The Role of Personality in Psychotherapy for Anxiety and Depression

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ABSTRACT A trait approach to personality has many implications for psychotherapy. Given that traits contribute to the expression of symptoms of common psychiatric disorders, are moderately heritable, and relatively stable (yet also dynamic to some extent), long-term change in symptoms is possible but is likely to be limited. Analogous to the manner in which genes set the reaction range for phenotype, standing on certain traits may set the patient’s “therapeutic range.” On the other hand, some of the same traits that may limit the depth of therapeutic benefits might also increase their breadth. In addition, taking the patient’s standing on different traits into account can inform the choice of therapeutic strategy and targets and can affect the formation of the therapeutic alliance and compliance with self-help exercises. Finally, other aspects of personality beyond traits, such as ego development and narrative identity, also appear to have important implications for psychotherapy.

A great deal of theory and research has focused on the implications of personality traits for the development and course of psychopathology. Indeed, questions concerning the associations between personality traits and the development and course of psychopathology were a central focus of the research programs developed by H. J. Eysenck (e.g., 1967) and J. A. Gray (e.g., Gray & McNaughton,
2000). Far less attention has been paid, however, to implications that personality traits have for psychotherapy. Yet a trait approach to personality has great potential to be useful in the psychological clinic. In this article, we explore the implications of personality traits for prognosis: choosing therapeutic strategies, identifying therapeutic targets, deciding whether medications are necessary, developing the therapeutic alliance, and ensuring compliance with between-session practice/self-help exercises.

Our primary focus is on four questions regarding the nature of personality traits and the relationship between personality traits and psychopathology. Two considerations guided our selection of questions to focus on. First, we believe that, relative to other questions we considered, these four questions either have more fundamental implications for psychotherapy or have been mistakenly taken to have such implications by laypeople and even experts in some cases. Second, given our commitment to empirically informed psychotherapy, we also limited ourselves to questions for which personality science has provided relatively firm answers.

The first, and most fundamental, of our four questions is “Do personality traits contribute to the development, maintenance, and/or expression of psychopathology?” If the answer to this question is “No,” then the implications of personality traits for psychotherapy would seem to be limited to whether certain traits might influence the therapeutic alliance or other aspects of the therapeutic process such as patient compliance. On the other hand, if traits do contribute to the development and/or maintenance of psychiatric symptoms, then characteristics of traits such as their degree of mutability would seem to be central to considerations of patient prognosis.

Our second question is “How stable are personality traits?” This question bears directly on mutability and therefore prognosis. For example, if traits contributing to symptoms are perfectly stable, it would seem that prognosis would be rather grim in many cases.

Our third question is “How heritable are personality traits?” As one’s genotype is invariant, it is assumed by some patients whom we work with—and perhaps by some therapists and researchers—that if much of the reliable variance in traits contributing to symptoms is heritable, then the prognosis for most psychotherapy cases would be rather grim, whereas biological interventions stand a better chance of working and are perhaps even necessary.
The final question we consider is “Are personality traits hierarchically structured?” If so, an important task confronting psychotherapists and their patients is to determine which levels of the hierarchy are the most relevant for each aspect of treatment planning and implementation.

In this article, we discuss the literatures bearing on the four questions posed above. After answering these questions, we discuss their implications for psychotherapy including challenging what we believe are two commonly held myths about personality and psychotherapy: that trait vulnerability factors cannot be altered by psychotherapy and that medications are necessary for symptom reduction. Given our expertise in anxiety and unipolar depressive disorders, almost all of our examples involve the treatment of these disorders. Finally, we will also briefly consider psychotherapeutic implications of aspects of personality beyond traits.

The current diagnostic system of the American Psychiatric Association posts several distinct acute clinical syndromes that are seen as reflecting deviations from the individual’s usual functioning and collectively comprise the Axis I disorders. It is important to note that according to this system, the Axis I disorders are distinct from personality disorders that collectively comprise the Axis II disorders and are believed to be more chronic and encompass the individual’s usual ways of interacting with the world. Given the distinct boundaries posited in the diagnostic system and that we focus only on the treatment of Axis I internalizing disorders, it might be thought that our hypotheses are necessarily limited to these disorders. Nonetheless, we believe that many of the hypotheses we discuss also apply to other disorders, including many of the Axis II disorders. The reason we say this is that empirical evidence exists that greatly blurs the boundary between Axis I and Axis II. First, Axis I and Axis II disorders are highly co-occurring. Those with a Cluster B or Cluster C personality disorder have a significantly greater chance of also having a mood or anxiety disorder (e.g., Coid, Yang, Tyrer, Roberts, & Ulrich, 2006; Lenzenweger, 2006). Second, some mood and anxiety disorders and Axis II disorders (particularly in Cluster C) share specific symptoms (e.g., social phobia and avoidant personality disorder, dysthymia, and depressive personality disorder), further blurring the Axis I-Axis II boundary (Krueger, 2005). Third, mood and anxiety disorders are associated with the same personality traits that characterize at least some personality
disorders (Widiger, 2003). Finally, Axis I and Axis II disorders are not distinguishable in many important ways, including stability, age of onset, degree of insight, or genetic versus psychosocial etiology (e.g., Krueger, 2005; Widiger, 2003).

Treatment response may be one of the areas where there may be important differences between Axis I and Axis II. Because there are many more treatment studies of Axis I than Axis II, this continues to be an area of debate (Beck, Freeman, & Davis, 2004; Krueger, 2005). While several uncontrolled clinical reports and single-case design studies yield positive results for cognitive-behavioral therapy across 9 of 10 personality disorders (excluding schizotypal), controlled treatment studies seem to exist only for antisocial, avoidant, and borderline personality disorder (Beck et al., 2004). These studies have yielded mixed results; at worst, treatment results in little change in symptomatology (e.g., those with antisocial personality disorder who were not depressed; Luborsky, McLellan, Woody, O’Brien, & Auerbach, 1985); at best, treatment results in a reduction of serious symptoms (for example, in dialectical behavior therapy for borderline personality disorder; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991). Some researchers are particularly optimistic about these results, especially those involving dialectical behavior therapy for borderline personality disorder (Krueger, 2005; Livesley, 2004). However, it is important to note that this therapy consists of 1 year of intensive treatment (much longer than many treatments of Axis I disorders) and often results in only moderate overall improvement, as opposed to complete recovery or high end-state functioning that is more commonly seen in treatments of anxiety (e.g., Borkovec & Whisman, 1996) and depressive disorders (e.g., Jacobson et al., 1996). Even though there appears to be a mean difference between Axis I and Axis II disorders in treatment responses, however, the many similarities between them suggest to us that the many of the psychotherapeutic implications of traits we discuss will also apply to Axis II disorders.

Four Basic Questions About the Nature of Personality Traits

Do personality traits contribute to the development, maintenance, and/or expression of psychiatric disorders?

