Quetiapine/Ritonavir: Clinically Significant Interaction

Drug interactions are common in patients receiving highly active antiretroviral therapy (HAART) and are of particular concern with the protease inhibitor ritonavir. The package insert for quetiapine includes a warning about potential interactions with protease inhibitors, but does not mention any specific agent. Quetiapine is metabolized by the CYP3A4 pathway. Because ritonavir and to a lesser extent atazanavir inhibit CYP3A4, quetiapine concentrations can dramatically increase with potentially toxic effects.

Two patients experienced symptoms of quetiapine toxicity while receiving a concomitant ritonavir-atazanavir regimen. The first patient, a 57-year-old male with HIV, had been treated with a regimen that included ritonavir-atazanavir for more than 1 year. He had also been treated with psychotropics for bipolar disorder, but experienced tardive dyskinesia (TD). His antipsychotic was replaced with quetiapine because it has less potential to induce TD. Within 6 months the patient had gained 50 lbs and had significant increases in appetite and serum glucose. Ritonavir-atazanavir was replaced with fosamprenavir, and quetiapine was stopped. One month later the patient had lost 9 lbs and his appetite and serum glucose were controlled. The second patient, a 32-year-old female with HIV, anxiety, and a history of IV drug use, presented to a methadone clinic confused and increasingly sedated within days of adding a HAART regimen that included ritonavir-atazanavir to psychotropics including quetiapine. Her mental status improved after she stopped quetiapine while continuing all other medications.

Psychiatric comorbidity is common in patients with HIV, and coadministration of atypical antipsychotics with ritonavir-based HAART regimens is often prescribed. Particular care should be taken with quetiapine and aripiprazole, which are mainly metabolized primarily via the CYP3A4 pathway. Because some other antipsychotics are metabolized via this pathway, they could also potentially interact with ritonavir-boostered regimens.


**Drug Trade Names:**
- aripiprazole—Abilify
- atazanavir—Reyataz
- fosamprenavir—Lexiva
- quetiapine—Seroquel
- ritonavir—Norvir

**PSYCHIATRY DRUG ALERTS** (ISSN 0894-4873) is published monthly by M.J. Powers & Co. Publishers, 65 Madison Ave., Morristown, NJ 07960. Telephone 973-898-1200. e-mail: psych@alertpubs.com. Periodical-class postage is paid at Morristown, NJ, and at additional mailing offices. POSTMASTER: Send address changes to Psychiatry Drug Alerts, 65 Madison Ave., Morristown, NJ 07960. © 2009 by M.J. Powers & Co. Publishers. Written permission from M.J. Powers & Co. is required to reproduce material from this publication. Subscription $89 a year in the U.S.; $97.50 Canada; $107.50 elsewhere; $141 institutional. Back issues and single copies are available for $10.00 each, prepaid. Subscribers may enroll in the 12-month CME program for $77.00 per year.
Fatty Acid Augmentation of Sertraline

Low levels of omega-3 fatty acids have been associated with depression and with mortality in heart disease. High dietary intake or use of supplements had been shown to improve depression and to reduce cardiac mortality in high-risk patients. The present study examined the value of adding fatty acids to sertraline (Zoloft) in patients with depression and heart disease.¹

Methods: Patients with documented coronary heart disease (≥50% stenosis in a major coronary artery, a history of revascularization, or hospitalization for an acute coronary syndrome) recruited from cardiology practices completed a standardized self-report of depressive symptoms. Those who reported symptoms underwent a structured clinical interview. Patients who met DSM-IV criteria for a major depressive episode without psychiatric comorbidity (n=178) received 25 mg/day sertraline for 2 weeks. The 122 patients who continued to meet criteria for major depression were then randomized to 10 weeks of double blind 50 mg/day sertraline plus 2 g/day omega-3 fatty acids or sertraline plus placebo. Fatty acid capsules contained 930 mg eicosapentaenoic acid (EPA) and 750 mg docosahexaenoic aid (DHA). About two-thirds of patients had a previous history of depression, and severity of cardiac disease did not differ between the groups. Blood was drawn before randomization and at 10 weeks to quantify fatty acid levels, and the Beck Depression Inventory (BDI) was the primary outcome measure.

