Prejudice along ethnic lines is often predicated on fallacious notions of the existence of fundamental difference between groups and of uniformity within them. Be it IQ or other biological or cognitive profiles, such markers carry the danger of concretizing perceptions of difference between groups and of sameness within them. In so doing they serve to justify the perception of difference between ethnic groups where such difference does not exist. In the area of race or ethnicity, this can have particular unwanted effects of validating social inequalities.

The most masterful exponent of such ideas was the late Stephen Jay Gould who articulated his ideas most lucidly in *The Mismeasure of Man*.[2] Gould enunciated the dangers of biological determinism in the context of race.

One of Gould's criticisms of *The Bell Curve*[3] was that, in championing the idea that inequality is subserved by innate differences, it understates the role of socio-political factors in its perpetuation. Gould asked, "What argument against social change could be more effective than the claim that established orders exist as an accurate reflection of innate intellectual capacities?" [1]

Heller et al.'s[1] findings need to be considered in this light. The authors strike an encouraging note of receptivity to possible explanations when they suggest that "as yet unmeasured variables" mediate the reported association and socioeconomic disadvantage may be a potent "etiological factor." Neuropsychiatry is biased toward locating the explanation for particular cognitive findings "in the brain." In the absence of an integrative model of cognition incorporating social factors, there is a tendency to misattribute etiology in a manner which minimizes sociogenic factors. When the message is that social inequalities between ethnic groups are underpinned by innate differences, the implications may be particularly invidious.

Sociologists are at pains to stress the complexities and ambiguities surrounding notions of ethnicity.[4] A common error is to assume homogeneity within an ethnic group. The authors fail to address these points to a depth that their complexity warrants. With no guarantee that successive generations are immune to repeating the mistakes of previous ones, it is incumbent upon new generations of neuroscientists and psychiatrists to be cognizant of the issues raised by Gould.

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Lamotrigine for the Treatment of Impulsive Aggression and Affective Symptoms in a Patient with Borderline Personality Disorder Comorbid with Body Dysmorphic Disorder

To the Editor: Many psychiatric disorders are characterized by impulsive auto-hetero-aggression and violence which are manifested as suicide attempts, self-injurious behavior, or physician-directed violence or domestic violence. The consequences of these behaviors significantly increase costs as they often lead to the patient's hospitalization and/or property destruction. Borderline personality disorder and body dysmorphic disorder share some similarities such as impulsive aggression, anger, hostility, and irritability associated with moderate to severe depressive symptoms.

Pharmacological approaches to borderline personality disorder have focused mostly on the mood and impulsive aggressive domains, thus leading to the frequent use of combination treatments, mainly selective serotonin reuptake inhibitors (SSRIs), atypical antipsychotics, and mood stabilizers such as carbamazepine or valproate.[1] As for body dysmorphic disorder, SSRIs like fluvoxamine have proven to be beneficial in reducing depressive symptoms, anxiety symptoms, and anger outbursts.[2]

Lamotrigine is a mood stabilizer with antidepressant properties which has been approved for acute treatment maintenance of bipolar depression; as an anticonvulsant it might possess an anti-aggressive effect.

Our patient was a 26-year-old man diagnosed with borderline personality disorder disorder at the age of 19. He was twice hospitalized mainly due to frequent episodes of inappropriate anger or rage, aggression toward parents, and self-injurious behavior. During our interview we also diagnosed presence of body dysmorphic disorder. His major complaints were depressed mood, loss of interest, irritability, impulsive behavior, and anger outbursts. His depressive symptoms were rated moderately severe—18 points on Hamilton Depression Rating Scale (HAM-D)—while his impulsive aggression was

measured with the Overt Aggression Scale (total score = 35). We initiated treatment with lamotrigine, 200 mg/day, in order to reduce both symptom domains. We made this decision as the patient responded poorly to the treatment with SSRI and serotonin-norepinephrine reuptake inhibitor antidepressants and second generation antipsychotics.

After 4 weeks of continuous lamotrigine, 200 mg/day, we observed a 50% decrease on the HAM-D, with prominent antiagressive effectiveness (Overt Aggression Scale total score = 9). Major benefits were noted on the verbal assault subscale and irritability (4 point decrease). There were no treatment-emergent adverse events.

Our case report suggests that lamotrigine might be an appropriate bimodal-action drug, targeting simultaneously impulsive aggression and depressive symptoms. Larger controlled trials are necessary to establish its exact value and effect size for each of these two symptom clusters often present together in various Axis I and Axis II disorders.

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Persistent Neurobehavioral Signs and Symptoms Following West Nile Fever

To the Editor: Symptomatic West Nile virus infections present with fever, headache, and flu-like constitutional and gastrointestinal symptoms, and less commonly with neurological signs and symptoms. The hallmark features of neuroinvasive West Nile virus infections[1,2] are meningitis, encephalitis, and/or poliomyelitis-like acute flaccid paralysis. In addition to these hallmark features, neuroinvasive West Nile virus infections may also entail other motor (e.g., tremor, parkinsonism, myoclonus, ataxia, etc.), cognitive, and neuropsychiatric impairments.[1–4] In the absence of the hallmark features of neuroinvasive West Nile virus, however, postinfectious neurobehavioral signs and symptoms are not expected. We describe here a patient with West Nile fever (i.e., without initially apparent neuroinvasive West Nile virus infection) who developed multiple persistent postinfectious neurobehavioral disturbances.

Case Report

A 54-year-old man with Crohn's disease (on a regimen of azathioprine, 150 mg/day) presented with fever (104°F), nausea, and vomiting, but no acute neurological signs or symptoms. Serological testing confirmed acute West Nile virus infection. Approximately 3 weeks later, he developed bilateral upper extremity kinetic and rest tremors, impaired concentration and "memory," and depressive and anxious symptoms. His motor, cognitive, and emotional symptoms persisted thereafter.

Neurological examination 2 years after West Nile virus infection demonstrated transient end-gaze nystagmus and saccadic pursuits; symmetrically increased bilateral upper extremity resistance to passive manipulation, bradykinesia, 4-Hz rest tremor with a postural component, kinetic and intention tremors, and dysdiadochokinesis; decreased vibratory sense in his feet; mild ataxia; and a narrow based gait with preserved tandem gait and no retropulsion. His Mini-Mental State Examination (MMSE) was normal but his Frontal Assessment Battery score was severely abnormal (Z-score = −4.5). His performance on the Repeatable Battery for the Assessment of Neuropsychological Status, Stroop Color and Word Test, Trail Making Test, the Controlled Oral Word Association Test (COWAT), and WAIS-III Digit Symbol subtest demonstrated impairments in attention, working memory, lexical fluency, and executive function. Depressive and anxious symptoms were evident on the SCL-90-R, and the Neuropsychiatric Inventory identified anxiety and apathy/indifference. His performance of basic and instrumental activities of daily living (using Functional Independence Measure, Functional Activities Questionnaire, and Disability Rating Scale assessments) was normal but the Medical Outcomes Study 36-item Short Form Survey Instrument identified significant impairments in multiple aspects of quality of life. Two family members corroborated the subacute postinfectious onset and subsequent static nature of the patient's neurobehavioral signs and symptoms.

Comment

The neurobehavioral signs and symptoms experienced by this patient are typical of neuroinvasive West Nile virus.[2–4] However, the onset and persistence of parkinsonism, cerebellar signs, impairments in frontally mediated cognition, and emotional dysregulation following West Nile virus fever alone (i.e., without meningitis, encephalitis, and/or acute flaccid paralysis) is highly unusual and has not been reported previously.

The patient's preinfection immu-