We are now studying the clinical and therapeutic features of BMS comorbid with major depressive disorder, because we hypothesize that in these cases BMS has a different course and prognosis. Our preliminary impression is also that in these patients the treatment of BMS is much more difficult. We did not find a relationship between previous or ongoing treatment with antidepressants and onset of BMS, but we now believe the issue merits investigation in light of the interesting report by Levenson.

*The authors report no financial affiliation or other relationship relevant to the subject matter of this letter.*

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## Nutritional Approach to Bipolar Disorder

**Sir:** Kaplan and colleagues[1] are to be commended for their pioneering nutritional approach to treating bipolar disorder. Their clinical report described the open-label trials of 14 adults with bipolar disorder who were treated with E.M.Power+ (EMP), a mixture of essential minerals, vitamins, and other nutrients, developed by David L. Hardy and Anthony F. Stephan, which is marketed by Evince International. While perhaps startling initially, this novel treatment approach appears to offer substantial benefit. Popper[2] has briefly described successful clinical use in some cases, and I report here on my own experience with this same nutrient supplement.

Impressed by a striking response in a patient who learned of this supplement on the Internet, I began to discuss this option with other patients in my private clinical practice. After discussing alternative available treatments, the nature of this new approach, and the lack of controlled data regarding its use, I gave some treatment-resistant patients the option to try EMP under careful observation. I have now worked with EMP in treating 19 adults (mean age = 38 years; range, 18–68) with DSM-IV-TR bipolar I (N = 14) or bipolar II (N = 5) disorder, who were followed for a mean of 13 months (range, 5–21). At the time of starting EMP, 16 patients were already receiving pharmacotherapy (mean = 2.7 psychiatric medications). After gaining some experience in using EMP, I elected to start 3 unmedicated (at the time; not medication-naive) patients on this supplement.

Following the usage described by Hardy and Stephan[3] for acute phase treatment of adults, the patients were started on 32 EMP capsules daily (taken as 8 q.i.d.). Most patients experienced mild transient gastrointestinal symptoms, including nausea (6 patients), loose bowels or diarrhea (7 patients), burning stomach pain (2 patients), and stomach ache (1 patient). Most patients experienced nausea if they took EMP without food. One patient developed apparent moderate gastritis with stomach ache that responded well to standard medical treatment. Two patients described mild transient headaches. Two patients switched from depressed mood to mild hypomania.

By clinical global estimate, 12 of the 19 patients showed marked clinical improvement, 3 showed moderate improvement, and 1 showed mild improvement. Thirteen patients (10 marked responders, 3 moderate responders) were able to completely discontinue psychiatric medications over a mean of 5.2 weeks (range, 3–10 weeks) and remain stable on EMP alone. Of the 12 showing marked improvement, 10 have remained on EMP (current follow-up mean length = 13 months; range, 5–21). One of the 3 moderate responders has also continued on EMP, so that 11 of 19 patients have chosen to remain on EMP rather than psychiatric medications.

Of the other 8 patients, 1 was lost to follow-up. Four discontinued EMP because of gastrointestinal problems. Three had recurrent symptoms, stopped EMP, and resumed psychiatric medication.

It is clear that the effectiveness and safety of EMP remain to be established in controlled trials, but this approach does appear to represent an exciting potential direction for new research in bipolar disorder.

*Dr. Simmons reports no financial or other relationships relevant to the subject matter of this letter.*

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## Drs. Simpson and Kaplan Reply

**Sir:** Dr. Simmons' observations confirm our report[1] that a micronutrient treatment has therapeutic effects in bipolar effects in bipolar patients, a finding supported also by Popper's clinical experience.[2] With a third observer describing a high response rate, this nutritional approach might begin to take on increasing credibility, but it is important to note that no controlled trials have been reported yet.

The conciseness of Dr. Simmons' report prevented his providing details of the transition from conventional medications to this micronutrient treatment, but the prior reports describe interactions between micronutrients and conventional psychiatric medications that are unexpectedly strong and significantly complicate the clinical management of drug-treated patients. We support Popper's advice[2] to physicians against use of E.M.Power+ in patients currently taking psychiatric medications, unless they have solid and ongoing consultation with an experienced advisor. Instead, physicians who are inexperienced in the use of micronutrient treatment would be wise to restrict its use to unmedicated patients.

While the accumulation of similar anecdotal observations from multiple clinicians should counter initial skepticism, con-

trolled studies are needed to clarify whether micronutrient treatment represents an important new direction for bipolar research.