Several theorists have hypothesized a trait vulnerability factor common to most, if not all, of the internalizing disorders (e.g., Cloninger,
This trait is defined slightly differently and given different labels by different theorists—harm avoidance (Cloninger), neuroticism (Eysenck), trait anxiety or behavioral inhibition system sensitivity (Fowles, Gray) and negative affectivity (Tellegen). However, in our opinion, the conceptual and empirical overlap among these conceptualizations far outweighs the differences, and a discussion of the subtle differences among these conceptualizations is beyond the scope of this article. Each of these conceptualizations implies a trait disposition to experience negative affect. The term neuroticism (N) will be used hereafter to acknowledge Eysenck’s pioneering work and to be consistent with the emergence of the Big Five model of personality—which includes N, Introversion/Extraversion (I), Conscientiousness (C), Agreeableness (A), and Openness to experience (O)—as the dominant model of personality trait structure (e.g., Goldberg, 1993; Watson, Clark, & Chmielewski, 2008).

In factor-analytic terminology, the hypothesis of a common risk factor implies that there should be a general factor that is common to depression and all of the anxiety disorders. Indeed, evidence for such a general factor has consistently been obtained across samples seeking psychiatric treatment (e.g., Zinbarg & Barlow, 1996), epidemiological samples (e.g., Fergusson, Horwood, & Boden, 2006; Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Vollebergh et al., 2001) and in a large, multinational sample comprising consumers of general health care service centers (Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003). There is also evidence not only of a general factor in cross-sectional analyses but also in longitudinal analyses (Ferguson et al., 2006; also see Gregory et al., 2007).

Though the evidence regarding the general factor common to the Axis I internalizing disorders is consistent with the hypothesis that there is a risk factor common to all of these disorders, there is an alternative explanation that these results are not capable of ruling out. That is, it is possible that the second-order factor may have arisen entirely as a consequence of demoralization resulting from developing an internalizing disorder rather than from a variable of etiologic significance (Frank, 1961; Link & Dohrenwend, 1980). Evidence with some bearing on this alternative explanation has come from multivariate, behavior-genetic studies conducted by Kendler and his colleagues (e.g., Hettema, Prescott, Myers, Neale,
& Kendler, 2005; Kendler, Prescott, Myers, & Neale, 2003). These results suggest strongly that a genetic factor is largely responsible for the pattern of symptom covariance that results in the frequently co-occurring symptoms of internalizing disorders. Furthermore, it seems likely that the genetic factor is at least partly, if not entirely, related to the vulnerability to these disorders. Indeed, data we review next from longitudinal studies demonstrate that N and related traits (e.g., behavioral inhibition and anxiety sensitivity) do prospectively predict the onset of new episodes of MDD and anxiety disorders (for a more detailed discussion of the studies on the phenotypic and genetic structure of the internalizing disorders, see Zinbarg & Yoon, 2008).

Clark, Watson, and Mineka (1994) reviewed several longitudinal studies showing N to be a predictor of both subsequent diagnoses and chronicity of major depression. Since that review, studies reported by Hayward, Killen, Kraemer, and Taylor (2000), Kendler and colleagues (e.g., Kendler, Kuhn, & Prescott, 2004), and Krueger, Caspi, Moffitt, Silva, and McGee (1996) obtained results consistent with the conclusions arrived at by Clark et al. (1994). Krueger et al. also found that N is an especially good marker of risk for multiple diagnoses of emotional disorders.

For anxiety disorders, Clark et al. (1994) also reviewed evidence that N is a risk factor for the development of post-traumatic stress disorder. Additional longitudinal studies have shown that children high on behavioral inhibition (thought to be a precursor to, and facet of, N; Turner, Beidel, & Wolff, 1996) are at greater risk for the development of multiple phobias and other anxiety disorders in later childhood (Biederman et al., 1990, 1993; Hirschfeld et al., 1992) and social phobia in adolescence (Hayward, Killen, Kraemer, & Taylor, 1998; Schwartz, Snidman, & Kagan, 1999). Hayward et al. (2000) also found N to be a predictor of panic attacks in a 4-year prospective study of adolescents. Thus, extant data are consistent with the hypothesis that N is a marker of vulnerability for both major depression and anxiety disorders.

Other studies have focused on more narrow facets of N. For example, negative beliefs about the consequences of experiencing anxiety, known as anxiety sensitivity (AS; Reiss, 1980), are believed to be facets of N that confer vulnerability for panic attacks and panic disorder and, to a lesser degree, other anxiety disorders. Laboratory studies supporting a role for AS in panic attacks entail panic provocation procedures. In nonclinical samples, AS predicts subjective
distress and reported symptomatology in response to CO₂ inhalation (Forsyth, Palav, & Duff, 1999), and hyperventilation (Sturges, Goetsch, Ridley, & Whittal, 1998), even after partialing trait anxiety (Rapee & Medoro, 1994). Several longitudinal studies now indicate that AS is a marker of vulnerability for the onset of panic attacks over 1- to 4-year intervals in adolescents (Hayward et al., 2000), college students (e.g., Li & Zinbarg, 2007), and community samples with specific phobias or no anxiety disorders (Ehlers, 1995). In addition, AS predicted the development of worry about panic (and anxiety more generally), during an acute military stressor (i.e., 5 weeks of basic training), even after partialing history of panic attacks and trait anxiety (e.g., Schmidt et al., 1999). Finally, whereas the clinical/practical importance of AS in symptom genesis has been questioned based on small effect size estimates (Schmidt, Lerew, & Jackson, 1999), Li and Zinbarg (2007) demonstrated that when AS is measured properly (i.e., in accordance with its hierarchical structure) it predicts meaningful proportions of variance in the onset of panic.

Given that low positive affect (PA) or anhedonia is relatively specific to depression (e.g., Clark & Watson, 1991), Fowles (1993) and others hypothesized low positive emotionality (PEM) to be a trait-vulnerability marker for depression. Despite different labels for this marker—PEM (Tellegen, 1985) and Introversion (I; DePue & Iacono, 1989)—similarities in these conceptualizations, in our view, outweigh the differences, and a discussion of the subtle differences among these conceptualizations is beyond the scope of this article. We will refer to this trait as I, again to acknowledge Eysenck’s pioneering work and to be consistent with the Big Five model of personality. Several longitudinal studies suggest that I is a predictor of subsequent depressive symptoms (e.g., Levenson, Aldwin, Bosse, & Spiro, 1988; Holahan & Moos, 1991) and diagnoses (Hirschfeld et al., 1989; Krueger et al., 1996), but other studies did not replicate these findings (e.g., Gershuny & Sher, 1988; Kendler et al., 1993). Gershuny and Sher (1998) found that I interacted with N in the prediction of symptoms of depression although Jorm et al. (2000) failed to replicate this interactive effect. A more consistent finding is that I has been shown to be positively associated with the chronicity of the course of major depression (Clark et al., 1994). There is also evidence that children at risk of depression by virtue of having a mother with a history of depression are low on observational measures of PEM (Durbin, Klein, Hayden, Buckley, & Moerk, 2005).
Structural modeling studies have also been informative about the relationship between normal and abnormal traits or characteristics. O'Connor (2002) meta-analyzed dozens of correlation and factor loading matrices published in English-language journals over the past 50 years. This included data on 37 measures, both normal (e.g., 16 Personality Factor Questionnaire, California Psychological Inventory) and abnormal (e.g., Beck Depression Inventory, MMPI) in content and with clinical and nonclinical populations. The results indicated that the same number of components existed in the measures for both clinical and nonclinical populations, with the factor structure looking essentially the same for the two groups. Another study completed a meta-analysis on 77 samples from 52 studies examining five different personality measures (Markon, Krueger, & Watson, 2005). Markon, Krueger, and Watson (2005) also found an integrated model of normal and abnormal personality, which they then replicated in a sample of college-age participants. Thus, these two meta-analyses converge in suggesting that psychopathology symptoms can often be explained as extreme variants of normal traits.

