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Dr. Buchanan and Colleagues Reply

TO THE EDITOR: Drs. de Haan and van Beveren raise two points concerning our recent article on the comparative efficacy of olanzapine and haloperidol for residual positive or negative symptoms. First, they note that in the abstract of our article we stated that "Olanzapine has limited differential benefit for either positive or negative symptoms in patients with treatment-resistant schizophrenia" (p. 124). They suggest that the proper conclusion should be that "olanzapine has no benefit over haloperidol." The qualification in our statement reflects the fact that our study is not the only one to have addressed this issue. In our Discussion section, we reviewed two studies that asserted a benefit for olanzapine in this population (1, 2). Although we believe that these studies have serious methodological flaws, they remain in contradistinction to our results. Second, Drs. de Haan and van Beveren suggest that the mean doses of haloperidol and olanzapine achieved in our study result in noncomparable D_2 receptor occupancy, with the haloperidol dose associated with increased D_2 receptor occupancy, which could potentially lead to increased extrapyramidal symptoms, dysphoria, and secondary negative symptoms. Although we agree with the theoretical concern, we note that the haloperidol-treated patients did not exhibit a mean worsening of either extrapyramidal symptoms or depressive symptoms (as measured by the Hamilton Depression Scale) nor a worsening of negative symptoms (Tables 2 and 3). The alternative concern, which we raised in our Discussion section, is that the olanzapine dose was relatively low for this population. A higher olanzapine dose may have led to increased symptomatic improvement.

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Origin of the Term "Schizophrenia"

TO THE EDITOR: The portrayal by Ernest L. Abel, Ph.D., of the 1857 "theory of degeneration" of Benedict-Augustin Morel (1) as a "parsimonious explanation for the etiology of insanity" dominating French psychiatry for "almost a century" overestimated its influence by almost half a century.

By 1911, Swiss psychiatrist Paul Eugen Bleuler had renamed Kraepelin's 1899 Latin form of Morel's earlier term *demence precoce,* "schizophrenia," emphasizing that the illness known as "dementia praecox" was not an actual dementia and did not always begin at an early age. Although the growing influence of German psychiatry in France induced negative reactions, whose main target was the work of Kraepelin, the nationalistic tone reached its apex on the French side during World War I, and only some faint traces remained for several years after 1920 (2).

Moreover, the 1950 discovery of chlorpromazine by physician Henri Laborit and the seminal work in 1952 by French psychiatrists Jean Delay and Pierre Deniker, which introduced the chemical as a treatment for schizophrenia (3), demonstrated French psychiatrists' much earlier acceptance of the Bleuler model.

Although today we can look to Morel's work as a progenitor of the current biological approach to psychiatric illnesses, Bleuler's characterization—"the group of schizophrenias"—remains the more parsimonious approach.

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