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Personality and Depression: Explanatory Models and Review of the Evidence

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Abstract

Understanding the association between personality and depression has implications for elucidating etiology and comorbidity, identifying at-risk individuals, and tailoring treatment. We discuss seven major models that have been proposed to explain the relation between personality and depression, and we review key methodological issues, including study design, the heterogeneity of mood disorders, and the assessment of personality. We then selectively review the extensive empirical literature on the role of personality traits in depression in adults and children. Current evidence suggests that depression is linked to traits such as neuroticism/negative emotionality, extraversion/positive emotionality, and conscientiousness. Moreover, personality characteristics appear to contribute to the onset and course of depression through a variety of pathways. Implications for prevention and prediction of treatment response are discussed, as well as specific considerations to guide future research on the relation between personality and depression.

Keywords

traits; temperament; mood disorders; neuroticism; extraversion

INTRODUCTION

The hypothesis that depression is linked to personality can be traced to antiquity, when Hippocrates, and later Galen, argued that particular “humors” were responsible for specific personality types and forms of psychopathology. In this article, we discuss the major conceptual models that have been proposed to explain the association between personality and depression, comment on some important methodological issues, and selectively review the empirical literature. Due to space limitations, we limit our review to nonbipolar forms of depression.

This literature has developed along several distinct lines: (*a*) early clinical psychiatrists’ descriptions of affective temperaments; (*b*) research on the structure and neurobiology of personality; (*c*) psychoanalytic and cognitive-behavioral theory and observations; and (*d*) developmental psychologists’ work on temperament. In recent years, there has been substantial convergence between these lines of work, and it is increasingly possible to view them within a single integrative framework. Understanding the associations between

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personality and depression has a number of potentially important implications for research and practice. First, personality traits associated with emotional experience, expression, and regulation may be intermediate phenotypes that provide more tractable targets for genetic and neurobiological research than depressive diagnoses (Canli 2008). Second, personality may be useful in identifying more homogeneous subgroups of depressive disorders that differ in developmental trajectories and etiological influences (e.g., Beck 1983). Third, tracing the pathways between personality and depressive disorders can help elucidate more proximal processes involved in the development of mood disorders (Compas et al. 2004, Klein et al. 2008a, Lahey 2009). Fourth, personality may be useful in tailoring treatment (Zinbarg et al. 2008) and predicting treatment response (Quilty et al. 2008a). Fifth, temperament/personality may provide a means to identify at-risk individuals who could benefit from prevention and early intervention efforts (Kovacs & Lopez-Duran 2010). Finally, there is substantial comorbidity between depressive disorders and other forms of psychopathology. Some personality traits, such as neuroticism, are associated with multiple psychiatric conditions. Thus, personality could help explain patterns of comorbidity and point toward more etiologically relevant classification systems (Brown & Barlow 2009, Kotov et al. 2007, Watson 2009).

THE CONSTRUCT OF PERSONALITY

Before addressing the relation between personality and depression, several conceptual issues regarding the construct of personality should be considered. First, personality has traditionally been conceptualized as having two components: temperament, which refers to biologically based, early-emerging, stable individual differences in emotion and its regulation, and character, which refers to individual differences due to socialization. However, the distinctions between these constructs are questionable, as a large body of evidence has accumulated indicating that personality traits have all the characteristics of temperament, including strong genetic and biological bases and substantial stability over the lifespan (Krueger & Johnson 2008, Watson et al. 2006). Hence, the terms “personality” and “temperament” are now often used interchangeably (Caspi & Shiner 2006, Clark & Watson 1999). As most research on personality in childhood has been conducted under the temperament rubric, in this review we refer to this work using the term “temperament” and reserve the term “personality” for discussing the literature on adolescents and adults. However, this is intended to reflect traditional usage rather than a conceptually meaningful distinction.

Second, a variety of personality classifications have been proposed over the past century, but in the 1980s they were integrated in a consensus taxonomy, the Five-Factor Model (FFM). The FFM recognized that personality is ordered hierarchically from a large number of specific traits to five general characteristics (Digman 1994, Goldberg 1993, Markon et al. 2005). These “Big Five” traits are neuroticism, extraversion, conscientiousness, agreeableness, and openness to experience. Importantly, the FFM can be further reduced to three dimensions of negative emotionality, positive emotionality, and disinhibition versus constraint that form the next level of the personality hierarchy (Clark & Watson 1999, Markon et al. 2005). This “Big Three” model is used in studies of temperament as well as personality, although disinhibition is often labeled as effortful control in the child literature (Caspi & Shiner 2006, Rothbart & Bates 2006). The Big Five and Big Three schemes are closely related, with neuroticism being essentially identical to negative emotionality and extraversion corresponding to positive emotionality (Clark & Watson 1999, Markon et al. 2005); we refer to these two dimensions as neuroticism/negative emotionality (N/NE) and extraversion/positive emotionality (E/PE), respectively. Disinhibition does not have an exact counterpart in the FFM but instead reflects a combination of low conscientiousness and low

agreeableness. Finally, openness to experience is outside the territory covered by the Big Three.

Third, there is increasing recognition that temperament and personality are not a fixed, static set of characteristics, but rather are dynamic constructs that develop over the lifespan and change in response to maturation and life circumstances (Fraley & Roberts 2005, Rothbart & Bates 2006). For example, although the rank-order stability of most personality traits is in the moderate range, it increases over the course of development (Roberts & DelVecchio 2000). In addition, mean levels of conscientiousness and some facets of E/PE increase, and levels of N/NE decrease, over time, particularly in young adulthood (Roberts et al. 2006). A number of processes contribute to stability and change of personality. For example, genes are a major influence on stability (Krueger & Johnson 2008, Kandler et al. 2010). In addition, people often select, create, and construe environments in ways that reinforce and maintain their initial trait dispositions (Caspi & Shiner 2006). However, life stressors and major shifts in social roles and relationships can contribute to personality change (Fraley & Roberts 2005, Kandler et al. 2010). We consider the implications of these processes for the relation between personality and depression below.

MODELS OF PERSONALITY AND DEPRESSION

Classical Models of Personality-Depression Relations

A variety of models of the relation between personality and mood disorders have been proposed (e.g., Akiskal et al. 1983, M.H. Klein et al. 1993, Krueger & Tackett 2003). These proposed relations include: (*a*) personality and depressive disorders have common causes; (*b*) personality and depressive disorders form a continuous spectrum; (*c*) personality is a precursor of depressive disorders; (*d*) personality predisposes to developing depressive disorders; (*e*) personality has pathoplastic effects on depression; (*f*) personality features are state-dependent concomitants of depressive episodes; and (*g*) personality features are consequences (or scars) of depressive episodes. The distinctions between some of these accounts are subtle (cf. Kendler & Neale 2010), and other models, as well as combinations of these scenarios, are plausible. However, these seven models provide a useful conceptual framework for approaching the issue.

These models can be divided into three groups. The first three models (common cause, continuum/spectrum, and precursor) view personality and depression as having similar causal influences but do not see one as having a causal influence on the other. The fourth and fifth models (predisposition and pathoplasticity) hold that personality has causal effects on the onset or maintenance of depression. Finally, the sixth and seventh models (concomitants and consequences) view depression as having a causal influence on personality. These models, and their unique predictions, are summarized in Table 1.

The common cause model views personality and depressive disorders as distinct entities that arise from the same, or at least an overlapping, set of etiological processes. From this perspective, personality and depression are not directly related; rather, the association is due to a shared third variable. The common cause model would be supported by evidence that personality traits and depression have shared etiological influences.

The continuum/spectrum model emphasizes the conceptual overlap between depressive disorders and certain personality traits and argues for a fundamental continuity between them. A depressive diagnosis is thought to simply identify individuals who have the most extreme scores on a relevant trait. Like the common cause model, the continuum/spectrum model assumes that personality and depression arise from a similar, if not identical, set of causal factors. However, the continuum/spectrum model goes further in positing that the

association between the trait and disorder should be fairly specific because they are on the same continuum.[1] Moreover, this association is expected to be nonlinear, so that almost nobody below the definitional threshold on the trait has the diagnosis but nearly everyone above the threshold meets the criteria. Thus, the continuum/spectrum model would be supported by evidence that the trait and depression are associated with the same etiological influences and that the trait-disorder relationship is fairly specific and nonlinear.

The precursor model views personality as an early manifestation or "forme fruste" of depressive disorder. Like the common cause and continuum/spectrum accounts, the precursor model posits that personality and depressive disorders are caused by similar etiologic factors. Also like the continuum/spectrum account, it implies considerable phenomenologic similarity between the relevant trait and depression. However, the precursor model differs from both of these other models in that it assumes a particular developmental sequence, with the personality traits being evident prior to the onset of depressive disorder. In other words, both the common cause and continuum/spectrum models assume a fixed clinical expression as traits or disorder, whereas the precursor model implies escalation from traits to disorder within individuals over time. Support for the precursor model would come from evidence that the trait and depression are associated with the same etiological influences and that individuals with high levels of the trait are at increased risk for developing the disorder over time.[2]

The common cause, continuum/spectrum, and precursor models do not posit causal relations between personality and depression. In contrast, the predisposition model holds that personality plays a causal role in the onset of depression. However, the predisposition model overlaps with the precursor model in that both propose that the relevant traits are evident prior to the onset of depressive disorder. The major difference between these two accounts is that the precursor model assumes that personality and depression derive from the same set of etiological processes, but the predisposition model posits that the processes that underlie personality differ from those that lead to depression. Thus, the predisposition account implies a complex interplay among risk factors involving moderation and/or mediation, and this is what distinguishes it from the precursor model.[3] The most common example—the diathesis-stress model—conceptualizes personality as the diathesis and stress as a moderator that precipitates the onset of depressive disorder. Alternatively, stress may be a mediator, so that personality vulnerability leads to negative experiences (e.g., interpersonal rejection, job loss), which in turn increase the probability of a depressive episode. A second difference between these models is that the predisposition model does not assume any phenomenological links between personality traits and depressive symptoms. Consequently, the predisposing trait may not have any phenotypic similarity to depression. Thus, the two most critical sources of support for the predisposition model would involve demonstrating that individuals with the trait are at increased risk for subsequently developing depression, and that other variables play a role in mediating or moderating this transition.

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[1]There may not be complete specificity owing to diagnostic heterogeneity. As discussed below, depression is a heterogeneous disorder with multiple etiological pathways (equifinality). A personality trait may be part of only one of the pathways. In contrast, multifinality, in which the trait is associated with multiple disorders, is less consistent with the continuum/spectrum view.

[2]Application of the continuum/spectrum and precursor models to depressive disorders is not straightforward. Personality traits are relatively stable, whereas depression is often episodic. Existing formulations of the continuum/spectrum model have not explained how stable trait characteristics manifest as an episodic illness. Similarly, the precursor model does not account for why a stable trait would subsequently develop into a nonstable depressive state. Thus, the continuum/spectrum and precursor models may provide a better explanation for chronic than episodic forms of depression.

[3]Although moderating and mediating variables play an explicit and central role in the predisposition model, it should be acknowledged that they are not incompatible with the precursor account. That is, the escalation from personality traits to depressive disorders in the precursor model implies that additional variables (e.g., maturational or environmental factors) must be involved to precipitate the change.

The pathoplasticity model is similar to the predisposition model in that it also views personality as having a causal influence on depressive disorder. However, rather than contributing to the onset of depression, the pathoplasticity model posits that personality influences the expression of the disorder after onset. This influence can include the severity or pattern of symptomatology, course, and response to treatment. The pathoplasticity model would be supported by evidence that personality explains variation among depressed individuals in their clinical presentation or outcome.