Similar structural questions have been studied in the area of personality disorders (PDs). O'Connor and Dyce (2001) reported a five-factor structure of PDs. Importantly, normal and abnormal personalities were distinguishable only by vector length, a measure of rigidity, and extremity of the trait. Miller, Lynam, Widiger, and Leukefeld (2001) also reported results that support the contention that psychopathy can be understood as extreme standing on several Big Five personality factors, most notably extremely low A and extremely low C. One possible exception to the conclusion that PD symptoms can be explained as variants of normal traits is the evidence presented by Watson, Clark, and Chmielewski (2008) that the Big Five needs to be expanded to incorporate a dimension—Oddity—relevant for the symptoms related primarily to the Cluster A PDs. On the other hand, Watson et al. (2008) also review evidence that the Big Five personality factors (with the exception of Openness) provide an adequate basis for the assessment of the characteristics related to the Cluster B and Cluster C PDs.

In concluding this section, it is clear that N-related and I-related traits are markers of mechanisms that contribute to the development and/or maintenance of anxiety and depressive disorders. Whereas there is a need for longitudinal research testing whether other traits are markers for risk of other disorders, cross-sectional research on
structural models suggests that four of the five Big Five personality factors (i.e., all but Openness) might contribute to the development, maintenance, and/or expression of PDs.

*How stable are traits?*

McCrae and Costa (1996) concluded that much research has shown that normal personality traits tend to remain relatively stable throughout a person’s lifetime. Consistent with this, a recent study by Watson and Humrichouse (2006) found rank-order stability in the .7 range (with little variability across different traits) for a 2-year interval for both self- and spouse ratings. Hampson and Goldberg (2006) found similar estimates of .70–.79 (.85–.98 corrected for unreliability) over 2.8 years in an adult sample. Indeed, based on the observation that the asymptotic values (as retest intervals lengthen) of adult trait temporal stability coefficients are above zero, Fraley and Roberts (2005) concluded that there must be a constant factor contributing to trait scores over time.

At the same time, it is important to note that a well-accepted principle among researchers in this area is that the longer the retest interval, the lower the level of rank-order stability (e.g., Roberts & DelVecchio, 2000). For example, Hampson and Goldberg also reported very modest temporal stability coefficients over a 40-year interval spanning elementary school and midlife including values of .16 for O, .08 for A, and .00 for N. Though it is almost certain that higher values would have been obtained if the 40-year retest interval had spanned early adulthood to older adulthood (e.g., Fraley & Roberts, 2005; Roberts & DelVecchio, 2000). Other studies have also demonstrated moderating variables that are associated with changes in personality, at least in the period of adolescence to young adulthood. For example, the introduction of a controlled stressor (12-month exposure to an intercultural environment) can decrease personality vulnerability in terms of trait anxiety, locus of control, and defensiveness (Andrews, Page, & Neilson, 1993). Another study showed that one’s work experience, including success, power, satisfaction, and security in young adulthood can modify basic personality dimensions, particularly N and E (Roberts, Caspi, & Moffitt, 2003). These results illustrate the point that personality traits, though relatively stable in the short- and midterm, are far from immutable.

Results have consistently shown that N and I scores significantly increase (sometimes up to one standard deviation) during a depres-
sive episode (see Costa, Bagby, Herbst, & McCrae, 2005; Ormel, Oldehinkel, & Vollebergh, 2004; Santor, Bagby, & Joffe, 1997). This has caused much confusion and controversy among personality researchers, with some concluding that personality measures are systematically biased by acute depression, therefore questioning the validity and usefulness of personality assessment during depressive episodes (e.g., Hirschfeld, Klerman, Clayton, & Keller, 1983). Other researchers have proposed that it is erroneous to believe that personality traits are immutable but that the changes in trait scores that occur during a depressive episode represents a “true” change in personality (Costa et al., 2005). Widiger and Trull (1992) discuss a third possibility of a spectrum relationship in which both personality and disorder stem from common causes and vary together.

While this debate is not yet settled, sophisticated methodological techniques may make it possible to do so in the future. Thus, a study might use informant reporting to rule out the possibility that reporting bias is entirely responsible for the changes in trait scores during a depressive episode. Of course, it is possible that the observed changes reflect both reporting bias and true trait or spectrum change. Teasing apart the trait change and spectrum change explanations is probably more challenging, and a full discussion of this issue is beyond the scope of this article. Briefly, though, the crux of the difference seems to be that the trait change explanation implies that N and depressive episodes are related yet distinct constructs, whereas the spectrum explanation implies that although they reflect a single trait, they reflect different location parameters along this trait. Thus, one could use confirmatory factor analysis for categorical items—equivalent to multidimensional Item Response Theory analysis (e.g., Muthen, 1984; Takane & de Leeuw, 1987—to test whether a diagnosis of depression and symptoms of depression define a separable factor from that defined by items tapping N. Certainly, regardless of how this debate is ultimately resolved, the evidence regarding changes in trait scores associated with depressive episodes is consistent with the conclusion based on trait stability studies in nonclinical samples that traits are not immutable.

How heritable are traits?

Behavioral genetic research has yielded consistent results indicating a moderate heritable component of traits. There appears to be little
differential heritability across traits with estimates falling in the moderate range of .20 to .60 (e.g., Jang, McCrae, Angleitner, Riemann, & Livesley, 1998; Keller, Coventry, Heath, & Martin, 2005; Loehlin, Neiderhiser, & Reiss, 2003; Saudino & Plomin, 1996). Shared environment (e.g., parenting style, socioeconomic status, neighborhood) accounts for, at most, a small portion of variance in most traits (e.g., McCrae, Jang, Livesley, Riemann, & Angleitner, 2001; Saudino, Plomin, & Defries, 1996). However, some studies have found that larger shared environmental effects are sometimes found when variables that moderate heritability are modeled (Krueger, South, Johnson, & Iacono, this issue). Nonshared environmental effects (e.g., peer group influence, birth order effects, accidents, illnesses) for personality traits are estimated to fall between .4 to .8 (e.g., Saudino & Plomin, 1996). It is important to remember that nonshared environment is computed as a residual score, which includes the variance, if any, associated with the interaction of the genotype by shared environment, systematic method variance, random error variance, and nonadditive genetic effects.

