80								
A		24	16.0		50.0	10.0	-2.36								
C		47	11.5		50.0	10.0	-0.29								
Trull & Sher (1994) ^a	21			280				1	3	2	2	2	0		
N		25.38	7.60		16.76	6.72	1.27								
E		28.00	6.92		31.57	5.88	-0.60								
O		30.24	5.59		28.17	6.10	0.34								
A		29.10	6.32		32.99	5.72	-0.67								
C		27.86	6.04		32.35	6.28	-0.72								
van Zelst et al. (2003)	13			312				3	2	1	2	1	0		
N		12.4	5.2		5	5	1.48								
D. Watson (2005a)	173			3,854				1	2	2	2	1	0		
N		24.65	6.40		18.45	4.98	1.23								
E		26.78	5.08		28.97	4.79	-0.46								
O		28.93	4.34		28.43	4.37	0.12								
Panic disorder															
Andrews & Slade (2002) ^a	210			9,538				2	2	1	2	2	0		
N		6.792	3.87		2.272	3.22	1.40								
Bienvenu et al. (2004) ^a	43			297				1	2	2	2	2	0		
N		94.00	26.23		73.52	18.47	1.04								
E		104.67	21.89		108.57	15.25	-0.24								
O		106.65	18.28		103.03	15.79	0.22								
A		123.67	15.90		123.30	15.35	0.02								
C		112.93	22.23		119.09	16.92	-0.35								
Bijl et al. (1998) ^a	165			5,492				3	2	2	2	2	0		
N		12.39	7.04		3.00	3.39	2.64								
Brown (2007) ^a ; Furnham & Miller (1997)	225			250				2	1	1	4	1	1		
N		15.88	5.04		8.12	4.37	1.65								
Carrera et al. (2006)	103			103				1	1	1	4	1	0		
N		29.7	11.8		19.2	9.0	1.00								
E		26.9	7.5		29.8	7.3	-0.39								
O		24.6	7.6		24.8	6.8	-0.03								
A		30.3	5.8		31.0	6.2	-0.12								
C		30.2	8.1		30.3	7.8	-0.01								
Chambless (1985) ^a ; McKenzie et al. (1997)	283			740				2	1	2	3	1	1		
N		17.9	4.14		11.58	5.33	1.26								
D		2.76	2.33		3.68	2.69	-0.35								
Corominas et al. (2002); Fullana et al. (2004) ^a	64			40				2	1	2	1	1	1		
N		19.25	3.88		8.97	5.47	2.26								
E		10.28	4.52		14.67	2.59	-1.13								
D		2.34	2.03		1.43	1.30	0.51								
Dammen et al. (2000)	33			40				2	3	1	1	1	0		
N		12.8	3.8		9.9	3.4	0.81								
Dammen et al. (2000)	39			77				2	3	1	1	1	0		
N		9.1	2.5		7.5	2.4	0.66								
Foot & Koszycki (2004) ^a ; Costa & McCrae (1992)	32			1,000				1	1	1	1	1	1		
N		113.42	21.71		79.1	21.2	1.62								
E		99.85	17.87		109.4	18.4	-0.52								

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
O		111.14	15.57		110.6	17.3	0.03							
A		118.68	19.36		124.3	15.8	-0.35							
C		111.38	21.17		123.1	17.6	-0.66							
Foot & Koszycki (2004) ^a ; Siegler & Brummett (2000)	69			2,379				1	1	1	1	1	1	1
N		63.88	12.15		48.9	10.4	1.43							
E		49.36	10.29		50.7	10.4	-0.13							
O		52.91	9.86		51.1	11.5	0.16							
A		51.39	10.37		49.3	10.0	0.21							
C		46.00	11.98		51.6	10.4	-0.54							
Freire et al. (2007)	77			30				3	1	1	1	1	1	0
N		34.75	9.36		23.90	10.70	1.11							
Gamez et al. (2007)	12			402				3	3	1	1	1	1	0
N		62.65	8.39		46.61	9.10	1.77							
E		39.55	10.33		49.18	9.64	-1.00							
D		52.73	8.51		48.05	8.36	0.56							
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	455			3,468				1	1	1	4	2	1	1
N		128.4	21.9		82.9	24.1	1.90							
E		87.1	24.5		118.5	20.0	-1.52							
O		100.9	23.7		118.0	19.5	-0.85							
A		128.8	19.7		121.2	15.6	0.47							
C		96.6	24.9		115.3	19.1	-0.94							
Hunt & Andrews (1998); Dunbar & Lishman (1984)	67			30				2	1	2	2	1	1	1
N		17.54	4.11		9.5	4.4	1.91							
R. J. King et al. (1988)	48			43				2	1	2	1	1	1	0
N		12.7	5.9		6.8	4.5	1.12							
E		5.9	3.0		7.2	3.0	-0.43							
D		3.9	2.0		3.9	1.8	0.00							
Krueger et al. (2001) ^a	94			1,007				3	2	2	1	2	2	0
N		84.67	13.65		78.16	12.53	0.51							
E		118.34	14.57		121.65	13.07	-0.25							
D		147.98	13.11		148.57	14.61	-0.04							
Lopes et al. (2005); Moreira et al. (1998)	57			137				2	1	1	1	1	1	1
N		17.72	4.52		12.32	4.46	1.20							
McGlashan et al. (2000) ^a ; Clark et al. (2009)	194			561				1	1	2	1	2	2	1
D		11.89	5.98		8.80	5.80	0.53							
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	194			1,638				1	1	2	1	2	2	1
N		125.34	22.54		74.55	20.25	2.48							
E		88.53	22.47		110.12	18.60	-1.13							
O		116.08	22.06		114.30	18.81	0.09							
A		112.78	18.68		124.50	15.35	-0.74							
C		99.42	24.67		123.30	17.81	-1.28							
Middeldorp et al. (2006) ^a	57			1,191				3	2	1	2	2	2	0
N		72.9	24.6		48.8	25.6	0.94							
E		54.5	13.9		60.2	15.9	-0.36							
Reich et al. (1986) ^b ; Hirschfeld et al. (1989)	56			370				3	3	2	1	1	1	1
N		10.57	6.45		22.3	5.0	2.25							
E		13.94	8.44		19.5	6.0	-0.87							
Roy-Byrne et al. (2002); Du et al. (2002)	58			53				1	1	1	2	1	1	1
N		32.4	10.8		13.2	7.25	2.07							
Sakado et al. (1997)	27			48				3	1	2	1	1	1	0
N		11.6	6.4		7.9	5.5	0.63							
E		10.5	6.8		11.8	5.3	-0.22							

(table continues)

Table 1 (*continued*)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
D. Watson (2005a)	88			3,854										
N		26.94	6.05		18.45	4.98	1.70							
E		26.28	5.83		28.97	4.79	-0.56							
O		28.88	4.30		28.43	4.37	0.10							
Arrindell & Emmelkamp (1987) ^a	32			38				3	3	2	3			0
N		25.97	4.99		12.00	7.56	2.15							
Bienvenu et al. (2007) ^a	418			6,574				2	2	2	2	2		0
N		6.42	3.56		3.30	3.08	1.00							
E		4.44	2.65		5.31	2.40	-0.36							
Bienvenu et al. (2004) ^a	47			297				1	2	2	2	2		0
N		95.89	23.65		73.52	18.47	1.16							
E		93.85	22.32		108.57	15.25	-0.90							
O		101.74	16.77		103.03	15.79	-0.08							
A		121.06	16.05		123.30	15.35	-0.14							
C		111.53	18.56		119.09	16.92	-0.44							
Bijl et al. (1998) ^a	115			5,492				3	2	2	2	2		0
N		10.33	6.34		3.00	3.39	2.11							
Gamez et al. (2007)	16			402				3	3	1	1	1		0
N		62.66	11.13		46.61	9.10	1.75							
E		41.40	10.90		49.18	9.64	-0.80							
D		54.65	9.43		48.05	8.36	0.79							
Harcourt et al. (1998); Strong (2003)	18			47				1	3	2	2	1		1
N		118.55	25.97		65.98	18.70	2.51							
E		98.07	24.23		116.06	16.35	-0.95							
O		127.00	21.21		124.23	19.03	0.14							
A		116.68	15.45		122.98	12.90	-0.46							
C		115.44	19.82		122.87	15.96	-0.43							
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	375			3,468				1	1	1	4	2		1
N		128.26	21.27		82.92	24.12	1.90							
E		84.03	23.85		118.50	20.00	-1.69							
O		99.48	23.19		117.97	19.48	-0.93							
A		131.22	17.82		121.22	15.56	0.63							
C		96.17	24.26		115.27	19.09	-0.97							
Krueger (1999) ^b	34			543				3	2	2	2	2		0
N		0.40	0.98		-0.29	0.87	0.78							
E		-0.11	0.87		0.09	0.99	-0.21							
D		-0.08	1.12		0.09	0.97	0.17							
Krueger et al. (2001) ^{a,b}	121			1,007				3	2	2	1	2		0
N		87.36	12.65		78.16	12.53	0.73							
E		117.02	14.75		121.65	13.07	-0.35							
D		148.27	13.83		148.57	14.61	0.02							
Mavissakalian (1985); Townsley (1993)	20			25				2	1	2	3	1		1
N		15.35	4.54		4.36	3.46	2.76							
McGlashan et al. (2000) ^a ; Clark et al. (2009)	16			561				1	1	2	1	2		1
D		9.00	4.83		8.80	5.80	0.03							
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	16			1,638				1	1	2	1	2		1
N		126.13	12.63		74.55	20.25	2.56							
E		98.25	15.48		110.12	18.60	-0.64							
O		112.56	17.29		114.30	18.81	-0.09							
A		119.06	19.60		124.50	15.35	-0.35							
C		110.56	32.61		123.30	17.81	-0.71							
Middeldorp et al. (2006) ^a	41			1,207				3	2	1	2	2		0
N		66.50	24.30		49.40	25.90	0.66							
E		53.00	16.20		60.20	15.80	-0.46							
Sams (1990)	60			30				2	1	2	4	2		0
N		14.80	5.49		13.23	5.19	0.29							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Trull & Sher (1994) ^a	18			280				1	3	2	2	2	0		
N		24.00	7.65		16.76	6.72	1.07								
E		25.17	5.39		31.57	5.88	-1.09								
O		31.72	4.86		28.17	6.10	0.59								
A		30.06	5.86		32.99	5.72	-0.51								
C		30.06	5.24		32.35	6.28	-0.37								
D. Watson (2005a)	120			3,854				1	2	2	2	1	0		
N		25.71	6.35		18.45	4.98	1.44								
E		26.04	5.79		28.97	4.79	-0.61								
O		27.76	5.24		28.43	4.37	-0.15								
Social phobia															
Andrews & Slade (2002) ^a	166			9,538				2	2	1	2	2	0		
N		7.75	3.62		2.27	3.22	1.70								
Bienvenu et al. (2007) ^a	583			6,409				2	2	2	2	2	0		
N		5.65	3.56		3.29	3.09	0.75								
E		4.14	2.66		5.37	2.38	-0.51								
Bienvenu et al. (2004) ^a	92			297				1	2	2	2	2	0		
N		90.96	19.88		73.52	18.47	0.93								
E		93.26	17.71		108.57	15.25	-0.96								
O		103.07	18.15		103.03	15.79	0.00								
A		123.13	14.35		123.30	15.35	-0.01								
C		111.25	15.71		119.09	16.92	-0.47								
Bijl et al. (1998) ^a	348			5,492				3	2	2	2	2	0		
N		10.35	6.94		3.00	3.39	1.99								
Brown (2007) ^a	252							2	1	1	4	1	0		
N		16.53	4.80												
Chavira (2000)	95			180				1	3	1	2	1	0		
N		58.42	9.81		47.36	9.70	1.14								
E		40.84	10.09		48.90	10.50	-0.78								
O		49.39	10.66		50.43	8.42	-0.11								
A		50.54	12.75		50.57	8.95	0.00								
C		45.34	9.11		47.89	9.38	-0.27								
Gamez et al. (2007)	28			402				3	3	1	1	1	0		
N		57.33	9.51		46.61	9.10	1.17								
E		40.02	11.39		49.18	9.64	-0.94								
D		50.50	10.66		48.05	8.36	0.29								
Heiser et al. (2003) ^a	20			180				2	3	1	2	2	0		
N		15.85	3.62		9.97	5.15	1.17								
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	508			3,468				1	1	1	4	2	1		
N		130.02	21.10		82.92	24.12	1.98								
E		76.20	21.82		118.50	20.00	-2.09								
O		100.24	23.91		117.97	19.48	-0.88								
A		132.50	18.36		121.22	15.56	0.71								
C		95.89	25.12		115.27	19.09	-0.97								
Hunt & Andrews (1998)	26			30				2	1	2	2	1	1		
N		18.70	4.10		9.50	4.40	2.16								
Krueger (1999) ^b	89			543				3	2	2	2	2	0		
N		0.52	1.09		-0.29	0.87	0.89								
E		-0.19	1.00		0.09	0.99	-0.28								
D		0.11	0.90		0.09	0.97	-0.02								
Krueger et al. (2001) ^{a,b}	282			1,007				3	2	2	1	2	0		
N		84.54	13.74		78.16	12.53	0.50								
E		117.43	13.07		121.65	13.07	-0.32								
D		148.89	14.65		148.57	14.61	-0.02								
McGlashan et al. (2000) ^a ; Clark et al. (2009)	171			561				1	1	2	1	2	1		
D		11.75	5.52		8.80	5.80	0.51								
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	171			1,638				1	1	2	1	2	1		
N		126.51	21.07		74.55	20.25	2.56								
E		83.26	19.96		110.12	18.60	-1.43								

(table continues)

Table 1 (continued)