The final two models also assume that there is a causal relation between personality and depression. However, these models reverse the direction of causality. In the concomitants (or state-dependent) model, assessments of personality are colored, or distorted, by the individual's mood state. This model implies that personality returns to its baseline form after recovery from the episode. In contrast, the consequences (or scar) model holds that depressive episodes have an enduring effect on personality, such that changes in personality persist after recovery. These models would be supported by evidence that depression alters levels of personality traits, either concurrently (concomitants model) or over the longer-term (consequences model).

Dynamic Models of Personality-Depression Relations

The models above consider traits to be perfectly stable. As noted earlier, there is now extensive evidence indicating that personality shows plasticity in childhood, with long-term test-retest correlations of $r \approx 0.35$, and continues to change across the lifespan, although personality consistency gradually increases up to $r \approx 0.75$ after the age of 50 (Roberts & DelVecchio 2000). Models of personality-psychopathology relations can be expanded to recognize the malleability of traits (e.g., Ormel et al. 2001). For example, one can posit a dynamic precursor model[4] in which early temperament defines the baseline level of risk but subsequent experiences modify personality liability to depression. This model explains variability in disorder onset as a function of the initial level of risk and steepness of the trait trajectory over time. Given the evidence on patterns of personality continuity and change (Roberts & DelVecchio 2000), it appears likely that trait vulnerability is more malleable early in life, but significant life events can alter its trajectory even in old age. A depressive disorder is thought to emerge when personality liability crosses the threshold. Thus, individuals who are born with an elevated personality liability or those with a rapidly increasing trait trajectory would have a childhood onset of the disorder, whereas those with a more slowly increasing trait trajectory would not cross the threshold until much later, if ever. Moreover, a pathological trait trajectory may be checked or reversed by positive experiences (Ormel & de Jong 1999). In fact, personality generally tends to change in a more adaptive direction with age (Roberts et al. 2006), although this pattern is not universal (Johnson et al. 2007). This may help to explain why the probability of first-episode depression peaks in adolescence, as trait deviance is more common at that age.

Similarly, the predisposition model can be expanded to recognize personality change. This dynamic predisposition model (Ormel & de Jong, 1999, Ormel et al. 2001) acknowledges transactions between personality and the environment and integrates them with the environmental moderation and mediation mechanisms of the classic predisposition model. In the environmental moderation version of this account, negative life experiences influence not only depression onset but also levels of trait vulnerability (Middledorp et al. 2008). This increase in personality liability may then lead to additional life stress. If this vicious cycle is perpetuated unchecked, personality liability would continue to increase, and at some point, a

[4]This could also be called the dynamic continuum model because once the dynamic element is introduced, it becomes virtually impossible to distinguish the continuum/spectrum and precursor models.

negative life event could overwhelm coping capabilities and elicit a depressive disorder. Importantly, and in contrast to the dynamic precursor model, in this account maladaptive traits alone are not sufficient to cause depression, and an environmental trigger is necessary.

The vicious cycle of increasing trait vulnerability and stress exposure does not necessarily indicate that personality per se influences depression onset. Indeed, certain traits may increase stress exposure but have no effect on depression otherwise (e.g., it is possible that low conscientiousness does not cause depression directly but leads to depressogenic experiences, such as academic difficulties, job loss, and relationship problems; Roberts et al. 2007), consistent with the environmental-mediation pathway.

Dynamic models offer richer and more complete accounts of the role of personality in the onset of depression. Moreover, it is important to recognize that depressive disorders have been linked to multiple traits (as reviewed below), and it is likely that different personality characteristics contribute through different pathways.

METHODOLOGICAL ISSUES

A number of methodological issues must be considered in evaluating the relation between personality and mood disorders, including (*a*) study design, (*b*) heterogeneity of depressive disorders, and (*c*) assessment of personality.

Study Design

A number of research designs can be useful in studying the relation between personality and depressive disorders. The common cause, continuum/spectrum, precursor, and predisposition models would all be supported by family studies demonstrating personality differences between nonaffected relatives of probands with and without a history of depression. The common cause, continuum/spectrum, and precursor models would be supported by twin and genetic association studies demonstrating that the same genes predispose to both personality and depressive disorders. The precursor and predisposition models posit that personality abnormalities are trait markers and hence should be present prior to the onset, and after recovery from, depressive episodes. Hence, these models can be tested by comparing individuals with a history of depression that is currently in remission to persons with no history of depression on relevant personality traits. An even stronger approach to testing the precursor and predisposition models is to use prospective longitudinal studies of persons with no prior history of mood disorder to determine whether particular personality traits predict the later onset of depressive disorder. Although no single design can distinguish among these four models, the combination of designs can bolster the case for particular accounts. For example, finding substantial common genetic variance in twin studies, but no evidence of developmental sequencing in longitudinal studies, would support the common cause and continuum/spectrum models. In turn, these two models could be compared by examining the specificity of the association between trait and disorder and whether there is a nonlinear relation between trait level and probability of disorder. On the other hand, if there were evidence of developmental sequencing in longitudinal studies as well as substantial common genetic variance in twin studies (or overlap of other etiological factors in other designs), it would support the precursor model (particularly if the trait was also phenomenologically similar to depression). In contrast, developmental sequencing but less shared genetic (or other etiological) variance would support the predisposition model. Also crucial for the predisposition model is evidence from longitudinal studies demonstrating that other variables (e.g., life stress) moderate or mediate the association between personality and subsequent depression.

The pathoplasticity model can be evaluated in longitudinal studies of persons with depressive disorders by examining the associations among personality traits and clinical features, course, and treatment response. Specifically, the pathoplasticity model posits that the trait would predict these outcomes even after controlling for initial illness severity and other prognostic factors. Of note, an alternative explanation of such results is that the personality trait is a marker for a more severe, chronic, or etiologically distinct subgroup, rather than having a causal influence on the expression of the disorder. A multiwave follow-up of individuals with a depressive disorder could be helpful in ruling out this possibility. If the trait influences the disorder course directly, rather than because it is an indicator of a latent disorder class, changes in personality scores should predict subsequent changes in outcomes.

The concomitants model can be tested through cross-sectional studies comparing persons who are currently depressed, persons who have recovered from depressive episodes, and healthy controls. An even better approach is to conduct longitudinal studies assessing individuals when they are in a depressive episode and again after they have recovered. If personality measures are abnormal during depressive episodes but not after recovery, it would suggest that they are concomitants of the depressed state. Multilevel analyses can also be used to separate personality variance into trait and state components and to test whether state variance is associated with concurrent measures of depression (e.g., Duncan-Jones et al. 1990).

The consequences (or scar) hypothesis can be evaluated by assessing persons before and after a first depressive episode. If personality deviance is much greater after the episode has remitted, it would suggest that scarring has occurred.

Testing dynamic theories requires longitudinal data with at least three assessment points. These assessments should measure relevant contextual factors (e.g., life stress) in addition to depression and personality to allow the examination of dynamic and transactional effects. Multilevel modeling and structural equation modeling offer powerful approaches to evaluating such effects with longitudinal data.

Heterogeneity of Depressive Disorders

The depressive disorders are almost certainly etiologically heterogeneous, reflecting the convergence of multiple developmental pathways. Hence, it is likely that the role of personality factors and, as suggested above, the applicability of different models of the relation between personality and depression differ for different forms of depression. The current classification system for depressive disorders is based on clinical features and is a poor approximation of etiological distinctions. Nonetheless, it is important to consider whether the role of personality varies as a function of the specific depressive diagnosis (e.g., major depressive disorder, dysthymic disorder), subtype (e.g., psychotic, melancholic, atypical), and clinical characteristics such as age of onset, recurrence, and chronicity. Failure to take heterogeneity into account may obscure important personality-depression associations. Conversely, personality may provide a basis for identifying more homogeneous subgroups within the depressive disorders. Unfortunately, few studies of personality and depressive disorders have attempted to take this heterogeneity into account.

It is important to note, however, that associations between personality and specific subtypes and clinical characteristics do not necessarily indicate etiological heterogeneity. Instead, they could reflect pathoplasticity, in which personality influences symptom presentation and/or course, but the primary etiological process is the same, or they could reflect differential severity of subtypes that results in quantitative differences in their trait profiles.

Finally, a significant source of heterogeneity in depression is comorbidity with other forms of psychopathology. Given the high rates of comorbidity, particularly with the anxiety disorders, associations between personality and depressive disorders may actually reflect the relation of personality with a co-occurring nonmood disorder. Indeed, personality may be a third variable that explains broad patterns of comorbidity among many disorders. For example, recent hierarchical models of classification posit that trait dispositions such as N/NE account for much of the comorbidity between depression and other disorders (Griffith et al. 2010, Kotov et al. 2007). Thus, it is important for researchers to consider whether traits have specific relations with depression over and above more general associations with the broader group of internalizing disorders.

Assessment

Temperament/personality can be assessed using a variety of methods, including self-report inventories, semistructured interviews, informants' reports, and observations in naturalistic settings and the laboratory. Unfortunately, most of the literature examining the association between personality and depressive disorders has assessed personality via self-report. This is potentially problematic because self-reports of personality can be complicated by current mood state, limited insight, response styles, and the difficulty of distinguishing traits from the effects of stable environmental contexts (Chmielewski & Watson 2009). In addition, when the same individual provides information on both personality and depression, as has been the case in almost all studies in this area, common method variance can inflate associations. Hence, there is a need for greater use of informant report and observational measures.

A second issue concerns the overlap between some personality constructs and psychopathology (Lahey 2004). For example, many items on N/NE scales are similar to depressive symptoms (Ormel et al. 2004b). This can inflate associations between measures of personality and depression. On the other hand, personality and symptom assessments usually have different time frames, with trait scales reflecting long-standing patterns and depression measures tapping more recent experiences (e.g., past week, past month). This trait versus state distinction parallels that between personality and other related constructs. For example, measures of N/NE and negative affect have nearly identical content but are distinguished by their time frames (Watson 2000). Thus, the degree to which this content overlap threatens the validity of personality-psychopathology research depends, at least in part, on the duration/chronicity of the disorders of interest. The extent to which this is a concern also depends on one's model of personality-depression relations. From the continuum/spectrum perspective, personality and depression are variants of the same phenomenon, so the two constructs should overlap. In contrast, the predisposition model views personality and depression as distinct domains, so from this perspective it is important to define and assess these sets of constructs as independently as possible and to judiciously delineate their time frames.

AFFECTIVE TEMPERAMENTS

The classical European descriptive psychopathologists in the late-nineteenth and early-twentieth centuries observed that many patients with mood disorders, as well as their relatives, exhibited particular patterns of premorbid personalities that appeared to be attenuated versions of their illnesses. For example, Kraepelin (1921) described four patterns of personality that he considered the "fundamental states" underlying manic-depressive illness: depressive, manic, irritable, and cyclothymic temperament. He believed that these were precursors or "rudimentary forms" of the major mood disorders. Schneider (1958) described similar types; however, he viewed them as personality disorders that were not necessarily related to the mood disorders. Two variants of these types, cyclothymic disorder

and dysthymic disorder, are included as mood disorder diagnoses in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; Am. Psychiatr. Assoc. 1994). However, these disorders are defined as fairly severe conditions, with the criteria emphasizing symptomatology rather than personality traits. As a result, these categories appear to be limited to the more severe, symptomatic manifestations of the affective temperaments described by Kraepelin and Schneider (Akiskal 1989).