Also, pertinent to this discussion is the heritability of psychopathology, with a focus on depressive and anxiety disorders. Research has shown that major depressive disorder (MDD) has a heritability estimate of approximately .4–.5, with the remaining variance accounted for by nonshared environmental effects (for review, see Levinson, 2005; Sullivan, Neale, & Kendler, 2000). Behavioral genetic analyses of anxiety disorders yield similar results, with estimates of heritability generally ranging from .3–.4 (see Hettema, Neale, & Kendler, 2001). Again, common environmental effects were nil, with nonshared environmental effects accounting for 60–70% of the variance. As mentioned above, multivariate behavior-genetic studies suggest at least one genetic factor common to MDD and the anxiety disorders (e.g., Hettema et al., 2005; Hettema, Neale, Myers, Prescott, & Kendler, 2006; Kendler et al., 2003). Moreover, N appears to be related to one of these common genetic factors (e.g., Hettema et al., 2006). For example, the association between N and MDD results largely from shared genetic factors; approximately 50% of the genetic effects on MDD are shared with N (Kendler, Gatz, Gardner, & Pedersen, 2006). For GAD, this number is approximately 35% (MacKintosh et al., 2006).

Behavioral genetic research not only studies the variance accounted for by genetics and the environment within traits, but
the covariation between traits. Multivariate factor analyses have been completed on genetic correlations of groups of traits, leading behavioral genetic research to support two-, three-, four-, and five-factor models of personality (Krueger & Johnson, 2007). This research also supports personality structure at the individual facet level beneath the Big Five (Jang et al., 1998). Taken together, this evidence suggests that genetic influences on personality traits are not confined to one specific level of the personality hierarchy but affect the entire hierarchy and all of its levels. The phenotypic structure of personality traits, as discussed below, appears to mirror this hierarchical organization (Krueger & Johnson, 2007).

*Are personality traits hierarchically structured?*

As noted earlier, the dominant model of normal personality trait structure is the Big Five (e.g., Goldberg, 1993). Many theorists have represented and emphasized the hierarchical structure of the Big Five (e.g., Costa & McCrae, 1997; Digman, 1997; Eysenck, 1947; Watson et al., 2008). Importantly, though different researchers have endorsed what might appear to be different structural models, Markon, Krueger and Watson (2005) present evidence that the structures put forward by these different theorists can be integrated and understood as focusing on different levels of the same hierarchical structure. Costa and McCrae (1997) present the Big Five at the highest level of the hierarchy, with each Big Five factor comprised of six lower order facets. Digman (1997) presents a five-level hierarchy with the fourth level consisting of the Big Five, with two additional factors, $\alpha$ (comprised of A, N, and C) and $\beta$ (comprised of O and I), at Level 5. Levels 1–3 in the Digman model are those proposed by Eysenck (1947): Level 3 is facets of the Big Five, Level 2 is habitual responses, and Level 1 is specific responses to situational cues. Eysenck (1992) advocated that Level 4 (represented by the Big Five in Digman’s model) consists of only three factors: I, N, and psychoticism (P). In Eysenck’s model, A and C reside on Level 3, as facets of P.

Based on both theory (e.g., Lilienfeld, Turner, & Jacob, 1993) and empirical evidence (e.g., McNally, 1996; Zinbarg & Barlow, 1996; Zinbarg, Barlow, & Brown, 1997; Zinbarg, Brown, Barlow, & Rapee, 2001), we view N-spectrum traits as comprising a four-tiered hierarchy with N as a fourth-order trait that is related to the general
tendency to react with negative affect to stressful stimuli. (It is worth noting, however, that we agree with Livesley, 2007, that we see no reason why nature should be ordered so neatly that all five Big Five traits comprise structures with the same numbers of levels or the same number of facets per level.) Trait anxiety is a third-order facet of N related to the slightly less general tendency to react anxiously to potentially threatening stimuli (with trait depression and hostility being examples of other third-order facets of N). AS is a second-order facet (perhaps corresponding to Eysenck’s habitual response level) of trait anxiety related to the more specific tendency to react anxiously to one’s own anxiety. Finally, AS is divisible into three first-order facets (corresponding to Eysenck’s level of specific responses to particular cues) relating to concerns about different domains: (a) AS-Physical Concerns (fear of the physical sensations of anxiety related to the belief that they will result in physical catastrophe), (b) AS-Mental Incapacitation Concerns (fear of the mental and emotional manifestations of anxiety related to the belief that they will result in emotional catastrophe), (c) AS-Social Concerns (fear of publicly observable sensations of anxiety related to the belief that they will result in social catastrophe). Whereas AS tends to be elevated in all anxiety disorders and major depression, profiles across the first-order facets of AS appear to be useful for discriminating among these disorders (Zinbarg et al., 1997).

Hierarchical models are important for studying personality and related constructs, partly because of the “bandwidth/fidelity trade-off” (Cronbach & Gleser, 1957). This refers to the notion that broad constructs (those at the top of a hierarchy) are useful in predicting diverse behaviors at moderate levels of accuracy, while narrow constructs (those at the bottom of a hierarchy) are useful at predicting a limited range of behaviors at a high level of accuracy. This might imply that a personality construct should ideally be studied at different levels of breadth (Hampson, John, & Goldberg, 1986). However, some researchers have identified a “basic level” of personality traits, which they considered to be the most useful and informative level. In a study by John, Hampson, and Goldberg (1991) this level consisted of the highest level of the hierarchy that is still behaviorally descriptive, the third level of a four-level hierarchy. This level was preferred by John and colleagues over the more abstract superordinate levels and the more basic and specific subordinate levels, illustrating a good compromise between bandwidth
and fidelity. When it comes to implications for psychotherapy, however, it seems to us that different levels of the hierarchy each have their own contributions to make. Our discussion of treatment planning to follow draws heavily on the second and first levels. In contrast, our discussion of the breadth of therapeutic benefits is most germane to the third and fourth levels.

**Myths Based on the Evidence Bearing on One or More of the Four Basic Questions About the Nature of Personality Traits**

*Trait vulnerability factors cannot be altered by psychotherapy*

Some researchers seem to have either implicitly or explicitly assumed that trait vulnerability factors cannot be altered by psychotherapy. For example, Mathews, Mogg, Kentish, and M. Eysenck (1995) suggest that their finding that cognitive-behavioral therapy (CBT) for generalized anxiety disorder (GAD) reduced an information processing bias is “consistent with two alternative views: either that cognitive bias depends on current state factors such as mood, or that vigilance represents an enduring characteristic that becomes apparent only when vulnerable Ss are under stress” (p. 302). Clearly, an implicit assumption underlying this argument is that trait vulnerabilities can’t be altered by psychotherapy (ergo, any characteristic, such as an information-processing bias, which appears to have been altered by therapy must neither be a trait vulnerability factor nor have been altered but rather is one not currently being expressed). In turn, this implicit assumption seems likely to stem from thinking of traits as being immutable. Notably absent from this discussion is the possibility that threat-processing biases are trait vulnerability markers that are relatively enduring but not immutable and that, therefore, are at least partly modifiable by experiences such as therapy. In contrast, the literature cited above clearly shows that traits can be vulnerability factors that are relatively enduring but far from immutable. Given the mutability of traits, we believe they can be modified to some degree by therapy.

