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The authors report no competing interests.

Dr. Milrod Replies

TO THE EDITOR: In clinical medicine, mechanisms of action are often opaque. Because we have yet to (may never, in fact) fully answer questions concerning mechanisms of effective treatments, should we avoid these treatments? To do so would be irresponsible, since these treatments have demonstrated efficacy from which patients should benefit. The late Gerald L. Klerman emphasized the primacy of outcome, pointing out that if a therapy lacks clinical benefit, its mechanism holds little interest (1). Our study demonstrated the efficacy of panic-focused psychodynamic psychotherapy for the treatment of panic disorder. It should not be confused with a study of mechanism, something we never claimed. The CBT model of treatment, whether scientifically sound or not, is irrelevant to the psychoanalytic model. Often, in the treatment of complex illnesses such as psychiatric disorders, more than one model can be useful—as Dr. McKay et al. note for depression.

Our study was more rigorously conducted than many oft-cited psychotherapy outcome studies, and this was demonstrated in its control and tracking of non-study interventions such as medications, which can blur apparent psychotherapy outcome, and in its maintenance of two levels of blindedness among independent evaluators (who were blinded to patient and therapist orientation) (2). Furthermore, our study has been prescient in evaluating the moderator effect of axis II pathology on panic outcome (3). No one should be blinded by ideology (4), a risk that cognitive theorists now face as much as psychoanalysts have in the past.

Without the equivalent of a pharmaceutical industry to provide financial backing, psychotherapy researchers must battle one another for ever-shrinking federal funds. A step forward for psychodynamic psychotherapy should not be a defeat for CBT. This has become as much of a "guild war" as any. In both cases, it is in our patients' best interest that the field remains open to and accepting of a range of treatment approaches.

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BK_{ca} Channel in Autism and Mental Retardation

TO THE EDITOR: We read with great interest the article by Frederic Laumonnier, Ph.D., et al. (1) in the Sept. 2006 issue of the *Journal*, as it potentially contributes to knowledge of the etiobiology of autism and may identify a novel treatment target. With the exception of two prior reports (2, 3), channel mutations have not been commonly observed in autism. Therefore, the study by Dr. Laumonnier et al. may represent one of the first on definitive mutations in a channel associated with autism. Yet, several complexities to the relationship between channel mutations, autism, and epilepsy are introduced by the data presented.

The most compelling finding of the article was the discovery and characterization of balanced translocation, which appeared to interrupt one allele of the KCNMA1 gene in the first intron in a patient with autistic disorder. The patients' parents do not carry the balanced translocation, and therefore, the fact that this translocation is *de novo* supports the notion that it may be pathogenic. Using semiquantitative reverse transcriptase-polymerase chain reaction, Dr. Laumonnier et al. showed that BK gene expression was decreased by approximately 50% in lymphoblastoid cell lines from the patient, which is consistent with the mutation leading to haploinsufficiency.

In Figure 2 of the article, the authors showed that the BK toxin, iberiotoxin (IbTx), blocked whole-cell current from the autistic patient significantly more than it did in the comparison subject, which suggests decreased activity of this receptor, presumably because of the haploinsufficiency of the genetic mutation. However, this analysis represents a somewhat ambiguous assessment of such a change, since the authors did not provide information regarding the amplitude of the