On the basis of Kraepelin's and Schneider's descriptions, Akiskal (1989) proposed formal criteria for the affective temperament types, and he and his colleagues developed interview and self-report measures of these constructs that have been applied in a number of settings and cultures (e.g., Akiskal et al. 2005). Akiskal's work also provided the basis for including depressive temperament as a personality disorder in the DSM-IV appendix. Of the four affective temperament types, depressive temperament has been the most systematically studied in relation to nonbipolar depressive disorders. The terms "depressive temperament," "depressive personality," and "depressive personality disorder" have been used interchangeably in the literature to refer to the following constellation of traits: introversion, passivity, and nonassertiveness; gloominess, cheerlessness, and joylessness; self-reproach and self-criticism; pessimism, guilt, and remorse; being critical and judgmental of others; conscientiousness and self-discipline; brooding and given to worry; and feelings of inadequacy and low self-esteem.

Data on the nature of the relation between depressive personality and depressive disorders are consistent with most of the causal models described above, illustrating the complexity of the associations between personality and depression. The strongest support for the common cause and continuum/spectrum models derives from twin and family studies. In a large twin study, Ørstavik et al. (2007) found that depressive personality and major depressive disorder (MDD) shared substantial genetic variance, although there was evidence for unique genetic factors as well. Family studies indicate that individuals with depressive personality have an increased rate of mood disorders in their first-degree relatives (e.g., Klein & Miller 1993). In addition, patients with MDD, particularly those with chronic forms of depression, have elevated levels of depressive personality traits in their first-degree relatives (Klein 1999).

Consistent with Kraepelin's (1921) retrospective observations, prospective longitudinal data indicate that depressive personality traits precede the onset of depressive disorders. Kwon et al. (2000) found that young women with depressive personality and no comorbid Axis I and II disorders had a significantly increased risk of developing dysthymic disorder (but not MDD) over the course of a three-year follow-up. Taken together with the twin and family studies, these findings provide compelling support for the precursor model. In addition, in light of the conceptual issue regarding traits and states raised above for the continuum/spectrum and precursor models, it is noteworthy that depressive personality is most closely associated with chronic forms of depression at both the family and individual levels.

Evidence also supports the predisposition, pathoplasticity, and consequences models. Rudolph & Klein (2009) reported that youth with elevated levels of depressive personality traits experienced a significant increase in depressive symptoms 12 months later. While consistent with the precursor model, it is noteworthy that this association was moderated by pubertal status and timing. Thus, youth with elevated levels of depressive personality traits and more advanced pubertal status and earlier pubertal timing experienced the greatest increase in depressive symptoms. This supports the predisposition model, suggesting that depressive personality traits confer vulnerability to depression in the presence of other maturational and psychosocial processes.

Depressive personality also appears to have a pathoplastic effect on the course of depressive disorders, predicting poorer outcomes and response to treatment (Laptook et al. 2006, Ryder et al. 2010). Moreover, Rudolph & Klein (2009) recently reported preliminary support for the consequences model, at least in youth. They found that in a sample of early adolescents, higher levels of depressive symptoms predicted an increase in depressive personality traits 12 months later. Finally, the limited evidence available suggests that semi-structured interview assessments of depressive personality traits are not influenced by a depressive episode (Klein 1990), arguing against the concomitants model.

Although the work on affective temperaments is important in understanding the development of depressive disorders, it is unlikely that these types actually reflect basic temperamental processes that originate in early childhood, as their defining features include a number of developmentally complex cognitive and interpersonal characteristics. Instead, these temperament types are more likely to be intermediate outcomes that reflect the interaction of more basic temperament traits that are elaborated over development in conjunction with early socialization and other environment influences.

In recent years, considerable evidence has accumulated indicating that depressive personality is associated with several of the basic personality trait dimensions discussed below, particularly high N/NE and low E/PE and a number of their facets (e.g., Huprich 2003, Vachon et al. 2009).

PERSONALITY TRAIT DIMENSIONS

The affective temperaments are conceptualized within a categorical framework. In contrast, most of the other work on personality and depression views personality in dimensional terms. In this section, we focus on the FFM, but we also briefly consider Gray's (1994) and Cloninger and colleagues' (1993) psychobiological models and several additional traits from the clinical literature (e.g., self-criticism, dependency, and rumination). This section focuses primarily on studies of adults and adolescents using self-report measures of personality. Studies of younger children using observational measures of temperament are reviewed in a later section.

The Five-Factor Model

Cross-sectional associations—In their influential theory of personality and depression, Clark & Watson (1999, Clark et al. 1994) posited that depressive disorders are characterized by high levels of N/NE and low levels of E/PE. A large number of cross-sectional studies have evaluated these relations as well as the links between depression and the other FFM dimensions. Kotov et al. (2010) recently conducted a meta-analysis of this literature, which revealed that MDD is associated with very high N/NE (Cohen's d =1.33) and low conscientiousness (d =−0.90). The link to low E/PE was more modest (d =−0.62) and inconsistent, with some studies finding positive effects. The associations with the other two traits were weak and unremarkable. The N/NE finding is consistent with expectations, but the effect for E/PE was smaller and that for conscientiousness was larger than anticipated. Dysthymic disorder exhibited a more extreme profile with remarkably strong and consistent links to E/PE (d =−1.47), N/NE (d =1.93), and conscientiousness (d =−1.24). This is not surprising as dysthymic disorder is thought to be more trait-like than MDD, and a greater contribution from personality might be expected.

To determine whether the observed personality links are specific to depression, Kotov et al. (2010) also examined personality profiles of anxiety disorders. They found that with the exception of specific phobia, which had relatively weak associations with all five traits, all anxiety disorders showed stronger effects on N/NE, E/PE, and conscientiousness (average d

=1.91, −1.05, −1.02, respectively) than did MDD. Several also scored above dysthymic disorder on N/NE. Dysthymia had stronger associations than anxiety disorders on the other two traits, but the differences were slight.

It is conceivable that more specific associations were not evident because these analyses focused on broad personality dimensions. Narrow traits that comprise the general dimensions may have stronger associations with depressive disorders. Indeed, self-harm—a component of N/NE that reflects propensity to self-deprecation and self-injury—was found to contribute to depression even after controlling for the broad traits, and this effect was specific relative to other common mental disorders (Watson et al. 2006). With regard to E/PE, evidence is emerging that the positive affectivity facet, but not the sociability/extraversion facet, is related to depression (Durbin et al. 2005, Naragon-Gainey et al. 2009). This may explain the surprisingly modest association between MDD and E/PE, if this general trait includes much variance not relevant to depression. Thus, facet-level research promises to yield stronger and more specific evidence of personality-depression links.

Evidence bearing on causal models—Because most attempts to tease apart explanatory models of the association between depression and personality have focused on N/NE and E/PE, we consider only these two traits in this section. The section is organized by the type of research design used to address the models.

Personality during and after a depressive episode: Studies of personality and psychopathology may be complicated by the influence of participants' mood states on reports of their personalities (the concomitants model). For example, many studies have found that individuals with MDD report higher levels of N/NE when they are depressed than when they are not depressed (Hirschfeld et al. 1983b, Kendler et al. 1993, Ormel et al. 2004a). In contrast, the evidence for mood state effects on E/PE is weaker and less consistent (de Fruyt et al. 2006, Kendler et al. 1993, Morey et al. 2010). However, the influence of mood state on personality should not be overstated. Even though levels of N/NE decline significantly after remission from a depressive episode (i.e., absolute stability), individuals' relative positions with respect to levels of N/NE (i.e., rank-order stability) tend to be moderately well preserved (de Fruyt et al. 2006, Morey et al. 2010). Moreover, clinical trials suggest that changes in depressive symptoms are not necessarily accompanied by changes in personality (Quilty et al. 2008b, Tang et al. 2009).

Cross-sectional comparisons of remitted patients and controls: A number of early studies used remission designs, comparing patients who had recovered from a depressive episode to never-depressed controls or population norms on self-rated personality traits. These studies found that E/PE is significantly lower in formerly depressed patients than in healthy controls (Hirschfeld et al. 1983a, Reich et al. 1987), arguing against the concomitants model and in favor of the precursor, predisposition, and/or consequences models. However, the results for N/NE were less consistent (Hirschfeld et al. 1983a, Reich et al. 1987). This inconsistency may be due to a number of factors, including insufficiently stringent criteria for recovery, thereby possibly confounding personality and residual symptoms; using normative data collected by other investigators, which may introduce demographic and sociocultural differences between the formerly depressed and comparison samples; and selection effects, as N/NE is associated with a poorer course (discussed below) and thus samples of remitted depressives may include a disproportionate number with low levels of this trait.

Personality before and after a depressive episode: Several studies have tested the consequences (or scar) hypothesis by comparing personality measures in depressed individuals before and after a MDD episode. The results of these studies have been

inconsistent. Kendler and colleagues reported increases in N/NE (but not E/PE) after a depressive episode in two separate samples (Fanous et al. 2007, Kendler et al. 1993); however, other studies have found that N/NE and E/PE do not change from before to after a MDD episode (e.g., Ormel et al. 2004a, Shea et al. 1996). Importantly, the studies reporting scarring used less stringent criteria for recovery and shorter follow-ups, suggesting that the findings may be due to residual symptoms and/or that the scars dissipate over time.

Personality in relatives of depressed individuals: A number of studies have tested the common cause, continuum/spectrum, precursor, and predisposition models by comparing personality traits in the never-depressed relatives of patients with mood disorders and never-depressed controls (e.g., Farmer et al. 2002, Hecht et al. 1998, Ouimette et al. 1996). The results have been mixed, with some studies reporting higher N/NE and/or lower E/PE in the never-depressed relatives of probands with mood disorders, and other studies reporting no differences. However, interpretation of these studies is complicated by two factors. First, personality traits may not play the same role in risk for depression among familial as nonfamilial forms of depression. Second, there may be selection biases in samples using well relatives who are already partly through the risk period for mood disorder. Thus, those relatives with the strongest personality vulnerabilities may have already developed the disorder and be excluded from the study.

Twin studies: As discussed above, a valuable approach to testing the common cause, continuum/spectrum, and precursor models is through twin studies. These studies indicate that there are substantial associations between the liabilities for N/NE and MDD, but only weak associations between the genetic liabilities for E/PE and MDD (Fanous et al. 2007; Kendler et al. 1993, 2006).

Prospective longitudinal studies: The most direct approach to testing the precursor and predisposition models is to conduct prospective studies of personality in never-depressed participants to determine whether personality characteristics predict the subsequent onset of depressive disorders. Several studies using large community samples have reported that higher levels of N/NE predict the onset of first lifetime MDD episodes (de Graaf et al. 2002; Fanous et al. 2007; Kendler et al. 1993, 2006; Ormel et al. 2004a). In addition, several studies using measures of other traits that overlap with N/NE or its facets have reported similar findings (Hirschfeld et al. 1989, Rorsman et al. 1993). Although there is some evidence that E/PE predicts the first onset of MDD (Kendler et al. 2006, Rorsman et al. 1993), it is much weaker, and several studies have failed to find an association (Fanous et al. 2007, Hirschfeld et al. 1989, Kendler et al. 1993).

Personality and the subsequent course of depression: Finally, there is evidence that both N/NE and E/PE have pathoplastic influences on the course of depression after the onset of the disorder. For example, many studies have reported that higher N/NE and lower E/PE predict a poorer course and response to treatment, although the findings regarding E/PE are slightly less consistent (de Fruyt et al. 2006, Duggan et al. 1990, Morris et al. 2009, Quilty et al. 2008a, Tang et al. 2009). As noted above, however, these findings are also consistent with diagnostic heterogeneity, such that personality dysfunction is a marker for a more severe or etiologically distinct group. Indeed, there is evidence that the nonmelancholic subtype is characterized by more vulnerable personality styles than is melancholia and that chronic depressions are associated with higher N/NE and lower E/PE than is nonchronic MDD (Klein 2008, Kotov et al. 2010).

