

## Inside

- 2 How to Succeed as a Resident  
David Hsu, M.D.  
Associate Editor
- 3 Alzheimer's Disease Therapeutics: New Targets and Strategies  
Yash B. Joshi, B.S.
- 6 Disparities in Clozapine Use in the Veterans Affairs Medical System  
Ryan S. Sultan, M.D.
- 7 A Case of Treatment-Resistant Schizoaffective Disorder With Dramatic Response to Asenapine Augmentation  
Mark B. Ting, M.D.
- 10 Residents' Attitudes Toward Dually Diagnosed Patients  
Jonathan Avery, M.D.
- 11 Therapy with Older Clients: Key Strategies for Success  
David Hsu, M.D.
- 12 Test Your Knowledge
- 13 Author Information for *The Residents' Journal* Submissions and Upcoming Themes

## In This Issue



This issue of the *Residents' Journal* begins with an article by Yash B. Joshi, B.S., on novel targets and strategies in the treatment of Alzheimer's disease. Next, Ryan S. Sultan, M.D., discusses the disparities in clozapine use in the Veterans Affairs system. Mark B. Ting, M.D., presents a case report of a woman with treatment-resistant schizoaffective disorder who responded well to asenapine augmentation. Jonathan Avery, M.D., enlightens us on residents' attitudes toward patients with comorbid severe mental illness and substance use disorders. Lastly, David Hsu, M.D., presents his review of the book *Therapy with Older Clients: Key Strategies for Success*.

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## How to Succeed as a Resident

David Hsu, M.D.  
Associate Editor

Residency in a sense is a lot like growing up. It has its own developmental trajectory, similar to Mahler's stages of development. Around the second year, trainees will find themselves undergoing a separation-individuation phase in which they begin to differentiate from the rest of medicine, as well as from their colleagues. Success is measured upon meeting the standards of care in an independent and autonomous fashion that fits the individual psychiatrist. My objective is to outline certain ways that residents can succeed in residency so that they can be in a better position to choose their desired career path.

Clinical work serves as the foundation of any residency program, and only successful residents are open to new clinical experiences. There are only two kinds of psychiatrists: those who enjoyed their internal medicine rotations and those who did not. It disappoints me when psychiatry residents are turned off when the discussion of patient care turns to anything medical. This places them at a disadvantage for gaining future promotions, since psychiatrists are often asked to serve as medical directors of units or clinics. Given the recent move toward Team Care and integration of the patient-care medical home, psychiatrists will need to interact with their medical colleagues sooner rather than later. Understanding

this fact early on in residency will better prepare future leaders in psychiatry.

*Residency is not easy, but it is the experience of a lifetime.*

A successful resident will pursue scholarly activities throughout his or her training. These include research projects, writing manuscripts, presenting at conferences, and serving on committees. Many trainees may forget that case reports, book reviews, review articles, newspaper articles, and blogs are easy ways to get notable published pieces for their curriculum vitae. These short pieces are easy to do and are more flexible during a high-impact residency program. There are also many national fellowships that residents should apply for in their second or third year of training. The Ginsberg fellowship, PRITE fellowship, APA Leadership fellowship, and Laughlin fellowship are just a few examples of highly regarded fellowships that residents should pursue.

Toward the end of residency, residents may be asked to serve as chief residents during their last year of training. If this

is the case, my recommendation is to seize the opportunity. Being chief is a lot like being junior faculty in that the chief gets to sit on certain faculty committees and to serve as a leader for the entire residency program. The chief position is a great opportunity to grow as an administrative and academic psychiatrist. Future employers and fellowship directors always look positively on previous chief Residents. Being chief resident stays with you everywhere you go, in any career direction you take. Someone who has served as chief before will be up for chief again.

Residency is not easy, but it is the experience of a lifetime. I cannot believe that residency for me was just 2 months ago, but it feels like forever. I hope that this editorial will resonate with psychiatry residents in all years with regard to how to approach the developmental trajectory of residency and how to succeed. Like psychotherapy, you only reap what you put in. Over the years, the *Residents' Journal* has served as a consistent reminder for me that residents have their own needs that are unique and meaningful in their own right. May you, dear reader, succeed in all your future endeavors!

*Dr. Hsu is a fellow in geriatric psychiatry at Massachusetts General Hospital/McLean/Harvard, Boston, and Associate Editor of the Residents' Journal.*

# Alzheimer's Disease Therapeutics: New Targets and Strategies

Yash B. Joshi, B.S.

Alzheimer's disease is the most prevalent aging-associated neurodegenerative dementia and is characterized by progressive memory loss and cognitive decline. The histopathological hallmarks of Alzheimer's disease include the presence of extraneuronal plaques composed of the amyloid  $\beta$  protein, as well as intraneuronal tangles of the microtubule-associated tau protein, in the brains of patients. Because the burden of Alzheimer's disease is expected to dramatically increase in the coming decades and current Food and Drug Administration (FDA)-approved cholinesterase inhibitors and *N*-methyl-D-aspartic acid receptor antagonists only offer temporary symptomatic benefit, significant effort has been made to identify therapeutic strategies that directly modify Alzheimer's disease-specific targets (1). In the present review, treatment strategies in clinical trials, as well as recent genetic discoveries that may provide new targets for Alzheimer's disease, are discussed.