Reference	Disorder			Control			d	Measure	Sample	System	Method	Time frame	Control	Drug
	N	M	SD	N	M	SD								
O		117.53	22.50		114.30	18.81	0.17							
A		114.10	20.83		124.50	15.35	-0.65							
C		96.40	25.22		123.30	17.81	-1.44							
Middeldorp et al. (2006) ^a	56													
N		78.30	23.70	1,192	48.60	25.30	1.18	3	2	1	2	2	0	
E		50.10	14.80		60.40	15.70	-0.66							
Townsley (1993)	67			25				2	1	2	4	1	0	
N		10.94	5.06		4.36	3.46	1.41							
Trull & Sher (1994) ^a	26			280				1	3	2	2	2	0	
N		23.92	8.69		16.76	6.72	1.04							
E		25.54	7.81		31.57	5.88	-0.99							
O		31.08	5.55		28.17	6.10	0.48							
A		31.23	5.85		32.99	5.72	-0.31							
C		28.62	7.09		32.35	6.28	-0.59							
van Velzen et al. (2000)	43							3	1	2	4	1	0	
N		26.92	12.34											
E		36.46	8.01											
D. Watson (2005a)	336			3,854				1	2	2	2	1	0	
N		24.12	6.01		18.45	4.98	1.12							
E		24.92	5.62		28.97	4.79	-0.83							
O		27.52	4.57		28.43	4.37	-0.21							
Specific phobia														
Bienvenu et al. (2007) ^a	1,219			5,774				2	2	2	2	2	0	
N		4.58	3.39		3.26	3.11	0.42							
E		5.26	2.46		5.26	2.42	0.00							
Bienvenu et al. (2004) ^a	175			297				1	2	2	2	2	0	
N		85.52	18.85		73.52	18.47	0.64							
E		104.53	16.21		108.57	15.25	-0.26							
O		104.77	15.72		103.03	15.79	0.11							
A		121.81	16.80		123.30	15.35	-0.09							
C		115.62	15.95		119.09	16.92	-0.21							
Bijl et al. (1998) ^a	517			5,492				3	2	2	2	2	0	
N		8.17	6.85		3.00	3.39	1.35							
Brown (2007) ^a ; Furnham & Miller (1997)	119			250				2	1	1	4	1	1	
N		14.00	5.74		8.12	4.37	1.21							
Gamez et al. (2007)	32			402				3	3	1	1	1	0	
N		55.38	11.76		46.61	9.10	0.94							
E		45.41	10.46		49.18	9.64	-0.39							
D		49.09	10.09		48.05	8.36	0.12							
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	66			3,468				1	1	1	4	2	1	
N		132.97	23.95		82.92	24.12	2.03							
E		88.03	25.25		118.50	20.00	-1.52							
O		104.62	26.05		117.97	19.48	-0.68							
A		125.71	19.35		121.22	15.56	0.29							
C		93.03	25.19		115.27	19.09	-1.16							
Krueger (1999) ^b	79			543				3	2	2	2	2	0	
N		0.44	1.07		-0.29	0.87	0.81							
E		-0.30	1.10		0.09	0.99	-0.39							
D		0.18	0.92		0.09	0.97	-0.09							
Krueger et al. (2001) ^{a,b}	182			1,007				3	2	2	1	2	0	
N		85.48	13.96		78.16	12.53	0.57							
E		120.82	12.27		121.65	13.07	-0.06							
D		151.61	14.31		148.57	14.61	-0.21							
Trull & Sher (1994) ^a	32			280				1	3	2	2	2	0	
N		20.75	8.08		16.76	6.72	0.58							
E		30.22	7.11		31.57	5.88	-0.22							
O		30.19	6.14		28.17	6.10	0.33							
A		31.28	6.12		32.99	5.72	-0.30							
C		27.97	7.00		32.35	6.28	-0.69							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
D. Watson (2005a)	379			3,854				1	2	2	2	1	0		
N		23.38	6.22		18.45	4.98	0.97								
E		27.36	5.48		28.97	4.79	-0.33								
O		28.12	4.49		28.43	4.37	-0.07								
Obsessive-compulsive disorder															
Andrews & Slade (2002) ^a	64			9,538				2	2	1	2	2	0		
N		6.61	4.43		2.27	3.22	1.34								
Bienvenu et al. (2004) ^a	14			297				1	2	2	2	2	0		
N		99.79	31.93		73.52	18.47	1.36								
E		100.71	24.02		108.57	15.25	-0.50								
O		115.93	25.67		103.03	15.79	0.79								
A		127.71	17.93		123.30	15.35	0.28								
C		113.93	21.38		119.09	16.92	-0.30								
Bijl et al. (1998) ^a	33			5,492				3	2	2	2	2	0		
N		13.93	6.43		3.00	3.39	3.20								
Brown (2007) ^a ; R. J. King et al. (1988)	75			43				2	1	1	4	1	1		
N		16.93	4.39		6.80	4.50	2.29								
Cath et al. (2001)	36			26				2	1	2	2	1	0		
N		14.35	4.86		7.50	4.50	1.45								
E		7.78	4.72		12.90	3.90	-1.17								
Fullana et al. (2004) ^a	56			40				2	1	1	3	1	0		
N		20.49	3.71		8.97	5.47	2.54								
E		8.63	4.83		14.67	2.59	-1.49								
D		4.27	2.18		1.43	1.30	1.52								
Gamez et al. (2007)	7			402				3	3	1	1	1	0		
N		60.16	14.76		46.61	9.10	1.47								
E		50.03	8.54		49.18	9.64	0.09								
D		48.29	6.23		48.05	8.36	0.03								
L. O. Gomez (1999) ^d	33			43				1	3	1	1	1	0		
N							1.03								
E							-0.56								
O							0.21								
A							0.01								
C							-0.55								
Hoehn-Saric & Barksdale (1983); Dunbar & Lishman (1984)	20			30				2	1	2	3	1	1		
N		15.45	5.07		9.50	4.40	1.27								
E		9.65	4.67		11.40	4.70	-0.37								
Hummelen et al. (2007) ^a	98			3,468				1	1	1	4	2	1		
N		132.34	22.09		82.92	24.12	2.05								
E		81.90	24.72		118.50	20.00	-1.82								
O		103.00	23.82		117.97	19.48	-0.76								
A		128.83	19.99		121.22	15.56	0.48								
C		98.60	25.07		115.27	19.09	-0.86								
Krueger (1999) ^b	62			543				3	2	2	2	2	0		
N		0.80	1.02		-0.29	0.87	1.23								
E		-0.15	1.00		0.09	0.99	-0.24								
D		-0.05	0.88		0.09	0.97	0.14								
Lal et al. (1987)	40			37				2	1	2	3	1	0		
N		14.42	4.27		11.47	2.67	0.82								
McGlashan et al. (2000) ^a ; Clark et al. (2009)	111			561				3	1	2	1	2	1		
D		11.94	6.31		8.80	5.80	0.53								
McGlashan et al. (2000) ^a ; Siegler & Brummett (2000)	111			2,379				1	1	2	1	2	1		
N		125.53	22.95		76.77	22.05	2.21								
E		94.25	22.91		110.69	19.14	-0.85								
O		120.25	23.27		112.50	19.90	0.39								
A		112.12	21.55		123.19	15.80	-0.69								
C		102.04	24.82		125.92	18.30	-1.28								

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Rector et al. (2002); Schinka et al. (1997)	98			400				1	1	1	1	1	0		
N		121.67	21.65		79.45	21.64	1.95								
E		90.69	19.49		114.25	18.55	-1.26								
O		113.09	18.58		113.70	18.67	-0.03								
A		120.81	21.65		120.85	16.35	0.00								
C		106.57	23.36		124.95	17.69	-0.97								
Rees et al. (2005) ^a ; Terracciano & Costa (2004)	21			1,638				1	1	1	1	1	1		
N		123.41	20.29		74.55	20.25	2.41								
E		98.45	26.83		110.12	18.60	-0.62								
O		122.47	21.47		114.30	18.81	0.43								
A		118.66	20.59		124.50	15.35	-0.38								
C		100.04	18.81		123.30	17.81	-1.31								
Samuels et al. (2000)	65			72				1	1	1	4	1	0		
N		64.00	12.50		49.80	11.00	1.21								
E		47.40	12.60		52.00	10.00	-0.41								
O		53.50	11.40		52.30	10.90	0.11								
A		48.90	11.30		43.80	12.50	0.43								
C		43.30	10.50		46.20	13.30	-0.24								
Scarrabelotti et al. (1995); Haidt et al. (1994) ^a	20			124				2	1	2	3	1	0		
N		17.65	3.60		11.70	5.10	1.21								
E		9.15	5.38		14.30	4.20	-1.18								
D		4.95	3.75		3.76	2.77	0.42								
Wu (2005); D. Watson et al. (2004)	52			580				3	1	2	3	2	0		
N		29.42	5.23		22.22	7.00	1.05								
E		24.60	6.80		28.12	6.46	-0.54								
O		33.68	8.35		38.87	6.00	-0.83								
A		35.21	4.64		35.36	5.31	-0.03								
C		32.72	6.56		34.17	5.89	-0.24								
Substance use disorders															
K. G. Anderson et al. (2007)	326			96				1	3	1	2	1	0	2	
N		22.96	7.46		20.51	7.44	0.33								
E		31.03	6.08		31.10	5.79	-0.01								
O		30.77	4.46		30.67	4.67	0.02								
A		32.57	5.27		35.30	5.26	-0.52								
C		29.62	6.09		31.60	5.94	-0.33								
Andrews & Slade (2002)	262			9,538				2	2	1	2	2	0	2	
N		4.36	4.29		2.27	3.22	0.64								
Ball et al. (1998); Murray et al. (2003)	360			527				1	1	2	1	1	1	3	
N		26.37	7.87		17.80	8.20	1.06								
E		26.13	7.70		28.30	6.60	-0.31								
O		25.01	5.37		28.70	6.60	-0.60								
A		27.78	6.03		32.60	5.60	-0.83								
C		28.12	6.82		34.10	6.50	-0.90								
Beaudoin et al. (1997); S. B. G. Eysenck et al. (1993)	96			615				2	3	2	2	1	1	1	
N		10.09	5.14		9.23	5.42	0.16								
D		4.79	2.99												
Bijl et al. (1998) ^a	489			5,492				3	2	2	2	2	0	2	
N		5.10	5.19		3.00	3.39	0.59								
Borman et al. (2006); Savla et al. (2007)	69			234				1	1	1	3	1	0	3	
N		119.00	25.14		85.68	16.17	1.79								
E		105.00	20.56		106.56	13.87	-0.10								
O		112.65	19.21		104.62	11.99	0.58								
A		115.39	18.40		122.06	13.40	-0.45								
C		94.39	21.77		116.73	14.68	-1.35								

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug	
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>									
Butler (2003); Strong (2003)	50			47											
N		60.88	8.06		43.81	8.82	2.02	1	1		3	1	0	2	
E		52.82	6.62		53.62	8.89	-0.10								
O		47.48	6.59		57.88	11.00	-1.16								
A		43.20	9.35		49.16	8.16	-0.68								
C		46.88	9.66		49.87	9.07	-0.32								
Chapman et al. (2007) ^a	90			343				1	3	1	1	2	0	1	
N		44.12	10.04		43.72	9.25	0.04								
E		51.91	11.33		52.19	9.49	-0.03								
O		49.38	11.05		49.88	9.43	-0.05								
A		50.27	9.27		56.23	9.28	-0.64								
C		48.70	9.90		50.18	9.62	-0.15								
Chassin et al. (2004) ^a	261			479				1	3	2	2	2	0	1	
N		2.87	0.64		2.63	0.67	0.37								
E		3.48	0.53		3.57	0.50	-0.18								
O		3.35	0.53		3.24	0.55	0.20								
A		3.43	0.45		3.71	0.54	-0.54								
C		3.59	0.56		3.79	0.58	-0.35								
Chinnian et al. (1994)	70			70				2	1	2	3	1	0	1	
N		14.11	4.53		12.53	4.62	0.35								
E		12.30	3.80		13.26	3.93	-0.25								
D		6.30	3.41		5.06	2.72	0.40								
Conner et al. (2004) ^a ; Han et al. (1996)	48			231				1	1			1	1	2	
N		148.30	21.30		97.00	22.60	2.29								
Cutrona et al. (2005) ^a	105			654				3	2	1	2	2	0	2	
N		5.45	3.93		4.23	3.54	0.34								
E		10.05	2.87		10.76	2.56	-0.27								
D		3.89	2.56		2.74	1.95	0.56								
Drummond & Phillips (2002) ^a ; Buckley et al. (1999)	78			15				2	1	1	3	1	1	1	
N		17.79	4.76		6.70	6.00	2.23								
E		10.47	5.62		12.60	6.10	-0.37								
D		5.56	4.15		3.10	2.70	0.62								
Gamez et al. (2007)	87			402				3	3	1	1	1	0	3	
N		53.53	10.40		46.61	9.10	0.74								
E		46.17	11.37		49.18	9.64	-0.30								
D		54.38	10.56		48.05	8.36	0.72								
J. Gomez (1984)	71			20				2	1	2	3	1	0	1	
N		18.80	3.00		10.60	3.40	2.65								
Goodyear (1991); Roy (1998)	48			56				2	1	2	3	1	1	2	
N		14.83	5.05		5.40	4.20	2.04								
E		13.27	5.19		13.60	4.40	-0.07								
D		5.44	3.00		3.00	2.10	0.95								
Gossop & Eysenck (1982); Riggio (1999)	221			226				2	1	2	3	1	1	3	
N		16.17	5.01		11.79	5.24	0.86								
E		10.64	4.76		13.38	4.75	-0.58								
D		7.28	3.57				0.85								
Heiser et al. (2003) ^a	37			163				2	3	1	2	2	0	2	
N		11.92	4.82		10.25	5.39	0.32								
Henderson et al. (1998) ^{a,b} ; Tellegen (1982)	149			1,350				3	1	2	4	1	1	1	
N		149.74	18.60		127.58	13.40	1.58								
E		149.69	14.87		151.07	12.82	-0.11								
D		164.26	12.82		167.37	13.31	0.23								
Hill et al. (1990) ^b	29			18				3	3	2	2	1	0	1	
N		130.30	13.60		116.60	10.70	1.09								
E		148.90	10.80		153.80	12.20	-0.43								
D		154.00	13.60		160.10	13.80	0.45								

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time frame	Control	Drug
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>								
Hummelen et al. (2007) ^a ; Martinsen et al. (2003)	174			3,468				1	1	1	4	2	1	2
N		126.61	23.45		82.92	24.12	1.81							
E		90.99	23.37		118.50	20.00	-1.36							
O		109.55	23.25		117.97	19.48	-0.43							
A		120.03	19.64		121.22	15.56	-0.08							
C		86.45	26.48		115.27	19.09	-1.48							
A. C. King et al. (2003); Roy et al. (1985)	67			11				2	1	2	3	1	1	1
N		11.86	8.22		4.30	3.40	0.98							
E		10.42	5.32		14.60	3.70	-0.82							
D		4.20	3.35		2.50	1.60	0.54							
Koller et al. (2006) ^a ; Borkenau & Ostendorf (1993)	416			1,908				1	1	1	1	1	1	1
N		21.31	7.83		19.44	7.44	0.25							
E		22.85	6.08		26.40	6.00	-0.59							
O		27.15	6.82		24.60	5.52	0.44							
A		31.40	5.07		30.48	5.64	0.17							
C		29.10	5.69		32.52	6.60	-0.53							
Kornør & Nordvik (2007)	65			65				1	1	1	2	2	0	3
N		64.00	8.00		49.00	8.90	1.77							
E		41.00	8.40		51.00	8.90	-1.16							
O		48.00	9.50		52.00	12.40	-0.36							
A		46.00	8.30		51.00	11.00	-0.51							
C		36.00	8.10		50.00	9.30	-1.61							
Krueger (1999) ^b	150			543				3	2	2	2	1	0	2
N		0.66	0.96		-0.29	0.87	1.06							
E		-0.17	0.96		0.09	0.99	-0.27							
D		-0.59	1.03		0.09	0.97	0.69							
Krueger et al. (2001) ^{a,b}	816			1,007				3	2	2	2	2	0	2
N		84.70	13.88		78.16	12.53	0.50							
E		119.62	12.74		121.65	13.07	-0.16							
D		142.75	13.89		148.57	14.61	0.41							
Lalone (2001)	74			199				1	1	1	4	1	0	1
N		62.00	11.30		50.00	10.20	1.14							
E		43.00	10.10		51.00	10.40	-0.78							
O		45.00	10.80		51.00	11.00	-0.55							
A		44.00	11.70		49.00	9.50	-0.49							
C		38.00	11.30		52.00	10.40	-1.31							
Larkins & Sher (2006) ^a	119			368				2	3	2	2	2	0	1
D		4.25	2.99		2.18	2.20	0.86							
Lejuez et al. (2007) ^b ; Patrick et al. (2002)	304			1,350				3	1	1	1	1	1	3
N		51.60	16.88		34.90	14.60	1.11							
E		65.80	15.08		67.60	14.70	-0.12							
D		80.05	12.41		85.30	14.50	0.37							
Luo et al. (2007) ^a	249			303				1	3	2	4	2	0	2
N		25.39	9.28		16.21	7.17	1.12							
E		26.01	6.68		30.76	6.35	-0.73							
O		26.31	5.92		30.17	6.32	-0.63							
A		28.53	6.00		33.01	6.46	-0.72							
C		28.78	7.29		34.28	7.39	-0.75							
McCormick et al. (1998); Costa & McCrae (1985)	2,676			363				1	3	2	1	1	1	2
N		97.05	24.08		74.20	19.40	0.97							
E		101.23	17.44		101.20	18.10	0.00							
O		103.17	15.16		108.10	16.70	-0.32							
A		41.57	7.25		48.30	9.20	-0.90							
C		42.35	8.83		53.20	9.20	-1.22							
McGlashan et al. (2000) ^a ; Clark et al. (2009)	365			561				3	1	2	1	2	1	2
D		13.37	6.08		8.80	5.80	0.77							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time		
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>						frame	Control	Drug
McGlashan et al. (2000) ^a ; Terracciano & Costa (2004)	365			1,638				1	1	2	1	2	1	2
N		121.69	23.28		74.55	20.25	2.26							
E		92.78	22.04		110.12	18.60	-0.90							
O		116.65	21.10		114.30	18.81	0.12							
A		111.28	18.97		124.50	15.35	-0.82							
C		96.81	23.92		123.30	17.81	-1.39							
Mckinnie (1996); Heiser et al. (2003)	200			158				2	1	2	3	1	1	1
N		11.95	5.50		9.53	4.95	0.46							
E		10.80	3.56											
Miller et al. (2004) ^{a,b}	86			332				3	3	2	1	1	0	2
N		48.71	8.13		42.39	9.27	0.70							
E		52.77	9.31		56.29	8.33	-0.41							
D		45.79	8.15		52.34	10.06	0.68							
Montes (1999); Ross et al. (2003)	21			251				1	1	2	3	1	1	1
N		110.81	20.72		97.57	22.07	0.60							
E		111.48	18.65		124.19	21.20	-0.60							
O		101.48	17.45		121.07	18.93	-1.04							
A		106.00	18.98		116.49	19.77	-0.53							
C		105.09	21.12		117.97	22.68	-0.57							
Muench (2005); Phillips et al. (2006)	252			112				1	1	1	3	2	0	2
N		51.77	10.61		52.16	10.47	-0.04							
E		41.15	8.95		54.39	11.23	-1.36							
O		50.59	8.73		54.74	11.73	-0.43							
A		47.51	9.98		47.95	12.35	-0.04							
C		36.43	8.78		49.88	11.12	-1.41							
O'Boyle (1995); Grace & O'Brien (2003)	97			40				2	1	2	1	1	1	2
N		13.70	6.00		6.10	4.01	1.38							
E		12.20	5.00		12.40	4.97	-0.04							
D		3.90	3.20				0.55							
Ogden et al. (1989); Pickering et al. (2003)	562			105				2	1	2	3	1	1	1
N		17.14	4.78		8.72	5.02	1.75							
E		10.87	5.22		14.98	4.92	-0.79							
D		4.31	2.92		4.24	2.97	0.03							
Ottomanelli (1995); Sen et al. (2004)	108			340				1	1	2	3	1	1	1
N		101.54	25.93		85.15	20.47	0.75							
E		106.79	20.69		108.07	17.52	-0.07							
O		106.24	16.87		103.63	17.15	0.15							
A		49.55	26.71		48.25	8.30	0.09							
C		44.13	9.10		47.16	6.45	-0.42							
Piedmont (2004) ^a ; Nowakowska et al. (2005)	73			47				1	1	2	3	1	1	2
N		63.20	9.65		43.82	8.82	2.08							
E		46.80	9.37		53.64	8.91	-0.74							
O		50.21	10.29		57.86	10.98	-0.72							
A		42.29	10.75		49.18	8.16	-0.70							
C		36.36	11.33		49.89	9.09	-1.29							
Rankin et al. (1982); Dunbar & Lishman (1984)	137			30				2	1	2	3	1	1	1
N		17.97	4.48		9.50	4.40	1.90							
E		10.77	5.57		11.40	4.70	-0.12							
D		5.09	3.40				0.67							
Reno (2004); Tran et al. (2006)	43			340				1	1	1	1	1	1	3
N		57.52	9.40		45.35	9.51	1.28							
E		49.14	8.70		50.53	15.73	-0.09							
O		50.30	8.70		51.66	16.10	-0.09							
A		41.27	10.44		41.87	18.71	-0.03							
C		43.34	10.55		41.21	17.01	0.13							