Evidence relevant to dynamic models—Transactions between N/NE and environmental contexts have received the most attention in the literature (Ormel & de Jong

1999, van Os & Jones 1999). N/NE shows reciprocal relations with a range of significant life experiences, such as initiation and break-up of a committed relationship, relationship quality, occupational attainment, and financial security (Neyer & Lehnart 2007, Roberts et al. 2003, Scollon & Diener 2006). Furthermore, N/NE has been repeatedly implicated in the generation of stressful life events (Kercher et al. 2009, Lahey 2009, Middeldorp et al. 2008), which suggests an environmentally mediated relationship between this trait and depression. The environmentally moderated mechanism has also received support, as several studies found that N/NE interacts with stressful life events to predict first onset of major depression (Kendler et al. 2004, Ormel et al. 2001, van Os & Jones 1999).

E/PE has demonstrated bidirectional effects with many significant social and occupational experiences (Neyer & Lehnart 2007, Roberts et al. 2003, Scollon & Diener 2006). In addition, a decrease in E/PE over time was found to predict future internalizing problems (Van den Akker et al. 2010). However, little attention has been given to mechanisms underlying the association between this trait and depression. Support for an environmentally mediated effect is limited and mixed (Middeldorp et al. 2008, Wetter & Hankin 2009), and the environmental moderation model is largely untested, although there is some evidence that positive affect moderates the effects of daily stressors on depressive symptoms (Wichers et al. 2007).

Finally, conscientiousness may play an important role in dynamic models of personality-depression relations. It has reciprocal associations with family support, divorce, occupational attainment, and job involvement (Roberts et al. 2003, Roberts & Bogg 2004). Conscientiousness is hypothesized to influence depression by increasing exposure to negative life events (Anderson & McLean 1997, Compas et al. 2004), but mediation and moderation effects have not been tested.

Interactions between temperament dimensions—Finally, personality-depression relations may be multivariate, rather than bivariate, with multiple traits interacting to influence depression. Indeed, in their influential model of personality and depression, Clark & Watson (1999, Clark et al. 1994) hypothesized that depression is characterized by high N/NE and low E/PE, raising the possibility that it is the combination of the two traits that is particularly important in depressive disorders. A growing number of studies have reported that the interaction of high N/NE and low E/PE predicts subsequent depressive symptoms or disorders in adults and youth (Gershuny & Sher 1998, Joiner & Lonigan 2000, Wetter & Hankin 2009), although several studies have not found such an interaction (Jorm et al. 2000, Kendler et al. 2006, Verstraeten et al. 2009). The interaction between N/NE and conscientiousness is also of interest, as the latter construct includes aspects of self-regulation and effortful control (Rothbart & Bates 2006) and may therefore reflect the ability to modulate one's affective reactivity. Indeed, there is cross-sectional evidence that effortful control moderates the association between N/NE and depressive symptoms in adolescents (Verstraeten et al. 2009).

Summary and discussion—Cross-sectional studies have documented strong links of depressive disorders to N/NE, conscientiousness, and E/PE, although the latter effect is substantial in dysthymic disorder but only moderate in MDD. In fact, personality generally appears to play a greater role in dysthymia. None of these relations are specific, however, as anxiety disorders have very similar trait profiles. This observation argues against the continuum/spectrum model at least with regard to these broad dimensions. It may be possible to find traits that are specific to depression by targeting lower-order personality dimensions. Narrower traits may also explain the surprisingly modest link between E/PE and MDD, as some, but not other, facets of this general dimension are relevant to depression.

The nature of relations between these personality traits and depression is complex, and our understanding is still limited. N/NE, the most widely studied personality trait in depression, raises challenging conceptual and methodological issues due to the overlap between some of its features and depressive symptoms (Ormel et al. 2004b). Nonetheless, this cannot completely explain the association between these constructs (Tang et al. 2009). N/NE is moderately influenced by clinical state (the concomitants model), shares common etiological influences with MDD (common cause, continuum/spectrum, and precursor models), predicts the subsequent onset of MDD (precursor and predisposition models), and influences the course of depression (pathoplasticity model). In addition, N/NE appears to contribute to subsequent stress and adversity and increases the risk of depression in the face of negative life events (predisposition model). Finally, it may also be changed by experience of MDD episodes (consequences model), but the evidence for this is weaker and less consistent.

The role played by E/PE in depression is less clear. Its cross-sectional association with dysthymia is substantial, but its relation to MDD is more modest. E/PE is not influenced by clinical state or changed by the experience of depressive episodes. It appears to be abnormally low even during remission, which is consistent with the continuation of trait deviance from the pre-morbid stage (precursor or predisposition accounts). Moreover, low E/PE tends to predict a poorer course of depression. However, the degree of shared etiological influences between E/PE and MDD is low, and the evidence that E/PE predicts the onset of MDD in prospective longitudinal studies is weak. As noted above, three possible reasons for the weaker and less consistent findings regarding E/PE are (*a*)it plays a greater role in some forms of depression than others (e.g., chronic depressions); (*b*) only some facets of the broader trait (e.g., low positive affective and approach motivation) are related to depression; and (*c*) E/PE may make a greater contribution to depression by moderating N/NE than as a main effect (Olino et al. 2010).

Finally, there appears to be a strong negative association between conscientiousness and depression, at least in cross-sectional studies. This may appear surprising in light of the positive associations discussed below between depression and a number of other constructs that are thought to be related to conscientiousness, such as behavioral inhibition system sensitivity, harm avoidance, perfectionism, and temperamental behavioral inhibition. It is important to note, however, that these latter constructs are more strongly associated with high N/NE, and in some cases, low E/PE, than with conscientiousness (de Fruyt et al. 2000, Muris et al. 2009a, Smits & Boeck 2006). Hence, it is likely that their positive correlations with depression are driven by their shared variance with high N/NE. Unfortunately, few studies testing causal models of personality and depression have considered conscientiousness. However, evidence indicating that this trait may moderate the effects of N/NE on depression and that it increases the likelihood of subsequent adversity that could then, in turn, produce depression suggests that further research on the role of conscientiousness is warranted.

Psychobiological Models

Gray's model—Gray's (e.g., Gray 1994) influential theory proposes that there are two major neurobehavioral systems that underlie behavior: the behavioral activation system (BAS), which responds to signals of reward, and the behavioral inhibition system (BIS), which is sensitive to cues for punishment. Although BAS and BIS differ conceptually and empirically from E/PE and N/NE, their relations with depression are thought to be similar. Thus, it has been hypothesized that depression is associated with reduced BAS and/or heightened BIS sensitivity (Depue & Iacono 1989, Gray 1994). Although much of this work has focused on bipolar disorder (e.g., Alloy et al. 2008, Johnson et al. 2008), several recent studies have examined self-report measures of BAS and BIS sensitivity in MDD. Consistent

with Gray's model, compared with healthy controls, currently depressed patients report lower levels of BAS and higher levels of BIS, and patients with a past history of MDD report lower levels of BAS (Pinto-Meza et al. 2006). In addition, lower BAS sensitivity, but not higher BIS sensitivity, is associated with a poorer course of MDD (e.g., Kasch et al. 2002, McFarland et al. 2006), suggesting that BAS may have a pathoplastic effect on depression.

Cloninger's model—Cloninger (e.g., Cloninger et al. 1993) has proposed a model of personality that includes four temperament and three character dimensions. The temperament dimensions include novelty seeking (an appetitive/approach system), harm avoidance (an inhibition/avoidance system), reward dependency (a system that is responsive to signals of social approval and attachment), and persistence. The character dimensions are self-directedness (responsible, goal-directed), cooperativeness (helpful, empathic versus hostile and alienated), and self-transcendence (imaginative, unconventional). Harm avoidance is conceptually and empirically associated with BIS, and novelty seeking and persistence are associated with BAS. Similarly, harm avoidance is positively correlated with N/NE and negatively associated with E/PE, self-directedness is negatively correlated with N/NE, and novelty seeking and persistence are associated with E/PE (e.g., de Fruyt et al. 2000).

A number of studies have reported that patients with MDD report higher levels of harm avoidance and lower levels of self-directedness than do healthy controls (e.g., Celikel et al. 2009). Most of the traits in Cloninger's system are influenced by the respondent's mood state (e.g., Farmer et al. 2003); however, abnormal levels of harm avoidance and self-directedness are present even after remission (e.g., Smith et al. 2005). Increased harm avoidance and lower self-directedness are also characteristic of most anxiety disorders, indicating that these effects are not specific to MDD (Öngür et al. 2005).

Few studies have explicitly tested the common cause, precursor, predisposition, and consequences hypotheses for Cloninger's model. Farmer et al. (2003) found that the never-depressed siblings of patients with MDD reported significantly greater harm avoidance and less self-directedness than did the never-depressed siblings of healthy controls. In addition, Cloninger et al. (2006) reported that in a large community sample, high harm avoidance and persistence and low self-directedness predicted an increase in self-reported depressive symptoms 12 months later. A larger number of studies have addressed the pathoplasticity hypothesis, albeit with mixed results. Low harm avoidance, self-directedness, and reward dependency have predicted a poorer response to treatment in some, but not all, studies; the other dimensions have generally not been associated with course and treatment outcome (Joyce et al. 2007, Kennedy et al. 2005, Morris et al. 2009).

Clinical Traits

Independent of the traditional personality field, clinical researchers have developed a number of trait-like constructs to describe dispositions to depression. These clinical traits are similar in scope to personality facets, and their stability is comparable to that of a typical personality dimension (e.g., Kasch et al. 2001, Zuroff et al. 2004). Also, factor analytic studies have shown that most of these clinical traits can be successfully incorporated in the personality taxonomy as components of neuroticism (Watson et al. 2006). Next, we briefly discuss three of the most studied constructs: ruminative response style, self-criticism, and dependency.

Ruminative response style, a tendency to dwell on sad mood and thoughts (Nolen-Hoeksema 1991), is correlated with concurrent depressive symptoms and predicts future symptoms as well as increases in symptoms over time (Rood et al. 2009). Also, one study reported that

ruminative response style prospectively predicts onset of MDD (Nolen-Hoeksema 2000). The trait has also been linked to anxiety disorders, but the association with depression is appreciably stronger (Cox et al. 2001, Nolen-Hoeksema et al. 2008).

Blatt's (1974, 1991) theory of depression focuses on two trait vulnerabilities: self-criticism (an inclination to feelings of guilt and failure stemming from unrealistically high expectations for oneself) and dependency (a disposition to feelings of helplessness and fears of abandonment resulting from a preoccupation with relationships). These constructs are similar, although not identical, to Beck's (1983) constructs of autonomy and sociotropy. Studies indicate that the link between dependency and depressive disorders is relatively weak and nonspecific, whereas self-criticism has been established as an important and specific factor in these conditions (Zuroff et al. 2004). Both traits have been conceptualized as dynamic predispositions to depressive disorders, and there is some support for this view, including evidence of transactions with life stress as well as environmental mediation and moderation of personality effects (Zuroff et al. 2004). Self-criticism, and to a lesser extent dependency, have also been found to predict future increases in depressive symptoms. In addition, there is evidence that dependency predicts the subsequent onset of major depression in older, but not younger, individuals (Hirschfeld et al. 1989, Rohde et al. 1990). The concomitants and pathoplasty models have also received empirical support (Zuroff et al. 2004). Finally, there is some research indicating that dependency may increase as a function of depressive episodes (consequences model) in youth but not adults (Rohde et al. 1990, 1994; Shea et al. 1996).

