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Research report

Can personality assessment predict future depression? A twelve-month follow-up of 631 subjects☆

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Abstract

Background: Personality assessment provides a description of a person's fundamental emotional needs and of the higher cognitive processes that modulate thoughts, feelings, and behavior. Prior studies by us examined personality and mood at the same time. Assessing personality may allow prediction of mood changes over time in a longitudinal study, as described in earlier prospective studies by Paula Clayton and others.

Method: A group of 631 adults representative of the general population completed the Temperament and Character Inventory (TCI) and Center for Epidemiological Studies depression scale (CES-D) at baseline and one year later.

Results: TCI scores at baseline accounted for gender differences in levels of depression. TCI personality scores were strongly stable (range in r=.78 to .85 for each of seven dimensions) whereas mood was only moderately stable (r=.62) over the twelve-month follow-up. Baseline personality scores (particularly high Harm Avoidance and low Self-Directedness) explained 44% of the variance in the change in depression. Baseline levels and changes in Harm Avoidance and Self-Directedness explained 52% of the variance in the change in depression at follow-up.

Limitations: The follow-up sample was representative of the target population except for slightly lower Novelty Seeking scores.

Clinical relevance: Observable personality levels strongly predict mood changes. Personality development may reduce vulnerability to future depression.

Keywords: Personality assessment; Depression; Mood changes

Under the leadership of Eli Robins and Samuel B. Guze the Department of Psychiatry at Washington University demonstrated the utility of systematic diagnosis in patient assessment and treatment (Goodwin and Guze, 1996). The work at Washington University on psychiatric diagnosis led to the adoption of explicit diagnostic criteria in the official diagnostic and statistical manual of the American Psychiatric Association in 1980 and subsequently by the World Health Organization. Ironically, the way psychiatric diagnosis is now done in practice following these official systems violates the scientific principles of diagnostic assessment that were advocated by its pioneers. In fact, there has been a steady accumulation of knowledge about diagnosis that now requires a fundamental paradigm shift as a result of the careful work on differential diagnosis at Washington University and elsewhere over the past few decades (Cloninger, 1999a, 2000a,b, 2004).

Here we will examine the observable personality variables that strongly modulate mood changes as an illustration of the way that personality assessment is a

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necessary foundation for any rigorous differential diagnosis of psychopathology. Paula Clayton and Robert Cloninger carried out a systematic follow-up and family study of 500 psychiatric outpatients in collaboration with their late colleagues, Samuel Guze, Robert Woodruff, and Ronald Martin (Clayton, 1974; Cloninger et al., 1985; Martin et al., 1985a,b; Guze et al., 1986). Paula Clayton was engaged in the assessment of the original patient series, and Cloninger carried out most of the follow-up interviews six to twelve years later. Clayton was also doing psychiatric assessments and follow-up studies of bereavement at the same time. Together these studies of bereaved subjects and psychiatric outpatients revealed much about the causes and course of depression that helped Cloninger develop a more adequate understanding of the way that personality modulates mood changes.

Eli Robins and Sam Guze taught that each patient had only one fundamental diagnosis. The presence of two or more syndromes suggested to them that the patient should be considered undiagnosed except in particular situations where the chronology was clear and predictable, such as a patient with antisocial personality disorder or panic disorder developing secondary alcoholism or secondary depression (Feighner et al., 1972). Patients with "primary" depressive disorders had no other recognized psychopathology prior to the onset of the major depressive disorder. As a consequence, patients with "primary" depressive disorders must be psychologically healthy before the onset of their first depressive episode. The Washington University investigators also observed that the first-degree relatives of patients with primary depression or panic disorder with secondary depression were less likely to have antisocial personality disorder, whereas the relatives of patients with depression secondary to somatization disorder had an increased risk of antisocial personality disorder and substance dependence (Guze et al., 1986). Essentially, differential diagnosis as done at Washington University involved assessment of multiple syndromes simultaneously to define groups of patients who were as clinically homogeneous as possible. This required careful attention to both inclusion and exclusion criteria for each diagnosis. Just having sufficient criteria to diagnose a major depression was not an adequate basis for differential diagnosis; evidence for other psychopathology prior to the onset of depression and in the family history had to be considered as possible exclusion criteria for rigorous differential diagnosis. The careful use of both inclusion and exclusion criteria at Washington University under Eli Robins, Sam Guze, and George Winokur is in marked contrast to the current practice of making multiple comorbid diagnoses based largely on heterogeneous inclusion criteria that often ignore the distinction between the primary psychiatric illness and other secondary phenomena (Winokur and Clayton, 1994). The current DSM systems do require some exclusions but these are minimal in a system with more than 300 diagnostic categories, which are often redundant and are too numerous in practice to assess in every patient. As a result, current diagnostic practice is usually unreliable because the primary diagnosis is largely determined by current presenting complaints, diagnostic biases, and subjective impressions. These impressions can be easily justified by listing loose inclusion criteria, which are actually heterogeneous in their psychological and biological basis (Cloninger, 2002b). In contrast, the original Washington University diagnostic system was based on a dozen or so categories that were each assessed in detail in every patient in order to identify the primary foundation from which other symptoms developed.

Experience with the Washington University approach led clinicians to recognize the semi-quantitative features of a clinical spectrum associated with each fundamental problem. Essentially the Washington University approach to psychiatric diagnosis required a multidimensional assessment — all patients should be systematically assessed in each of the descriptive dimensions underlying personality and psychopathology. People using this system usually continued to assume that diagnoses referred to categories of discrete diseases, but this assumption was not necessary in an empirical approach: there has never been objective evidence for such discreteness (Kendell, 1982; Cloninger, 2002b). Nevertheless, clinicians using categorical diagnoses for descriptive purposes frequently recognized partial expressions of disorders that obviously varied in severity and degree of functional impairment (Goodwin and Guze, 1996). Longitudinal research on antisocial personality became the exemplar at Washington University for distinguishing the primary diagnosis upon which other syndromes developed as secondary complications (Robins, 1966; Robins and Price, 1991; Robins et al., 1995). For example, a patient might not satisfy the full inclusion criteria for any other major diagnosis prior to a major depression, but they might be so aloof, asocial, or asexual that they would be noted to have "cluster A" traits even if they did not have definite schizophrenia. Alternatively, they might be so antisocial or impulsive that they would be noted to have "cluster B" traits even if they did not have antisocial personality disorder or somatization disorder (Cloninger, 1986). They might be so anxiety-prone, fearful, shy, or fatigable that they would be noted to have "cluster C" traits even if they did not have sufficient impairment to diagnose panic disorder or generalized anxiety disorder.

Later Cloninger systematized such observations in his description of the structure of personality in the