(table continues)

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time		
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>						frame	Control	Drug
Rosenthal et al. (1990); McKenzie et al. (1997)	297			740				2	1	2	3	1	1	3
N		14.71	5.68		11.58	5.33	0.58							
E		12.53	4.78		13.57	4.78	-0.22							
D		4.89	3.07				0.43							
Roy (2003a) ^a ; H. J. Eysenck & Eysenck (1975)	270			5,574				2	1	1	3	1	1	1
N		15.39	5.28		11.53	5.39	0.72							
E		9.71	5.27		12.84	4.87	-0.64							
D		5.73	3.22				0.95							
Roy (2003b) ^a ; Damas-Mora et al. (1982)	134			57				2	1	1	3	1	1	3
N		17.44	4.54		7.78	5.06	2.05							
E		8.96	5.45		13.19	4.83	-0.80							
D		6.10	3.60				0.63							
Ruiz et al. (2003) ^a	115			85				1	3	1	1	2	1	1
N		93.39	20.47		82.56	20.03	0.53							
E		122.60	19.14		122.12	21.77	0.02							
O		127.99	19.73		127.86	19.55	0.01							
A		111.39	20.21		116.85	21.14	-0.26							
C		103.40	24.11		118.91	21.17	-0.68							
Schadé et al. (2007) ^a ; Hoekstra et al. (1996)	90			2,415				1	1	1	1	1	1	1
N		43.95	6.62		31.10	8.20	1.58							
E		27.12	5.76		40.10	6.60	-1.97							
O		32.79	5.42		35.90	6.40	-0.49							
A		33.64	4.91		44.10	5.20	-2.01							
C		31.79	5.76		45.30	5.60	-2.41							
Schuckit et al. (1994)	18			58				2	3	2	3	1	0	1
N		8.60	3.71		7.90	4.20	0.17							
Slutske et al. (2002) ^a	598			4,722				2	3	2	3	2	0	1
N		2.48	1.70		2.17	1.65	0.19							
E		2.66	1.66		2.32	1.59	0.21							
D		2.35	1.36		1.65	1.28	0.54							
Small & Bennett (2004)	56			12				2	3	1	3	2	0	3
N		14.00	5.80		10.80	7.10	0.53							
E		15.50	5.00		16.20	4.90	-0.14							
D		11.30	4.80		9.70	4.90	0.33							
Swendsen et al. (2002) ^b	205			120				3	1	2	4	2	0	3
N		140.60	17.90		127.50	15.00	0.78							
E		145.80	13.10		148.30	11.30	-0.20							
D		161.30	13.00		170.60	12.00	0.74							
Tarter et al. (2007) ^a	39			73				3	3	1	1	2	0	2
N		28.52	24.66		20.55	20.66	0.36							
Trull & Sher (1994) ^a	141			280				1	3	2	2	2	0	1
N		19.53	7.85		16.76	6.72	0.39							
E		30.21	6.50		31.57	5.88	-0.22							
O		20.07	6.22		28.17	6.10	-1.32							
A		29.38	6.46		32.99	5.72	-0.60							
C		28.91	6.89		32.35	6.28	-0.53							
Walker (2001); Bienvenu et al. (2004)	669			297				1	1	1	1	1	1	1
N		62.44	12.47		47.37	8.71	1.32							
E		45.71	16.07		49.55	8.29	-0.27							
O		45.98	11.02		45.62	9.13	0.03							
A		48.65	14.55		49.37	9.72	-0.05							
C		39.93	12.38		47.72	9.61	-0.67							
Ward & Hemsley (1982); Lamey et al. (2006)	15			18				2	1	2	3	1	1	3
N		15.70	5.20		7.55	5.22	1.56							
E		9.70	5.90		10.33	5.88	-0.11							
D		5.90	3.90				1.56							

Table 1 (continued)

Reference	Disorder			Control			<i>d</i>	Measure	Sample	System	Method	Time		
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>						frame	Control	Drug
D. Watson (2005a)	769			3,854				1	2	2	2	1	0	1
N		21.27	5.74		18.45	4.98	0.55							
E		28.56	5.15		28.97	4.79	-0.08							
O		28.59	4.38		28.43	4.37	0.04							
Weijers et al. (2001); Allemand et al. (2007)	40			455				1	1	1	3	1	1	1
N		22.80	6.98		17.76	6.95	0.72							
E		26.40	5.89		28.51	5.67	-0.37							
O		27.00	5.65		29.63	6.43	-0.41							
A		28.20	4.78		29.83	5.33	-0.31							
C		31.80	5.40		35.19	5.39	-0.63							
Young & Schinka (2001) ^a ; Siegler & Brummett (2000)	118			2,379				1	3	1	4	1	1	1
N		65.14	12.28		48.90	10.40	1.55							
E		42.33	11.45		50.70	10.40	-0.80							
O		45.83	10.17		51.10	11.50	-0.46							
A		42.53	12.08		49.30	10.00	-0.67							
C		35.97	12.14		51.60	10.40	-1.49							
Zilberman et al. (2003); Schinka et al. (1997)	95			200				1	1	1	3	1	1	2
N		127.30	24.80		82.40	22.90	1.91							
E		104.30	23.60		115.80	18.60	-0.57							
O		112.30	17.60		115.90	18.90	-0.19							
A		117.10	18.80		123.80	13.80	-0.43							
C		92.50	26.60		125.20	17.40	-1.57							

Note. Second reference indicates the study from which the control group was obtained. Measure = NEO family (1), Eysenck's inventories (2), or other (3); sample = patient (1), epidemiologic (2), or other (3); system = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*)/*International Classification of Diseases* (10th ed.; 1) or earlier (2); method = Structured Clinical Interview for *DSM* (1), completely structured interview (2), informal (3), or other (4); time frame = current (1) or broader (2); control = control group (0) or no internal control group (1); drug = primarily alcohol (1) or mixed (2) or primarily drugs (3); N = neuroticism; E = extraversion; O = openness; A = agreeableness; C = conscientiousness; D = disinhibition.

^a Some of the presented information was obtained from the authors, as it was not presented in the article. ^b Some study scales measure the low end of the trait, and signs of the corresponding effect sizes were reversed. ^c Effect sizes were computed from Pearson's *r*, as means and standard deviations were not available. ^d Effect sizes were computed from *F* statistic, as means and standard deviations were not available.

within each stratum, which resulted in 454 additional analyses. All computations were conducted with the Hunter-Schmidt Meta-Analysis Programs Package (Schmidt & Le, 2004). They were adjusted for unequal sizes of disorder and control groups.

Results

Eight hundred fifty-one effect sizes based on 175 articles were cumulated in this review (see Table 3; SUD subgroups are not included in this count, as they are parts of the overall SUD category). The number of studies (*K*) ranged from three to 63 (*M* = 12.9, *SD* = 13.2) across the 66 primary analyses. The associated total sample size (*N*) ranged from 1,076 to 75,229 (*M* = 16,517, *SD* = 15,772). Most of the data came from control groups. In fact, control groups were 10.8 times larger on average than disorder groups. Nevertheless, all analyses included at least 187 individuals with the diagnosis, and the average was much higher (*M* = 2,235, *SD* = 3,331). SUD subgroup analyses were based on a similar number of studies (*M* = 11.6, *SD* = 6.1) but had slightly smaller total sample size (*M* = 12,153, *SD* = 9,686).

Strength of Trait-Disorder Links

Average effect sizes corrected for unreliability of personality scales are reported in Table 4. Neuroticism clearly showed the

strongest links to psychopathology (mean *d* = 1.65) as hypothesized. The associations were uniformly positive and large in magnitude (all *ds* ≥ 0.92), and none of the CrIs included zero. The results for specific disorders provided limited support for our predictions, however. As expected, SUD and specific phobia had the weakest links to the trait, with elevations that were just below one standard deviation. All other disorders showed very large effect sizes (*d* = 1.33 to 2.25) and failed to conform consistently to the predicted pattern of higher elevations for distress disorders than for fear disorders. In fact, the average effect for the former was only 19% larger than the average *d* for the latter group (see Figure 2).

Associations between extraversion and psychopathology were uniformly negative but considerably smaller in magnitude (mean *d* = -0.90). In fact, the CrIs for specific phobia, SUD, and MDD included zero, which indicates that the findings were mixed, with more than 10% of effect sizes being truly positive. Moreover, the effect sizes for specific phobia and SUD were quite small (*d* < |0.40|) and can be considered null results. These analyses provided mixed support for our predictions. Of the four disorders that we hypothesized to have particularly low extraversion scores, two (dysthymic disorder and social phobia) exhibited the largest negative effect sizes on the trait, but the other two (MDD and agoraphobia) produced relatively weak associations.

Table 2
Summary of Reliability Estimates for Personality Scales (Cronbach's alpha)

Reference	N	E	D	C	A	O	Diagnostic group
Aggen et al. (2005) ^a	.84	.83					MDD
K. W. Anderson & McLean (1997)				.62			MDD
Andrews & Slade (2002) ^a	.72						MDD
Andrews & Slade (2002) ^a	.66						Dysthymia
Andrews & Slade (2002) ^a	.68						GAD
Andrews & Slade (2002) ^a	.80						PTSD
Andrews & Slade (2002) ^a	.81						Panic disorder
Andrews & Slade (2002) ^a	.79						Social phobia
Andrews & Slade (2002) ^a	.82						OCD
Andrews & Slade (2002) ^a	.81						SUD
Andrews & Slade (2002) ^a	.75						Control
Angst (1998)	.68	.76			.76		MDD
Angst (1998)	.65	.80			.76		Control
Angst (1998)	.71	.61			.79		Dysthymia
Auerbach & Pegg (2002) ^a	.84	.79		.84	.75	.74	Control
Ball et al. (1998)	.78	.72		.77	.62	.50	SUD
Barelids (2005) ^a	.86	.85					Control
Bienvenu et al. (2007) ^a	.84	.83					Agoraphobia
Bienvenu et al. (2007) ^a	.84	.83					Social phobia
Bienvenu et al. (2007) ^a	.84	.83					Specific phobia
Bijl et al. (1998)	.80						Dysthymia
Bijl et al. (1998) ^a	.80						MDD
Bijl et al. (1998) ^a	.80						GAD
Bijl et al. (1998) ^a	.80						Panic disorder
Bijl et al. (1998) ^a	.80						Agoraphobia
Bijl et al. (1998) ^a	.80						Specific phobia
Bijl et al. (1998) ^a	.80						OCD
Bijl et al. (1998) ^a	.80						SUD
Booij et al. (2007) ^a	.85	.81					MDD
Brieger et al. (2003) ^a	.84	.78		.77	.56	.60	MDD
Brown (2007) ^a	.75						MDD
Brown (2007) ^a	.68						Dysthymia
Brown (2007) ^a	.62						GAD
Brown (2007) ^a	.83						Panic disorder
Brown (2007) ^a	.83						Social phobia
Brown (2007) ^a	.86						Specific phobia
Brown (2007) ^a	.78						OCD
Carter et al. (2001) ^a	.72	.68		.71	.72	.74	SUD
Chapman et al. (2007) ^a	.88	.77		.81	.68	.73	MDD
Chapman et al. (2007) ^a	.88	.77		.81	.68	.77	SUD
Chassin et al. (2004) ^a	.77	.81		.79	.68	.76	SUD
Chassin et al. (2004) ^a	.86	.84		.87	.83	.80	Control
Cheng & Furnham (2001) ^b	.82	.83	.66				Control
Cheng & Furnham (2001) ^c	.84	.86	.80				Control
Clara et al. (2003) ^a	.80				.78		MDD
Clark et al. (2003)	.86	.86	.82				MDD
Conner et al. (2004) ^a	.91						SUD
Costa & McCrae (1992)	.92	.89		.90	.86	.87	Control
Cuijpers et al. (2005) ^a	.80	.78		.75	.71	.69	MDD
Cuijpers et al. (2005) ^a	.80	.78		.75	.71	.69	Dysthymia
Cuijpers et al. (2005) ^a	.80	.78		.75	.71	.69	GAD
Cuijpers et al. (2005) ^a	.80	.78		.75	.71	.69	Panic disorder
Cuijpers et al. (2005) ^a	.80	.78		.75	.71	.69	Social phobia
Cuijpers et al. (2005) ^a	.80	.78		.75	.71	.69	Social phobia
Cutrona et al. (2005) ^a	.87	.70	.62				MDD
Cutrona et al. (2005) ^a	.86	.71	.66				SUD
De Fruyt et al. (2006) ^a				.84		.71	Control
Drummond & Phillips (2002) ^a	.41	.73	.44				SUD
Ellenbogen & Hodgins (2004) ^a	.95	.86		.94	.87	.77	MDD
Ellenbogen & Hodgins (2004) ^a	.91	.81		.83	.82	.84	Control
Enns & Cox (2005)	.76						MDD
H. J. Eysenck & Eysenck (1975) ^d	.85	.84	.68				Control
H. J. Eysenck & Eysenck (1975) ^e	.84	.85	.74				Control
S. B. G. Eysenck et al. (1993)	.87	.84	.66				Control

Table 2 (continued)