## Antiamyloid $\beta$ Therapies

Genetic analyses have revealed rare but highly penetrant mutations that lead to the development of early-onset Alzheimer's disease in some families. These mutations are in genes that code for the amyloid  $\beta$  precursor protein and in the proteins of  $\gamma$ -secretase, a protein complex that catalyzes the final step for amyloid  $\beta$  production. Based on these observations, as well as significant preclinical work on the amyloid  $\beta$  peptide (for a review, see reference [2]), many ongoing clinical trials focus on agents or mechanisms to reduce amyloid  $\beta$

from the brain. Two treatment strategies have received recent attention:  $\gamma$ -secretase inhibitors and immunotherapy targeting amyloid  $\beta$  peptides and plaques.

Initially,  $\gamma$ -secretase inhibitors showed great promise as amyloid  $\beta$ -lowering agents in early-phase clinical trials but did not show any therapeutic benefit in large-phase III studies. They were associated with increased cancer risk, and in addition to lowering amyloid  $\beta$ , they also reduced the  $\gamma$ -secretase-mediated signaling of Notch, an important regulator of proliferation and differentiation (3). Despite these setbacks, Notch-sparing Alzheimer's disease secretase inhibitors are currently in development, with the hope that more selective agents may be of greater clinical utility (4).

In addition to reducing amyloid  $\beta$  production, clearance of amyloid  $\beta$  using immunotherapy is also the focus of several clinical trials. The central strategy with immunotherapy is to either make amyloid  $\beta$  antigenic or deliver anti-amyloid  $\beta$  immunoglobulin. Immunotherapy is thought to clear amyloid  $\beta$  from the brain by increasing phagocytosis of plaques by microglia, disrupting amyloid  $\beta$  aggregation or shifting the brain amyloid  $\beta$  load to the periphery (4). However, of the several anti-amyloid  $\beta$  antibodies that have been developed, only solanezumab has shown modest reduction in cognitive decline in mild cases of Alzheimer's disease; larger-scale trials are under way to see whether solanezumab is indeed effective in early Alzheimer's disease (5). While promising, immunotherapy has been associated with several adverse effects, including me-

ningoencephalitis, vasogenic cerebral edema, and microhemorrhages, although more selective targeting of amyloid  $\beta$  is being pursued to reduce these effects.

## New Genetic Discoveries and Targets

Several recent discoveries in human and animal models, if properly validated and reproduced, also hold promise as attractive targets for Alzheimer's disease therapy. First is the recent discovery of the  $\gamma$ -secretase-activating protein, which was found to mediate amyloid  $\beta$ -lowering properties of imatinib in an animal model of Alzheimer's disease (6). It is thought that  $\gamma$ -secretase-activating protein facilitates the interaction between  $\gamma$ -secretase and partially processed amyloid  $\beta$  precursor protein, making it easier for amyloid  $\beta$  to be formed. Although directly using imatinib as an Alzheimer's disease treatment would not be prudent because of its side effects, modification of imatinib or creation of an imatinib-like compound that is brain specific may hold potential.

Advances in neurobiology of insulin signaling in the brain have also led to enthusiasm about using insulin and insulin-sensitizing agents in Alzheimer's disease. Insulin signaling has been known to be disrupted in the brain of Alzheimer's disease patients, and preliminary human data have suggested that the risk of Alzheimer's disease is increased in those who have diabetes mellitus type 2 (7). Restoration of appropriate insulin signaling in Alzheimer's disease delays reduces Alzheimer's disease-associated neurodegeneration (8). Recent data

describing cognitive improvement with intranasal insulin in a small cohort of patients with amnesic mild cognitive impairment and early mild Alzheimer's disease suggest that such therapies could be clinically valuable (9). Additionally, since insulin and insulin-sensitizing agents have been FDA approved for diabetes, adverse effects for these classes of drugs are already well-known, making potential translation to a frontline therapy easier.

Beyond the classical hallmarks of amyloid  $\beta$  and tau tangles, chronic inflammation is also present in the Alzheimer's disease brain. Disruption of proinflammatory molecules and pathways have attenuated Alzheimer's disease pathology in animal models of the disease, and some observational data have shown a protective effect with chronic anti-inflammatory agent use in humans, but prospective trials using anti-inflammatory agents have thus far been disappointing (10). However, a new genetic association in Alzheimer's disease risk and specific variants in the TREM2 protein (triggering receptor expressed on myeloid cells-2), which is found on many immune cells, is likely to renew interest in anti-inflammatory drugs in Alzheimer's disease (11). TREM2 expression rises with amyloid  $\beta$  levels in the brains of Alzheimer's disease mice, and TREM2 is upregulated in plaque-associated immune cells (12). Further characterization of TREM2 is required, but an anti-inflammatory drug targeting TREM2-positive immune cells, either alone or in concert with other therapy, could be helpful in the treatment of Alzheimer's disease.

