

## Protecting Psychiatrists' Reputations on the Internet

TO THE EDITOR: The reputation of physicians is now at risk on the Internet. Dr. Gabbard's clinical case conference in the May issue (1) provides a striking example. A resident providing good clinical care seems helpless to protect his professional reputation in the face of critical web postings by the patient's mother, becoming a victim of "the Internet era." Dr. Gabbard suggests that guidelines on "how to respond to [such] attacks should be developed." We would like to offer some ideas about how to begin that process.

Although there are services (e.g., [emerit.biz](http://emerit.biz) [2]) that offer to help doctors establish and protect their reputation by being proactive, these entities apparently have no effective remedy against negative postings. Indeed, one web site has a "wall of shame" that lists physicians who have tried to prevent postings of negative comments (3). Sites are protected by federal law against suits for defamation, and anecdotal evidence indicates that physicians who initiate legal action against the posting individual are likely to make matters worse.

It seems to us that this is a problem that requires some institutional remedy; self-help is apparently unavailing. What can the aggrieved psychiatrist ethically do given the constraints of confidentiality? Confidentiality is waived when ethics complaints or malpractice suits are initiated by patients; psychiatrists have the right to defend themselves. Perhaps an attack on the Internet might be considered to constitute a "limited waiver." As in this case, it was ethically appropriate, even without consent or a waiver, to have a clinical case conference and to publish what we take to be a disguised (unidentifiable) account. We suggest the creation of an Internet ombudsman—a mental health professional not involved in the care of the patient or the family. An aggrieved psychiatrist acting under the partial waiver and the accepted practice of consulting a colleague would identify the derogatory posting. The ombudsman would review the patient's care and the posting and when appropriate would go to the web site and post a limited response (e.g., "I have reviewed this posting in my capacity as ombudsman [for the clinic, for the hospital, for the medical school, for the District Branch of APA] and am of the opinion that this posting misrepresents the quality of the clinical care provided by the doctor." In the Internet age, it may well become necessary for every organized mental health setting to have a page on their website to which the ombudsman could refer those accessing the derogatory posting for further information about the psychiatrist and the provision of treatment.

In this case report, the postings included "antipsychiatry" material. In responding to these allegations, an institutional site might do a service not only for aggrieved psychiatrists but for the entire profession. These are tentative suggestions, but one thing is clear: one cannot deal with the challenges of the Internet without becoming part of it.

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## Response to Stone and Appelbaum Letter

TO THE EDITOR: I welcome the thoughtful suggestion by my two esteemed colleagues, Drs. Stone and Appelbaum. They note that we need to develop responses to patients' Internet postings designed to tarnish the reputation of psychiatrists, and their suggestion of an ombudsman is one systematic approach to addressing the problem. Drs. Stone and Appelbaum have started a dialogue that needs to continue in the profession of psychiatry. While their approach should be seriously debated, I would argue that it has a drawback that may be unavoidable. Those who file complaints and the public in general might well be skeptical of the objectivity and fairness of the ombudsman, whom they would see as representing a veiled attempt to silence or dismiss patient complaints. Moreover, it might paradoxically bring greater attention to the original complaint and serve to publicize the negative comments about the psychiatrist to a wider audience of readers. Perhaps we can trust our existing professional networks as the principal source of our reputations and ignore the periodic random attacks from the web until further alternatives are considered.

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## Celiac Disease and Schizophrenia

TO THE EDITOR: In the June issue, Karlsson et al. (1) report an association between high levels of antigliadin immunoglobulin (Ig) G in maternal circulation and elevated risk of nonaffective psychosis in offspring. The authors propose several possible mechanisms to explain this association, and I wish to suggest an alternative explanation. Because gliadin must cross the intestinal epithelium to evoke IgG antigliadin antibodies, greater intestinal permeability, both paracellular and transcellular, has been hypothesized as an early event in the development of celiac disease (2). Estrogens are described as playing a key role in the development and maintenance of the intestinal barrier (3). Bisphenol-A (BPA), an estrogenic endocrine disruptor, can prematurely and permanently close the barrier in perinatally exposed female but not male rats (3). As some degree of permeability is required for maturation of the immune system through the development of tolerance, the perinatally BPA-exposed female rat, lacking immunological tolerance,

develops enhanced colonic inflammatory responses in adulthood (3). This would set the stage so that when the perinatally BPA-exposed female rat becomes pregnant, the pregnancy may be marked by enhanced inflammation. Paradoxically, estrogenic exposure may have anti-inflammatory effects in the exposed adult, but inappropriate estrogen exposure may have pro-inflammatory effects in the perinatally exposed offspring. These effects were observed at levels of BPA exposure previously believed to be too low for observed adverse effects in humans (3).

I have proposed elsewhere an estrogenic endocrine disruption theory of schizophrenia, in which inappropriate dosage, timing, or duration of prenatal estrogen exposure causes schizophrenia (4, 5). Within this theoretical framework, inappropriate estrogen exposure occurring in the brain could also be occurring in the colon so that an association of celiac disease or some other inflammation and schizophrenia may be observable not from a genetic link per se but rather a transgenerational effect of prenatal estrogen exposure.

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## Alternative Interpretation for the Early Detection of Psychosis Study

TO THE EDITOR: In the April issue, the Treatment and Intervention in Psychosis (TIPS) early-detection study reports 10-year results in a manner that overstates the impact of reducing the duration of untreated psychosis (1). The authors dismissed a 50% increase in hospitalization in the treatment group after 5 years as the result of regional policy differences. They did not describe the policy differences or analyze the effects of this impressive confound on the small difference in symptoms, instead claiming to have demonstrated “positive effects on clinical and functional status” (2, 3). They omit hospitalization results altogether at 10 years, despite this being by far the most impressive result at 5 years (1).

Perhaps because at 5 years the researchers reported a nonsignificant advantage in remission for the control group (2), at 10 years they introduce a new recovery metric, based largely on work function, which showed a significant advantage for the treatment group (1). Although they acknowledge a significant attrition bias by 10 years, they do not report that at 5 years there was no difference in work function, or suggest how reducing the duration of untreated psychosis at baseline would not improve work function at 5 years but double work function at 10 years.

The authors reported that the control group achieved independent living significantly more often at the 10-year mark, but dismiss this evidence of worse function in the treatment group, suggesting that independent living is not evidence of recovery because it is not included in the new metric. They do not analyze the possibility that failure to achieve independent living is evidence of poor function (1).

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## Response to Amos Letter

TO THE EDITOR: Dr. Amos raises several points of criticism regarding the TIPS study and our interpretation of the data, as he did previously (1) in response to abstracts from our group. We thank the *Journal* for the opportunity to respond.

First, Dr. Amos points out that patients from the health care area practicing early detection had significantly higher rates of hospitalization at the 5-year follow-up, and he is critical of the fact that we did not thoroughly investigate this possible confounder. This is a valid concern; however, he seems to miss the point that it is the group of patients *not* in symptom remission (a prerequisite of recovery) who received more inpatient care in the early-detection area. For recovered patients, there was no difference between early and usual detection. Knowing that more hospital time did not lead to better recovery, hospitalization cannot be a confounder.

Second, Dr. Amos questions the finding that while there apparently were no differences in work function at the 5-year follow-up, the early-detection patients had double the chance of full-time employment at 10 years. He goes on to imply that we might have chosen a new measure of “recovery” out of convenience, having made sure that this measure would yield us more favorable results. At 5 years, we used “working at least