Reference	N	E	D	C	A	O	Diagnostic group
Furnham & Cheng (1999)	.85	.78	.62				Control
Furnham et al. (2003) ^a	.88	.89		.88	.79	.80	Control
Griffin et al. (2004) ^b				.90		.90	Control
Griffin et al. (2004) ^c				.91		.87	Control
Hayden & Klein (2001) ^a	.71	.86	.66				Dysthymia
Heiser et al. (2003) ^a	.82	.85					Unipolar
Heiser et al. (2003) ^a	.68	.73					Social phobia
Heiser et al. (2003) ^a	.80	.69					SUD
Holden et al. (2006) ^a	.87	.76		.81	.78	.73	Control
Hummelen et al. (2007) ^a	.74	.75		.81	.67	.71	MDD
Hummelen et al. (2007) ^a	.78	.71		.81	.67	.67	Dysthymia
Hummelen et al. (2007) ^a	.73	.74		.83	.69	.69	GAD
Hummelen et al. (2007) ^a	.77	.73		.81	.71	.76	PTSD
Hummelen et al. (2007) ^a	.75	.77		.81	.67	.72	Panic disorder
Hummelen et al. (2007) ^a	.74	.77		.80	.63	.72	Agoraphobia
Hummelen et al. (2007) ^a	.74	.72		.81	.64	.73	Social phobia
Hummelen et al. (2007) ^a	.79	.77		.79	.62	.76	Specific phobia
Hummelen et al. (2007) ^a	.76	.75		.77	.65	.72	OCD
Hummelen et al. (2007) ^a	.79	.74		.84	.65	.69	SUD
Hunt & Andrews (1998)	.85	.80		.84	.77	.74	Social phobia
Ignjatovic & Svrakic (2003) ^a	.93	.89				.88	MDD
Kendler et al. (2007) ^a	.75	.62					MDD
Kendler et al. (2007) ^a	.75	.62					GAD
Kitamura et al. (2002)	.84	.82					Control
Koller et al. (2006) ^a	.84	.72		.65	.62	.81	SUD
Larkins & Sher (2006) ^a			.57				SUD
Measelle et al. (2006)	.79						MDD
Middeldorp et al. (2006) ^a	.89	.82					MDD
Middeldorp et al. (2006) ^a	.89	.82					Dysthymia
Middeldorp et al. (2006) ^a	.89	.82					GAD
Middeldorp et al. (2006) ^a	.89	.82					Panic disorder
Middeldorp et al. (2006) ^a	.89	.82					Agoraphobia
Middeldorp et al. (2006) ^a	.89	.82					Social phobia
Miles et al. (1999)	.83	.86	.61				Control
Miller et al. (2004) ^a	.85	.85	.65				PTSD
Miller et al. (2004) ^a	.89	.82	.66				Control
Moerk (2003)	.89	.88					Unipolar
Mongrain & Leather (2006) ^a	.83	.86		.82	.78	.80	MDD
Mooradian & Nezlek (1996) ^a	.84	.75		.83	.75	.74	Control
Muench (2005)				.89		.62	SUD
Neuman & Kickul (1998) ^a		.94		.91	.87		Control
Pedersen et al. (1988)	.75	.66					Control
Piedmont (2004) ^a	.88	.85		.90	.84	.87	SUD
Ross et al. (2003)	.91	.91		.91	.89	.88	Control
Ross et al. (2004) ^a	.91			.86			Control
Saucier (1998) ^a	.88	.80		.83	.76	.79	Control
Savla et al. (2007)	.86	.79		.85	.81	.74	Control
Schadé et al. (2007) ^a	.81	.80		.77	.81	.79	SUD
Tarter et al. (2007) ^a	.72						SUD
Taylor & MacDonald (1999) ^a	.90	.87		.89	.88	.87	Control
Tokar et al. (1999) ^a	.85	.79		.82	.74	.74	Control
Trull & Sher (1994) ^a	.85	.80		.84	.77	.74	MDD
Trull & Sher (1994) ^a	.85	.80		.84	.77	.74	PTSD
Trull & Sher (1994) ^a	.85	.80		.84	.77	.74	Agoraphobia
Trull & Sher (1994) ^a	.85	.80		.84	.77	.74	Specific phobia
Trull & Sher (1994) ^a	.85	.80		.84	.77	.74	SUD
van Oppen et al. (1995)	.79	.80	.46				OCD
Verkerk et al. (2005) ^a	.85	.87					MDD
D. Watson (2005a)	.86	.80				.84	MDD
D. Watson (2005a)	.87	.77				.84	Dysthymia
D. Watson (2005a)	.84	.81				.85	GAD
D. Watson (2005a)	.88	.80				.81	PTSD
D. Watson (2005a)	.85	.84				.82	Panic disorder

(table continues)

Table 2 (continued)

Reference	N	E	D	C	A	O	Diagnostic group
D. Watson (2005a)	.86	.82				.86	Agoraphobia
D. Watson (2005a)	.87	.81				.82	Social phobia
D. Watson (2005a)	.88	.82				.82	Specific phobia
D. Watson (2005a)	.87	.81				.81	SUD
D. Watson (2005a)	.85	.79				.83	Control
D. Watson et al. (2004)	.85	.83		.76	.79	.82	Control
R. Watson et al. (2007) ^a	.87	.74		.84	.74	.72	Control
Wu (2005)	.70	.81		.79	.55	.84	OCD
Yang et al. (1999) ^a	.91	.87		.88	.80	.77	MDD
Yang et al. (1999) ^a	.91	.87		.88	.80	.77	OCD
Yang et al. (1999) ^a	.91	.87		.88	.80	.77	SUD
Young & Schinka (2001) ^a	.92	.89		.91	.88	.87	SUD
<i>M</i>	.82	.80	.64	.82	.74	.77	
<i>SD</i>	.07	.06	.10	.06	.08	.07	

Note. Diagnostic group indicates the sample for which the estimate was computed. N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; MDD = major depressive disorder; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

^a Some of the presented information was obtained from the authors, as it was not presented in the article. ^b Study 1. ^c Study 2. ^d Female sample. ^e Male sample.

The analyses of disinhibition showed much greater specificity and were more consistent with predictions. As hypothesized, SUD were substantially elevated on this trait ($d = 0.72$). Obsessive-compulsive disorder and dysthymic disorder also showed notable effect sizes ($d = 0.63$ and 1.09 , respectively), but these estimates were based on a small number of studies and had wide CrIs. All remaining effect sizes were small, and many were equivocal (i.e., the CrIs included zero).

Unexpectedly, conscientiousness produced strong and consistently negative effect sizes, none of which included zero in the CrI. All estimates were in a narrow range between -0.90 and -1.24 with the exception of specific phobia, which had a somewhat weaker effect ($d = -0.67$). Overall, conscientiousness emerged as

the second most powerful general trait correlate of psychopathology, with a mean effect size of -1.01 .

All links of agreeableness and openness were equivocal, except for the moderate negative associations of openness with agoraphobia and dysthymic disorder. However, both of these CrIs came very close to zero. This pattern of results was consistent with our predictions for openness, but we anticipated a stronger link between SUD and agreeableness. Of note, the latter association was moderately negative ($d = -0.60$) and fairly consistent across studies, as the CrI just barely included zero. In fact, analyses of SUD subgroups produced stronger effects for mixed and primarily drug groups, both of which were unambiguous, thus providing qualified support for our hypothesis. However, differences be-

Table 3
Number of Observations Cumulated in the Meta-Analysis

Disorder	Neuroticism			Extraversion			Disinhibition			Conscientiousness			Agreeableness			Openness		
	N_d	N_c	K	N_d	N_c	K	N_d	N_c	K	N_d	N_c	K	N_d	N_c	K	N_d	N_c	K
MDD	14,653	60,576	63	12,916	43,907	55	2,549	5,060	18	4,850	15,897	25	4,938	16,144	25	5,141	19,745	26
Unipolar	1,453	13,401	18	1,284	9,789	15	334	742	5	422	2,970	6	422	2,970	6	422	2,970	6
Dysthymic disorder	1,578	36,011	13	799	16,999	9	213	6,146	3	495	5,403	3	531	5,686	4	609	9,257	4
GAD	1,674	44,570	14	1,023	28,042	10	220	2,513	4	498	6,144	3	498	6,144	3	611	9,998	4
PTSD	1,714	22,174	16	1,501	10,758	10	1,023	1,411	5	523	6,054	5	523	6,054	5	696	9,908	6
Panic disorder	2,556	32,227	24	1,419	15,870	15	695	2,793	6	896	8,885	6	896	8,885	6	984	12,739	7
Agoraphobia	1,451	24,902	15	1,224	19,317	11	187	2,513	4	474	5,730	5	474	5,730	5	594	9,584	6
Social phobia	3,188	36,165	18	2,309	20,650	12	570	2,513	4	892	5,863	5	892	5,863	5	1,228	9,717	6
Specific phobia	2,800	21,367	10	2,164	15,625	8	293	1,952	3	273	4,045	3	273	4,045	3	652	7,899	4
OCD	905	25,152	18	733	10,079	15	256	2,161	5	492	8,877	8	492	8,877	8	492	8,877	8
SUD	13,550	54,525	58	12,290	38,177	49	5,231	19,056	26	6,940	16,871	25	6,940	16,871	25	7,709	20,725	26
Alcohol	5,257	26,031	26	4,872	25,180	22	2,175	12,878	11	2,143	9,431	12	2,143	9,431	12	2,912	13,285	13
Mixed	6,437	24,403	20	5,562	8,906	15	1,667	3,193	7	4,260	6,274	9	4,260	6,274	9	4,260	6,274	9
Drugs	1,856	4,091	12	1,856	4,091	12	1,319	2,925	8	537	1,166	4	537	1,166	4	537	1,166	4

Note. N_d = pooled number of patients; N_c = pooled number of controls; K = number of studies; MDD = major depressive disorder; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

Table 4
Average Effect Sizes Corrected for Unreliability of Personality Scales

Disorder	Neuroticism		Extraversion		Disinhibition		Conscientiousness		Agreeableness		Openness	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD	1.33	[0.44, 2.23]	-0.62	[-1.36, 0.13]	0.28	[-0.09, 0.65]	-0.90	[-1.42, -0.39]	-0.14	[-0.78, 0.49]	-0.21	[-0.88, 0.47]
Unipolar	1.54	[0.92, 2.17]	-0.92	[-1.54, -0.30]	0.25	[0.25, 0.25]	-1.13	[-1.88, -0.39]	-0.17	[-0.46, 0.11]	-0.12	[-0.40, 0.17]
Dysthymic disorder	1.93	[1.01, 2.84]	-1.47	[-2.47, -0.47]	1.09	[0.39, 1.78]	-1.24	[-1.39, -1.09]	0.26	[-0.69, 1.21]	-0.57	[-1.13, -0.01]
GAD	1.96	[1.33, 2.60]	-1.02	[-1.86, -0.18]	0.44	[0.22, 0.65]	-1.13	[-1.51, -0.76]	0.18	[-0.67, 1.04]	-0.40	[-1.04, 0.25]
PTSD	2.25	[1.23, 3.27]	-0.79	[-1.55, -0.03]	-0.02	[-0.68, 0.63]	-1.02	[-1.50, -0.54]	-0.70	[-2.38, 0.99]	-0.30	[-0.99, 0.39]
Panic disorder	1.92	[1.12, 2.72]	-1.07	[-1.81, -0.34]	0.05	[-0.54, 0.65]	-0.98	[-1.43, -0.53]	0.08	[-0.64, 0.81]	-0.41	[-1.09, 0.26]
Agoraphobia	1.61	[0.86, 2.36]	-0.98	[-1.82, -0.13]	0.15	[-0.11, 0.41]	-0.96	[-1.20, -0.73]	0.52	[-0.02, 1.05]	-0.70	[-1.32, -0.08]
Social phobia	1.63	[0.76, 2.49]	-1.31	[-2.54, -0.08]	0.19	[-0.16, 0.54]	-1.06	[-1.52, -0.61]	0.32	[-0.50, 1.14]	-0.47	[-1.09, 0.16]
Specific phobia	0.92	[0.30, 1.53]	-0.20	[-0.65, 0.26]	-0.17	[-0.17, -0.17]	-0.67	[-1.25, -0.08]	0.00	[-0.25, 0.25]	-0.10	[-0.44, 0.23]
OCD	2.07	[1.25, 2.90]	-1.12	[-1.85, -0.39]	0.63	[0.04, 1.22]	-0.97	[-1.46, -0.47]	-0.06	[-0.69, 0.57]	-0.14	[-0.87, 0.60]
SUD	0.97	[0.13, 1.81]	-0.36	[-1.02, 0.29]	0.72	[0.36, 1.08]	-1.10	[-1.84, -0.36]	-0.60	[-1.30, 0.10]	-0.16	[-0.72, 0.40]
Alcohol	0.77	[0.02, 1.51]	-0.32	[-0.98, 0.35]	0.71	[0.32, 1.11]	-0.90	[-1.77, -0.03]	-0.44	[-1.33, 0.44]	-0.04	[-0.62, 0.55]
Mixed	1.14	[0.20, 2.09]	-0.39	[-0.93, 0.14]	0.71	[0.51, 0.92]	-1.34	[-1.79, -0.89]	-0.74	[-1.18, -0.30]	-0.30	[-0.71, 0.10]
Drugs	1.13	[0.63, 1.63]	-0.33	[-0.61, -0.04]	0.68	[0.40, 0.97]	-1.02	[-1.62, -0.42]	-0.75	[-1.11, -0.39]	-0.38	[-0.98, 0.23]
<i>M</i>	1.65		-0.90		0.33		-1.01		-0.03		-0.32	

Note. Bold indicates that credibility interval (CrI) does not include zero. Mean excludes substance use disorders (SUD) subgroups (i.e., based only on the 11 diagnostic groups). MDD = major depressive disorder; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder.

tween the subgroups on this and other traits were equivocal, and hence no firm conclusions can be made regarding differential personality profiles for specific substances.

Given our interest in the fear and distress clusters, we plotted profiles of these groups along with SUD by averaging effect sizes across relevant disorders (see Figure 2). The distress and fear

curves were essentially parallel, with the former scoring slightly higher on all traits except openness. Contrary to our hypotheses, no personality dimension clearly distinguished these two clusters. The SUD, however, showed a rather distinct profile with relatively low elevations on neuroticism and introversion but appreciably stronger effects on disinhibition and (low) agreeableness.

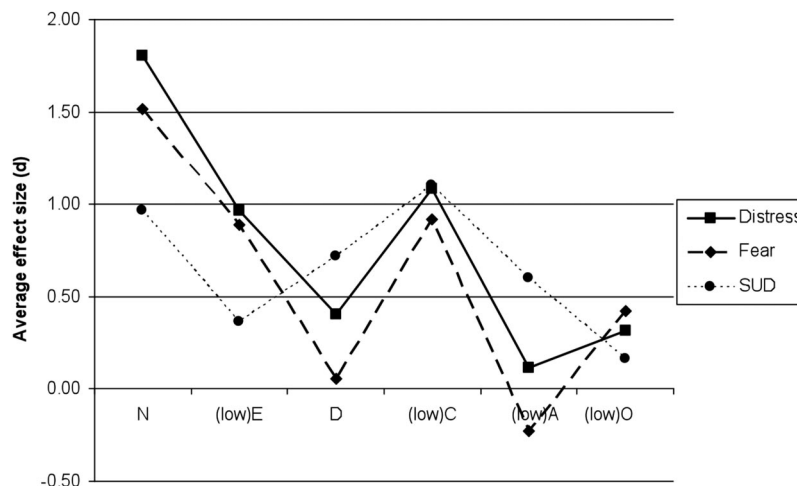


Figure 2. Average profiles of the three disorder types. Averages are not sample size weighted. Distress group includes major depressive disorder, dysthymic disorder, unipolar depression, generalized anxiety disorder, and posttraumatic stress disorder. Fear group includes panic disorder, agoraphobia, social phobia, and specific phobia. Extraversion (E), conscientiousness (C), agreeableness (A), and openness (O) scores were reversed so that higher values indicate greater pathology. SUD = substance use disorders; N = neuroticism; D = disinhibition.

Trait–Disorder Associations Controlling for Effects of Neuroticism

In evaluating these data, it is important to recognize that many higher order personality traits are at least somewhat correlated (Markon et al., 2005). We were most concerned about the potentially distorting effects of neuroticism, as our previous analyses showed that it easily is the strongest predictor of psychopathology. Other trait–disorder links may appear to be significant simply because the trait shares relevant variance with neuroticism. To assess the magnitude of this overlap precisely, we reanalyzed the data of Markon et al. (2005), who performed a meta-analysis of associations among 44 personality scales. Markon et al. cumulated data across multiple studies to estimate each correlation among these 44 variables. We selected 11 of their scales that map clearly on the target six traits. This yielded as many as nine meta-analytic estimates per trait pair because many traits were assessed by multiple measures. We computed sample size weighted averages of these estimates to obtain a single correlation coefficient for each trait pair, thus pooling information across different instruments as well as samples. We found that neuroticism correlated $-.19$, $.10$, $-.33$, $-.22$, and $.00$ with extraversion, disinhibition, conscientiousness, agreeableness, and openness, respectively.