Finally, interventional approaches are also being investigated. For example, intravenous injection of human adipose-derived stem cells

have been shown to improve the Alzheimer's disease phenotype in transgenic Alzheimer's disease mice (13). Adipose-derived stem cells preclude many of the technical and ethical challenges posed by other stem-cell therapies, which may lead to faster entry into the clinic. Additionally, deep brain stimulation, under investigation for other psychiatric illnesses, may also be useful in Alzheimer's disease treatment, through stimulation of the nucleus basalis of Meynert (14).

## The Future of Clinical Trials in Alzheimer's Disease: Selecting Patients Using Biomarkers

Inextricably linked to the development of new treatment strategies in Alzheimer's disease is the development of useful Alzheimer's disease biomarkers that can accurately predict disease course. The vast majority of intervention trials to this point have been conducted when the symptoms of the disease or mild cognitive impairment has been clinically detectable. Yet the growing consensus is that in Alzheimer's disease, pathological changes occur far earlier than can be clinically detectable and that intervening when symptoms manifest may not be useful in delaying or reversing cognitive decline (15–16). With the increasing accuracy of PET studies utilizing radioligands for amyloid  $\beta$  and tau to predict Alzheimer's disease risk, along with characterization of amyloid  $\beta$  and tau species in CSF fluid, intervening in patients at high risk for development of Alzheimer's disease before symptom onset will be the future paradigm of treatment trials (17). Only with early presymptomatic intervention can the importance and usefulness of any therapy be properly evaluated.

## Conclusions

While currently no disease-specific therapeutics exist for Alzheimer's disease, many new strategies are being developed that could have significant clinical impact. Among these are anti-amyloid  $\beta$  therapies, which include  $\gamma$ -secretase inhibitors and immunotherapy against amyloid  $\beta$ . Discovery of  $\gamma$ -secretase-activating protein, recognition of the importance of insulin-signaling pathways in the brain, and new genetic associations between Alzheimer's disease and TREM2 offer additional avenues of hope for patients with Alzheimer's disease. Regardless of treatment option, the highest likelihood of clinical success will be in patients at high risk for Alzheimer's disease identified very early on in the disease process using biomarkers.

*Yash B. Joshi is an M.D./Ph.D. candidate in the Department of Pharmacology, Center for Translational Medicine, Temple University School of Medicine, Philadelphia.*

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## CALL FOR PAPERS

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Would you like the opportunity to have your work appear in *Psychiatric News*? Here's your chance! *Psychiatric News* is inviting members-in-training to participate in a new feature focusing on renowned psychiatrists who are well established in the field or coming to the end of their careers, as well as psychiatrists who have served as outstanding mentors to residents. The articles should capture the essence of the subject (that of a personal perspective of the subject), along with information about the subject's career and his or her accomplishments. The format can vary—for example, it can be written in paragraph form and incorporate quotes from the subject, or it can be written in a Q&A format. The length of each submission should be about 750 words.

This opportunity is being offered to readers of the *Residents' Journal* only. If you are interested in participating in this series, please contact Cathy Brown at *Psychiatric News* at [cbrown@psych.org](mailto:cbrown@psych.org).

We look forward to sharing our pages with psychiatry's newest members and getting them involved in a project that will help educate fellow members about individuals who have truly made a difference in the lives of patients and trainees.

# Disparities in Clozapine Use in the Veterans Affairs Medical System

Ryan S. Sultan, M.D.

Clozapine remains the most efficacious medication in the treatment of schizophrenia (1). Its robust efficacy for positive and negative symptoms prompted the development of an entire second generation of antipsychotics meant to replicate this effect. Data have been published supporting clozapine's protective effects on suicide, as well as long-term cost savings by reducing hospitalization (1). Despite this, the frequency of its use remains lower than expected.

The present study analyzed the outpatient prescribing records for antipsychotics in the Veterans Integrated Service Network 7 of the Veterans Affairs system between 1999 and 2012. This includes eight medical centers in Georgia, Alabama, and South Carolina. Out of a total of 15,953 patients who had a diagnosis of schizophrenia (ICD-9: 295.X), only 246 patients (1.54%) had ever had a trial of clozapine.

In comparison, Medicaid prescribing data demonstrates clozapine to represent as much as 11% of total antipsychotic prescriptions. An APA survey published in 2002 revealed that 7% of patients receiving an antipsychotic were taking clozapine (2).