Results: Despite increased fatty acid blood levels, the augmentation group did not have better depression outcomes than the sertraline monotherapy group. Depressive symptoms improved in both groups to a comparable degree. BDI scores decreased from 29 to 15 with sertraline plus placebo and from 28 to 16 with fatty acid augmentation. Rates of depression response (nearly 50%) and remission (about 28%) did not differ between the groups. Stomach upset was significantly more common with placebo.

Discussion: These results contradict 2 previous studies that found omega-3 supplements significantly improved antidepressant efficacy.²,³ It is possible that a higher ratio of EPA to DHA in the supplements or a higher sertraline dose could have been more effective.

Study Rating—17 (100%): This study met all criteria for a randomized trial.


Adjunctive Aripiprazole for Schizophrenia

A multicenter manufacturer-sponsored study found adding aripiprazole did not benefit patients with incomplete quetiapine or risperidone response. Because it has a unique receptor profile, it had been hypothesized that aripiprazole might augment other atypical antipsychotic efficacy.

Methods: Outpatients (n=323) aged ≥18 years with chronic schizophrenia or schizoaffective disorder who had been receiving either risperidone or quetiapine monotherapy for at least 4 weeks but continued to experience at least moderately-severe symptoms were enrolled in the double-blind trial. Participants received 16 weeks of randomized adjunctive aripiprazole at 2–15 mg/day (mean, 10 mg/day) or adjunctive placebo. Change in Positive and Negative Syndrome Scale (PANSS) score was the primary outcome.

Results: At 16 weeks there was no significant difference between the adjunctive aripiprazole and placebo groups in PANSS total score reduction. Treatment response (≥20% decrease in
PANSS score or a Clinical Global Impression-Improvement rating of much improved or very much improved) occurred in 41% of each treatment group. Secondary outcomes including depressive symptoms, fatigue, cognition, and well-being also did not differ between the groups.

**Discussion:** The large placebo response in this study could suggest that patients had not been treated with the initial antipsychotic long enough to attain maximal benefits, thus obscuring a positive effect of aripiprazole. In addition, aripiprazole dosages may have been too low.

**Study Rating—17 (100%):** This study met all criteria for a controlled trial.

Kane J, Correll C, Goff D, Kirkpatrick B, et al: A multicenter, randomized, double-blind, placebo-controlled, 16-week study of adjunctive aripiprazole for schizophrenia or schizoaffective disorder inadequately treated with quetiapine or risperidone monotherapy. *Journal of Clinical Psychiatry* 2009;70 (October):1348–1357. From The Zucker Hillside Hospital, Glen Oaks, N.Y.; and other institutions. **Funded by Bristol-Myers Squibb; and Otsuka Pharmaceutical Co. Most of the study authors disclosed commercial relationships that could pose conflicts of interest.**

**Drug Trade Names:**
- aripiprazole—*Abilify*
- quetiapine—*Seroquel*
- risperidone—*Risperdal*

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**Citalopram and Ziprasidone in Pregnancy**

Both citalopram and ziprasidone are labeled as Pregnancy Category C because animal studies have shown adverse fetal effects and there are no adequate human studies.\(^1\) For category C drugs, the potential benefits may warrant use despite potential risks. There is some evidence for the use of citalopram in pregnancy, but this appears to be the first case of continued ziprasidone use during pregnancy and lactation.\(^2\)

A 26-year-old woman with psychotic depression and posttraumatic stress disorder had been successfully treated with 40 mg/day ziprasidone and 60 mg/day citalopram for several months before and then throughout pregnancy. At 39 weeks, she delivered a healthy 6-lb infant with no apparent physical or neurological complications. The infant was breastfed from birth to 6 months while the mother continued psychotropic therapy. Periodic evaluations by a pediatrician found no untoward drug effects or withdrawal symptoms up to 18 weeks. At 6 months, the infant was judged healthy with normal growth and development.