To control for neuroticism, we converted average effect size estimates (see Table 4) into Pearson's r s. In these calculations, we took into account the aggregate size of the disorder group relative to that of the control group (N_d and N_c in Table 3) using the conversion formula for unequal split (Hunter & Schmidt, 2004, p. 280). The general pattern of the resulting correlations was similar to that of the effect sizes, but the estimates were only low to moderate ($r < .50$; see Table 5) because their magnitude was reduced by the unequal sizes of the disorder and control groups. Neuroticism correlated most strongly with PTSD and MDD. Extraversion had its closest link with social phobia. Conscientiousness, disinhibition, and agreeableness correlated mostly highly with SUD. Specific phobia generally showed the weakest effects

across the six traits. Next, we computed partial correlations between traits and disorders according to the standard formula (J. Cohen & Cohen, 1983). One input for this formula is the correlation between the target trait and the controlled variable (i.e., neuroticism), for which we used the meta-analytic estimates described earlier. Finally, we transformed partial correlations back to Cohen's d s. We applied the same procedure to the ends of CrIs to compute adjusted CrIs. The results are presented in Table 6.

The largest change in the effect sizes was observed for analyses of conscientiousness, which was expected because conscientiousness was the strongest correlate of neuroticism. The estimates declined 38% on average (from a mean $d = -1.01$ to -0.63), and four CrIs now included zero. Nevertheless, several disorders continued to show moderate to strong effect sizes. Disinhibition's effects decreased 30% on average, but only one link became equivocal; SUD and dysthymic disorder continued to show notable elevations on this trait. Controlling for neuroticism reduced extraversion effects from mean $d = -0.90$ to -0.69 , and four additional CrIs included zero. Only unipolar depression, dysthymic disorder, obsessive-compulsive disorder, and panic disorder produced unequivocal adjusted associations with extraversion. Social phobia still exhibited one of the strongest effects, but it had a wide CrI, which now included zero. In contrast, estimates for agreeableness increased from average $d = -0.03$ to 0.29 , and two associations became unequivocal. The mixed and primarily drug SUD subgroups continued to show unambiguous negative associations with this trait. Finally, no noteworthy changes were observed for openness.

Moderating Variables

To stratify studies for the moderator analyses in a meaningful way, we generally needed at least 10 samples because they had to be split into as many as four groups. Neuroticism had the requisite number of samples in every analysis. Extraversion fulfilled this criterion also, except for its associations with dysthymic disorder

Table 5
Effect Sizes in Metric of Pearson's r

Control	Neuroticism	Extraversion	Disinhibition	Conscientiousness	Agreeableness	Openness
MDD	0.47	-0.25	0.13	-0.36	-0.06	-0.08
Unipolar	0.42	-0.28	0.11	-0.35	-0.06	-0.04
Dysthymic disorder	0.36	-0.29	0.19	-0.33	0.07	-0.14
GAD	0.34	-0.18	0.12	-0.29	0.05	-0.09
PTSD	0.49	-0.25	-0.01	-0.27	-0.19	-0.07
Panic disorder	0.45	-0.28	0.02	-0.27	0.02	-0.11
Agoraphobia	0.34	-0.23	0.04	-0.25	0.14	-0.16
Social phobia	0.41	-0.37	0.07	-0.34	0.11	-0.15
Specific phobia	0.28	-0.07	-0.06	-0.16	0.00	-0.03
OCD	0.35	-0.27	0.19	-0.21	-0.01	-0.03
SUD	0.36	-0.16	0.28	-0.44	-0.27	-0.07
Alcohol	0.28	-0.12	0.24	-0.33	-0.17	-0.02
Mixed	0.42	-0.19	0.32	-0.55	-0.34	-0.15
Drugs	0.46	-0.15	0.30	-0.43	-0.33	-0.17
<i>M</i>	0.39	-0.24	0.10	-0.30	-0.02	-0.09

Note. Effect sizes were computed from value in Table 4 while taking into account the relative size of disorder and control groups. Bold indicates $r > .20$. Mean excludes substance use disorders (SUD) subgroups (i.e., based only on the 11 diagnostic groups). MDD = major depressive disorder; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder.

Table 6
Effect Sizes Controlling for Neuroticism

Disorder	Extraversion		Disinhibition		Conscientiousness		Agreeableness		Openness	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD	-0.46	[-1.26, 0.40]	0.20	[-0.22, 0.62]	-0.59	[-1.13, -0.03]	0.12	[-0.60, 0.89]	-0.24	[-1.01, 0.54]
Unipolar	-0.73	[-1.39, -0.06]	0.18	[0.18, 0.18]	-0.77	[-1.55, 0.03]	0.12	[-0.20, 0.44]	-0.13	[-0.44, 0.19]
Dysthymic disorder	-1.21	[-2.25, -0.15]	0.95	[0.20, 1.68]	-0.87	[-1.02, -0.71]	0.61	[-0.44, 1.75]	-0.61	[-1.22, -0.01]
GAD	-0.71	[-1.59, 0.19]	0.33	[0.10, 0.56]	-0.75	[-1.15, -0.36]	0.51	[-0.41, 1.52]	-0.42	[-1.11, 0.27]
PTSD	-0.57	[-1.42, 0.31]	-0.14	[-0.96, 0.61]	-0.46	[-0.99, 0.10]	-0.33	[-2.20, 1.80]	-0.34	[-1.15, 0.46]
Panic disorder	-0.84	[-1.64, -0.03]	-0.07	[-0.75, 0.60]	-0.52	[-1.00, -0.01]	0.49	[-0.33, 1.40]	-0.46	[-1.23, 0.29]
Agoraphobia	-0.75	[-1.62, 0.16]	0.02	[-0.26, 0.29]	-0.57	[-0.83, -0.33]	0.90	[0.29, 1.53]	-0.74	[-1.41, -0.08]
Social phobia	-1.13	[-2.43, 0.20]	0.09	[-0.29, 0.47]	-0.72	[-1.19, -0.23]	0.67	[-0.26, 1.72]	-0.51	[-1.20, 0.18]
Specific phobia	-0.04	[-0.51, 0.45]	-0.27	[-0.27, -0.27]	-0.31	[-0.92, 0.34]	0.27	[0.01, 0.54]	-0.10	[-0.46, 0.24]
OCD	-0.91	[-1.66, -0.13]	0.55	[-0.08, 1.17]	-0.48	[-1.00, 0.06]	0.32	[-0.37, 1.03]	-0.15	[-0.93, 0.65]
SUD	-0.25	[-0.89, 0.41]	0.67	[0.29, 1.06]	-0.87	[-1.62, -0.08]	-0.47	[-1.22, 0.29]	-0.17	[-0.76, 0.42]
Alcohol	-0.19	[-0.87, 0.53]	0.65	[0.25, 1.07]	-0.70	[-1.58, 0.23]	-0.30	[-1.21, 0.65]	-0.04	[-0.65, 0.57]
Mixed	-0.25	[-0.83, 0.35]	0.68	[0.46, 0.91]	-1.11	[-1.56, -0.65]	-0.60	[-1.06, -0.12]	-0.33	[-0.79, 0.11]
Drugs	-0.16	[-0.47, 0.17]	0.65	[0.34, 0.98]	-0.75	[-1.37, -0.10]	-0.59	[-0.97, -0.19]	-0.43	[-1.14, 0.26]
<i>M</i>	-0.69		0.23		-0.63		0.29		-0.35	

Note. Bold indicates that credibility interval (CrI) does not include zero. Mean excludes substance use disorders (SUD) subgroups (i.e., based only on the 11 diagnostic groups). MDD = major depressive disorder; Unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder.

and specific phobia, which were based on nine and eight studies, respectively. We examined moderator effects for all links between these two traits and psychopathology. The remaining personality variables had sufficient *K* only in analyses of MDD and SUD; thus, these eight effects were included as well. SUD subgroups were not examined in moderator analyses given the limited *K* and lack of unequivocal differences among substances. For each of the 30 selected associations, artifacts (sampling error and unreliability) accounted for no more than 20% of the variance in observed effect sizes. This indicates large true differences among the studies and implies the presence of moderators. Hence, we proceeded to evaluate effects of the six study characteristics.

To assess the extent of overlap among these variables, we compared them across the 851 samples. For each pairing we computed the uncertainty coefficient, which is a measure of the association between two nominal variables and can be interpreted analogously to r^2 as the proportion of variance in the dependent variable accounted for by the independent variable (Agresti, 2002). The uncertainty coefficient is a directional measure, and its

value changes depending on which variable in the pair is treated as the predictor. We considered overlap notable only if the uncertainty coefficient was above .10 in both directions. The moderators overlapped only weakly (uncertainty coefficients < .10), except for the sample type, which had moderate links to method, time frame, and control group status (see Table 7). Detailed examination of these effects revealed a few points of overlap that accounted for the observed associations. Specifically, nearly all epidemiologic studies used completely structured interviews (84.1% vs. 12.4% in other studies) and broad time frames (81.1% vs. 28.5%). Also, patient studies only rarely had a control group (36.9% vs. 84.9%). These patterns have to be considered when interpreting the results of the moderator analyses.

We stratified data for the 30 selected trait-disorder links by each moderator variable in turn and performed meta-analyses within each stratum. We examined the CrI overlap among all pairs of strata. If neither CrI included the average *d* for the other stratum, then the two strata were considered distinct, as such a pattern

Table 7
Associations Among Moderator Variables

Variable	Measure	Sample	System	Method	Time frame	Control
Measure	—	.07	.00	.07	.03	.06
Sample	.06	—	.04	.19	.14	.21
System	.00	.03	—	.02	.01	.00
Method	.10	.26	.05	—	.12	.13
Time frame	.02	.11	.01	.06	—	.02
Control	.04	.15	.00	.07	.02	—

Note. Associations are expressed as uncertainty coefficients and indicate proportion of variance in a variable at the top of the column accounted by a variance at the beginning of the row. Overlap greater than 10% is bolded.

indicates that the average estimates likely came from different populations.

Comparisons of measures revealed consistently weaker results for Eysenck's instruments (see Table 8). Indeed, the NEO inventories produced stronger effects than Eysenck's scales in nine comparisons (36% of analyses), and other measures did the same in four cases (15%). In contrast, Eysenck's instruments outperformed either of the others in only two analyses (9%). Similarly, epidemiologic samples frequently yielded weaker effects than other recruitment strategies (see Table 9). Patient studies outperformed them in 15 comparisons (58%), and other samples did so nine times (36%), as opposed to one comparison that favored epidemiologic studies (4%). Diagnostic system had little impact on

effect sizes, except for somewhat stronger results for *DSM-IV/ICD-10* in analyses of specific phobia, agoraphobia, and PTSD (see Table 10). Comparison of diagnostic methods revealed that completely structured interviews produced consistently weaker results, as this category had the smallest effects in 14 comparisons (47%) relative to three for informal diagnosis (13%), three for the SCID (10%), and one for other methods (3%; see Table 11). Contrary to our predictions, current diagnoses were not associated with stronger results. In fact, broader time frames outperformed them in four cases (13%; see Table 12). We also found that studies without internal control groups produced larger effects in 10 comparisons (33%; see Table 13). Importantly, observed differences were not due to control groups. We compared internal

Table 8
Breakout of Effect Sizes by Measure Family

Disorder	NEO		Eysenck		Other	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD						
N	1.97_a	[1.48, 2.46]	0.87 _b	[0.23, 1.50]	1.29 _{a,b}	[0.43, 2.15]
E	-1.23_a	[-1.80, -0.67]	-0.20 _b	[-0.55, 0.14]	-0.42 _b	[-0.82, -0.02]
D	—	—	0.37 _a	[-0.24, 0.98]	0.26 _a	[0.00, 0.53]
C	-1.04_a	[-1.39, -0.70]	—	—	-0.19 _b	[-0.31, 0.07]
A	-0.07 _a	[-0.74, 0.61]	—	—	-0.46 _a	[-0.46, -0.46]
O	-0.13 _a	[-0.77, 0.51]	—	—	-0.64 _a	[-1.24, -0.05]
Unipolar						
N	1.77 _a	[1.17, 2.37]	1.43 _a	[0.87, 2.00]	1.18 _a	[0.56, 1.80]
E	-0.90 _a	[-1.37, -0.44]	-0.77 _a	[-1.17, -0.37]	-2.12_b	[-2.48, -1.76]
Dysthymic disorder						
N	1.70 _a	[0.82, 2.58]	1.72 _a	[1.38, 2.05]	2.72_b	[2.20, 3.25]
E	-1.74_a	[-2.40, -1.08]	-1.02_b	[-1.02, -1.02]	-0.93 _c	[-0.93, -0.93]
GAD						
N	2.17 _a	[1.78, 2.55]	1.78 _a	[1.05, 2.51]	1.97 _a	[1.40, 2.54]
E	-1.33_a	[-2.02, -0.64]	-0.47 _b	[-1.14, 0.20]	-0.55 _b	[-1.07, -0.02]
PTSD						
N	2.35 _a	[1.24, 3.47]	1.63 _a	[1.63, 1.63]	2.36 _a	[1.52, 3.21]
E	-1.05_a	[-1.67, -0.44]	—	—	-0.21 _b	[-0.71, 0.28]
Panic disorder						
N	2.09_a	[1.55, 2.63]	1.54 _b	[1.18, 1.90]	2.05 _{a,b}	[0.83, 3.27]
E	-1.23 _a	[-1.94, -0.52]	-0.88 _a	[-1.27, -0.49]	-0.52 _a	[-0.82, -0.21]
Agoraphobia						
N	1.96_a	[1.54, 2.38]	1.12 _b	[0.71, 1.53]	1.58_a	[0.64, 2.52]
E	-1.52_a	[-2.17, -0.88]	-0.40 _b	[-0.40, -0.40]	-0.43 _c	[-0.43, -0.43]
Social phobia						
N	1.93 _a	[1.16, 2.70]	1.27 _a	[0.60, 1.93]	1.61 _a	[0.68, 2.53]
E	-1.69_a	[-2.50, -0.87]	-0.59 _b	[-0.70, -0.49]	-1.22 _{a,b}	[-3.19, 0.74]
Specific phobia						
N	1.17_a	[0.56, 1.78]	0.53 _b	[0.24, 0.82]	1.25_a	[0.81, 1.69]
E	-0.54 _a	[-1.14, 0.06]	0.00 _a	[0.00, 0.00]	-0.21 _a	[-0.38, -0.04]
OCD						
N	2.20 _a	[1.77, 2.62]	1.72 _a	[1.04, 2.40]	2.13 _a	[0.76, 3.49]
E	-1.30_a	[-1.99, -0.60]	-1.22_a	[-1.54, -0.89]	-0.39 _b	[-0.50, -0.28]
SUD						
N	1.16 _a	[0.23, 2.08]	0.73 _a	[-0.03, 1.49]	0.85 _a	[0.38, 1.32]
E	-0.53 _a	[-1.23, 0.16]	-0.24 _a	[-0.77, 0.29]	-0.21 _a	[-0.21, -0.21]
D	—	—	0.79 _a	[0.37, 1.21]	0.64 _a	[0.40, 0.89]
C	—	—	—	—	—	—
A	—	—	—	—	—	—
O	—	—	—	—	—	—

Note. Dashes indicate absence of relevant studies. Values that do not share subscripts do not fall into one another's credibility intervals (CrIs). The larger of two values that do not share a subscript is shown in bold. MDD = major depressive disorder; N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

