An institutional review board approved this study, and the subjects provided written informed consent. Six subjects (50% women, mean age=29.3 years, SD=11.9) were first treated with fluoxetine for ≥12 weeks (mean dose=70.0 mg/day, SD=11.0). Olanzapine was then added to fluoxetine (the fluoxetine dose was unchanged) if the fluoxetine response was inadequate (i.e., the subjects still met DSM-IV body dysmorphic disorder criteria, had a body dysmorphic disorder score ≥20 on the Yale-Brown Obsessive Compulsive Scale Modified for Body Dysmorphic Disorder (4), and were no more than minimally improved on the Clinical Global Impression scale [CGI]). Two subjects received olanzapine under double-blind conditions; four were treated openly with olanzapine after failing to respond to placebo. Exclusion criteria were standard for efficacy studies. The subjects took no other psychotropic medications. Olanzapine was begun at 2.5 mg/day, with an attempt to raise the dose to 15 mg/day over 8 weeks if it was tolerated.

With olanzapine treatment, body dysmorphic disorder symptoms on the CGI were minimally improved in two patients and unchanged in four. Olanzapine was received for a mean duration of 5.3 weeks (SD=3.1); the mean endpoint dose was 4.6 mg/day (SD=3.3). Two patients experienced fatigue, and three gained weight.

These results must be considered preliminary because of the small sample size. Nonetheless, they are consistent with the only placebo-controlled SRI augmentation study in body dysmorphic disorder, to my knowledge, in which a typical neuroleptic (pimozide) was not efficacious (5). In what is to my knowledge the only report of SRI augmentation with atypical antipsychotics in body dysmorphic disorder (a chart review study, reference 3), only two of nine subjects responded, although the effect size was large. In one case report (6), olanzapine monotherapy was efficacious. These somewhat mixed results underscore the need for further studies of atypical antipsychotics as augmentation agents and monotherapy. Because clinical experience suggests that atypical antipsychotics can be very helpful for associated anxiety and agitation, this also warrants investigation.

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Pilocarpine Treatment of Xerostomia Induced by Psychoactive Medications

TO THE EDITOR: Dry mouth (xerostomia) is a frequent complication of psychoactive medications with antimuscarinic and anticholinergic side effects. The lack of saliva is annoying to patients, impairs their ability to masticate and digest food, and is a potential source of dental morbidity, including increased risk for caries and oral infection. Pilocarpine is a cholinergic muscarinic agonist. It has been used to treat xerostomia induced in cancer patients by head and neck radiotherapy (1). It has recently been found to be effective in doses of 20 mg/day in a randomized, placebo-controlled dose-adjustment study in the treatment of dry mouth and dry eyes in patients with Sjogren's syndrome (2). It has been used to treat dry mouth as a complication of opioid treatment (3). Toxicity has been infrequently reported (4). However, it is contraindicated in patients with angle-closure glaucoma.

We have empirically used pilocarpine in doses of 10–30 mg/day, divided into dosing of two or three times a day. We have used it with our acute psychiatric inpatients, ages 20–69, who complained of dry mouth after they had been started on psychoactive medication. These included atypical antipsychotic agents, particularly clozapine and olanzapine; anticholinergic agents, primarily benztropine; and antidepressants, particularly tricyclic antidepressants and mirtazapine. Substantial relief of dry mouth was achieved in most patients. Side effects were mainly sweating and increased urination. We did not observe any adverse impact on psychiatric symptoms. The patients were generally pleased that their dry mouth symptoms responded rapidly, usually within 1 day, to pilocarpine treatment. Further investigation into the use of pilocarpine for the treatment of xerostomia induced by psychoactive medication seems warranted.

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