As noted above, all of these constructs are strongly linked to N/NE (Cox et al. 2001, Kasch et al. 2001), and some (particularly rumination and self-criticism) can be considered facets of this broader trait (Watson et al. 2006). Lower-order facets can account for variance over and above that of higher-order traits (Paunonen & Ashton 2001), and several cross-sectional studies have supported the incremental validity of ruminative response style and self-criticism (Cox et al. 2004, Muris et al. 2009b) in associations with depressive symptoms. However, this issue requires more research, particularly using longitudinal designs.

CHILD TEMPERAMENT

Most of the literature on personality and depression has focused on adolescents and adults. Research that is grounded in the child temperament literature in developmental psychology has the potential to extend existing work on personality in depression by (*a*) providing the strongest test of the precursor and predisposition models; (*b*) more precisely delineating the behavioral manifestations of temperamental vulnerabilities to mood disorders in young children; (*c*) tracing the development and continuity of trait vulnerabilities across the lifespan; and (*d*) examining the neurobiological, cognitive, and interpersonal processes that may mediate the association between early temperament traits and the subsequent development of depressive disorder (Compas et al. 2004, Klein et al. 2008a, Kovacs & Lopez-Duran 2010).

The early childhood temperament dimensions that have received the greatest attention with respect to depression are N/NE, E/PE, and behavioral inhibition (BI). BI refers to wariness, fear, and low exploration in novel situations (Kagan et al. 1987). It combines aspects of N/NE (fear and anxiety), E/PE (low approach), and conscientiousness (constraint/ effortful control) that do not have a direct analog in most models of adult personality.

Cross-sectional and longitudinal studies of older children and adolescents using self-report measures have generally reported associations of low E/PE and high N/NE with depression similar to those in the adult literature (e.g., Lonigan et al. 2003).[5] Observational studies of younger children of depressed mothers also indicate that these traits may be associated with

risk for depression (Kovacs & Lopez-Duran 2010). For example, in a community sample of 100 three-year-olds, Durbin et al. (2005) reported that children of mothers with a history of mood disorder exhibited low PE in emotion-eliciting laboratory tasks. Importantly, this effect was limited to the affective (positive affect) and motivational (approach/engagement), rather than the interpersonal (sociability), components of PE. Furthermore, low PE at age 3 predicted depressotypic cognition and memory biases at age 7 (Hayden et al. 2006) and parent-reported depressive symptoms at age 10 (Dougherty et al. 2010).

Subsequently, using a larger community sample (N = 543), Olino et al. (2010) found that preschool-aged children of parents with a history of depression had higher levels of NE and BI. However, both main effects were qualified by interactions with child PE. At high and moderate (but not low) levels of child PE, higher levels of NE and BI were each associated with higher rates of parental depression. Conversely, at low (but not high and moderate) levels of child NE, low PE was associated with higher rates of parental depression. Taken together, these results suggest that children of depressed parents may exhibit diminished PE or elevated NE and BI. In this latter sample, low PE was also associated with elevated levels of cortisol shortly after awakening, an index of hypothalamic-pituitary-adrenal axis dysregulation that has been shown to predict MDD in adolescents and adults (Dougherty et al. 2009).

In both the Durbin et al. (2005) and Olino et al. (2010) studies, the child temperament–parental psychopathology associations were specific to depression. However, other work suggests that children of parents with anxiety disorders may also exhibit elevated BI. For example, Rosenbaum et al. (2000) assessed BI using laboratory measures in 2-to 6-year-old children of parents with a history of MDD and/or panic disorder and parents with no history of mood or anxiety disorders. Children of patients with both MDD and panic disorder exhibited significantly greater BI than did children of parents with no history of mood or anxiety disorder. Children of parents with panic disorder alone and children of parents with MDD alone had intermediate levels of BI that did not differ significantly from children of parents in the comorbid and no-psychopathology groups.

Finally, there is some direct evidence that personality traits assessed in childhood predict the development of depressive disorders in adults. Caspi et al. (1996) reported that children who were rated as socially reticent, inhibited, and easily upset at age 3 had elevated rates of depressive (but not anxiety or substance use) disorders at age 21. Moreover, van Os et al. (1997) found that physicians' ratings of behavioral apathy at ages 6, 7, and 11 were predictive of both adolescent mood disorder and chronic depression in middle adulthood. However, BI appears to predict the development of anxiety disorders at least as strongly as depression (Hirshfeld-Becker et al. 2008).

CLINICAL IMPLICATIONS

Prevention

Personality research has important implications for the prevention of depression. Meta-analytic evidence indicates that existing preventive interventions can reduce the incidence of depressive disorders by 25% (Cuijpers et al. 2008). However, the available strategies are a mix of universal (intervention is administered to the entire population), selective (to a well-defined at-risk group), and indicated (to those with subthreshold disorder) approaches. Universal interventions are costly, lack a personalized focus, and require very large samples to yield detectable effects, whereas indicated interventions may be better described as

[5]Few studies have examined the association of conscientiousness/effortful control with depression in youth, but analogous to the adult literature, there is cross-sectional evidence that effortful control is negatively correlated with depression (Verstraeten et al. 2009).

treatment than prevention (Muñoz et al. 2010). In contrast, selective interventions are true preventive measures that are cost effective and can be tailored to a specific mechanism of risk. However, implementation of selective strategies requires knowledge of risk factors and causal processes that lead from the vulnerability to the disorder.

The majority of established risk factors for depressive disorders are either immutable (e.g., demographic characteristics, family history) or predict onset only in the short term (e.g., stressful life events). In contrast, personality is at least somewhat malleable, especially in youth, but may forecast the onset of depression years in advance, which makes traits a potentially attractive means of identifying individuals at risk and informing selection of interventions. Different trait-disorder pathways would point to different preventive strategies; hence, further research on the nature of personality-depression relations can significantly facilitate development of preventive interventions. Another advantage of traits is that they can be assessed relatively easily and efficiently and thus are ideal for screening.

Treatment Response

Personality also can inform treatment of depressive disorders post onset. In particular, traits can predict response to treatment. Substantial evidence has accumulated that individuals with lower N/NE have better treatment outcomes across modalities (Kennedy et al. 2005, Mulder 2002, Tang et al. 2009). Other Big Five traits have been studied less and their role is not yet certain. However, a recent large investigation of a combination intervention (medication plus psychotherapy) found that low N/NE and high conscientiousness predicted who would respond to treatment, and although high E/PE did not contribute directly, it amplified the effect of high conscientiousness (Quilty et al. 2008a). As discussed above, investigations of Cloninger's traits have produced inconsistent results (Joyce et al. 2007, Kennedy et al. 2005, Mulder 2002). Few studies have examined personality facets, but preliminary evidence suggests that lower-order traits can add substantially to the prediction of treatment response (Bagby et al. 2008). Among clinical traits, self-criticism, but not dependency, was found to forecast poor treatment outcomes (Blatt et al. 1995). Furthermore, personality may be useful in matching patients to interventions. For instance, Bagby et al. (2008) reported that patients high on N/NE or low on some agreeableness facets respond better to antidepressant medication than to psychotherapy.

The processes underlying these predictive associations are not entirely clear. One hypothesis is that personality change mediates the effect of treatment on depression. Indeed, there is a fair amount of evidence that depression treatment reduces N/NE and increases E/PE (Zinbarg et al. 2008) and that this effect is not due to confounding by the depressive state (Tang et al. 2009). Quilty et al. (2008b) tested a mediation model and found direct support for this hypothesis. Other possibilities need to be ruled out, however, particularly the hypotheses that traits predict poorer response because they indicate a more severe form of depression or that they interfere with treatment compliance and the therapeutic relationship, thus reducing the efficacy of the intervention.

CONCLUSIONS AND FUTURE DIRECTIONS

The literature on the relation between personality and depression is large, but it has many gaps and inconsistent findings. Nonetheless, it is possible to draw a number of conclusions. First, there are moderate-to-large cross-sectional associations between depression and three general personality traits—N/NE, E/PE, and conscientiousness—as well as with a variety of related traits (e.g., harm avoidance, rumination, and self-criticism) and personality types (depressive personality). Second, most of the personality traits associated with depression also are related to other forms of psychopathology, particularly anxiety disorders. This may reflect the phenomenon of multifinality, in which variables early in the causal chain lead to

multiple outcomes depending on subsequent events in the causal pathway. On the other hand, many of the disorders that are currently classified as distinct conditions are closely related; hence, research on personality-psychopathology associations can provide important information for revising our nosological system. Third, reports of some traits (e.g., N/NE and harm avoidance) are influenced by clinical state, whereas other traits (e.g., E/PE) appear to be independent of mood state. However, state effects cannot fully account for the associations between personality and depression. Fourth, shared etiological factors (e.g., genes) account for a portion of the association between N/NE and depression. Fifth, depressive personality and some traits, particularly N/NE, predict the subsequent onset of depressive disorders. However, it is unclear at this point whether they are best conceptualized as precursors or predispositions, as it is difficult to tease these models apart, and there is evidence supporting both accounts. In either case, there is growing evidence that temperamental risk factors are evident at an early age, suggesting a promising approach to identifying young children at risk for depression. Sixth, there is evidence suggesting that other traits, such as low E/PE and low conscientiousness/effortful control, may moderate the relationship between N/NE and depression. Seventh, it appears unlikely that depressive episodes produce enduring changes in most personality traits. Finally, personality traits predict, and may in fact influence, the course and treatment response of depression.

To make further progress in elucidating the relation between personality and mood disorders, future studies should be guided by six broad considerations. First, most of the literature on personality and depression has focused on the broad traits of N/NE and E/PE. There is a need for further work on conscientiousness and on lower levels in the trait hierarchy (i.e., facets). It is important to determine whether a more specific level of analysis will yield more powerful effects and increase the specificity of associations between personality constructs and particular forms of psychopathology. Clinical traits, such as ruminative response style and self-criticism, need to be included in these studies and evaluated jointly with traditional personality dimensions. Finally, it is important to continue to explore interactions between traits.

Second, there is a critical need for prospective, longitudinal studies. Most existing longitudinal studies have begun in late adolescence or adulthood. However, a substantial proportion of mood disorders have already developed by mid-adolescence. Therefore, in order to further test the precursor and predisposition models, and to trace the developmental pathways between personality and depression, it is necessary to conduct longitudinal studies that start as early as possible in order to obtain a sufficient number of first-onset cases and avoid selection biases caused by excluding participants who already have a history of mood disorder at initial assessment.

Third, depression researchers have treated personality as static. However, personality changes over the course of development. Future work must begin to consider the complex personality-environment transactions that can strengthen or attenuate personality trajectories and predispositions for depressive disorder. In addition, as understanding of epigenetics increases, it will be important to explore epigenetic influences on personality change and their relation to depression.

Fourth, if personality is a precursor of, or predisposes to, the development of depressive disorders, it is critical to identify the moderating factors and mediating processes involved in these pathways. There is some evidence suggesting that moderators may include gender, early adversity, and life stress, and mediators may include interpersonal deficits, depressotypic cognitions, maladaptive coping, and behavioral and neurobiological stress reactivity (Klein et al. 2008a). There is a need for more systematic research examining these moderators and mediators in a longitudinal framework.

Fifth, self-reports have borne the brunt of most research in this area and have made important contributions. However, like all methods, they have limitations and cannot be applied in all contexts (e.g., young children). Thus, there is a need for further work using complementary methods such as informant reports and observations in naturalistic and laboratory settings.