Table 9
Breakout of Effect Sizes by Sample Type

Disorder	Patient		Epidemiologic		Other	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD						
N	1.88_a	[1.26, 2.50]	0.91 _b	[0.22, 1.60]	1.21 _b	[0.56, 1.86]
E	-1.11_a	[-1.79, -0.43]	-0.19 _b	[-0.32, -0.06]	-0.83_a	[-1.20, -0.45]
D	0.38 _a	[-0.12, 0.87]	0.17 _a	[0.07, 0.27]	0.38 _a	[0.38, 0.38]
C	-0.96_a	[-1.42, -0.50]	-0.39 _b	[-0.39, -0.39]	-0.35 _{a,b}	[-0.97, 0.26]
A	-0.12 _a	[-0.78, 0.54]	-0.15 _a	[-0.44, 0.14]	-0.46 _a	[-0.74, -0.17]
O	-0.27 _a	[-0.97, 0.44]	0.04 _a	[-0.01, 0.09]	0.20 _a	[-0.16, 0.57]
Unipolar						
N	1.62 _a	[0.95, 2.28]	—	—	1.31 _a	[0.99, 1.64]
E	-0.90 _a	[-1.08, -0.71]	—	—	-0.98 _a	[-2.12, 0.17]
Dysthymic disorder						
N	2.08_a	[1.46, 2.71]	2.30_a	[1.56, 3.03]	0.87 _b	[0.76, 0.98]
E	-1.79_a	[-2.35, -1.23]	-0.86 _b	[-0.86, -0.86]	—	—
GAD						
N	2.28_a	[2.11, 2.44]	1.68 _b	[1.20, 2.15]	2.75 _{a,b}	[1.55, 3.96]
E	-1.60_a	[-1.91, -1.28]	-0.34 _b	[-0.47, 0.21]	-1.52_a	[-2.12, -0.91]
PTSD						
N	2.53_a	[2.08, 2.98]	1.44 _b	[1.44, 1.44]	2.49_a	[0.87, 4.10]
E	-0.90 _a	[-1.85, 0.05]	-0.51 _a	[-0.51, -0.51]	-0.70 _a	[-0.70, -0.70]
Panic disorder						
N	1.97 _a	[1.36, 2.57]	1.86 _a	[0.86, 2.87]	1.80 _a	[0.85, 2.75]
E	-1.28_a	[-1.96, -0.60]	-0.42 _b	[-0.50, -0.33]	-1.00_a	[-1.00, -1.00]
Agoraphobia						
N	2.09_a	[1.59, 2.60]	1.34 _b	[0.70, 1.97]	2.00_a	[1.40, 2.59]
E	-1.85_a	[-2.10, -1.59]	-0.48 _b	[-0.66, -0.30]	-1.05_c	[-1.05, -1.05]
Social phobia						
N	2.29_a	[1.89, 2.69]	1.31 _b	[0.57, 2.06]	1.27 _b	[1.27, 1.27]
E	-2.36_a	[-3.32, -1.41]	-0.64 _b	[-0.92, -0.37]	-0.93_c	[-0.93, -0.93]
Specific phobia						
N	1.82_a	[1.28, 2.36]	0.85 _b	[0.32, 1.38]	0.84 _b	[0.84, 0.84]
E	-1.70_a	[-1.70, -1.70]	-0.12 _b	[-0.32, 0.08]	-0.34_c	[-0.34, -0.34]
OCD						
N	2.10_a	[1.50, 2.70]	2.10 _{a,b}	[0.87, 3.33]	1.27 _b	[1.27, 1.27]
E	-1.26_a	[-1.92, -0.61]	-0.32 _b	[-0.32, -0.32]	-0.43_c	[-0.48, -0.37]
SUD						
N	1.34 _a	[0.42, 2.26]	0.64 _a	[0.47, 0.81]	0.63 _a	[0.05, 1.22]
E	-0.65 _a	[-1.29, -0.01]	-0.15 _a	[-0.21, -0.10]	-0.12 _a	[-0.57, 0.32]
D	0.77 _a	[0.29, 1.24]	0.60 _a	[0.48, 0.73]	0.72 _a	[0.60, 0.85]
C	-1.20 _a	[-1.97, -0.42]	—	—	-0.93 _a	[-1.54, -0.32]
A	-0.47 _a	[-1.32, 0.38]	—	—	-0.80 _a	[-1.01, -0.59]
O	-0.11 _a	[-0.70, 0.47]	0.05 _a	[0.05, 0.05]	-0.37 _a	[-0.96, 0.22]

Note. Dashes indicate absence of relevant studies. Values that do not share subscripts do not fall into one another's credibility intervals (CrIs). The larger of two values that do not share a subscript is shown in bold. MDD = major depressive disorder; N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

controls to assigned controls and aggregated the resulting effect sizes across samples and measures. On average, both types of control groups had similar scores on all traits except for neuroticism, which actually was lower in internal controls ($d = 0.53$). Hence, the observed moderator effect reflects differences in the diagnostic groups.

Discussion

Patterns of Personality–Psychopathology Associations

The present study sought to quantify the links between broad personality traits and common Axis I disorders. The results are best interpreted as estimates of concurrent associations rather than

causal effects because the analyses were based almost entirely on cross-sectional data. We found that common mental illnesses are very strongly connected to personality, with some effect sizes surpassing a d of 2.0. All disorders examined were defined by high neuroticism and low conscientiousness, most exhibited low extraversion, and some were elevated on disinhibition. In contrast, agreeableness showed notable (negative) links only with SUD, whereas openness was largely unrelated to the analyzed conditions.

Malouff et al.'s (2005) meta-analysis arrived at similar conclusions, although their effect size estimates were considerably lower. That study did not examine disinhibition, but for neuroticism, conscientiousness, and extraversion their estimates were 46% lower on average. It appears that three factors contributed to this

Table 10
Breakout of Effect Sizes by Diagnostic System

Disorder	DSM-IV/ICD-10		Earlier systems	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD				
N	1.19 _a	[0.36, 2.02]	1.64 _a	[0.73, 2.55]
E	-0.59 _a	[-1.40, 0.22]	-0.67 _a	[-1.24, -0.11]
D	0.35 _a	[-0.35, 1.05]	0.27 _a	[0.00, 0.54]
C	-0.84 _a	[-1.39, -0.30]	-1.04 _a	[-1.43, -0.65]
A	-0.01 _a	[-0.67, 0.66]	-0.44 _a	[-0.81, -0.06]
O	-0.43 _a	[-1.04, 0.18]	0.20 _b	[-0.20, 0.60]
Unipolar				
N	1.50 _a	[1.12, 1.88]	1.59 _a	[0.77, 2.41]
E	-0.96 _a	[-1.27, -0.66]	-0.86 _a	[-1.73, 0.02]
Dysthymic disorder				
N	1.76 _a	[1.36, 2.16]	2.05 _a	[0.91, 3.20]
E	-2.05 _a	[-2.40, -1.70]	-1.09 _b	[-1.40, -0.77]
GAD				
N	1.85 _a	[1.24, 2.45]	2.23 _a	[1.66, 2.79]
E	-1.17 _a	[-2.05, -0.29]	-0.70 _a	[-1.28, -0.12]
PTSD				
N	1.72 _a	[1.39, 2.06]	2.54 _a	[1.47, 3.62]
E	-1.42_a	[-1.98, -0.87]	-0.53 _b	[-1.08, 0.03]
Panic disorder				
N	1.74 _a	[1.28, 2.20]	2.12 _a	[1.12, 3.11]
E	-1.26 _a	[-2.04, -0.48]	-0.82 _a	[-1.33, -0.32]
Agoraphobia				
N	1.98 _a	[1.52, 2.44]	1.43 _a	[0.68, 2.19]
E	-1.74_a	[-2.25, -1.22]	-0.51 _b	[-0.75, -0.28]
Social phobia				
N	1.96 _a	[1.59, 2.33]	1.46 _a	[0.50, 2.42]
E	-2.03 _a	[-2.79, -1.27]	-0.99 _a	[-2.16, 0.19]
Specific phobia				
N	1.70_a	[1.10, 2.31]	0.85 _b	[0.32, 1.37]
E	-1.36_a	[-2.04, -0.68]	-0.12 _b	[-0.32, 0.07]
OCD				
N	2.05 _a	[1.52, 2.59]	2.10 _a	[1.05, 3.15]
E	-1.45 _a	[-2.20, -0.70]	-0.77 _a	[-1.11, -0.43]
SUD				
N	1.05 _a	[0.22, 1.87]	0.91 _a	[0.10, 1.71]
E	-0.69 _a	[-1.41, 0.03]	-0.24 _a	[-0.67, 0.18]
D	0.82 _a	[0.47, 1.18]	0.69 _a	[0.34, 1.04]
C	-1.16 _a	[-2.05, -0.27]	-1.05 _a	[-1.56, -0.54]
A	-0.38 _a	[-1.23, 0.46]	-0.82 _a	[-1.16, -0.49]
O	-0.06 _a	[-0.57, 0.46]	-0.22 _a	[-0.78, 0.34]

Note. Values that do not share subscripts do not fall into one another's credibility intervals (CrIs). The larger of two values that do not share a subscript is shown in bold. *DSM-IV/ICD-10* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.)/*International Classification of Diseases* (10th ed.); MDD = major depressive disorder; N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

discrepancy. First, Malouff et al.'s analysis also included studies of clinical symptoms, which consistently reported smaller effect size estimates than investigations comparing diagnostic and control groups (47% weaker effect sizes on these three traits). Second, in addition to the diagnoses examined here (depressive, anxiety, and substance use disorders), the previous meta-analysis included seven other classes of mental illness, and their links to the three traits were 32% weaker on average. Third, Malouff et al. did not

adjust effect sizes for unreliability, and hence their estimates are inherently conservative. Indeed, we found that the reliability of personality scales used in psychopathology studies varies a great deal and often is well below .80 (see Table 2). Studies that employ weaker measures reduce average effect sizes, unless adjustments for unreliability are made (Hunter & Schmidt, 2004, pp. 75–136; Schmidt, Le, & Ilies, 2003).

With regard to specific trait–disorder associations, we obtained mixed support for our hypotheses. As expected, neuroticism had the strongest links to studied disorders. However, the individual diagnoses displayed less specificity than we had anticipated. All effect sizes were large, and the average elevation did not differ between the fear and distress clusters. This pattern reaffirms Widiger and Costa's (1994) conclusion that “neuroticism is an almost ubiquitously elevated trait within clinical populations” (p. 81). Neuroticism clearly is a crucial dimension that needs to be considered in any studies examining trait characteristics of psychopathology. Consistent with the hypotheses, effect sizes for extraversion were somewhat weaker, dysthymic disorder and social phobia had the strongest links to this trait, and observed associations were mostly independent from neuroticism. However, effect sizes for MDD, unipolar depression, and agoraphobia were smaller than expected. The biggest surprise involved conscientiousness, as it yielded consistently strong effects, the majority of which remained unambiguous after adjusting for neuroticism, and showed little evidence of specificity. This finding highlights and extends the observations of Malouff et al. (2005), who also noted the important role of low conscientiousness in psychopathology. Furthermore, it parallels research on physical health, which identified low conscientiousness as the strongest personality predictor of mortality and an important correlate of behaviors that contribute to poor health (Bogg & Roberts, 2004; Roberts et al., 2007).

The results for disinhibition were consistent with predictions: SUD exhibited a substantial elevation on the trait, which was fairly specific and persisted after controlling for neuroticism. Agreeableness and openness produced weak and equivocal associations. The only exception was SUD, as they showed moderate effects on agreeableness, some of which were unambiguous. Thus, findings for these two traits were broadly consistent with our predictions.

Nevertheless, a surprising number of our hypotheses were not supported. A likely reason for this discrepancy is that existing theories are based largely on studies that used Pearson's *r* to quantify personality–psychopathology associations, and our analyses indicate that such data present a different picture of these associations than Cohen's *d*. When we converted *d* to *r* (see Table 5), the results aligned appreciably better with our hypotheses. In these analyses, MDD and unipolar depression emerged among the strongest correlates of neuroticism. Their links to extraversion also became notable relative to other disorders. SUD were the leading correlates of conscientiousness, disinhibition, and agreeableness. In fact, the latter two traits showed no other notable effects. This change in apparent associations is due to the base rates of the corresponding conditions (i.e., the size of the diagnostic group relative to the control group). Specifically, because low base rates reduce Pearson's *r* but do not affect Cohen's *d*, a trait may show a stronger correlation with a more prevalent disorder even though it is more elevated in a less common condition. In the general population, SUD and MDD are the most prevalent disorders (Compton et al., 2005; Kessler, Berglund, et al., 2005), and they

Table 11
Breakout of Effect Sizes by Method of Diagnosis

Disorder	SCID		Structured		Informal		Other	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD								
N	1.46 _{a,b}	[0.64, 2.29]	1.00 _a	[0.25, 1.75]	1.80 _{a,b}	[0.94, 2.66]	2.03_b	[1.56, 2.51]
E	-0.82 _{a,b}	[-1.44, -0.19]	-0.21 _a	[-0.44, 0.01]	-0.82 _{a,b}	[-1.65, 0.01]	-1.41 _b	[-2.07, -0.75]
D	0.26 _a	[-0.06, 0.58]	0.36 _a	[0.36, 0.36]	0.30 _a	[-0.38, 0.99]	0.32 _a	[0.13, 0.52]
C	-1.00 _a	[-1.52, -0.49]	-0.60 _a	[-1.00, -0.20]	-0.57 _a	[-1.13, -0.01]	-1.00 _a	[-1.16, -0.84]
A	-0.31 _{a,b}	[-0.63, 0.01]	-0.05 _a	[-0.34, 0.24]	-0.51_b	[-0.61, -0.41]	0.41 _a	[-0.33, 1.14]
O	0.14 _a	[-0.22, 0.51]	0.10 _a	[-0.15, 0.34]	-0.76_b	[-1.00, -0.52]	-0.64_b	[-1.29, 0.02]
Unipolar								
N	2.07_a	[1.74, 2.39]	1.30 _b	[1.30, 1.30]	1.34 _{a,b}	[0.55, 2.12]	1.57 _{a,b}	[0.84, 2.31]
E	-1.02 _a	[-1.61, -0.43]	-0.75 _a	[-1.24, -0.26]	-0.82 _a	[-0.82, -0.82]	-1.08 _a	[-1.80, -0.36]
Dysthymic disorder								
N	2.33 _{a,b}	[1.36, 3.30]	2.31_a	[1.56, 3.07]	2.90_a	[2.90, 2.90]	1.50 _b	[0.83, 2.17]
E	-1.25_a	[-1.54, -0.96]	-0.85 _b	[-0.95, -0.74]	-0.80 _b	[-0.80, -0.80]	-1.98_c	[-2.42, -1.54]
GAD								
N	2.42 _a	[1.97, 2.86]	1.69 _b	[1.21, 2.16]	4.53_c	[4.53, 4.53]	2.16 _{a,b}	[2.16, 2.16]
E	-1.04_a	[-1.54, -0.55]	-0.37 _b	[-0.37, -0.37]	-2.25_c	[-2.25, -2.25]	-1.77_d	[-1.77, -1.77]
PTSD								
N	2.68_a	[2.24, 3.12]	2.68_a	[2.68, 2.68]	1.79 _b	[1.79, 1.79]	2.47 _{a,b}	[1.13, 3.80]
E	-0.51 _a	[-1.20, 0.17]	-0.51 _a	[-0.51, -0.51]	—	—	-1.31_b	[-1.93, -0.69]
Panic disorder								
N	1.91 _{a,b}	[0.86, 2.95]	2.02 _{a,b}	[1.17, 2.87]	1.39 _a	[1.39, 1.39]	1.98_b	[1.67, 2.29]
E	-0.79 _a	[-1.34, -0.24]	-0.48 _a	[-0.50, -0.47]	—	—	-1.58_b	[-2.05, -1.10]
Agoraphobia								
N	1.30 _a	[0.32, 2.28]	1.42 _a	[0.78, 2.07]	2.61_b	[2.61, 2.61]	2.02 _a	[1.58, 2.47]
E	-0.49 _a	[-0.56, -0.41]	-0.53 _a	[-0.77, -0.28]	—	—	-1.89_c	[-1.89, -1.89]
Social phobia								
N	1.66 _{a,b}	[0.25, 3.07]	1.41 _a	[0.72, 2.09]	—	—	2.17_b	[2.17, 2.17]
E	-0.92 _a	[-1.68, -0.17]	-0.72 _a	[-0.97, -0.47]	—	—	-2.63_b	[-3.49, -1.77]
Specific phobia								
N	0.70 _a	[0.58, 0.82]	0.86 _a	[0.32, 1.40]	—	—	1.82_b	[1.28, 2.36]
E	-0.13 _a	[-0.21, -0.04]	-0.13 _a	[-0.33, 0.08]	—	—	-1.70_b	[-1.70, -1.70]
OCD								
N	2.28_a	[1.90, 2.66]	2.07 _{a,b}	[0.88, 3.27]	1.47 _b	[0.71, 2.24]	2.14 _{a,b}	[1.73, 2.56]
E	-1.03_a	[-1.39, -0.66]	-0.50 _b	[-0.91, -0.09]	-0.96 _{a,b}	[-1.43, -0.49]	-1.70_a	[-2.50, -0.91]
SUD								
N	1.22 _{a,b}	[0.32, 2.12]	0.61 _a	[0.33, 0.89]	0.86 _{a,b}	[-0.06, 1.77]	1.60_b	[1.16, 2.04]
E	-0.50 _{a,b}	[-1.18, 0.18]	-0.18 _a	[-0.38, 0.02]	-0.32 _{a,b}	[-0.91, 0.27]	-0.83_b	[-1.48, -0.19]
D	0.74 _a	[0.50, 0.98]	0.64 _a	[0.43, 0.84]	0.79 _a	[0.36, 1.23]	0.52 _a	[0.16, 0.87]
C	-1.12 _{a,b}	[-1.90, -0.34]	-0.55 _a	[-1.02, -0.08]	-1.33 _{a,b}	[-1.77, -0.49]	-1.41_b	[-1.83, -0.99]
A	-0.68 _a	[-1.54, 0.18]	-0.63 _a	[-0.63, -0.63]	-0.32 _a	[-0.66, 0.02]	-0.53 _a	[-0.93, -0.13]
O	-0.06 _a	[-0.56, 0.44]	-0.09 _{a,b}	[-0.68, 0.51]	-0.26 _{a,b}	[-0.93, 0.42]	-0.58_b	[-0.58, -0.58]

Note. Dashes indicate absence of relevant studies. Values that do not share subscripts do not fall into one another's credibility intervals (CrIs). The larger of two values that do not share a subscript is shown in bold. MDD = major depressive disorder; N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

also had the two highest base rates in our analyses. Hence, past findings of remarkably strong links of these conditions to personality appear to be, in part, artifacts of differential base rates. These results underscore the value of using Cohen's *d*—rather than Pearson's *r*—when examining associations between traits and dichotomous diagnoses.