Another study, published in 2005, compared rates of clozapine prescriptions in Victoria, Australia, and Maryland (3). In Victoria, 49% of the prescriptions for an atypical antipsychotic were for clozapine, compared with 19% in Maryland. The

authors attributed this gap to the more stringent clozapine monitoring guidelines in the United States. A recent, large study in Quebec found that only 6.7% (N=29/155) of individuals with schizophrenia received a trial of clozapine (1). The most recent Veterans Affairs annual report showed that the overall number of patients receiving clozapine was 3.3% (N=84/715) (4).

These data support generally low rates of clozapine use among patients with schizophrenia in the Veterans Affairs system compared with the rest of the country and the world. These rates are particularly low in our Veterans Integrated Service Network group of hospitals.

We suspect that the generally low rates of treatment with clozapine can be linked to the many barriers involved in treatment. The frequency of blood draws, severe weight gain, or metabolic consequences, as well as risk for agranulocytosis, contribute to clozapine's nonpalatability among patients and providers. Concerning the low rates at our specific Veterans Integrated Service Network, we suspect that this stems from a shortage of clozapine-based clinics, a paucity of public transportation options to allow for weekly visits, and concerns on the part of clinicians and patients about risks of metabolic consequences and agranulocytosis.

Schizophrenia remains a severe, persistent, and life-changing illness. Patients

with this disease have significant lifetime risk of completed suicide. Given the data on clozapine's efficacy, we believe strongly that initiatives need to be undertaken to address this health disparity among the veterans receiving care at our medical centers.

*Dr. Sultan is a third-year resident in the Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta.*

*For further information about prescribing clozapine and other antipsychotics, see the article by [Jessica L. Gören et al.](#) in the June 2013 issue of Psychiatric Services.*

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# A Case of Treatment-Resistant Schizoaffective Disorder With Dramatic Response to Asenapine Augmentation

Mark B. Ting, M.D.

Schizoaffective disorder is a construct that addresses individuals with prominent features of both schizophrenia and major mood disorders (1). The lifetime prevalence of schizoaffective disorder ranges from 0.2%–1.1% (2). Treatment for schizoaffective disorder is mostly symptom-guided with antipsychotics, mood stabilizers, and antidepressants (2). Treatment-resistant schizoaffective disorder is a therapeutic challenge. Treatment with clozapine, lithium, and ECT are generally recommended (1). The present case report is of a woman with treatment-resistant schizoaffective disorder, bipolar type, who responded dramatically to asenapine augmentation.

## Case

“Ms. A” is a 56-year-old African American woman with a history of schizoaffective disorder who was admitted to the hospital with increasingly disorganized behavior, agitation, and psychosis. She had a history of multiple combination psychotropic treatments (haloperidol, risperidone, olanzapine, aripiprazole, quetiapine, divalproex, and lithium) for extended periods of time. Despite this, she continued to exhibit significant psychotic and mood symptoms necessitating frequent hospitalizations. She had a history of elevated liver enzymes while taking divalproex and adverse effects with lithium, hence she refused these medications. Her medical comorbidities included asthma, hypertension, and uterine fibroids. Her vital signs were stable. Laboratory work-up, including complete blood count, comprehensive metabolic panel, lipid profile, glycosylated hemoglobin, urine toxicology, and ECG, were not clinically significant. The patient was medically cleared for admission to the psychiatric unit.

On admission, Ms. A was agitated and hyperactive. Her speech was pressured, and her affect was expansive and labile. Her thought process was tangential and illogical, and she exhibited grandiose and unusual delusions. She actively responded to internal stimuli. On admission, her score on the Clinical Global Impression–Severity (CGI-S) scale was 6 (Figure 1). Clozapine was initiated at 50 mg/day, while olanzapine was gradually tapered from 20 mg/day. The patient exhibited improvement in irritability, agitation, and overt delusional thinking. However, the illogical and tangential thought process persisted. Clozapine was discontinued after 3 days due to neutropenia (absolute neutrophil count,  $1.4 \times 10^3/\mu\text{L}$ ). She was then started on oral paliperidone at 6 mg/day, with the aim of initiating long-acting therapy. Olanzapine was continued at 10 mg/day. Since discontinuing clozapine, the patient gradually worsened, and her symptoms reverted back to baseline. At the end of the first week, her score on the CGI–Improvement (CGI-I) scale was 4. Medication dosages were increased over a 2-week period as follows: paliperidone, 12 mg daily, olanzapine, 15 mg daily, and clonazepam, 0.5 mg twice daily. The patient had no response and continued to exhibit agitated and aggressive behavior. Her CGI-I score at the end of the second and third weeks was still 4. Asenapine was then considered, since she had not responded to other medications.

Asenapine was selected because 1) it is an atypical antipsychotic with high affinities for dopamine and serotonin receptors, preferred over typical agents in cases with significant mood components; 2) it is indicated for schizophrenia and bipolar mania; 3) it has lesser metabolic burden, as olanzapine was still part of the regimen; and 4) it has rapid absorption and distribution, potentially minimiz-

ing the length-of-stay in a patient with treatment-resistant symptoms.