\(^1\)Facts and Comparisons Online 4.0. Available at http://online.factsandcomparisons.com.

**Drug Trade Names:**
- citalopram—*Celexa*
- ziprasidone—*Geodon*

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**Treating Severe Personality Disorders**

Data on pharmacotherapy of severe personality disorders is limited and the results have been inconsistent. A Cochrane review of studies conducted before 2002 did not find good evidence for pharmacologic treatment of borderline personality disorder because the number of included studies was small and there were few intervention options. A new meta-analysis included more recent studies and found symptom-targeted pharmacotherapy can be beneficial.

**Methods:** The study authors identified 35 randomized placebo controlled trials of pharmacotherapy in patients with borderline personality disorder and/or schizotypal personality disorder and no comorbid affective disorders. Fourteen studies were excluded because of methodological flaws (n=11) or because they investigated drugs other than antidepressants, antipsychotics, or mood stabilizers. Outcomes were categorized in 3 domains: cognitive-perceptual symptoms; impulsive-behavioral dyscontrol; and affective dysregulation, which was subdivided into depressed mood, anxiety, anger, and mood lability. Global functioning was also assessed.

**Results:** Cognitive-perceptual symptoms were moderately improved with antipsychotic treatment, but not other medication classes (see table, next page). Antipsychotics also improved anger, but not other affective symptoms. Only mood stabilizers significantly
improved impulsive-behavioral dyscontrol, and their effects were statistically significant and very large. They also had large positive effects on depressed mood, anxiety, and anger. Conversely, antidepressants did not improve depressive symptoms in the absence of an affective disorder. They did, however, have limited effects on anger and anxiety. Global functioning did not improve with antidepressant treatment, but both antipsychotics and mood stabilizers produced significant improvements. Atypical antipsychotics do not appear to outperform older conventional agents, and SSRIs were not found to be more effective than TCAs or MAOIs. Adverse effects were not evaluated in the analysis.

| Effect Sizes* for Active Pharmacotherapy vs Placebo in Personality Disorders |
|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
|                             | Cognitive-Perceptual Symptoms | Impulsive-Behavioral Dyscontrol | Depressed Mood | Anxiety | Anger | Mood Lability | Global Functioning |
| Antipsychotics (n=144)      | 0.56*                         | 0.26                        | 0.46                   | 0.23  | 0.69*          | 0.41                      | 0.37†                     |
| Antidepressants (n=133)     | 0.11                          | 0.10                        | 0.29                   | 0.30* | 0.34*          | 0.64                      | 0.22                      |
| Mood Stabilizers (n=142)    | 0.42                          | 1.51†                       | 0.55†                  | 0.80† | 1.33†          | Not Assessed              | 0.79†                     |

*p<0.05; †p<0.01 ‡p<0.001

Discussion: According to the authors, these results call into question several common and/or recommended practices in the treatment of personality disorders. In their opinion, mood stabilizers "deserve a more prominent position."

Ingenhoven T, Lafay P, Rinne T, Passchier J, et al: Effectiveness of pharmacotherapy for severe personality disorders: meta-analyses of randomized controlled trials. Journal of Clinical Psychiatry. Published online September 22, 2009 at www.psychiatrist.com; doi 10.4088/JCP.08r04526gre. From the Centers for Mental Healthcare, Amersfoort, The Netherlands; and other institutions. Funded by the Netherlands Expertise Center for Forensic Psychiatry. One study author disclosed a commercial relationship that might pose a conflict of interest; the other authors report no competing interests relevant to this article.

*Reference Guide Item.

Effect Size: Represents the amount of change in outcome that can be attributed to treatment, where 0.2 indicates a small effect, 0.5 a medium effect, and 0.8 a large effect. It is relatively independent of clinical significance, and large effect sizes do not ensure treatment efficacy.
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