Consistent with our belief, there are several studies that have provided evidence for a change in personality trait scores over the course of therapy, with most concentrating on the Big Five traits over the course of treatment for depression (e.g., Clark, Vittengl, Kraft, & Jarrett, 2003; De Fruyt, Van Leeuwen, Bagby, Rolland, & Rouillon,
As mentioned earlier, the major finding is that N and I scores tend to decrease over the course of treatment for depression. This has been supported regardless of whether the treatment is psychotherapy (Clark et al., 2003) or psychotropic medication (Bagby, Levitan, Kennedy, Levitt, & Joffe, 1999; Costa et al., 2005; Du et al., 2002; Santor et al., 1997). Moreover, whereas most of the medication studies failed to include the use of a placebo control group, we are aware of at least one exception that did find greater personality change associated with the active medication than with a placebo (Tang, DeRubeis, Hollon, Shelton, & Schalet, in preparation).

The basic effect, that of the change in N and I scores, is fairly consistent among studies (although De Fruyt et al., 2006, found only a change in N), and there are two other important findings from this body of research. First, though some studies have noted that change in personality traits is correlated with change in depression (e.g., Clark et al., 2003; Du et al., 2002), other studies have found that personality trait change occurs largely independent of change in depression. From these studies we might conclude that treatment for depression can have a significant effect on personality traits, independent of the effect the treatment has on depression (De Fruyt et al., 2006; Santor et al., 1997; Tang et al., in preparation). Second, studies have also demonstrated that personality trait variables not only demonstrate absolute change in the form of decreasing N and I but also rank order stability (De Fruyt et al., 2006; Santor et al., 1997). We interpret these results to imply that (a) the rank order stability found in these studies reflects a constant source of variance in personality traits that remains unchanged throughout adulthood (see Fraley & Roberts, 2005), and (b) the absolute change in personality trait measures seen throughout depression treatment represents an actual altering of the traits of N and I (for an alternative view, see Clark et al., 2003).

**Medications are necessary for symptom reduction**

Some of our patients (and according to their accounts, some of their psychiatrists and former psychotherapists) hold the belief that the heritability of traits likely involved in the development, maintenance, and/or expression of common psychiatric disorders implies that medications are necessary for successful treatment. A simple version of
this argument might go something like this: if there is a biological basis to psychopathology, then effective treatment must also involve a biological intervention. A more sophisticated version might recognize that traits associated with symptoms are not immutable but assume that the genetic influences on these traits are and will continue to produce symptoms if not addressed directly (i.e., biologically). Indeed, it is certainly true that the genotype remains constant over the lifespan and will not be altered by psychotherapy. Yet both versions of this argument are fallacious for four reasons.

First, heritability estimates are far from perfect. Thus, the environment clearly does influence traits even if the shared environment does not.

Second, despite the fact that genotype remains constant over the life span, heritability estimates can, and sometimes do, change as a function of age perhaps due to variability in gene expression over time and/or to gene-environment transactional processes (e.g., McGue, Bouchard, Iacono, & Lykken, 1993; Plomin & Spinath, 2004). Similarly, estimates of the influence of the shared, familial environment can, and sometimes do, change as a function of age probably due to the potency of more recent environment influences relative to more distal environmental influences (McGue et al., 1993; Plomin & Spinath, 2004). That is, genetic influences are clearly not constant over time.

Third, heritability estimates are specific to the range of environments represented in the samples they are based on. Even if the range of sampled environments is large, heritability estimates still only represent an average of these without any information regarding the effect of a specific environment. For example, Krueger and colleagues (this issue) found that genetic shared environmental and nonshared environmental effects were moderated by the participants' perception of their parental relationship. That is, when participants rated the relationship low in Regard, this diminished genetic effect and enhanced nonshared environmental effects. When participants rated the relationship high in Conflict, this diminished the genetic effect and enhanced shared environmental effects. It is possible that with further advances in psychotherapeutic theory and technique that psychotherapies for some conditions will represent systematic environmental perturbations larger than any represented in extant behavioral genetic studies and large enough to lead to larger trait differences than those obtained to date.
Finally, we know that experience changes the structure of the brain (e.g., Merzenich, 1998). Thus, psychotherapy is capable of leading to brain reorganization such that medications are not necessary even to effect changes in the biological bases of traits.

**Psychotherapeutic Implications of the Research Bearing on the Four Basic Questions About the Nature of Personality Traits**

If the basic research findings concerning the nature of traits does not support the inferences that trait vulnerability factors cannot be altered by psychotherapy or that medication is necessary, what then are the implications of this research? We next turn our attention to some of the implications that we believe traits can have for psychotherapy.

**Prognosis: The depth of psychotherapeutic benefits**

Given that personality traits such as N, AS, and I appear to be involved in the development or maintenance of symptoms, the patient's standing on these traits may place constraints either on the amount of therapeutic improvement he or she might experience or on the durability of such improvements. Indeed, if these traits had been shown to be entirely heritable and perfectly temporally stable in test-retest studies, then it might seem reasonable to argue that the benefits of psychotherapy would be fleeting at best. Even then, however, if few participants in the studies producing the perfect heritability and stability coefficients had been in psychotherapy, such an argument would not be unassailable. Fortunately, we do not need to debate such issues as it is clear that traits are not immutable given that they are neither completely stable nor entirely heritable. Thus, whereas some might argue that if traits are involved in symptom development, maintenance, and/or expression then long-term change in symptoms as a result of psychotherapy would be impossible, such a view is clearly not warranted by the evidence. That is, given that traits are not immutable, the fact that there are trait influences on symptom development, maintenance, and/or expression certainly does not imply that long-term change in symptoms should be impossible.

What then are the psychotherapeutic implications of the findings that personality traits are partially heritable and of those that led
Fraley and Roberts (2005) to conclude that there is a factor (i.e., one’s genotype) that provides a constant contribution to variance in personality traits? These findings are certainly consistent with our belief that the benefits of psychotherapy are likely to be at least somewhat limited in magnitude both in terms of effects on symptoms and on traits (for a related discussion, see Harkness & Lilienfeld, 1997). That is, the evidence suggests that there is a limit to the benefits of even the most effective of our currently available treatments. For example, in a trial of CBT for GAD Zinbarg, Lee, and Yoon (2007) obtained large effect sizes (on average symptom scores at post-test in the treatment group were 1.48 standard deviations (SDs) lower than in the wait-list group and 1.71 SDs lower than their own pretest scores). Despite these statistically large effects, the clinical significance of the findings were much more limited. Only 50% of the patients achieved high end-state functioning (i.e., being brought within 1 SD of the normal mean) on four of five outcome measures, and we are not aware of any studies that have obtained meaningfully better high-end state functioning results with GAD. Moreover, consistent with the findings cited above that whereas N and I tend to decrease during treatment they often remain elevated relative to the normal population (Costa et al., 2005; De Fruyt et al., 2006; Du et al., 2002), none of the patients scored more than a half of a SD below the normal mean on at least four of the measures.