Medications were cross-titrated: asenapine (5 mg daily) was started, and paliperidone was gradually tapered off. The patient was initially noted to be sedated, and thus the asenapine dosage was changed to be taken at nighttime. Her thought disorder persisted. On the third day, she was more alert, and her hyperactivity had resolved. Her thought process was mostly logical and goal-directed, with only mild derailments, but the delusions persisted. On day 4, she was alert, with normal psychomotor activity. Her affect was stable, and her thought process was again mostly logical and goal-directed. She had minimal internal preoccupation. Although mild grandiose delusions were present, no unusual delusions were elicited. The patient continued to improve. At the end of the fourth week, her CGI-I score was 2. She was able to interact well and maintain extended conversations. She had minimal psychotic symptoms, and her mood symptoms had resolved. She reported that she felt positive about the treatment regimen and would adhere to it. She was discharged at week 5 with asenapine (5 mg daily), olanzapine (15 mg daily), and clonazepam (0.5 mg twice daily), with the goal of tapering olanzapine on an outpatient basis. On discharge, her CGI-S score was 3, and her CGI-I score was 1.

## Discussion

The patient in the above case met DSM-IV-TR criteria for schizoaffective disorder and treatment-resistant illness based on the presenting history and review of symptoms (3, 4). DSM-5 was not published at that time. On review, the patient met DSM-5 criteria for schizoaffective disorder (5).

Her CGI-S scores at the beginning and endpoint, as well as weekly CGI-I scores,

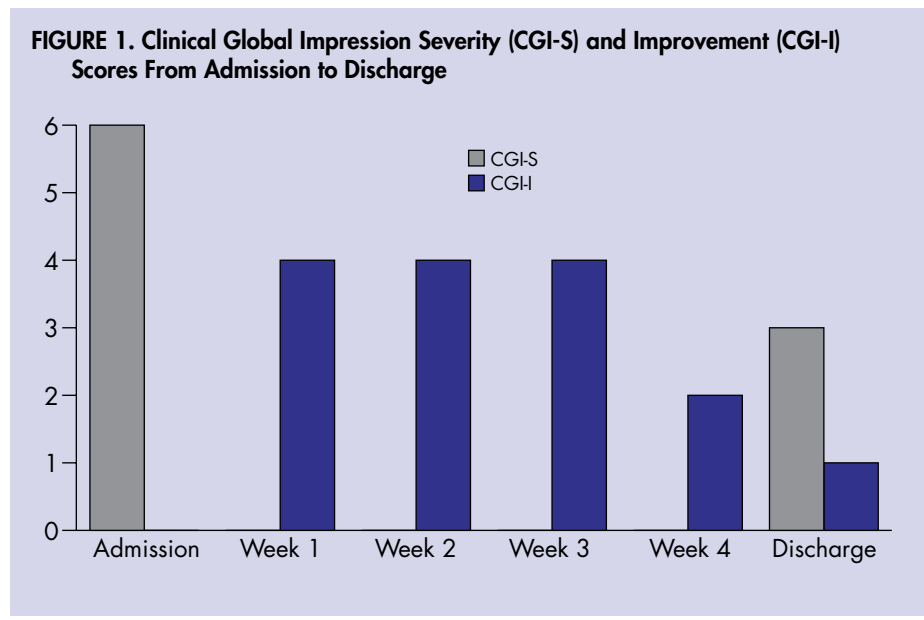
were used to measure the clinical course. CGI-S measures illness severity, ranging from 1 (normal) to 7 (extremely ill). CGI-I measures change, ranging from 1 (very much improved) to 4 (no change) to 7 (very much worse) (6).

Following the temporal course, the improvement appears to be due to the addition of asenapine. The supporting reasons are that 1) the patient was on olanzapine up to 30 mg/day in the past with limited response; 2) paliperidone was given for over 2 weeks with no response; 3) response occurred within 3 days of initiating asenapine with tapering of paliperidone; and 4) improvement was sustained for a week prior to discharge on the combination.

Limitations of this study include the fact that it is a single case report; clozapine rechallenge could have been considered but was not feasible at the time due to neutropenia; and the duration of treatment with paliperidone was not maximized due to the absence of any response during the 2-week titration to the maximum recommended daily dose. However, a dramatic response was noted with the addition of 5 mg of asenapine to the regimen (5).

Treatment approaches to schizoaffective disorder are not specific. Recommendations refer to atypical antipsychotics as firstline agents, owing to their additional thymoleptic properties (2). Asenapine is a novel antipsychotic, which is an antagonist with high binding affinities at dopaminergic (D<sub>2</sub>, D<sub>3</sub>, and D<sub>4</sub>), serotonergic (5-HT<sub>2A</sub>, 5-HT<sub>2B</sub>, 5-HT<sub>2C</sub>, 5-HT<sub>6</sub>, and 5-HT<sub>7</sub>), adrenergic (α<sub>1</sub>, and α<sub>2</sub>), and histamine (H<sub>1</sub>) receptors. It has a high hepatic first-pass mechanism, hence the development of sublingual administration. Asenapine is primarily metabolized by the cytochrome P450-1A2 enzyme into metabolites that do not have any significant effects and do not cross the blood-brain barrier. The recommended starting dosage is 5 mg twice daily, and the most common dosage in the maintenance phase is 10 mg twice daily (7).

