ample, if a subject had four visits, with HAM-D scores of 23, 10, 10, and 10, and her symptoms that met SCID criteria were 8, 1, 1, and 1, then the proportion of her visits that met SCID criteria would be 0.25. In comparison, another subject may have had HAM-D scores of 23, 20, 20, and 20, and her number of symptoms that met SCID criteria may have been 5, 4, 4, and 4. Thus, the second patient would have the same 0.25 proportion as the first. It may be problematic that these two patients would have different mean HAM-D scores (20.7 versus 13.2), yet their maximum scores and the proportion of their visits that meet SCID criteria for depression would be the same. Third, the authors used a regression model control for depression status represented by the proportion of visits that met SCID criteria. Given that the HAM-D is the widely-used tool for the assessment of depression in many studies, we suggest that it is more proper to control for the mean HAM-D score rather than the proportion of visits meeting SCID criteria to represent the degree of depression.

In summary, we feel that Dr. Suri et al. may have potentially underestimated the differences in the two depressed groups. Nevertheless, the study has shown that antidepressants might play an important role in increasing the risks at birth.

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TSUO-HUNG LAN, M.D.
BO-JIAN WU, M.D.
HSIEN-JANE CHIU, M.D.
TSUNG-MING HU, M.D.
YUNG-CHANG TUAN, M.D.
Yuli Township, Hualien County, Taiwan

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Dr. Suri Replies

TO THE EDITOR: Drs. Lan, Wu, Chiu, Hu, and Tuan raise important points that we appreciate and would like to address. We agree that the question of the effect of prenatal depression on birth outcome needs to be further studied. In fact, we initially hypothesized that active depression during pregnancy would be associated with an increased risk of preterm birth. Therefore, we were also surprised to find that depressive symptoms *per se* did not seem to influence outcome. We selected the proportion of monthly visits in which subjects met SCID criteria for depression and maximum scores on the HAM-D in order to prospectively determine the effect of the 1) time spent in a major depressive episode during pregnancy and 2) severity of depressive symptoms on outcome measures. Dr. Lan et al. raise concern that our assessment of depression may not fully reveal the extent of prenatal maternal depression and suggest that the average HAM-D score across pregnancy may more adequately reflect mood. In our study, the mean 28-item HAM-D scores were 15.1 for Group 1 (depressed subjects receiving medication), 15.5 for Group 2 (depressed subjects not receiving medication), and 8.8 for Group 3 (comparison subjects). In re-examining the data with mean HAM-D scores used in a hierarchical linear regression model, as Dr. Lan et al. suggest, we did not find that the overall model significantly predicted gestational age at birth (R_2=0.07; F=1.25, df=5, 83, p=0.29). The individual contribution of depression as measured by the average HAM-D scores over all time points, after controlling for other risk factors, was not significant (change in R_2=0.01; F=1.0, df=1, 83, p=0.32). Thus, in our study depression (which was defined as average depressive symptoms across pregnancy) was not associated with lower gestational age at birth or increased risk of preterm birth.

Dr. Lan et al. question the clinical significance of a mean gestational age for antidepressant-exposed subjects of 38.5 weeks compared with 39.4 to 39.7 weeks for untreated and healthy comparison subjects, respectively. While this mean difference of 1 week is quite large by conventional standards of effect size (Cohen's d=0.8), we agree with Dr. Lan et al. that the relevance for infant outcome is not clearly known. However, our study also found that the rate of preterm birth, defined as less than 37 weeks of gestation and a known cause of neonatal morbidity (1–3), was almost three times greater in the antidepressant-treated group (14.3% versus 0 and 5.3% for the other two groups, p=0.05). We feel that such a high rate of prematurity in medically healthy subjects with good prenatal care and few preterm risk factors is of concern.

Our findings suggest that prenatal antidepressant use is not neutral, and if antidepressants *per se* or antidepressants in interaction with depression impact the timing of delivery, this should be known for informed decision making. Further investigation in this important area is clearly necessary, and our research group plans to conduct larger-scale studies.

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RITA SURI, M.D.
Los Angeles, Calif.

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Psychotic Symptoms Following Buprenorphine Withdrawal

TO THE EDITOR: Buprenorphine reduces the risk of signs and symptoms of opioid withdrawal by its partial agonist property and is thought to potentially lead to a ceiling effect, particularly with respect to euphoria and respiratory depression (1). Delirium but not psychotic symptoms has been reported following buprenorphine discontinuation (2). We present a case of a patient who developed psychotic symptoms following buprenorphine withdrawal that disappeared only after its reintroduction.