Thus, it is possible to bring patients who begin therapy with symptom scores outside of the normal distributions back into the normal range as a result of therapy. We also believe it is possible to improve on our current therapies such that even higher proportions of our patients might achieve this high end-state functioning status in the future. However, we do not think it is realistic to expect to bring many high N patients to the low end of either symptom distributions or the N distribution as a result of psychotherapy. In an analogous fashion to the manner in which genes set the “reaction range” (Gottesman, 1963) for phenotype, we offer the speculation that standing on a trait such as N may be said to set the patient’s “therapeutic range.”

A weak version of the “therapeutic range” hypothesis does not necessarily predict that pretreatment personality predicts the amount of improvement a patient experiences. Rather, it may be that most patients will begin therapy with high enough elevations on N (or other relevant traits) that they all have a somewhat restricted
“therapeutic range” and/or a restricted statistical range of trait scores. (That is, it may be that there is an asymptotic function between N and “therapeutic range” such that further increases in N above some threshold value are not associated with further decreases in “therapeutic range.”) Thus, a weak version merely predicts that very few patients will ever be brought more than approximately a half of a SD below the normal mean on a majority of outcome measures.

Interestingly, however, some (though certainly not all) studies have shown that pretreatment personality traits have an impact on treatment outcome consistent with a stronger version of the “therapeutic range” hypothesis. For example, some researchers have explored the role of one facet of N—perfectionism—as measured by the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978) in the Treatment of Depression Collaborative Research Program (TDCRP). Over the course of treatment, patients with high to moderate levels of perfectionism at pretreatment made no significant therapeutic gain after the eighth treatment session (Blatt & Zuroff, 2005). Using video recordings of a sampling of treatment sessions and a measure that codes therapist and patient participation in the therapeutic alliance (Krupnick et al., 1996), results indicated that perfectionist patients showed a disengagement in therapy after the eighth treatment session (Shahar, Blatt, Zuroff, Krupnick, & Sotsky, 2004; Zuroff et al., 2000). One possible explanation of these results is that these patients may have tended to apply their perfectionistic beliefs to the therapy, which would be certainly be consistent with a trait perspective. If so, and if this tendency to apply perfectionistic standards to their work in therapy were not treated directly, it would have left the patients vulnerable to feeling they were “failing” in therapy and hopeless. Hopelessness about therapy, in turn, would be expected to lead to disengagement (but see Shahar et al., 2004, for an alternative explanation).

Some might argue that the nonzero heritability estimates of personality traits and the tendency for temporal stability coefficients to asymptote at values above zero (as the follow-up interval increases) necessarily dictates that the upper limit to therapeutic benefit must fall short of bringing patients to the low end of symptom and trait-risk factor distributions. If so, we believe such an argument would be just as logically fallacious—and for many of the same reasons—as the arguments that traits cannot be altered by psychotherapy or that
medications are necessary. Also, as in an example provided by Hampson and Goldberg (2006) of the number of hair follicles per square centimeter of scalp in men, characteristics with even higher heritabilities than personality traits undergo radical changes. That is, if men with genetically determined “thick” hair can, and sometimes do, go bald, then genetic influences on traits do not preclude radical change in standing on a trait. Thus, though we believe bringing patients to the low end of symptom and trait-risk factor distributions is unlikely, it is not logically impossible.

To summarize, we believe it is logically fallacious to argue against the possibility of therapy leading to radical change in personality traits based on evidence of their temporal stability and heritability. At the same time, we believe that until good evidence for such radical transformation is presented, it is best (and certainly most realistic) for patients and therapists alike to set more modest expectations for therapy given that many of the traits that contribute to seeking psychotherapy in the first place may limit a patient’s “therapeutic range.”

Prognosis: The breadth of psychotherapeutic benefits

Despite the limits to the depth of change we can realistically expect from psychotherapy, we believe the personality trait literature gives us more reason to be optimistic about the breadth of change. That is, the fact that there appear to be broad risk factors that confer risk for many forms of psychopathology offers some hope that when therapy is beneficial, it might have broad effects beyond the specifically targeted symptoms. To the extent that psychotherapy focused on the principal diagnosis leads to some reduction in one of those broad factors, then we should expect improvement in comorbid conditions even if they are not explicitly targeted in treatment.

To illustrate this point, consider the case of N and internalizing disorders. As discussed above, N appears to be a broad risk factor for MDD and the anxiety disorders. Thus, psychotherapy for any one of these disorders that produces change in one or more the processes (e.g., biases in the processing of threatening and/or ambiguous information, physiological hyper-reactivity, avoidance behaviors) that likely link N to symptoms should also be associated with reductions in symptoms of comorbid anxiety disorders or MDD that aren’t explicitly targeted in the therapy. Indeed, whereas
there is a need for more research on the impact of psychotherapy on comorbid conditions, the available evidence in the literature on CBT for anxiety disorders supports the expectation of broad improvement that extends to comorbid conditions not explicitly targeted in therapy (Borkovec, Abel, & Newman, 1995; Brown & Barlow, 1992).

Therapeutic strategy and targets

The bandwidth/fidelity trade-off may have specific implications in the clinical setting, especially in terms of treatment planning. First, personality trait measures can identify higher order traits that are related to a wide range of thoughts, feelings, and behaviors that may be causing distress in the patient (e.g., high N and high I) or that might interfere with treatment (e.g., low C, low or extremely high A). They give the therapist a broad, general view of how a patient thinks, feels, and behaves. The therapist now has a starting point from which to begin to understand the patient, develop strategies for building a strong alliance, and think of possible interventions that will be useful. For example, a therapist should be wary of a highly agreeable patient for signs of false enthusiasm or possible alliance tears that the patient does not verbalize because he or she is overly motivated to please the therapist (Miller, 1991; Muten, 1991). Similarly, a therapist should be prepared to be highly reinforcing of a low-conscientious patient for homework completion. However, specific treatment planning based solely on the higher order traits may be unwise; closer examination of lower order facets will predict patients’ behaviors with higher accuracy, allowing for a more specific treatment plan. Therefore, a second step involves examination of lower order facets to more specifically tailor a therapeutic approach. The lower order facets that comprise a higher order factor are often distinct enough in kind to warrant different treatment approaches. In other instances, lower order facets may not be this distinct but may be distinct enough to inform the choice of the specific target that a treatment approach is applied to. In general, it is likely that a person scoring high on one facet of a higher order factor will score high on the rest. A closer examination of the facets, however, may reveal informative data about the specific patient.

Let us also take the example of Introversion. A therapist with two patients who are extremely introverted might deem them both to be
better candidates for individual therapy than for group therapy (especially if the therapy group were relatively unstructured, in which case their participation would be expected to be minimal; for a related discussion see Miller, 1991). Consistent with this, Ogrodniczuk, Piper, Joyce, McCallum, and Rosie (2003) found that I and N were related to poor outcome in outpatient group therapy, whereas O and A were related to good outcome. However, each patient might have a distinct I facet profile that is informative to treatment planning. For example, Patient #1 is especially low on the I facets of warmth and gregariousness but average on the I facet of trait positive emotionality. In contrast to his average standing on trait positive emotionality, however, he is especially low on a measure of current state positive emotions, indicating an anhedonic depressive state in an individual who has at least average capacity for pleasure. A treatment plan might focus on behavioral activation (e.g., Martell, Addis, & Jacobson, 2001). On the other hand, Patient #2 is especially low on the I facets of assertiveness and excitement seeking, indicating that this person avoids situations that might involve increased emotional arousal, even if the end result is potentially rewarding. A treatment plan might focus on assertiveness training and behavioral exposures to increase tolerance of emotional arousal.