While there have been no studies, to our knowledge, regarding augmentation of olanzapine with asenapine, it is



possible that this combination has synergistic effects. Olanzapine binds with high affinity at serotonergic (5-HT<sub>2A</sub>, 5-HT<sub>2C</sub>, 5-HT<sub>6</sub>), dopaminergic (D<sub>1-4</sub>), histamine (H<sub>1</sub>), and adrenergic (α<sub>1</sub>) receptors. It is an antagonist with moderate affinity at serotonergic 5-HT<sub>3</sub> and muscarinic M<sub>1-5</sub> receptors (8). It is possible that the addition of low-dose asenapine may provide a boost in the antagonism of D<sub>2</sub> receptors required for antipsychotic activity, owing to its higher affinity (expressed as Ki [equilibrium constant] in nM [asenapine, 1.3; olanzapine, 11–31]). Increased antagonism of the other dopaminergic receptors may provide synergistic effects in decreasing psychosis, enhancing cognition, and improving negative symptoms (9). Enhanced serotonergic antagonism, especially at 5-HT<sub>2A</sub> (Ki: asenapine, 0.06; olanzapine, 4), also decreases psychosis, modulates mood, enhances cognition, and decreases anxiety (9). However, the physiological roles of 5-HT<sub>6</sub> and 5-HT<sub>7</sub> are still being determined (9).

It is generally not recommended for patients to be taking multiple antipsychotics. However, there may be cases in which multiple antipsychotics may be used, especially for treatment-resistant symptoms (10). In the present case, the patient was discharged on two antipsychotics, with the aim of continuing cross-titration as an outpatient.

## Conclusions

Augmentation with asenapine resulted in dramatic and sustained improvement in psychotic and mood symptoms in our patient. Although the exact reason for the dramatic improvement is unclear, it is possible that asenapine may provide a synergistic effect with olanzapine. Further controlled studies of asenapine augmentation in individuals with treatment-resistant symptoms are recommended (7).

*Dr. Ting is a third-year resident in the Department of Psychiatry, St. John's Episcopal Hospital, Far Rockaway, N.Y.*

*The author thanks his mentors, Dr. Subramoniam Madhusoodanan and Dr. Krishna Bezawada, Associate Chair and Unit Chief, respectively, of the Department of Psychiatry, St. John's Episcopal Hospital, for their guidance in the formulation of this article.*

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## PSYCHIATRIC SERVICES

### New Benefit for American Psychiatric Association Members-in-Training (MITs)

# Free Online Subscription to Psychiatric Services

Beginning with the January 2012 issue, APA Members-in-Training (MITs) will receive a [free online subscription to \*Psychiatric Services\*](#).

Simply visit [ps.psychiatryonline.org](http://ps.psychiatryonline.org) for full-text access to all of the content of APA's highly ranked, peer-reviewed monthly journal. *Psychiatric Services* focuses on service delivery in organized systems of care, evolving best practices, and federal and state policies that affect the care of people with mental illnesses.

**Please visit [ps.psychiatryonline.org](http://ps.psychiatryonline.org) and log in with your American Psychiatric Association username and password.**

Psychiatry residents who are not currently an APA Member-in-Training should consider membership in the American Psychiatric Association. The benefits provided to residents are an example of how the APA serves the needs of its members throughout their careers. The low introductory dues APA extends to MITs are even waived for the first year. Please visit <http://www.psych.org/joinapa> for more information.



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The *First and Last* Word in Psychiatry  
AH1216A

# Residents' Attitudes Toward Dually Diagnosed Patients

Jonathan Avery, M.D.

Psychiatry residents are often called upon to take care of individuals diagnosed with comorbid serious mental illness and substance use disorders. After all, approximately one-half of individuals diagnosed with alcohol use disorders have been diagnosed with a comorbid psychiatric disorder, as have nearly 75% of individuals diagnosed with a drug use disorder (1). Approximately one-half of individuals diagnosed with schizophrenia also have been diagnosed with a comorbid substance use disorder (1). While psychiatry programs seem to be increasing the amount of didactics and supervision on diagnosing and treating dually diagnosed patients, heightened attention needs to be given to resident attitudes toward these patients. Negative and stigmatizing attitudes from clinicians have been shown to decrease help seeking and to worsen psychological distress among individuals diagnosed with mental illness (2), and it is important that residents do not develop these attitudes.