Finally, the role of personality/temperament may differ for different forms of depressive disorder. Personality appears to play an especially important role in early-onset, chronic, and recurrent depressive conditions (e.g., Klein 2008, Kotov et al. 2010, van Os et al. 1997). Focusing on broad diagnostic categories such as MDD may obscure important associations with particular forms of depression; hence, future studies need to give greater consideration to the heterogeneity of depressive disorders.

Glossary

Temperament	generally used to describe personality in childhood; largely interchangeable with the term “personality”
Neuroticism	a tendency to cope poorly with stress and to experience feelings of sadness, anxiety, and anger
Five-Factor Model (FFM)	hierarchical personality taxonomy with five general traits at its apex, neuroticism, extraversion, conscientiousness, agreeableness, and openness to experience
Extraversion	a tendency to engage the environment and other people with vigor and enthusiasm
Conscientiousness	a tendency to approach tasks in a planful and deliberate manner and to be reliable and self-disciplined
N/NE	neuroticism/negative emotionality
E/PE	extraversion/positive emotionality
Precursor model	considers personality an early manifestation of the disorder
Predisposition model	posits that personality is distinct from psychopathology and plays a causal role in its development
Pathoplasticity model	posits that personality influences the expression of the disorder after onset
Dysthymic disorder	a condition defined by chronic but relatively mild feelings of depression lasting at least two years
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition
Major depressive disorder (MDD)	a condition characterized by episodes of depressed mood or loss of interest or pleasure lasting at least two weeks
BAS	behavioral activation system
BIS	behavioral inhibition system
BI	behavioral inhibition

LITERATURE CITED

Akiskal, HS. Validating affective personality types. In: Robins, L.; Barrett, J., editors. The Validity of Psychiatric Diagnosis. New York: Raven; 1989. p. 217-227.

Akiskal HS, Akiskal KK, Haykal RF, Manning JS, Connor PD. TEMPS-A: progress towards validation of a self-rated clinical version of the Temperament Evaluation of the Memphis, Pisa, Paris, and San Diego Autoquestionnaire. J. Affect. Disord. 2005; 85:3–16. [PubMed: 15780671]

Akiskal HS, Hirschfeld RMA, Yerevanian BI. The relationship of personality to affective disorders: a critical review. Arch. Gen. Psychiatry. 1983; 40:801–810. [PubMed: 6344834]

Alloy LB, Abramson LY, Walshaw PD, Cogswell A, Grandin LD, et al. Behavioral approach system and behavioral inhibition system sensitivities and bipolar spectrum disorders: prospective prediction of bipolar mood episodes. Bipolar Disord. 2008; 10:310–322. [PubMed: 18271911]

Am. Psychiatr. Assoc.. Diagnostic and Statistical Manual of Mental Disorders. 4th ed.. Arlington, VA: Am. Psychiatr. Assoc.; 1994.

Anderson KW, McLean PD. Conscientiousness in depression: tendencies, predictive utility, and longitudinal stability. Cogn. Ther. Res. 1997; 21:223–238.

Bagby RM, Quilty LC, Segal ZV, McBride CC, Kennedy SH, Costa PT. Personality and differential treatment response in major depression: a randomized controlled trial comparing cognitive-behavioural therapy and pharmacotherapy. Can. J. Psychiatry. 2008; 53:361–370. [PubMed: 18616856]

Beck, AT. Cognitive therapy of depression: new approaches. In: Clayton, P.; Barrett, J., editors. Treatment of Depression: Old and New Approaches. New York: Raven; 1983. p. 265-290.

Blatt SJ. Levels of object representation in anaclitic and introjective depression. Psychoanal. Study. Child. 1974; 29:107–157. [PubMed: 4445397]

Blatt SJ. A cognitive morphology of psychopathology. J. Nerv. Ment. Dis. 1991; 179:449–458. [PubMed: 1856707]

Blatt SJ, Quinlan DM, Pilkonis PA, Shea MT. Impact of perfectionism and need for approval on the brief treatment of depression: the National Institute of Mental Health Treatment of Depression Collaborative Research Program revisited. J. Consult. Clin. Psychol. 1995; 63:125–132. [PubMed: 7896977]

Brown TA, Barlow DH. A proposal for a dimensional classification system based on the shared features of the DSM-IV anxiety and mood disorders: implications for assessment and treatment. Psychol. Assess. 2009; 21:256–271. [PubMed: 19719339]

Canli T. Toward a neurogenetic theory of neuroticism. Ann. NY. Acad. Sci. 2008; 1129:153–174. [PubMed: 18591477]

Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 years predict adult psychiatric disorders. Arch. Gen. Psychiatry. 1996; 53:1033–1039. [PubMed: 8911226] Prospective birth-cohort study of relation of temperament in preschoolers and subsequent psychopathology in early adulthood.

Caspi, A.; Shiner, RL. Personality development. In: Damon, W.; Lerner, R.; Eisenberg, N., editors. Handbook of Child Psychology: Social, Emotional, and Personality Development. 6th ed.. New York: Wiley; 2006. p. 300-365.

Celikel FC, Kose S, Cumurcu BE, Erkorkmaz U, Sayar K, et al. Cloninger's temperament and character dimensions of personality in patients with major depressive disorder. Compr. Psychiatry. 2009; 50:556–561. [PubMed: 19840594]

Chmielewski M, Watson D. What is being assessed and why it matters: the impact of transient error on trait research. J. Personal. Soc. Psychol. 2009; 97:186–202.

Clark, LA.; Watson, D. Temperament: a new paradigm for trait psychology. In: Pervin, LA.; John, OP., editors. Handbook of Personality. 2nd ed.. New York: Guilford; 1999. p. 399-423.

Clark LA, Watson D, Mineka S. Temperament, personality, and the mood and anxiety disorders. J. Abnorm. Psychol. 1994; 103:103–116. [PubMed: 8040472] Reviews theories of, and data on, relations between personality and the mood and anxiety disorders.

Cloninger CR, Svrakic DM, Przybeck TR. A psychobiological model of temperament and character. Arch. Gen. Psychiatry. 1993; 50:975–990. [PubMed: 8250684]

Cloninger CR, Svrakic DM, Przybeck TR. Can personality predict future depression? A twelve-month follow-up of 631 subjects. J. Affect. Disord. 2006; 92:35–44. [PubMed: 16442638]

Compas BE, Connor-Smith J, Jaser SS. Temperament, stress reactivity, and coping: implications for depression in childhood and adolescence. J. Clin. Child. Adolesc. Psychol. 2004; 33:21–31. [PubMed: 15028538]

Cox BJ, Enns MW, Walker JR, Kjernisted K, Pidlubny SR. Psychological vulnerabilities in patients with major depression versus panic disorder. Behav. Res. Ther. 2001; 39:567–573. [PubMed: 11341253]

Cox BJ, McWilliams LA, Enns MW, Clara IP. Broad and specific personality dimensions associated with major depression in a nationally representative sample. Compr. Psychiatry. 2004; 45:246–253. [PubMed: 15224266]

Cuijpers P, van Straten A, Smit F, Mihalopoulos C, Beekman A. Preventing the onset of depressive disorders: a meta-analytic review of psychological interventions. Am. J. Psychiatry. 2008; 165:1272–1280. [PubMed: 18765483]

de Fruyt F, van de Wiele L, van Heeringen C. Cloninger's psychobiological model of temperament and character and the five-factor model of personality. Personal. Individ. Differ. 2000; 29:441–452.

de Fruyt F, van Leeuwen K, Bagby RM, Rolland JP, Rouillon F. Assessing and interpreting personality change and continuity in patients treated for major depression. Psychol. Assess. 2006; 18:71–80. [PubMed: 16594814]

de Graaf R, Bijl RV, Ravelli A, Smit F, Vollenbergh WAM. Predictors of first incidence of DSM-III-R psychiatric disorders in the general population: findings from the Netherlands Mental Health Survey and Incidence Study. Acta Psychiatr. Scand. 2002; 106:303–313. [PubMed: 12225498]

Depue RA, Iacono WG. Neurobehavioral aspects of affective disorders. Annu. Rev. Psychol. 1989; 40:457–492. [PubMed: 2648983]

Digman, JM. The Developing Structure of Temperament and Personality from Infancy to Adulthood. Hillsdale, NJ: Erlbaum; 1994. Child personality and temperament: Does the five-factor model embrace both domains?; p. 323-338.

Dougherty LR, Klein DN, Durbin CE, Hayden EP, Olino TM. Temperamental positive and negative emotionality and children's depressive symptoms: A longitudinal prospective study from age three to age ten. J. Soc. Clin. Psychol. 2010; 29:462–488.

Dougherty LR, Klein DN, Olino TM, Dyson M, Rose S. Increased waking salivary cortisol and depression risk in preschoolers: the role of maternal history of melancholic depression and early child temperament. J. Child Psychol. Psychiatry. 2009; 50:1495–1503. [PubMed: 19686335]

Duggan CF, Lee AS, Murray RM. Does personality predict long-term outcome in depression? Br. J. Psychiatry. 1990; 157:19–24. [PubMed: 2397360]

Duncan-Jones P, Fergusson DM, Ormel J, Horwood LJ. A model of stability and change in minor psychiatric symptoms: results from three longitudinal studies. Psychol. Med. Monogr. Suppl. 1990; 18:1–28. [PubMed: 2236383]

Durbin CE, Klein DN, Hayden EP, Buckley ME, Moerk KC. Temperamental emotionality in preschoolers and parental mood disorders. J. Abnorm. Psychol. 2005; 114:28–37. [PubMed: 15709809]

Fanous AH, Neale MC, Aggen SH, Kendler KS. Alongitudinal study of personality and major depression in a population-based sample of male twins. Psychol. Med. 2007; 37:1163–1172. [PubMed: 17407614]

Farmer A, Redman K, Harris T, Mahmood A, Sadler S, et al. Neuroticism, extraversion, life events and depression: the Cardiff Depression Study. Br. J. Psychiatry. 2002; 181:118–122. [PubMed: 12151281]

Farmer A, Mahmood A, Redman K, Harris T, Sadler S, McGuffin P. A sib-pair study of the temperament and character scales in major depression. Arch. Gen. Psychiatry. 2003; 60:490–496. [PubMed: 12742870]

Fraley RC, Roberts BW. Patterns of continuity: a dynamic model for conceptualizing the stability of individual differences in psychological constructs across the life course. Psychol. Rev. 2005; 112:60–74. [PubMed: 15631588]

Gershuny BS, Sher KJ. The relation between personality and anxiety: findings from a 3-year prospective study. J. Abnorm. Psychol. 1998; 107:252–262. [PubMed: 9604554]

Goldberg LR. The structure of phenotypic personality traits. Am. Psychol. 1993; 48:26–34. [PubMed: 8427480]

Gray, JA. Framework for a taxonomy of psychiatric disorder. In: van Goozen, SHM.; Van de Poll, NE., editors. Emotions: Essays on Emotion Theory. Hillsdale, NJ: Erlbaum; 1994. p. 29-59.

