consequences. Drs. Fishbain and Gallagher cite recent articles that they believe refute our thesis. We do not feel that this is the case.

Multidisciplinary pain clinics did not report that they were formed to prevent addiction, to our knowledge. Rather, the early clinics postulated that behavioral issues contributed to chronic pain complaints, which often seemed out of proportion to medical pathology. By not rewarding these complaints, either socially or with medication, they reported many treatment successes (1, 2).

The reference cited (Lusher et al.) does not describe cases of pseudoaddiction, but rather cases "at risk of pseudoaddiction." The cases selected were not opioid pain medication-seeking chronic pain patients, but rather patients undertreated for acute sickle-cell pain. The article's introduction and discussion reveals how problematic the concept of pseudoaddiction is.

The articles by Eisenberg et al., Furlan et al., and Devulder et al. review studies of opioid treatment for chronic pain. They reveal that the studies have been of short-term treatment, mostly funded by pharmaceutical companies, and the magnitude of pain relief, when present, was often small and of questionable clinical meaningfulness. Subjects in the trials may not be representative of the broader chronic pain population seen in clinical practice. They conclude that adequately designed trials have not been done and long-term studies are needed.

In a recent, detailed review of the pertinent literature, Clark et al. (3) conclude that "there remains no well-controlled, empirically sound evidence supporting the long-term effectiveness of opioid therapy for chronic noncancer pain."

In summary, reasonably well-designed studies of opioid treatment of chronic pain are almost all short-term treatment studies, usually using relatively low doses of opioids. We believe the current state of the evidence is as described by Ballantyne and Mao (4), that long-term, high-dose opioids for chronic pain have not been shown to be safe or effective. The case we presented details many of the problems associated with long-term prescription opioid intake.

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JON STRELTZER, M.D.
Honolulu, Hawaii
LUTHER JOHANSEN, M.D.
Landstuhl, Germany

Oxcarbazepine and Pediatric Bipolar Disorder

TO THE EDITOR: In reference to the article by Karen Dineen Wagner, M.D., Ph.D., et al. (1), two issues need clarification regarding 1) generalizability of the study results and 2) issues of comorbidity and combination therapy.

First, in Table 1, the authors reported that the mean number of manic/hypomanic episodes ranged between 15.1–17.1 in the past year of the illness in the groups randomly assigned to oxcarbazepine and placebo, respectively. This seems to be in significant variance with the cohort recruited for the basic descriptive psychopathology study by Geller et al. (2), where, using arguably the best available state-of-the-art diagnostic methods and operationalized definitions of episodes and "cycling," the index episode of mania at intake had a mean duration of 3.5 years, and there were no subjects characterized as "rapid cyclers" (four or more mood episodes per year), but the vast majority of subjects had ultrarapid cycling or ultradian cycling within a single prolonged episode. The average age of the study subjects is not dissimilar, and the male predominance in the two studies is quite similar. Are we left to conclude that the study subjects are in fact composed of different phenotypes of mania or bipolar disorder? Or is there a difference in the way terms such as "episodes" or "cycles" are being used, reflecting disagreement between research groups? Do the authors have suggestions regarding terminology so that interested readers can make informed comparisons between studies regarding the description of basic research subject characteristics?

Second, it is noted that about one-third of subjects in each group remained on "stable stimulant therapy" from screening through the end of the study. Was any consideration given to a concern that stimulant therapy may have been making the mood episode worse or more unstable? If so, why, as a preliminary step, weren't subjects required to discontinue from stimulant therapy prior to randomization to observe the effect of this intervention on the overall mood state? Many clinicians believe that safe and effective use of psychostimulant therapy in pediatric patients with comorbid ADHD and mania requires effective mood stabilization prior to use of stimulant therapy (3). Could the authors review their data and report on whether concurrent treatment with stimulants affected baseline ratings of psychopathology or had a predictive, moderating, or mediating effect on the data through the course of the trial?

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BRUCE WASLICK, M.D.
Springfield, Mass.

Dr. Waslick has received research support in the last 3 years from Eli Lilly and Co. and Johnson and Johnson.