Residents' attitudes toward individuals diagnosed with substance use disorders appear to be more negative compared with their attitudes toward individuals diagnosed with other disorders, and these attitudes may worsen over time (3). One study of medical student and resident attitudes toward caring for and

working with patients with alcohol and drug abuse diagnoses found that satisfaction achieved in caring for these patients consistently diminished over years in training, and the belief that these patients overutilize health care resources increased (3). Resident attitudes toward individuals diagnosed with serious mental illness and comorbid serious mental illness and substance use disorders have not been studied extensively, but some studies have found that clinicians do have more negative attitudes toward these patients (2, 4).

During their clinical work and supervision, psychiatry residents typically learn about common attitudes and countertransferences toward individuals diagnosed with mental illness. It is rare to have dedicated didactic time to learn about attitudes, but such training is critical to make residents aware of common attitudes. Educational reflection techniques may be helpful in supplementing the didactics. Ballon and Skinner (5), for example, utilized reflection discussion times, reflection journaling, and mandatory end-of-rotation reflection papers during a 4-week addiction psychiatry rotation in order to improve attitudes.

It can be challenging to care for individuals diagnosed with comorbid serious mental illness and substance use disorders. In order to provide the best

care for these patients, residents need a comprehensive education that highlights the challenges and attitudes that develop when treating dually diagnosed individuals.

*Dr. Avery is an addiction psychiatry fellow at New York University School of Medicine.*

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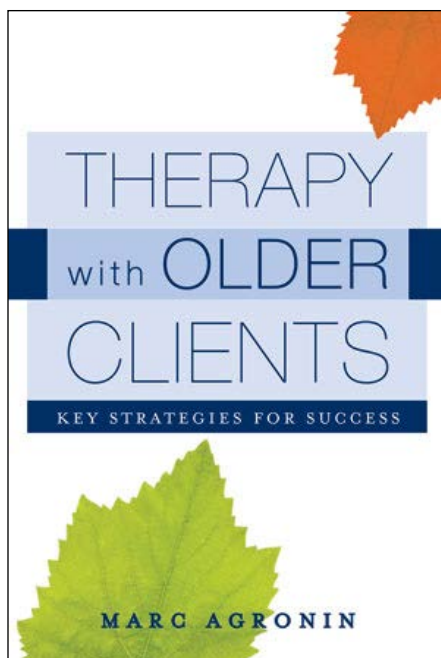
## Therapy with Older Clients: Key Strategies for Success

by Marc E. Agronin, M.D.

David Hsu, M.D.

I have read almost everything Dr. Agronin has written, and in my opinion, this is his best work to date. Similar to *How We Age*, *Therapy with Older Clients* is full of inspiring stories from clients Agronin has cared for. From beginning to end, his discussions on conducting therapy with older clients are philosophical and compassionate. He frames his approach to therapy as the “three Cs,” namely, curiosity, caring, and courage. I believe this perspective is apt for working with older patients, who have gained wisdom over time and are able to demonstrate “post-formal thinking,” which Agronin defines as the integration of logical and irrational elements.

Agronin helps to prepare the therapist for the first session by discussing potential countertransference. It is important for the therapist to reflect on how he or she feels about death, dying, and dementia. Can someone with dementia still have meaning in life? He also helps the therapist to prepare the client for the first session by making sure that enough time is allotted for the session, that the client has his or her entire medical history, and that the client has used the restroom prior to the session. Agronin clearly advocates that psychotherapy can be performed



by Marc E. Agronin, M.D. New York, W. W. Norton and Company, 2010, 320 pp., \$27.50

with clients who have cognitive impairment, and he gives plenty of examples.

The book features nine thought-provoking chapters. My favorite two chapters focus on the different forms of psychotherapy that can be adapted to older clients. Illus-

trating how therapy would proceed with two patients named “Sarah” and “Abe,” Agronin delves into psychodynamic psychotherapy, cognitive-behavioral therapy, interpersonal therapy, problem-solving therapy, and couples and family therapy. He also devotes much time to group psychotherapy. A poignant example includes how an older woman was at first hesitant to join a group but later warmed up to it and even led the group in song.

Agronin’s book on psychotherapy is accessible and practical. Each chapter is organized and filled with easy-to-use tables to help guide the therapist in proceeding with therapy. This introduction to psychotherapy with the older client serves as a much needed adjunct to the already plethora of geriatric psychiatry literature that is focused on psychopharmacology. Agronin reminds us that psychotherapy can make a difference in the lives of our clients, and sometimes it may be the only thing.

*Dr. Hsu is a fellow in geriatric psychiatry at Massachusetts General Hospital/McLean/Harvard, Boston, and Associate Editor of the Residents’ Journal.*

*For further information on therapies for older adults, see the recent article by Patricia A. Arean et al. in Psychiatric Services.*



If you will be completing your residency this year, we would like your help in recruiting new subscribers by encouraging an incoming resident or fellow to subscribe to our monthly e-publication. Also, if you'd like to continue receiving e-mail notification alerts when each issue of the AJP Residents' Journal is published, send your new e-mail address to [ajp@psych.org](mailto:ajp@psych.org) with the subject line "New e-mail address post-residency."