Griffith JW, Zinbarg RE, Craske MG, Mineka S, Rose RD, et al. Neuroticism as a common dimension in the internalizing disorders. Psychol. Med. 2010; 40:1125–1136. [PubMed: 19903363]

Hayden EP, Klein DN, Durbin CE, Olino TM. Low positive emotionality at age three predicts depressotypic cognitions in seven-year-old children. Dev. Psychopathol. 2006; 18:409–423. [PubMed: 16600061]

Hecht H, van Calker D, Berger M, von Zerssen D. Personality in patients with affective disorders and their relatives. J. Affect. Disord. 1998; 51:33–43. [PubMed: 9879801]

Hirschfeld RMA, Klerman GL, Clayton PJ, Keller MB. Personality and depression: empirical findings. Arch. Gen. Psychiatry. 1983a; 40:993–998. [PubMed: 6615162]

Hirschfeld RMA, Klerman GL, Clayton PJ, Keller MB, Mc Donald-Scott P, Larkin BH. Assessing personality: effects of the depressive state on trait measurement. Am. J. Psychiatry. 1983b; 140:695–699. [PubMed: 6846626]

Hirschfeld RMA, Klerman GL, Lavori P, Keller MB, Griffith P, Coryell W. Premorbid personality assessments of first onset of major depression. Arch. Gen. Psychiatry. 1989; 46:345–350. [PubMed: 2649038]

Hirshfeld-Becker DR, Micco J, Henin A, Bloomfield A, Biederman J, Rosenbaum J. Behavioral inhibition. Depress. Anxiety. 2008; 25:357–367. [PubMed: 18412062]

Huprich SK. Evaluating facet-level predictions and construct validity of depressive personality disorder. J. Personal. Disord. 2003; 17:219–232.

Johnson SL, Cueller AK, Ruggero C, Winett-Perlman C, Goonick P, et al. Life events as predictors of mania and depression in bipolar I disorder. J. Abnorm. Psychol. 2008; 117:268–277. [PubMed: 18489203]

Johnson W, Hicks BM, McGue M, Iacono WG. Most of the girls are alright, but some aren't: personality trajectory groups from ages 14 to 24 and some associations with outcomes. J. Personal. Soc. Psychol. 2007; 93:266–284.

Joiner TE, Lonigan CJ. Tripartite model of depression and anxiety in youth psychiatric inpatients: relations with diagnostic status and future symptoms. J. Clin. Child Psychol. 2000; 29:372–382. [PubMed: 10969421]

Jorm AF, Christensen H, Henderson AS, Jacomb PA, Korten AE, Rodgers B. Predicting anxiety and depression from personality: Is there a synergistic effect of neuroticism and extraversion? J. Abnorm. Psychol. 2000; 109:145–149. [PubMed: 10740946]

Joyce PR, McKenzie JM, Carter JD, Rae AM, Luty SE, et al. Temperament, character, and personality disorders as predictors of response to interpersonal psychotherapy and cognitive-behavioural therapy for depression. Br. J. Psychiatry. 2007; 190:503–508. [PubMed: 17541110]

Kagan J, Reznick JS, Snidman N. The physiology and psychology of behavioral inhibition in children. Child Dev. 1987; 55:1459–1473. [PubMed: 3691195]

Kandler C, Bleidorn W, Riemann R, Spinath FM, Thiel W, Angleitner A. Sources of cumulative continuity in personality: a longitudinal multiple-rater twin study. J. Personal. Soc. Psychol. 2010; 98:995–1008.

Kasch KL, Klein DN, Lara ME. A construct validation study of the Response Styles Questionnaire Rumination Scale in participants with a recent-onset major depressive episode. Psychol. Assess. 2001; 13:375–383. [PubMed: 11556274]

Kasch KL, Rottenberg J, Arnow BA, Gotlib IH. Behavioral activation and inhibition systems and the severity and course of depression. J. Abnorm. Psychol. 2002; 111:589–597. [PubMed: 12428772]

Kendler KS, Kuhn J, Prescott CA. The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. Am. J. Psychiatry. 2004; 161:631–636. [PubMed: 15056508]

Kendler KS, Neale MC. Endophenotype: a conceptual analysis. Mol. Psychiatry. 2010; 15:789–797. [PubMed: 20142819]

Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ. A longitudinal twin study of personality and major depression in women. Arch. Gen. Psychiatry. 1993; 50:853–862. [PubMed: 8215811]

Kendler KS, Gatz M, Gardner CO, Pederson NL. Personality and major depression. Arch. Gen. Psychiatry. 2006; 63:1113–1120. [PubMed: 17015813] Tests models of personality and MDD in a prospective study of a large twin sample.

Kennedy, SH.; Farvolden, P.; Cohen, NL.; Bagby, RM.; Costa, PT. The impact of personality on the pharmacological treatment of depression. In: Rosenbluth, M.; Kennedy, SH.; Bagby, RM., editors. Depression and Personality. Arlington, VA: Am. Psychiatr. Publ.; 2005. p. 97-119.

Kercher AJ, Rapee RM, Schniering CA. Neuroticism, life events and negative thoughts in the development of depression in adolescent girls. J. Abnorm. Child Psychol. 2009; 37:903–915. [PubMed: 19437113]

Klein DN. Depressive personality: reliability, validity, and relation to dysthymia. J. bnorm. Psychol. 1990; 99:412–421.

Klein DN. Depressive personality in the relatives of outpatients with dysthymic disorder and episodic major depressive disorder and normal controls. J. Affect. Disord. 1999; 55:19–27. [PubMed: 10512602]

Klein, DN. Dysthymia and chronic depression. In: Craighead, WE.; Miklowitz, DJ.; Craighead, LW., editors. Psychopathology: History, Theory, and Diagnosis. Hoboken, NJ: Wiley; 2008. p. 329-365.

Klein, DN.; Dougherty, LR.; Laptook, RS.; Olino, TM. Temperament and risk for mood disorders in adolescents. In: Allen, N.; Sheeber, L., editors. Adolescent Emotional Development and the Emergence of Depressive Disorders. Cambridge, UK: Cambridge Univ. Press; 2008a. p. 238-261.

Klein DN, Miller GA. Depressive personality in nonclinical subjects. Am. J. Psychiatry. 1993; 150:1718–1724. [PubMed: 8214182]

Klein, DN.; Torpey, DC.; Bufferd, SJ.; Dyson, MW. Depressive disorders. In: Beauchaine, TP.; Hinshaw, SP., editors. Child and Adolescent Psychopathology. Hoboken, NJ: Wiley; 2008b. p. 477-509.

Klein, MH.; Wonderlich, S.; Shea, MT. Models of relationships between personality and depression: toward a framework for theory and research. In: Klein, MH.; Kupfer, DJ.; Shea, TM., editors. Personality and Depression: A Current View. New York: Guilford; 1993. p. 1-54.

Kotov R, Gamez W, Schmidt FL, Watson D. Linking “Big” personality traits to anxiety, depressive, and substance use disorders: a meta-analysis. Psychol. Bull. 2010; 136:768–821. [PubMed: 20804236] Documents personality profiles of common mental disorders through a systematic meta-analysis.

Kotov R, Watson D, Robles J, Schmidt NB. Personality traits and anxiety symptoms: the Multilevel Trait Predictor Model. Behav. Res. Ther. 2007; 45:1485–1503. [PubMed: 17240351]

Kovacs M, Lopez-Duran N. Prodromal symptoms and atypical affectivity as predictors of major depression in juveniles: implications for prevention. J. Child. Psychol. Psychiatry. 2010; 51:472–496. [PubMed: 20202041]

Kraepelin, E. Fundamental states. In: Robertson, GM., editor. Manic-Depressive Insanity and Paranoia. Edinburgh, Scotland: Livingstone; 1921. p. 117-132.

Krueger, RF.; Johnson, W. Behavioral genetics and personality: a new look at the integration of nature and nurture. In: John, OP.; Robins, RW.; Pervin, LA., editors. Handbook of Personality: Theory and Research. 3rd ed.. New York: Guilford; 2008. p. 287-310.

Krueger RF, Tackett JL. Personality and psychopathology: working toward the bigger picture. J. Personal. Disord. 2003; 17:109–128.

Kwon JS, Young-Me K, Chang-Gok C, Byung-Joo P, Kim L. Three-year follow-up of women with the sole diagnosis of depressive personality disorder: subsequent development of dysthymia and major depression. Am. J. Psychiatry. 2000; 157:1966–1972. [PubMed: 11097962] Shows that depressive personality predicts the later first-lifetime onset of dysthymic disorder.

Lahey BB. Commentary: role of temperament in developmental models of psychopathology. J. Clin. Child. Adolesc. Psychol. 2004; 33:88–93. [PubMed: 15028544]

Lahey BB. Public health significance of neuroticism. Am. Psychol. 2009; 64:241–256. [PubMed: 19449983]

Laptook RS, Klein DN, Dougherty LR. Ten-year stability of depressive personality disorder in depressed outpatients. Am. J. Psychiatry. 2006; 163:865–871. [PubMed: 16648328]

Lonigan CJ, Phillips BM, Hooe ES. Relations of positive and negative affectivity to anxiety and depression in children: evidence from a latent-variable longitudinal study. J. Consult. Clin. Psychol. 2003; 71:465–481. [PubMed: 12795571]

Markon KE, Krueger RF, Watson D. Delineating the structure of normal and abnormal personality: an integrative hierarchical approach. J. Personal. Soc. Psychol. 2005; 88:139–157.

McFarland BR, Shankman SA, Tenke CE, Bruder GE, Klein DN. Behavioral activation system deficits predict the six-month course of depression. J. Affect. Disord. 2006; 91:229–234. [PubMed: 16487598]

Middledorp CM, Cath DC, Beem AL, Willemsen G, Boomsma DI. Life events, anxious depression and personality: a prospective and genetic study. Psychol. Med. 2008; 38:1557–1565. [PubMed: 18294422]

Morey LC, Shea MT, Markowitz JC, Stout RL, Hopwood CJ, et al. State effects of major depression on the assessment of personality and personality disorder. Am. J. Psychiatry. 2010; 167:528–535. [PubMed: 20160004]

Morris BH, Bylsma LM, Rottenberg J. Does emotion predict the course of major depressive disorder? A review of prospective studies. Br. J. Clin. Psychol. 2009; 48:255–273. [PubMed: 19187578]

Mulder RT. Personality pathology and treatment outcome in major depression: a review. Am. J. Psychiatry. 2002; 159:359–371. [PubMed: 11869996]

Muñoz RF, Cuijpers P, Smit F, Barrera AZ, Leykin Y. Prevention of major depression. Annu. Rev. Clin. Psychol. 2010; 6:181–212. [PubMed: 20192789]

Muris P, Bos AER, Mayer B, Verkade R, Thewissen V, Dell'Avvento V. Relations among behavioral inhibition, Big Five personality factors, and anxiety disorder symptoms in nonclinical children. Personal. Individ. Differ. 2009a; 46:525–529.

Muris P, Fokke M, Kwik D. The ruminative response style in adolescents: an examination of its specific link to symptoms of depression. Cogn. Ther. Res. 2009b; 33:21–32.

Naragon-Gainey K, Watson D, Markon K. Differential relations of depression and social anxiety symptoms to the facets of extraversion/positive emotionality. J. Abnorm. Psychol. 2009; 118:299–310. [PubMed: 19413405]

Neyer FJ, Lehnart J. Relationships matter in personality development: evidence from an 8-year longitudinal study across young adulthood. J. Personal. 2007; 75:535–568.

Nolen-Hoeksema S. Responses to depression and their effects on the duration of depressive episodes. J. Abnorm. Psychol. 1991; 100:569–582. [PubMed: 1757671]

Nolen-Hoeksema S. The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. J. Abnorm. Psychol. 2000; 109:504–511. [PubMed: 11016119]

Nolen-Hoeksema S, Wisco BE, Lyubomirsky S. Rethinking rumination. Perspect. Psychol. Sci. 2008; 3:400–424.

Olino TM, Klein DN, Dyson MW, Rose SA, Durbin CE. Temperamental emotionality in preschool-aged children and depressive disorders in parents: associations in a large community sample. J. Abnorm. Psychol. 2010; 119:468–478. [PubMed: 20677836] Examines relation of parental depression with observations of temperament in large sample of preschool-aged offspring.