*The open-label trial[1] was supported in part by the Alberta Children's Hospital Foundation and the Alberta Science and Research Authority, Edmonton, Alberta, Canada; and Evince International, Farmington, Utah (who provided the E.M.Power+ supplement free of charge).*

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## A Case Report of Olanzapine-Induced Fecal Incontinence

**Sir:** Fecal incontinence is a socially devastating and embarrassing condition. We report a case of primary insomnia that did not respond to various anxiolytics and sedative drugs. Eventually, olanzapine was added to the patient's regimen of minor tranquilizers. Although the patient showed improvement in sleep duration, he developed fecal incontinence. Withdrawal of olanzapine resulted in complete recovery from the incontinence.

***Case report.*** Mr. A, a 65-year-old man, presented with a history of primary insomnia (DSM-IV criteria) of 20 years' duration. The patient claimed that he would sleep for only 1 to 2 hours per night, and, in the daytime, due to lack of sleep, he would feel restless, irritable, and anxious and had poor concentration and decreased memory. During a 5-year period, he was treated with various anxiolytic and sedative drugs with no improvement in sleep. In July 2001, he was put on treatment with lorazepam, 2 mg, and zolpidem, 10 mg, at night for 15 days. Due to lack of response, olanzapine, 2.5 mg, was added at nighttime.

With this combination, Mr. A's total sleep time was increased from 2 hours to 4 hours with no daytime symptoms, but he noticed passage of stool in his clothes without being able to control the motion before reaching the toilet. This fecal incontinence would occur mostly in the morning, even after the patient had attended to proper toilet activities. The frequency of incontinence varied from 1 to 3 times per day, and it was so obvious that others could notice the patient's soiled clothes. The patient continued the same drug regimen for 20 days and continued to have fecal incontinence during this period.

In August 2001, during follow-up, oral olanzapine treatment was stopped. The next day, Mr. A observed complete recovery of fecal incontinence. He was seen by a gastroenterologist on the second day after stopping olanzapine to rule out any organic cause for the fecal incontinence. The findings of a per rectal examination were normal. No rectal prolapse was found, and anal sphincter tone was normal. No hemorrhoids or excoriations were found. A sigmoidoscopic examination showed normal rectal and sigmoid colon. A laboratory examination also did not suggest organic lesions as a cause of the incontinence. Mr. A had no history of urinary incontinence and was not suffering from any medical or neurologic disorder.

The literature contains reports of clozapine- and olanzapine-induced urinary incontinence that were treated successfully by using an α-adrenergic agonist (ephedrine).[1,2] To our knowledge, this case report is the first in which fecal incontinence was observed in association with olanzapine treatment. The specific mechanism of incontinence in this case is difficult to determine. The internal anal sphincter receives a stimulatory adrenergic innervation. A previous study[3] showed that abnormalities in the adrenergic innervation of the internal anal sphincter were seen in cases of idiopathic fecal incontinence. Recent research has successfully demonstrated the feasibility of an adrenergic agonist (topical phenylephrine) in raising resting anal tone in patients with fecal incontinence.[4] Olanzapine also possesses significant α-adrenergic antagonist effects,[5] which may be a possible explanation for the occurrence of fecal incontinence.

One can argue that the combined effect of olanzapine and other sedative drugs could have caused fecal incontinence, but complete recovery from incontinence following withdrawal of olanzapine is sufficient evidence to document that olanzapine can cause fecal incontinence. Our case would have been more convincing had we reexposed the patient to experimentally prove the point, but we believed this approach to be unethical.

*The authors report no financial affiliation or other relationship relevant to the subject matter of this letter.*

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## Adjunctive Quetiapine Treatment of the Polydipsia, Intermittent Hyponatremia, and Psychosis Syndrome: A Case Report

**Sir:** Excessive fluid intake by psychotic patients can produce significant morbidity and possibly death due to water intoxication and hyponatremia. In patients with chronic schizophrenia, the prevalence of polydipsia is estimated between 6% and 20%.[1] In a subset of these patients, fluid intake overwhelms the kidney's normal excretory capacity and produces symptomatic, dilutional hyponatremia, known as the *polydipsia, intermittent hyponatremia, and psychosis syndrome* (PIP).[2] The symptoms of PIP range from mild cognitive deficits to seizures, coma, and death. While the pathophysiology is poorly under-