Another example involves N. A therapist, noting that there are two patients high on the higher order trait of N, might first hypothesize that these people are especially likely to have a mood or anxiety disorder, be prone to experiencing negative affect, and might benefit from CBT. However, closer examination of their N facet profiles may indicate that different therapeutic approaches are necessary. Patient #3 is especially elevated on the anxiety and self-consciousness facets, indicating that cognitive restructuring and behavioral exposures should be aimed at fear of evaluation. Patient #4 is especially elevated on the impulsiveness and angry hostility facets, indicating that relaxation training and anger management training might be helpful.

A third example involves simultaneous consideration of I facets and N facets. Patients #5 and #6 are both high on N, with especially high elevations on the anxiety facet, thereby suggesting that exposure therapy may be beneficial. Patient #5 has at least average scores on the I facet of PEM, whereas Patient #6 is low on the I facet of PEM. Thus, one might be able to facilitate the exposure therapy for Patient #5 with motivation interviewing techniques designed
to increase approach motivation for exposures (Zinbarg, 1998). In contrast, such an approach might be of more limited value for Patient #6, and the therapist should instead focus primarily on decreasing avoidance motivation via cognitive restructuring to facilitate the exposure therapy.

A fourth example involves the second-order and first-order facets in our four-tiered N hierarchy. Patients #7 and #8 are both high not only on the fourth-order trait of N but are also both high on the third-order facet of anxiety. Thus, they would both be likely to benefit from CBT including exposure therapy. However, Patient #7 is also high on the second-order facet of AS and the first-order facet of AS-Physical Concerns, whereas Patient #8 is not. Rather, in addition to an elevation on the third-order facet of trait anxiety, Patient #8 is also high on the second-order facet of Specific Fears and the first-order facet of Fear of Heights. Thus, exposure would be indicated for both patients, but for Patient #7 we would recommend interoceptive exposure whereas we would obviously recommend in vivo or virtual exposure to heights for Patient #8 (see Zinbarg et al., 1997). From these oversimplified examples, one can see how an understanding of the hierarchical structure of personality and utilization of personality measures that take this structure into account can be helpful for treatment planning.

Alliance

There is evidence that even in CBT, which emphasizes specific therapeutic techniques and deemphasizes the importance of the therapeutic relationship relative to many other schools of psychotherapy, the strength of the therapeutic alliance is a predictor of outcome (e.g., Castonguay et al., 1996; Klein et al., 2003; Krupnick et al., 1996; Loeb et al., 2005). Thus, the patient’s standing on A might be expected to predict outcome as mediated by its impact on the therapeutic alliance (Miller, 1991). Consistent with this hypothesis, Gurtman (1996) found that the patient’s standing on hostile-dominance predicts poor therapeutic alliance and outcome. This association might be moderated, however, by therapist skill at working with disagreeable patients or by amount of attention to alliance building paid by the therapist. That is, disagreeable patients should require greater interpersonal skill, greater skill at repairing alliance ruptures, and/or effort on the part of the therapist to achieve the
same level of benefit compared with agreeable patients with equally severe initial symptoms.

**Compliance**

The best available evidence suggests that compliance with self-help exercises in between sessions is a predictor of outcome in CBT (e.g., Addis & Jacobson, 2000; Burns & Spangler, 2000; Kazantzis, Deane, & Ronan, 2000). Thus, the patient’s standing on C, A, and O at initial assessment might be expected to predict outcome as mediated by compliance with self-help exercises (Miller, 1991). Knowledge of a patient’s C level could be useful to a therapist in order to build in extra structure when negotiating self-help exercises with a low C patient (negotiating not only what exercises the patient will work on between sessions but also when and what external cues will be used to remind the patient when it is time to work on a given exercise). Note that we use the verb negotiating rather than assigning; we believe it is always best practice to adopt a collaborative stance and offer a menu of choices when it comes to self-help exercises. Knowledge of a patient’s A level could be helpful, however, to encourage a therapist to be particularly mindful of these principles from the outset of therapy with a highly disagreeable patient (Miller, 1991). Finally, as most self-help exercises will involve the patient experimenting with behaviors, cognitions, or feelings, O may also be related to compliance (McCrae, 1991; Miller, 1991). Thus, knowledge of a patient’s O level could help a therapist adopt a more incremental approach to negotiating self-help exercises to gradually build up to responses that deviate more and more from the patient’s usual repertoire.

**Beyond Traits**

It is worth mentioning that there are components of personality beyond traits that have important implications for psychotherapy. One increasingly influential theory of personality suggests that traits form the foundation or “dispositional signature” of personality (McAdams, 1995, p. 372), onto which are added two other distinct levels of personality: characteristic adaptations and narrative identity (e.g., McAdams, 1995, 2001; McAdams & Pals, 2006). Characteristic adaptations include a wide range of dynamic motivational, social-cognitive, and developmental aspects of human individuality. These
components of personality are contextualized within situations and evolve over time. In addition, people vary with respect to the life stories that they construct to provide their lives with a sense of unity and purpose and to make meaning out of their experiences, a level of personality termed **narrative identity** (e.g., McAdams, 1995, 2001; Singer, 2004). These three levels of personality are sometimes related to each other in predictable ways, and sometimes they are not, suggesting that characteristic adaptations and narrative identity are not simply emergent properties of traits (e.g., McAdams, 1995; McAdams & Pals, 2006). Individual differences at each of these additional levels of personality have also been studied in relation to psychotherapy. Indeed, Singer (2005), adopting and expanding upon this three-level approach to personality, strongly advocates that taking all components of personality into account, what he calls an “integrative, person based personality psychology, forms a scientific and ethical basis for the treatment of individuals in psychotherapy” (p. 158). Westen, Gabbard, and Blagov (2006) echo this assertion in their claim that a multileveled conception of personality structure should serve as a context for understanding psychopathology and therefore treatment. A comprehensive overview of these literatures is well beyond the scope of the present article, but for completeness it is important to acknowledge certain key findings that incorporate these other levels of personality.

**Characteristic adaptations—Ego development**

One characteristic adaptation that has implications for psychotherapy is ego development (ED). ED is a life span developmental construct that concerns the complexity individuals use when making meaning out of their experiences (e.g., Loevinger, 1976; Westenberg & Block, 1993). Individuals’ stage of ED does not tend to show strong correlation with their profile of dispositional traits, although ED is sometimes observed to correlate with the trait of Openness (Westenberg & Block, 1993). We have selected ED as an example because it is an important, if sometimes overlooked, characteristic adaptation among those that have been operationalized well for systematic empirical study (Westen, Gabbard, & Blagov, 2006). (For example, a PsychInfo search for ego development yielded 2,897 results, while searches for other characteristic adaptations, such as attachment, yielded many more [17,855].) There are at least three ways
in which the study of ED can inform the study and practice of psychotherapy.