Öngür D, Farabaugh A, Iosifescu DV, Perils R, Fava M. Tridimensional Personality Questionnaire factors in major depressive disorder: relationship to anxiety disorder comorbidity and age of onset. Psychother. Psychosom. 2005; 74:173–178. [PubMed: 15832068]

Ormel, J.; de Jong, A. On vulnerability to common mental disorders. An evidence-based plea for a developmental perspective. In: Tansella, M.; Thornicroft, G., editors. Common Mental Disorders in Primary Care: Essays in Honour of Professor Sir David Goldberg. London: Routledge; 1999. p. 34-51.

Ormel J, Oldehinkel AJ, Brilman EI. The interplay and ecological continuity of neuroticism, difficulties, and life events in the etiology of major and subsyndromal, first and recurrent depressive episodes in later life. Am. J. Psychiatry. 2001; 158:885–891. [PubMed: 11384895] Prospectively tested a dynamic predisposition model of neuroticism-depression relations in a sample of older adults.

Ormel J, Oldehinkel AJ, Vollebergh W. Vulnerability before, during, and after a major depressive episode. Arch. Gen. Psychiatry. 2004a; 61:990–996. [PubMed: 15466672] Prospectively tested competing models of personality-depression relations in a large community sample.

Ormel J, Rosmalen J, Farmer A. Neuroticism: a noninformative marker of vulnerability to psychopathology. Soc. Psychiatry Psychiatr. Epidemiol. 2004b; 39:906–912. [PubMed: 15549243]

Ørstavik RE, Kendler KS, Czajkowski N, Tambs K, Reichborn-Kjennerud T. The relationship between depressive personality disorder and major depressive disorder: a population-based twin study. Am. J. Psychiatry. 2007; 164:1866–1872. [PubMed: 18056242]

Ouimette PC, Klein DN, Pepper CM. Personality traits in the first degree relatives of outpatients with depressive disorders. J. Affect. Disord. 1996; 39:43–53. [PubMed: 8835653]

Paunonen SV, Ashton MD. Big Five factors and facets and the prediction of behavior. J. Personal. Soc. Psychol. 2001; 81:524–539.

Pinto-Meza A, Caseras X, Soler J, Puigdemont D, Pèrez V, Torrubia R. Behavioral inhibition and behavioral activation systems in current and recovered major depression participants. Personal. Individ. Differ. 2006; 40:215–226.

Quilty LC, De Fruyt F, Rolland JP, Kennedy SH, Rouillon PF, Bagby RM. Dimensional personality traits and treatment outcome in patients with major depressive disorder. J. Affect. Disord. 2008a; 108:241–250. [PubMed: 18067975]

Quilty LC, Meusel LAC, Bagby RM. Neuroticism as a mediator of treatment response to SSRIs in major depressive disorder. J. Affect. Disord. 2008b; 111:67–73. [PubMed: 18384882]

Reich J, Noyes R, Hirschfeld R, Coryell W, O'Gorman T. State and personality in depressed and panic patients. Am. J. Psychiatry. 1987; 144:181–187. [PubMed: 3812784]

Roberts BW, Bogg T. A 30-year longitudinal study of the relationships between conscientiousness-related traits, and the family structure and health-behavior factors that affect health. J. Personal. 2004; 72:325–354.

Roberts BW, Caspi A, Moffitt T. Work experiences and personality development in young adulthood. J. Personal. Soc. Psychol. 2003; 84:582–593.

Roberts BW, DelVecchio WF. The rank-order consistency of personality from childhood to old age: a quantitative review of longitudinal studies. Psychol. Bull. 2000; 126:3–25. [PubMed: 10668348]

Roberts BW, Kuncel NR, Shiner R, Caspi A, Goldberg LR. The power of personality: the comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. Perspect. Psychol. Sci. 2007; 2:313–345.

Roberts BW, Walton KE, Viechtbauer W. Patterns of mean-level change in personality traits across the life course: a meta-analysis of longitudinal studies. Psychol. Bull. 2006; 132:1–25. [PubMed: 16435954]

Rohde P, Lewinsohn PM, Seeley JR. Are people changed by the experience of having an episode of depression? A further test of the scar hypothesis. J. Abnorm. Psychol. 1990; 99:264–271.

Rohde P, Lewinsohn PM, Seeley JR. Are adolescents changed by an episode of major depression? J. Am. Acad. Child Adolesc. Psychiatry. 1994; 33:1289–1298. [PubMed: 7995795]

Rood L, Roelofs J, Bögels SM, Nolen-Hoeksema S, Schouten E. The influence of emotion-focused rumination and distraction on depressive symptoms in non-clinical youth: a meta-analytic review. Clin. Psychol. Rev. 2009; 29:607–616. [PubMed: 19682781]

Rorsman B, Grasbeck A, Hagnell O, Isberg PE, Otterbeck L. Premorbid personality traits and psychometric background factors in depression: the Lundby Study 1957–1972. Neuropsychobiology. 1993; 27:72–79. [PubMed: 8515830]

Rosenbaum JF, Biederman J, Hirshfeld-Becker DR, Kagan J, Snidman N, et al. A controlled study of behavioral inhibition in children of parents with panic disorder and depression. Am. J. Psychiatry. 2000; 157:2002–2010. [PubMed: 11097967]

Rothbart, MK.; Bates, JE. Temperament. In: Eisenberg, N.; Damon, W.; Lerner, RM., editors. Handbook of Child Psychology: Vol. 3. Social, Emotional, and Personality Development. 6th ed.. Hoboken, NJ: Wiley; 2006. p. 99-166.

Rudolph KD, Klein DN. Exploring depressive personality traits in youth: origins, correlates, and developmental consequences. Dev. Psychopathol. 2009; 21:1155–1180. [PubMed: 19825262]

Ryder AG, Quilty LC, Vachon DD, Bagby RM. Depressive personality and treatment outcome in major depressive disorder. J. Personal. Disord. 2010; 24:392–404.

Schneider, K. Psychopathic Personalities. London: Cassell; 1958.

Scollon CN, Diener E. Love, work, and changes in extraversion and neuroticism over time. J. Personal. Soc. Psychol. 2006; 91:1152–1165.

Shea MT, Leon AC, Mueller TI, Solomon DA, Warshaw MG, Keller MB. Does major depression result in lasting personality change? Am. J. Psychiatry. 1996; 153:1404–1410. [PubMed: 8890672]

Smith DJ, Duffy L, Stewart ME, Muir WJ, Blackwood DHR. High harm avoidance and low self-directedness in euthymic young adults with recurrent, early-onset depression. J. Affect. Disord. 2005; 87:83–89. [PubMed: 15967233]

Smits DJM, Boeck PD. From BIS/BAS to the Big Five. Eur. J. Personal. 2006; 20:255–270.

Tang TZ, DeRubeis RJ, Hollon SD, Amsterdam J, Shelton R, Schalet B. Personality change during depression treatment: a placebo-controlled trial. Arch. Gen. Psychiatry. 2009; 66:1322–1330. [PubMed: 19996037] Finds that antidepressants influenced personality independent of their effects on depression in a placebo-controlled trial.

Vachon DD, Sellbom M, Ryder AG, Miller JD, Bagby RM. A five-factor model description of depressive personality disorder. J. Personal. Disord. 2009; 23:447–465.

Van denAkker A, Deković M, Prinzie P. Transitioning to adolescence: how changes in child personality and overreactive parenting predict adolescent adjustment problems. Dev. Psychopathol. 2010; 22:151–163. [PubMed: 20102653]

van Os J, Jones PB. Early risk factors and adult person–environment relationships in affective disorder. Psychol. Med. 1999; 29:1055–1067. [PubMed: 10576298]

van Os J, Jones P, Lewis G, Wadsworth M, Murray R. Developmental precursors of affective illness in a general population birth cohort. Arch. Gen. Psychiatry. 1997; 54:625–631. [PubMed: 9236546]

Verstraeten K, Vasey MW, Raes F, Bijttebier P. Temperament and risk for depressive symptoms in adolescence: mediation by rumination and moderation by effortful control. J. Abnorm. Child Psychol. 2009; 37:349–361. [PubMed: 19107592]

Watson, D. Mood and Temperament. New York: Guilford; 2000.

Watson D. Differentiating the mood and anxiety disorders: a quadripartite model. Annu. Rev. Clin. Psychol. 2009; 5:221–247. [PubMed: 19327030]

Watson, D.; Kotov, R.; Gamez, W. Basic dimensions of temperament in relation to personality and psychopathology. In: Krueger, R.; Tackett, J., editors. Personality and Psychopathology. New York: Guilford; 2006. p. 7-38.

Wetter EK, Hankin BL. Mediational pathways through which positive and negative emotionality contribute to anhedonic symptoms of depression: a prospective study of adolescents. J. Abnorm. Child Psychol. 2009; 37:507–520. [PubMed: 19184402] Examines mediators and moderators of relation between personality and depressive symptoms in adolescents.

Wichers MC, Myin-Germeys I, Jacobs N, Peeters F, Kenis G, et al. Evidence that moment-to-moment variation in positive emotions buffer genetic risk for depression: a momentary assessment twin study. Acta Psychiatr. Scand. 2007; 115:451–457. [PubMed: 17498156]

Zinbarg RE, Uliaszek AA, Adler JM. The role of personality in psychotherapy for anxiety and depression. J. Personal. 2008; 76:1649–1688.

Zuroff DC, Mongrain M, Santor DA. Conceptualizing and measuring personality vulnerability to depression: comment on Coyne and Whiffen (1995). Psychol. Bull. 2004; 130:489–511. [PubMed: 15122935]

SUMMARY POINTS

1. There are moderate-to-large cross-sectional associations between depression and three general personality traits—N/NE, E/PE, and conscientiousness—as well as with a variety of related traits (e.g., harm avoidance, rumination, and self-criticism) and personality types (e.g., depressive personality).
2. Most of the personality traits associated with depression also are related to other forms of psychopathology, particularly anxiety disorders. This may reflect the phenomenon of multifinality, in which variables early in the causal chain lead to multiple outcomes depending on subsequent events in the causal pathway. On the other hand, many of the disorders that are currently classified as distinct conditions are closely related; hence, research on personality-psychopathology associations can also provide important information for revising our nosological system.
3. Reports of some traits (e.g., N/NE and harm avoidance) are influenced by clinical state, whereas other traits (e.g., E/PE) appear to be independent of mood state. However, state effects cannot fully account for the associations between personality and depression.
4. Shared etiological factors (e.g., genes) account for a portion of the association between N/NE and depression.
5. Depressive personality and some traits, particularly N/NE, predict the subsequent onset of depressive disorders. However, it is unclear at this point whether they are best conceptualized as precursors or predispositions, as it is difficult to tease these models apart, and there is evidence supporting both accounts. In either case, there is growing evidence that temperamental risk factors are evident at an early age, suggesting a promising approach to identifying young children at risk for depression.
6. There is evidence suggesting that other traits, such as low E/PE and low conscientiousness/effortful control, may moderate the relationship between N/NE and depression.
7. It appears unlikely that depressive episodes produce enduring changes in most personality traits.
8. Personality traits predict, and may in fact influence, the course and treatment response of depression.

Table 1

Summary of key predictions of the classic models

Model	Predictions about a target trait and its relation to depression
Common cause	Shared etiology accounts for the observed association
Continuum/spectrum	Similar etiology; association is fairly specific and nonlinear
Precursor	Similar etiology; predicts depression onset
Predisposition	Predicts depression onset; other variables mediate or moderate this link
Pathoplasticity	Predicts variation in presentation or outcome of depression above and beyond other baseline characteristics
Concomitants	Is altered during a depressive episode but returns to premorbid level after
Consequences/scars	Is altered during and after a depressive episode