# TEST *YOUR* KNOWLEDGE

In preparation for the PRITE and ABPN Board examinations, test your knowledge with the following questions.  
(answers will appear in the next issue)

This month's questions are courtesy of David Hsu, M.D., a fellow in geriatric psychiatry at Massachusetts General Hospital/McLean/Harvard, Boston, and Deputy Editor of the Residents' Journal.

## Question 1

According to Freud, what is "abreaction?"

- A. The removal of symptoms by recovery and verbalization of associated feelings that have been suppressed.
- B. The method of having the patient say whatever came into his or her mind.
- C. The transformation of latent dream content into manifest dream content.
- D. The unconscious reluctance to describe one's feelings.

## Question 2

What is true about acute intermittent porphyria?

- A. Patients have decreased porphobilinogen.
- B. It often presents in men over 65 years of age.
- C. Psychiatric symptoms may include visual hallucinations, paranoia, and depression.
- D. Peripheral neuropathy is uncommon in this syndrome.

## ANSWERS TO AUGUST QUESTIONS

### Question #1

**Answer:** B. 35%–60% of substance users meet criteria for antisocial personality disorder.

It is important for psychiatrists to be aware of comorbid psychiatric disorders in substance use disorders, including personality disorders. Answer A is incorrect because people with substance use disorders actually have a higher rate of suicide. Answer C is incorrect because in classic theory, users of substances actually have more difficulty defending against anxious impulses. Answer D is incorrect because men actually use more drugs than women.

### Reference

1. Sadock BJ, Sadock VA: Kaplan and Sadock's Synopsis of Psychiatry, 10th ed. Philadelphia, Lippincott Williams and Wilkins, pp 384–388

### Question #2

**Answer:** A. Duty, deviation, damage, direct causation

In order for a doctor to be found guilty of medical malpractice, the prosecution must demonstrate that the doctor had a duty to the patient, that there was a deviation of practice, that there was damage done to the patient, and that the doctor's actions had a direct causation of the harm.

### Reference

1. Sadock BJ, Sadock VA: Kaplan and Sadock's Synopsis of Psychiatry, 10th ed. Philadelphia, Lippincott Williams and Wilkins, p 1371

We are currently seeking residents who are interested in submitting Board-style questions to appear in the Test Your Knowledge feature. Selected residents will receive acknowledgment in the issue in which their questions are featured.

Submissions should include the following:

1. Two to three Board review-style questions with four to five answer choices.
  2. Answers should be complete and include detailed explanations with references from pertinent peer-reviewed journals, textbooks, or reference manuals.
- \*Please direct all inquiries and submissions to Dr. Hsu: davidhsu222@gmail.com.

# Author Information for *The Residents' Journal* Submissions

*The Residents' Journal* accepts manuscripts authored by medical students, resident physicians, and fellows; manuscripts authored by members of faculty cannot be accepted. To submit a manuscript, please visit <http://mc.manuscriptcentral.com/appi-ajp>, and select "Residents" in the manuscript type field.

- 1. Commentary:** Generally includes descriptions of recent events, opinion pieces, or narratives. Limited to 500 words and five references.
- 2. Treatment in Psychiatry:** This article type begins with a brief, common clinical vignette and involves a description of the evaluation and management of a clinical scenario that house officers frequently encounter. This article type should also include 2-4 multiple choice questions based on the article's content. Limited to 1,500 words, 15 references, and one figure.
- 3. Clinical Case Conference:** A presentation and discussion of an unusual clinical event. Limited to 1,250 words, 10 references, and one figure.
- 4. Original Research:** Reports of novel observations and research. Limited to 1,250 words, 10 references, and two figures.
- 5. Review Article:** A clinically relevant review focused on educating the resident physician. Limited to 1,500 words, 20 references, and one figure.
- 6. Letters to the Editor:** Limited to 250 words (including 3 references) and three authors. Comments on articles published in *The Residents' Journal* will be considered for publication if received within 1 month of publication of the original article.
- 7. Book Review:** Limited to 500 words and 3 references.

Abstracts: Articles should not include an abstract.

## Upcoming Themes

*Please note that we will consider articles outside of the theme.*

### Forensic Psychiatry

If you have a submission related to this theme, contact the Section Editor, Tobias Wasser, M.D. ([tobias.wasser@yale.edu](mailto:tobias.wasser@yale.edu)).

### Adolescent Psychiatry

If you have a submission related to this theme, contact the Section Editor, Justine Wittenauer ([jwittenauer@challiance.org](mailto:jwittenauer@challiance.org)).

### Mental Health Disparities

If you have a submission related to this theme, contact the Section Editor, Ijeoma Chukwu, M.D. ([ichukwu@uci.edu](mailto:ichukwu@uci.edu)).