First, King and her colleagues (e.g., King & Napa, 1998) found that lay conceptions of “the good life” include both subjective well-being as well as leading a life of complex meaning (high ED). They therefore suggest that clinicians broaden their conception of the desired outcomes of therapy to include an increase in the nuance and depth of a client’s self-understanding. Ego stage is unlikely to be related to the client’s trait profile, while their level of happiness might be predicted by neuroticism and extraversion.

Second, a client’s stage of ED has implications for their treatment. Dill and Noam (1990) found that individuals at higher stages of ED tended to request psychodynamic, insight-oriented therapy, whereas individuals at lower stages of ED tended to request more behavioral treatments and triage-type interventions that focused on immediate problem solving. As behavior therapy has become increasingly integrated with cognitive techniques and theory (e.g., Zinbarg, 1990), however, CBT may have gained some ground in preferences among those at higher stages of ED since the time that Dill and Noam collected their data, especially among those aware of the emphasis on meaning in contemporary CBT. Once in treatment, theory suggests that individuals at varying stages of ED might also bring different concerns to therapy. For example, Noam (1998) suggests that each stage of ED might bring with it certain specific vulnerabilities that could not be predicted from knowing the client’s trait profile.

Finally, variation in ED is related to the stories clients tell about their treatment experience, above and beyond that explained by traits (e.g., Adler & McAdams, 2007, in press). For example, Adler, Wagner, and McAdams (2007) found that ED was significantly positively correlated with the coherence of individuals’ stories about their experiences in therapy, even after partialing O. As an increase in coherence of narratives over the course of treatment may correlate with outcome (e.g., Baerger & McAdams, 1999; Foa, Molnar, & Cashman, 1995), therapists might do well to assist their clients at lower stages of ED to develop more coherent narratives. Thus, ED, a relatively stable construct in the adult years, may build upon traits’ contribution to the development and maintenance of psychiatric disorders by influencing their expression in individuals at different ED stages and may suggest varying treatment strategies.
Narrative identity

There has been increasing theoretical and empirical attention paid to the relationship between narrative identity and psychotherapy beyond the influence of traits as well, leading some to declare “narrative as a nontrivial point of convergence for the therapy field” (Angus & McLeod, 2004, p. 373; see also Singer, 2005). There is also evidence that narrative themes relate to psychopathology even after partialing relevant personality traits. For example, Adler, Kissel, and McAdams (2006) found that the narrative theme of contamination was strongly related to individuals’ depression and low life satisfaction, even partialing N. Within the narrative tradition, psychotherapy has been conceived of in two ways: as a process that is fundamentally concerned with modifying people’s self-narratives (e.g., White & Epston, 1990) and also as an important source of personality development that must itself get storied and incorporated into one’s evolving sense of self (e.g., Lieblich, 2004).

The first perspective holds that, regardless of the specific content of the treatment, therapy is, in essence, about helping clients modify their self-stories. This viewpoint has led to the development of therapeutic techniques that are explicitly focused on clients’ self-narratives (e.g., White & Epston, 1990). In addition, various components of clients’ narratives have been operationalized as important process and outcome indices, independent of traditional symptom-based measures (e.g., Hayes, Beevers, Feldman, Laurenceau, & Perlman, 2005; Lysaker, Lancaster, & Lysaker, 2003). For example, Lysaker and colleagues (2003) found that the coherence of the personal narratives of adults with schizophrenia increased over the course of treatment. While certain traits might predispose people to craft certain types of stories about their lives, the themes assessed at the level of narrative identity have been shown to be correlated with certain forms of psychopathology, such as depression, after partialing traits like N (Adler et al., 2006).

From the second perspective, the experience of going to therapy is itself conceived of as an important event that must be incorporated into the client’s evolving self-story (e.g., Lieblich, 2004). In a classic work on psychotherapy, Frank (1961) suggested that weaving the “myth” of the therapeutic experience is fundamental to the individual’s continued optimal functioning once treatment has ended (p. 327). Yet there are significant individual differences in the
narration of psychotherapy and these differences correlate with variations in mental health (e.g., Adler, Wagner et al., 2007; Baerger & McAdams, 1999). For example, Adler and McAdams (2007, in press) found that individuals high in psychological well-being (compared to those low) tended to recall their therapy as the story of a victorious battle from the past, wherein a personal problem rises to become (temporarily) a fierce antagonist, only to be defeated once and for all by a reenergized self (for a related discussion of client agency, see Combs & Freedman, 2004). In doing CBT for anxiety, our experience suggests that for many patients it is important to transform a self-story of being cowardly because of the experience of intense anxiety to one of being courageous because of action in the face of anxiety. The thematic content of clients’ narratives and their evolution over treatment cannot be predicted well from their trait profiles, yet they serve an important psychological function (e.g., Frank, 1961).

**CONCLUSION**

We have argued that taking personality into account may be useful for psychotherapists and psychotherapy researchers. Specifically, individual differences in personality traits as well as personality constructs beyond traits such as ego development and narrative identity appear to have implications for such diverse aspects of psychotherapy as prognosis, treatment-seeking preferences, choice of therapeutic strategies, the focus of therapeutic interventions, and the ways in which clients regard their treatment and narrate their experiences in therapy. Clearly, many of the implications we have drawn involve some degree or other of speculation. It is just as clear to us, however, that our conjectures are imminently testable. Thus, time and empirical studies will tell which of the hypotheses generated here are valid or clinically useful and which are not.

Given that the principles of learning theory provide the conceptual foundation for behavior therapy and that much of Eysenck’s work centered around hypothesized effects of heritable traits in moderating learning processes and outcomes (e.g., Eysenck, 1967), it is certainly the case that the perspective adopted here is far more consistent with the Eysenckian/European school of behavior therapy than the school that developed in the United States in the 1950s.
(Krueger & Piasecki, 2002). A potentially interesting historical note, however, is that whereas Eysenck in many ways pioneered the modern study of traits (e.g., Eysenck, 1947), the trait-psychopathology interface (e.g., Eysenck, 1944), behavior therapy (e.g., Eysenck, 1959, 1960a), and the study of the efficacy of psychotherapy (e.g., Eysenck, 1952), we are not aware of much research conducted by Eysenck himself at the trait-psychotherapy interface. In fact, whereas a PsycINFO search yielded 663 publications authored or coauthored by Eysenck, including 360 hits using the keyword personality and another 82 using the keywords psychotherapy and behavior therapy, we are aware of only three articles or chapters in which he explicitly discussed the implications of traits for psychotherapy (Eysenck, 1960b, 1969, 1985). Regardless of the ultimate fate of many of our conjectures, we are convinced that further merging of these two research streams pioneered by Eysenck will lead to enhanced understanding and improved practice of psychotherapy.

REFERENCES