Of note, our basic findings parallel research on the links between Axis I and personality disorders. Large epidemiologic studies have consistently found the two domains to be strongly related (Coid, Yang, Tyrer, Roberts, & Ullrich, 2006; Grant et al., 2005, 2004; Huang et al., 2009; Lenzenweger, Lane, Loranger, & Kessler, 2007). This can be expected, given that recent meta-analyses established close links between personality disorders and

normal personality (O'Connor, 2005; Samuel & Widiger, 2008; Saulsman & Page, 2004). Indeed, these higher order traits are thought to lie at the core of personality disorders (Clark, 2007; Widiger & Trull, 2007). Thus, evidence from trait and diagnostic perspectives are converging to support an important role of personality functioning in Axis I illnesses.

Specificity of Observed Associations

There was little specificity in personality profiles among the disorders (in particular, all conditions were associated with both high neuroticism and low conscientiousness). This finding is not altogether surprising given high levels of comorbidity among

Table 12
Breakout of Effect Sizes by Time Frame

Disorder	Current		Broader	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD				
N	1.79 _a	[1.17, 2.41]	1.09 _a	[0.23, 1.96]
E	-0.93 _a	[-1.51, -0.35]	-0.45 _a	[-1.19, 0.29]
D	0.29 _a	[-0.26, 0.84]	0.28 _a	[0.06, 0.50]
C	-0.90 _a	[-1.45, -0.35]	-0.91 _a	[-1.37, -0.46]
A	-0.32 _a	[-0.64, 0.01]	0.10 _a	[-0.72, 0.91]
O	-0.12 _a	[-0.74, 0.51]	-0.37 _a	[-1.08, 0.35]
Unipolar				
N	1.58 _a	[0.94, 2.22]	1.47 _a	[0.88, 2.06]
E	-0.90 _a	[-1.66, -0.13]	-0.96 _a	[-0.96, -0.96]
Dysthymic disorder				
N	1.21 _a	[0.70, 1.73]	2.35_b	[1.72, 2.98]
E	-0.89 _a	[-1.00, -0.78]	-1.84_b	[-2.35, -1.33]
GAD				
N	2.18 _a	[1.45, 2.92]	1.90 _a	[1.32, 2.48]
E	-0.80 _a	[-1.55, 0.05]	-1.06 _a	[-1.91, -0.22]
PTSD				
N	1.95 _a	[1.06, 2.85]	2.20 _a	[1.57, 2.83]
E	-0.32 _a	[-0.74, 0.11]	-1.37_b	[-1.81, -0.94]
Panic disorder				
N	1.64 _a	[1.13, 2.16]	2.08 _a	[1.22, 2.94]
E	-0.59 _a	[-0.91, -0.26]	-1.30_b	[-1.99, -0.62]
Agoraphobia				
N	1.80 _a	[1.31, 2.29]	1.57 _a	[0.80, 2.34]
E	-0.74 _a	[-0.74, -0.74]	-1.01 _a	[-1.91, -0.12]
Social phobia				
N	1.52 _a	[1.01, 2.04]	1.66 _a	[0.72, 2.59]
E	-1.53 _a	[-3.15, 0.09]	-1.24 _a	[-2.30, -0.18]
Specific phobia				
N	1.12 _a	[1.12, 1.12]	0.87 _a	[0.20, 1.54]
E	-0.37 _a	[-0.37, -0.37]	-0.15 _a	[-0.65, 0.35]
OCD				
N	1.93 _a	[1.25, 2.62]	2.15 _a	[1.27, 3.02]
E	-1.05 _a	[-1.56, -0.55]	-1.17 _a	[-2.02, -0.32]
SUD				
N	1.06 _a	[0.34, 1.77]	0.85 _a	[-0.11, 1.81]
E	-0.40 _a	[-0.93, 0.13]	-0.41 _a	[-1.15, 0.32]
D	0.73 _a	[0.27, 1.20]	0.70 _a	[0.51, 0.90]
C	-1.09 _a	[-1.87, -0.32]	-1.11 _a	[-1.79, -0.44]
A	-0.59 _a	[-1.43, 0.24]	-0.62 _a	[-1.01, -0.23]
O	-0.11 _a	[-0.62, 0.40]	-0.27 _a	[-0.91, 0.36]

Note. Values that do not share subscripts do not fall into one another's credibility intervals (CrIs). The larger of two values that do not share a subscript is shown in bold. MDD = major depressive disorder; N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

mental illnesses, which means that different diagnoses largely capture the same people. Given such an overlap, the similarity of profiles is to be expected. In this regard, it is noteworthy that the vast majority of studies in our review did not exclude cases because of comorbidity with anxiety, depressive, or substance use disorders. As the result, the diagnostic groups that we analyzed included many cases that could have been assigned to other diagnostic groups just as easily. In fact, in primary studies that examined multiple disorders, some of the same cases were included in different analyses. Hence, comorbidity certainly contributed to observed low specificity.

Unfortunately, high comorbidity is inherent in the *DSM-IV* (Clark et al., 1995; Krueger & Markon, 2006; D. Watson, 2009). It may be possible to reveal specific trait links by removing overlapping cases, but the remaining cases will hardly be representative of the target disorder. Indeed, only 23% of people diagnosed with a common mental illness do not have at least one other Axis I condition (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). In other words, "pure" cases actually represent a small minority of all people with a given disorder. It appears that unless the diagnostic system is thoroughly reorganized, we will not be

Table 13
Breakout of Effect Sizes by Control Group Status

Disorder	Control group		No control group	
	<i>d</i>	80% CrI	<i>d</i>	80% CrI
MDD				
N	1.05 _a	[0.24, 1.85]	1.82 _a	[1.16, 2.49]
E	-0.30 _a	[-0.73, 0.13]	-1.11_b	[-1.81, -0.41]
D	0.24 _a	[0.13, 0.34]	0.38 _a	[-0.22, 0.98]
C	-0.83 _a	[-1.29, -0.37]	-0.91 _a	[-1.43, -0.39]
A	-0.35 _a	[-0.75, 0.05]	-0.12 _a	[-0.77, 0.54]
O	-0.06 _a	[-0.44, 0.33]	-0.24 _a	[-0.95, 0.47]
Unipolar				
N	1.40 _a	[0.94, 1.86]	1.61 _a	[0.93, 2.28]
E	-0.64 _a	[-1.19, -0.09]	-1.06 _a	[-1.63, -0.50]
Dysthymic disorder				
N	2.30 _a	[1.57, 3.04]	1.68 _a	[0.80, 2.57]
E	-0.86 _a	[-0.86, -0.86]	-1.79_b	[-2.35, -1.24]
GAD				
N	1.84 _a	[1.23, 2.45]	2.44 _a	[2.25, 2.63]
E	-0.99 _a	[-1.90, -0.09]	-1.19 _a	[-1.19, -1.19]
PTSD				
N	2.18 _a	[1.37, 2.99]	2.42 _a	[1.06, 3.77]
E	-0.54 _a	[-1.12, 0.04]	-1.27_b	[-1.95, -0.59]
Panic disorder				
N	1.92 _a	[0.90, 2.95]	1.92 _a	[1.54, 2.30]
E	-0.71 _a	[-1.20, -0.22]	-1.41_b	[-2.07, -0.74]
Agoraphobia				
N	1.34 _a	[0.68, 2.01]	2.19_b	[1.97, 2.40]
E	-0.51 _a	[-0.73, -0.28]	-1.82_b	[-2.12, -1.53]
Social phobia				
N	1.38 _a	[0.63, 2.13]	2.34_b	[1.92, 2.75]
E	-0.90 _a	[-2.06, 0.27]	-2.12_b	[-2.57, -1.68]
Specific phobia				
N	0.85 _a	[0.32, 1.37]	1.82_b	[1.28, 2.36]
E	-0.13 _a	[-0.32, 0.07]	-1.70_b	[-1.70, -1.70]
OCD				
N	1.86 _a	[0.87, 2.85]	2.37 _a	[2.25, 2.49]
E	-0.88 _a	[-1.46, -0.31]	-1.41 _a	[-2.15, -0.68]
SUD				
N	0.67 _a	[0.09, 1.25]	1.29 _a	[0.43, 2.16]
E	-0.28 _a	[-0.94, 0.39]	-0.52 _a	[-1.03, 0.00]
D	0.72 _a	[0.44, 1.00]	0.72 _a	[0.30, 1.15]
C	-1.07 _a	[-2.08, -0.06]	-1.11 _a	[-1.69, -0.54]
A	-0.84 _a	[-1.60, -0.09]	-0.49 _a	[-1.11, 0.14]
O	-0.21 _a	[-0.84, 0.41]	-0.12 _a	[-0.62, 0.39]

Note. Values that do not share subscripts do not fall into one another's credibility intervals (CrIs). The larger of two values that do not share a subscript is shown in bold. MDD = major depressive disorder; N = neuroticism; E = extraversion; D = disinhibition; C = conscientiousness; A = agreeableness; O = openness; unipolar = broad diagnosis of unipolar depression; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; SUD = substance use disorders.

able to achieve a high level of specificity in trait profiles of mental illnesses. Extensive comorbidity has been complicating research on other processes involved in mental illness, which underscores the need to develop an empirically based taxonomy that addresses this and other structural problems (D. Watson & Clark, 2006).

New classification schemes have sought to organize disorders according to comorbidity patterns (Krueger & Markon, 2006; D. Watson, 2005b), and these taxonomies are expected to show more distinct correlates. In fact, the SUD personality profile was notably distinct from the profiles of the anxiety and depressive disorders. This is significant because the above-mentioned new taxonomies classify SUD as externalizing, whereas the depressive and anxiety disorders are viewed as internalizing conditions. Within the internalizing spectrum, however, we found no appreciable differences between the profiles of the fear and distress disorders. Thus, our data support the utility of the internalizing–externalizing distinction with regard to trait correlates, but suggest that the internal structure of the internalizing spectrum may need to be refined further to achieve greater specificity.

With regard to individual disorders, three exhibited notably distinct profiles. First, specific phobia showed a strikingly normative profile, which remained within one standard deviation of the general population mean on all traits. Of note, specific phobia is generally considered to be one of the least severe Axis I disorders (Mineka, Watson, & Clark, 1998; D. Watson et al., 2005), and the severity of a disorder appears to be correlated with the extremity of its profile (Krueger et al., 2001; Ruiz et al., 2008). Alternatively, given that conditioning plays an important role in the development of phobias (e.g., Mineka & Sutton, 2006; Öhman & Mineka, 2001), specific phobia may be more externally determined and less contingent on personality dysfunction than other common mental disorders.

At the other extreme, dysthymic disorder had perhaps the most pathological profile. Indeed, it showed more extreme extraversion, conscientiousness, and disinhibition scores than all other conditions considered. We cannot be completely confident in these findings, as they were based on a fairly small number of studies. However, they are consistent with the argument that dysthymic disorder can be best viewed as a form of personality pathology (e.g., D. Watson & Clark, 1995). Indeed, this condition tends to be chronic and often is lifelong (e.g., D. N. Klein, Shankman, & Rose, 2006). Hence, prominent personality disturbance can be expected to manifest in dysthymic disorder.

The SUD profile was marked by high disinhibition, low conscientiousness, and low agreeableness but relatively weak effects on neuroticism and extraversion. This pattern is consistent with the previous meta-analysis of this condition: Ruiz et al. (2008) did not examine disinhibition but found a similar pattern for the other four traits. Moreover, meta-analyses of antisocial personality disorder—another externalizing condition—found that it has substantial and specific links to disagreeableness and unconscientiousness (Samuel & Widiger, 2008; Saulsman & Page, 2004). These results support recent models that argue for a special role of disinhibition-related traits (including conscientiousness and agreeableness) in SUD and other externalizing conditions (Clark & Watson, 2008; Krueger et al., 2007; Sher & Trull, 1994). Indeed, there is evidence that these traits and SUD share genetic roots and are shaped by some of the same environmental factors (Krueger et al., 2002; Roberts, Jackson, Burger, & Trautwein, 2009). Internalizing dis-

orders, on the other hand, were found to share genetic variance with neuroticism (Kendler, Gatz, Gardner, & Pedersen, 2006; Middeldorp, Cath, Van Dyck, & Boomsma, 2005). These etiological data are consistent with the distinct personality profile of SUD, as compared with internalizing disorders, observed in the present study. Indeed, our results demonstrate that disinhibition and related traits rival neuroticism as key personality features of SUD.

Implications

Our findings have important implications for clinical psychology. Most notably, we found much stronger associations between personality and mental health than existing theories have acknowledged or anticipated. Indeed, we observed effects of a magnitude that is rarely seen in social sciences (note that eight neuroticism effects in Table 4 are $d > 1.5$). Dimensions of normal personality are not emphasized in many theories of psychopathology (Clark, 2005; D. Watson et al., 2006), but it is clear from the present results that no model of anxiety, depressive, or substance use disorders will be complete without some consideration of these traits. Furthermore, theories that include personality usually focus on neuroticism and its components. Our findings indicate that although neuroticism is the strongest correlate of common mental disorders, other traits have independent links to these conditions and should not be ignored.

Similarly, normal personality is rarely assessed in clinical practice. Our findings suggest that traits can be helpful in case conceptualization and making prognosis, although more longitudinal research is needed to confirm this. In fact, there is emerging evidence of the utility of personality assessment in treatment planning (Bagby et al., 2008; Quilty et al., 2008). Traits also can be used to guide prevention efforts, and previous research found them to be especially useful for identifying individuals at risk for onset of mental illness (Smit, Beekman, Cuijpers, de Graaf, & Vollebergh, 2004; Tokuyama, Nakao, Seto, Watanabe, & Takeda, 2003; Verkerk, Denollet, Van Heck, Van Son, & Pop, 2005). With regard to applied clinical work, one noteworthy advantage of the traits we have examined here is that they can be assessed easily in only a few minutes.

Our results also inform several theories directly. Most notably, we and others have argued that individual differences in neuroticism/negative emotionality are central to understanding patterns of comorbidity among these disorders (e.g., Clark et al., 1994; Kotov et al., 2007; D. Watson et al., 2005). Specifically, it has been posited that comorbidity primarily reflects the shared influence of neuroticism/negative emotionality and that diagnostic co-occurrence can be reduced dramatically by controlling for scores on this trait (see Kotov et al., 2007). Our nonspecific findings, however, strongly suggest that comorbidity patterns cannot be primarily attributed to the influence of neuroticism.

For example, D. Watson (2009, Table 1) computed weighted mean tetrachoric correlations among depressive and anxiety disorders based on four national epidemiological studies. He found that the diagnosis of MDD was strongly comorbid with GAD (weighted mean tetrachoric $r = .64$), but overlapped less with social phobia ($r = .50$) and agoraphobia ($r = .48$). If neuroticism is largely responsible for these associations, then major depression and GAD should have particularly strong links to this trait. Our

data, however, indicate that this is not the case. Indeed, the average effect size for MDD and GAD ($d = 1.65$) was virtually identical to that for social phobia and agoraphobia ($d = 1.62$). More generally, our data provide little support for the argument that the prominent comorbidities among the distress disorders (MDD, dysthymic disorder, GAD, PTSD) are due to their exceptional links to neuroticism.

