Only recently have personality disorders been appreciated as having substrates in the central nervous system (CNS). These disorders have been understood in psychodynamic terms and, as a result, have not been extensively studied from a neurobiologic vantage point. However, it is now increasingly clear that individual differences in CNS functioning can confer vulnerability to some of the major building blocks of the severe personality disorders, such as impulsivity and affective instability. The articles in this issue of CNS Spectrums reflect just such a dimensional approach to the personality disorders and their CNS correlates.

This recognition was reflected in an initiative by the National Institute of Mental Health (NIMH), which called for applications to translate basic scientific theories, methods, and findings to borderline personality disorder (BPD). At a recent NIMH follow-up meeting, the need for the identification of underlying brain mechanisms for core dimensions of BPD was acknowledged. One question that emerged was the continuity of these dimensions from normal variability or individual differences in personality to pathologic personality traits as seen in severe personality disorders such as BPD. This issue of CNS Spectrums focuses particularly on BPD and other impulsive personality disorders, and illustrates the importance of examining genetic and biologic mechanisms as well as psychosocial influences and their potential interaction on symptom clusters and dimensions of personality disorder.

In the first article, Vivian Mitropoulou, MA, and colleagues explore the hypotheses that personality traits may be linked with specific neurotransmitter system variability and utilize cerebrospinal fluid measures in monoamine neurotransmitters and responses to serotonergic and noradrenergic probes in relation to Cloninger’s Tridimensional Personality Questionnaire. Limited support was obtained for a relationship between the catecholamines and novelty-seeking and reward dependence, although many hypothesized relationships were insignificant. Given the limitations of measurement of neurotransmitter function, complexity of personality, and dependence of brain function on more specific circuitry rather than global neurotransmitter function, the fact that these limited relationships were found at all is noteworthy.

Emil F. Coccaro, MD, and colleagues address an index of noradrenergic function, plasma free 3-methoxy-4-hydroxyphenylglycol, in relation to a pathologic personality trait, aggression. In studies to date, pathologic traits have been correlated more with neurotransmitter dysfunction in the serotonergic and noradrenergic system than have temperamental traits in normal personality. These results are consistent with the more specific model of reduced pre-synaptic noradrenergic output coupled with excessive adrenergic receptor responsiveness, leading to the susceptibility to aggressive behaviors.

Neurobiologic variables have been considered seriously in the etiology of the personality disorders only recently, but there is a longstanding tradition of clinically generated hypotheses that early trauma may lead to later personality disorder. Linda M. Bierer, MD, and colleagues examine these hypotheses with a particular reference to BPD, which has been conceptionalized as a complex posttraumatic stress disorder. They found that a mixed personality disorder cohort reported a greater degree of emotional abuse and neglect in their childhood than comparison subjects, but that the subset of patients with BPD did not significantly differ from the rest of this cohort. Childhood abuse significantly predicted paranoid and antisocial personality disorders but only histrionic personality disorder was predicted by trauma scores, particularly sexual abuse.

The last two articles, by Daphne Simeon, MD, and Marianne Goodman, MD, and their colleagues, respectively, explore the relationship between childhood trauma and specific symptom dimensions of BPD: affective instability in the Goodman article and dissociation in the Simeon paper.

The Goodman study examined the relationship of self-reported histories of childhood trauma to measures of affective instability. Emotional abuse is the only trauma variable significantly correlated with affective instability measures. This correlation was greatly attenuated in patients with BPD raising the possibility that nontrauma-related factors may play a larger role in affective dyscontrol in individuals with BPD.

Simeon and colleagues evaluated childhood trauma and explored its relationship to dissociation, temperaments, and defensive style.

These studies suggest that the development of personality and its disorders are a complex multifactorial process, including genetic and environmental factors. Approaches to the neurobiology that emphasize pathologic traits may be more likely to identify brain behavioral associations in these populations. Symptom clusters seem not to be related so much to specific environmental traumas as they are to ongoing characteristics of the family and interpersonal environment in childhood. Bidirectional effects should also be investigated. Such of formulations may help refine our investigative approaches to the personality disorders in future studies.