reluctance of many pregnant women to take medication influenced this decision.

Finally, antidepressants are used to treat a range of disorders in addition to depression, particularly anxiety disorders. Psychological treatments are often appropriate in these disorders and should be offered in place of antidepressants where appropriate (2).

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Dr. Payne Replies

TO THE EDITOR: I greatly appreciate the thoughtful comments expressed by Ms. Burbeck, Mr. Pilling, and Drs. Tomson and McDonald. I completely agree that babies exposed to antidepressants during pregnancy should be monitored for symptoms of withdrawal and serotonergic toxicity and psychological treatments are appropriate during both pregnancy and the postpartum period. The space limitations of my article did not allow for a fuller discussion of such treatments, but I would like to note the recent publication of another Cochran review, which specifically investigated psychological treatments for postpartum depression (1). Unlike the review of psychological treatments for the *prevention* of postpartum depression (2) discussed in my article, this review found supportive evidence for the use of psychological interventions, including cognitive behavioral therapy and interpersonal psychotherapy, in the treatment of postpartum depression. The review also emphasized the lack of methodologically strong research in this area and the need for further trials. Finally, although I agree that the use of paroxetine during pregnancy is limited by concerns over its safety as well as the difficulties many experience during withdrawal, I would like to reemphasize the point that each case—when treating women who are or will be pregnant or breast-feeding—should be considered individually. The risks and benefits of any drug used during pregnancy or breast-feeding will need to be weighed against the risks and benefits of no treatment. There may in fact be cases in which the benefits of using paroxetine will outweigh the risks for a particular individual.

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Antidepressants and Manic Symptoms

TO THE EDITOR: In the September 2007 issue of the *Journal*, Joseph F. Goldberg, M.D., et al. (1) asserted that the use of antidepressant medications among depressed patients who have some manic symptoms may cause an increase in manic symptoms at 3 months. I have several concerns about the way the data were presented and described. First, the assessment of outcome at 3 months included the Young Mania Rating Scale (2) scores, but the authors did not indicate whether any patients actually had a switch into frank hypomania or mania. Second, in Table 2, data were presented indicating that in the group of non-antidepressant-treated patients, baseline Young Mania Rating Scale scores averaged 13.0 in those patients with *no manic* symptoms. How could patients free of manic symptoms have such high Young Mania Rating Scale scores, which are actually higher than those in patients with 1 or ≥2 manic symptoms?

Third, in the body of the article, the reference to Table 2 indicated that Young Mania Rating Scale outcomes were presented for those patients with 0, 1, 2, or ≥3 manic symptoms at baseline. However, Table 2 in fact only included these outcome data for patients with 0, 1, or ≥2 symptoms at baseline. The reason I bring this up is that in Figure 3, Young Mania Rating Scale outcomes were presented separately for patients with 2 versus ≥3 symptoms (as well as 0 or 1), and it appeared

as though patients with ≥3 baseline manic symptoms actually had lower 3-month Young Mania Rating Scale scores if they were given antidepressants, leading to the appearance that for these more severely mania-ridden patients, antidepressants actually had antimanic effects! Additionally, in that same figure, it appeared that the entirety of the supposed deleterious effect of antidepressants on 3-month Young Mania Rating Scale scores was accounted for by patients with only 1 baseline manic symptom. Patients with 0 or 2 baseline symptoms did not have differential 3-month Young Mania Rating Scale scores based on antidepressant treatment status. This rather odd set of findings seems to cast doubt on the confidence of the authors' conclusions about antidepressants and manic symptom outcomes.

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Dr. Rasmussen reports no competing interests.

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Dr. Goldberg Replies

TO THE EDITOR: We appreciate the comments by Dr. Rasmussen regarding our recent study of adjunctive antidepressants plus mood stabilizers for bipolar depression with concomitant mania symptoms. However, a few clarifications appear in order.

First, we did not assess discrete switches to frank mania or hypomania because our main goal was to determine whether adjunctive antidepressants were effective for bipolar patients when accompanied by any degree of mania. The observation that such usage worsened mania was a secondary result. We reported those results as worsening Young Mania Rating Scale scores because such dimensional outcomes are more sensitive measures of change than categorical outcomes. Many clinicians feel that mood destabilization only involves discrete "switching" from one affective pole to another, but such a categorical distinction is less meaningful when patients already manifest signs of both poles. In fact, if the "switch" phenomenon were categorical rather than dimensional, then antidepressant-induced "switching" from mixed to pure mania would, by definition, involve merely the retention of mania symptoms alongside reduction of depressive symptoms. This was not seen in our study.

Second, in Table 2, among subjects with no DSM-IV-defined mania symptoms, baseline Young Mania Rating Scale scores were higher in those who were antidepressant-free than antidepressant-treated. One must remember that the Young Mania Rating Scale was designed to assess change in inpatients rather than diagnose mania. It includes many nonspecific symptoms related to agitation and aggression and assigns lower-range scores on individual items for behaviors that are not necessarily pathological (whereas DSM-IV criteria are defined as falling outside the norm). Thus, our results may simply suggest that clinicians avoided antidepressants in those with nonspecific agitation/aggression despite the absence of DSM-IV mania criteria.

Third, as noted in the editorial accompanying our article, the observed significant interaction effect between baseline mania symptoms and antidepressant use that we depicted using a box plot (Figure 3) is complex: the slopes of the lines are different within each subgroup of patients with differing numbers of mania symptoms. Because of these changing relationships, it would have been a misinterpretation of the interaction effect to assume a simple linear relationship between the number of baseline mania symptoms and Young Mania Rating Scale severity score at follow-up. Rather, the interaction effect means that in the presence of any mania symptoms at baseline, Young Mania Rating Scale scores were higher after 3 months when antidepressants were added to mood stabilizers. Furthermore, in Figure 3, it would have been an overinterpretation (in a post hoc stratification within a subgroup analysis) to assert that there was any notable antidepressant-related improvement in mania in those with more than 3 baseline manic symptoms. The changes were not meaningfully different in magnitude between the two groups; their confidence intervals greatly overlapped.

We reiterate that the main finding of our study was the lack of efficacy of antidepressants for the treatment of bipolar depression, in this case when accompanied by mania symptoms. Consistent with findings reported previously from the STEP-BD randomized comparison of mood stabilizers with or without antidepressants for pure bipolar depression, our results contradict assumptions that antidepressants effectively treat bipolar depressive symptoms.

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Prevalence and Recovery From Anorexia Nervosa

TO THE EDITOR: In the August 2007 issue of the *Journal*, Anna Keski-Rahkonen, M.D., Ph.D., et al. (1) reported substantially higher lifetime prevalence and recovery rates from anorexia nervosa than rates reported in previous studies. To reach these conclusions, the authors diagnosed their subjects retrospectively after interviewing them by telephone. The diagnosis and assessment of recovery relied on the estimation of body mass index. The authors reported values of body mass index 5.9 to 10.2 years earlier, with a precision of 0.1 kg/m^2 (Table 1, Table 2), and a rate of recovery that was almost the same at 5 out of 6 points in time (six, eight, seven, seven, seven, and two patients [Figure 1]). Thus, in six subjects who recovered at least 4.5 years before the telephone interview, body mass index increased from approximately 16 to normal in 6 months. Assuming that their height was 1.6 m and their