Perhaps our most surprising finding was the unexpectedly weak link between MDD and extraversion. This result is inconsistent with the multilevel trait predictor model and other theories that originated from the tripartite model of anxiety and depression (e.g., Clark et al., 1994; Kotov et al., 2007). The present data suggest that extraversion is not specific to major depression. On the other hand, dysthymic disorder had the lowest score on this trait of all disorders considered, so extraversion clearly plays an important role in some forms of depression. In interpreting these observations, it also is important to keep in mind that our data reflect associations of the broad higher order trait. In this regard, Naragon-Gainey, Watson, and Markon (2009) recently found that the specific facets of extraversion relate differently to symptoms of depression and social anxiety. In their study, social anxiety was associated with all four analyzed facets of the trait (sociability, ascendance, positive emotionality, fun-seeking), whereas depressive symptoms correlated strongly only with low positive emotionality. These results illustrate the importance of moving beyond the “big” traits and conducting hypothesis-driven examinations of the lower level of the personality hierarchy. Furthermore, they suggest that analyses focused specifically on positive emotionality would provide stronger support for theoretical schemes based on the tripartite model (see also D. Watson & Naragon-Gainey, 2009).

MDD also had a relatively small elevation on neuroticism, which suggests that personality is less central to this disorder in general. One possible explanation of these observations is that the MDD diagnosis is quite broad and includes not only chronically ill individuals but also people who experienced a single episode of MDD and will never become depressed again (Eaton et al., 2008). Findings for dysthymic disorder, which represents long-standing depression, suggest that the chronicity of illness is linked to the extremity of the trait profile. Hence, it is possible that single-episode cases of MDD diluted associations between depression and personality. This likely was not apparent in previous studies because the relatively high prevalence of MDD offset this diluting effect and resulted in prominent correlations as compared with less common disorders. Our analytic approach is not affected by base rates and thus was able to reveal the relatively low trait profile of MDD. In sum, we can conclude that extraversion contributes to depression, but its effect is more important for chronic forms of this illness.

We also obtained intriguing results for GAD. This condition is often viewed as an extreme form of neuroticism (e.g., Mineka et al., 1998; D. Watson et al., 2005), so we expected a prominent elevation on that trait and negligible effects on others. GAD was, in fact, strongly associated with neuroticism ($d = 1.96$). However, it also showed notable links to conscientiousness ($d = -1.13$) and disinhibition ($d = 0.44$), which remained unequivocal even after neuroticism was controlled. These results suggest that the trait correlates of GAD merit closer scrutiny in future research.

Nature of Personality–Disorder Associations

The nature of the observed links is not yet clear. Our findings established strong concurrent associations between common mental disorders and general personality traits, but we cannot infer causality from these data. Previous research has provided some support for each of the three basic causal patterns: (a) Traits influence disorders (vulnerability and pathoplasty models), (b) disorders influence personality (scar and complication models), and (c) both are influenced by another variable (common cause model; Bienvenu & Stein, 2003; Christensen & Kessing, 2006; Clark et al., 1994; M. H. Klein, Wonderlich, & Shea, 1993; Ormel et al., 2004). Hence, it is probable that links observed in the present study reflect the joint effects of multiple causal processes. Importantly, some of the effects—especially those involving neuroticism—were so strong that direct causation is unlikely to account for them fully. It seems likely that observed associations to some extent reflect shared roots and perhaps conceptual overlap between the two domains (e.g., an episode of mental illness may be an exacerbation of a stable pathological trait), as proposed by the spectrum model. On the other hand, even the strongest effect sizes were far from identity, which suggests that spectrum relations are not the sole mechanism involved. Moreover, we found little evidence of the specific trait–disorder links implied by this model. Our data indicate that the same constellation of basic traits (i.e., high neuroticism, low conscientiousness, and low extraversion) contributes to all internalizing disorders; consequently, other factors (e.g., environmental influences) have to account for differences among the syndromes within this spectrum.

Beyond these general considerations, our study provided directly relevant evidence only with regard to the complication model. We did not find support for this model, given that current diagnosis was not associated with a more pathological personality profile. In fact, in some cases a broad time frame was associated with greater personality deviance (see Table 12). These results could be potentially due to the scar effect, so that profiles remain elevated for many years after the offset of active illness. Importantly, our analyses were cross-sectional and so did not allow for a clean differentiation between current and past cases, because studies with broad diagnostic time frames capture a mix of remitted and nonremitted participants. Also, current diagnoses may have included some individuals who recently entered remission. Thus, a more rigorous test of the complication model requires longitudinal data. Few such studies have been performed to date, and they have produced inconsistent results, with some reporting differences between remitted and nonremitted individuals (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Ormel et al., 2004; Reich, Noyes, Hirschfeld, Coryell, & O’Gorman, 1987) but others failing to find the predicted association between changes in psychopathology and personality (De Fruyt, Van Leeuwen, Bagby, Rolland, & Rouillon, 2006; Quilty et al., 2008; Santor, Bagby, & Joffe, 1997). Hence, the status of the complication model remains an open question.

With regard to specific traits, our findings for conscientiousness are especially notable, because strong links between this personality dimension and internalizing conditions were not anticipated. In fact, theories of anxiety and depressive disorders largely ignore conscientiousness, and thus there is little conceptual framework for interpreting the present results. However, two possibilities seem

most likely (K. W. Anderson & McLean, 1997). On the one hand, the demoralization and negative self-perceptions common in internalizing psychopathology may lead to lower conscientiousness scores, as this trait reflects a sense of self-efficacy and goal-related striving. This account is consistent with scar and complication models. On the other hand, individuals low in conscientiousness are prone to failures and poor coping, which can contribute to psychopathology. This interpretation reflects a vulnerability conceptualization of the trait. Several theoreticians have argued in favor of this second, vulnerability-based account (K. W. Anderson & McLean, 1997; Lonigan, Vasey, Phillips, & Hazen, 2004). Unfortunately, longitudinal data are too limited to clarify the nature of these intriguing associations.

Heterogeneity of Effect Sizes

Our discussion so far has been concerned with the average personality profiles of mental disorders. However, we also found that the size of these elevations varied considerably across the studies. Hence, the current report focused on CrIs rather than confidence intervals. Indeed, in most cases true heterogeneity was much larger than sampling error. Reporting of CrIs helped us to describe the distribution of true effects, rather than just average estimates and their precision. The majority of the CrIs were quite large, which implies the existence of noteworthy moderators. We examined six such variables.

With regard to personality measures, we found that Eysenck's scales show consistently weaker effects than the other instruments, especially the NEO inventories. This observation agrees with the conclusion of Malouff et al. (2005) that the NEO produces stronger results. The reasons for this discrepancy are unclear. One possibility is that it reflects differences in item content. In this regard, the NEO contains a broader range of explicit anxiety- and depression-related questions. In fact, the NEO-PI and NEO-PI-R both specifically include anxiety, depression, and positive emotionality facets. These results underscore the fact that even strongly correlated scales that target the same constructs can show differential associations with other measures.

The near absence of notable differences between *DSM-IV/ICD-10* and earlier psychiatric nosologies reinforces the contention that conceptualizations of common diagnoses have remained fairly constant since 1980. The few differences that were observed consistently indicated stronger effects for more recent conceptualizations, which may reflect an increase in the precision of the diagnoses.

We also observed weaker effects in epidemiologic samples, which replicated the results of Ruiz et al. (2008). In epidemiologic studies, diagnostic groups mostly consist of individuals who are not in treatment (e.g., Wang et al., 2007); treatment seeking, in turn, is closely linked to the severity of the illness (P. Cohen & Cohen, 1984; Wang et al., 2007). Hence, this moderator effect probably reflects differential severity of disorders in community and patient populations. It is noteworthy that Ruiz et al. arrived at the same conclusion.

With regard to the method of diagnosis, our analyses revealed weaker effects for completely structured interviews. However, this ascertainment method was largely confined to epidemiologic studies; hence, this result simply may be another consequence of lesser disorder severity in community samples.

Finally, we observed higher elevations in studies without a control group and thus confirmed the finding of Malouff et al. (2005). As described earlier, this effect did not result from differences between assigned and internal control groups. On the other hand, nearly all samples that needed a control group came from the patient population. Furthermore, almost all epidemiologic studies had an internal control group. Hence, this apparent moderator likely is another manifestation of the severity effect.

Unfortunately, we could not fully test our explanations of the ascertainment method and control group findings. Considerable overlap with sample type and the modest number of available studies precluded hierarchical analysis, in which data are stratified along multiple moderators simultaneously. Because of these limitations of the available data, hierarchical analysis would have resulted in a large number of empty cells. Another alternative is to employ a weighted least squares regression to jointly predict effect sizes with the six moderators (Hunter & Schmidt, 2004, p. 389). However, this approach requires more samples than we were able to obtain even for the best-studied association (MDD with neuroticism). Moreover, many of our moderators were nominal variables with three or four levels and could not be used in regression analyses as such. These powerful approaches to the investigation of moderator effects will become possible in the future as the literature continues to grow. Nevertheless, it appears that links between personality traits and illness severity are of considerable practical and theoretical importance, and thus deserve greater attention in this literature. For instance, primary studies can stratify diagnostic groups into mild, moderate, and severe categories—using the Global Assessment of Functioning or another index of impairment—rather than simply compare cases with and without a diagnosis.

Limitations

We have already mentioned three important limitations of the present investigation. First, the number of prospective studies was too small to examine them separately and begin to infer the direction of causality. This topic should be a top priority for future research. Second, the size of the literature was sufficient to examine moderator effects for only 30 of the 66 trait-disorder pairs. Moreover, we could evaluate the moderators only one at a time, as a larger K is required for hierarchical and regression analyses. Third, because of comorbidity among disorders, some of the same people were included in different analyses in studies reporting on multiple diagnoses. As a result, we could not perform formal statistical comparisons of diagnostic groups as we did with moderators.

We should mention several other caveats. One is that certain control groups were drawn from the general population and thus probably included at least some individuals with a targeted disorder. This concern is consistent with our finding that internal controls had lower neuroticism scores than the general population controls. The level of resulting misclassification appears to be relatively small, but it is important to recognize that our estimates are somewhat conservative. Second, reported associations are probably somewhat inflated because personality and psychopathology assessments were derived from the same source (participant) in some studies. This was especially true for epidemiologic studies. Many others, however, based diagnoses on multiple

sources and lengthy periods of observation. Other limitations concern our adjustment for unreliability. We corrected for unreliability of personality scales using Cronbach's α , but this index does not reflect transient error, which is the random error associated with a given occasion (Schmidt et al., 2003). Transient error can be corrected by taking into account the test-retest reliability of measures, but unfortunately such data are rarely reported (Chmielewski & Watson, 2009; D. Watson, 2004). Indeed, so few of the reviewed studies provided test-retest correlations that we could not create a corresponding artifact distribution. Similarly, we were unable to control for unreliability in diagnostic assignment, as interrater reliability was almost never reported. For these three reasons, our results underestimate the true associations between personality and psychopathology. It is critical for future primary studies on this topic to report data on all sources of unreliability: the internal consistency of the personality measures, the retest reliability of these scales, and the interrater reliability of diagnoses.

Future Directions

Our review indicated that quite a bit is already known about associations between common mental disorders and general traits, such that further cross-sectional studies of this same type are not needed for MDD and SUD. Instead, future research can most profitably focus on several specific issues. As discussed earlier, stratifying diagnostic groups on severity can help to elucidate its effects on personality profiles. Prospective longitudinal studies can clarify the direction of causality between personality and mental illness. This question is of particular interest for both theory building and treatment development (Bienvenu & Stein, 2003; Clark et al., 1994). Moreover, we know less about the role of temperament in childhood disorders than about personality-psychopathology associations in adults (Tackett, 2006). Research in this age group is particularly promising because it may help to explicate the basic etiology of mental illness.

Another understudied question concerns the links between psychopathology and specific lower order traits. A focus on this topic is important because, as was noted earlier, specific and powerful correlates may emerge from this fine-grained level of analysis (Naragon-Gainey et al., 2009; D. Watson et al., 2006; D. Watson & Naragon-Gainey, 2009). In fact, the Ruiz et al. (2008) meta-analysis reviewed several facet-level studies and found stronger effects for some facets than for general traits. For example, SUD had a closer link with a lower order dimension of trust ($r = -.36$) than with the broader construct of agreeableness ($r = -.20$). Similarly, SUD essentially were unrelated to extraversion ($r = -.06$) but showed significant—and, it is interesting to note, opposite—effects on two of its components, warmth ($r = -.23$) and excitement seeking ($r = .17$).

Having said this, however, we also must acknowledge two problems that researchers face in adopting a lower order, facet-level approach. First, we currently lack a consensus regarding the component traits within each of these higher order domains (for discussions, see Naragon-Gainey et al., 2009; Roberts, Walton, & Bogg, 2005). Thus, although researchers typically adopt a particular instrument-based facet scheme (e.g., the NEO-PI-R) as a matter of convenience, it is unclear how well this actually captures the true structure of personality. For research in this area to progress, it therefore will be important to clarify the lower levels

of the trait hierarchy. Second, many current facet scales have problematic psychometric properties, showing inadequate reliability and uncertain convergent and discriminant validity. For instance, several NEO-PI-R facet scales have coefficient alphas below .70 (Costa & McCrae, 1992). Our review indicates that this problem is by no means limited to lower order scales, as several of the higher order trait measures included in our analyses also displayed poor reliability (see Table 2). Consequently, it will be important to improve the assessment of key personality constructs at all levels of the hierarchy.

The large majority of the studies in our meta-analysis used self-ratings to assess personality. Thus, it will be informative for future studies to move beyond self-report and employ other methods to assess traits. For example, it is clear that informant ratings contain important incremental information that can enhance the prediction of psychopathology in many contexts (e.g., De Los Reyes & Kazdin, 2005; Gizer et al., 2008; Oltmanns & Turkheimer, 2009; Ready & Clark, 2002). It therefore would be valuable to conduct comparative analyses that examine how specific disorders relate to (a) self-rated traits, (b) informant-rated traits, and (c) personality scores that are aggregated across multiple raters.

Finally, future personality research should also pay more attention to less prevalent disorders. Indeed, such work may reveal novel patterns of association that would expand our theories in fruitful ways. For example, there is emerging evidence that bipolar disorder has a strong positive association with openness (Tackett et al., 2008), which is in contrast to the generally negative (and weak) link between this trait and depression. If this effect is replicated, it would suggest important differential processes among mood disorders.

Conclusions

Perhaps the most important finding of the present study is that several "big" personality traits were highly correlated with anxiety, depressive, and substance use disorders. Indeed, these effects were substantially stronger than had been anticipated. It appears that greater attention to personality dimensions is warranted across clinical psychology. These traits may be helpful in directing prevention efforts, developing case conceptualizations, and making clinical prognoses.

Importantly, neuroticism was not the only important correlate of these disorders. Conscientiousness and extraversion were associated with a range of conditions independently of neuroticism. Disinhibition and agreeableness also showed some notable links. Contrary to existing theories, we found that the connection between MDD and extraversion is relatively weak. We also were surprised to discover that conscientiousness is strongly and broadly related to internalizing disorders. Both of these findings suggest important new directions for etiologic research.

Another important observation was the lack of specificity in personality profiles. In part, weak specificity reflects high comorbidity among these disorders. It also suggests, however, that higher order personality constructs are not uniquely linked to specific conditions, but rather are meaningfully related to broad classes of psychopathology (e.g., internalizing or externalizing). Nevertheless, some specific effects were observed, especially for disinhibition and agreeableness as well as for SUD and specific phobia. With regard to potential etiologic connections, the following find-

ings are especially notable. Disinhibition and agreeableness were primarily relevant to SUD. Extraversion had its strongest associations with social phobia and dysthymic disorder. Neuroticism was at the core of all disorders but was much less prominent in SUD and specific phobia. These leads are worth pursuing in future longitudinal studies. Even if the links turn out to be noncausal in nature, these traits may still prove to be valuable for identifying individuals at risk for mental disorders.

Overall, our results indicate that there is a great deal of interaction between the domains of personality and psychopathology. This interface needs to be thoroughly mapped out, and our study is a significant step in that direction. We hope that our review stimulates a new generation of research that explicates the nature of the important links between major personality traits and psychopathology.

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