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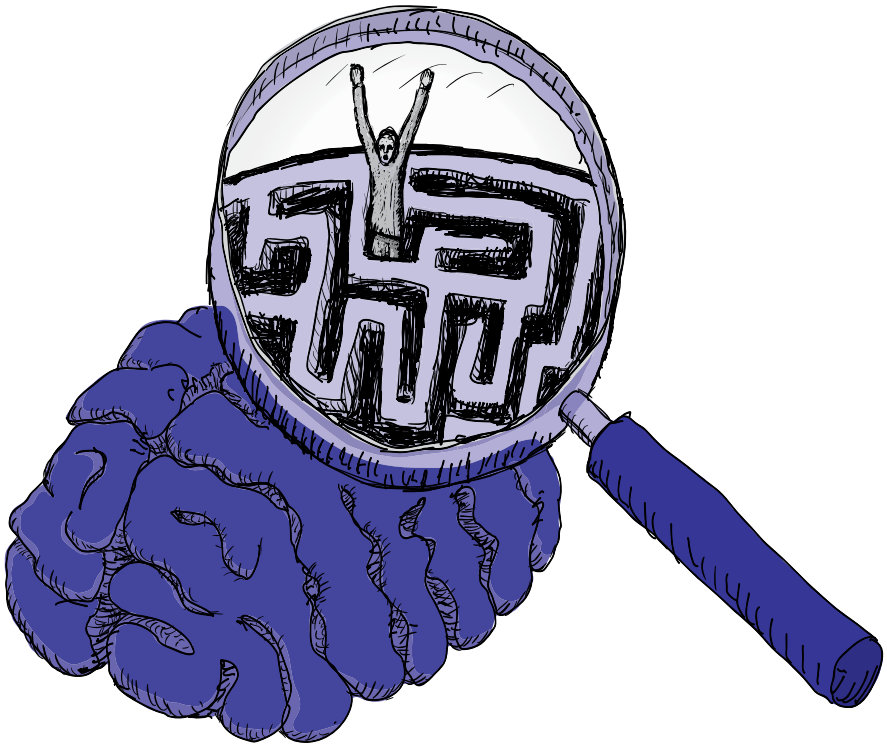

This issue of the *Residents' Journal* begins with an informative article by David Ryan Goldsmith, M.D., on the use of metformin adjuvant therapy to treat the metabolic side effects, including weight gain, of atypical antipsychotics. Next, Vivek Jayadeva, B.S., provides an overview of recent advances in psychiatric biomarkers for mood disorders, autism spectrum disorders, and schizophrenia. Megan Mroczkowski, M.D., presents a case report of a 4-year-old boy with gender dysphoria. Misty C. Richards, M.D., M.S., discusses the advancement of women in academic psychiatry. In a perspective, Carmen Casasnovas, M.D., shares her experience providing care during Hurricane Sandy. Last, David Hsu, M.D., reviews the book *How Doctors Think*.

Metformin Adjuvant Therapy to Counteract Metabolic Side Effects of Atypical Antipsychotics

David Ryan Goldsmith, M.D.

With the introduction of second-generation atypical antipsychotic medications to treat schizophrenia and other psychotic disorders, there is less risk of extrapyramidal side effects compared with first-generation medications. The atypical class may also offer improvement in cognition, negative symptoms, and overall quality of life (1). Nonetheless, these newer medications are not without risks, posing unique challenges to prescribers, including weight gain, elevated lipids, abnormal glucose metabolism, and subsequent increased cardiovascular risk. This is especially important in a population whose cardiometabolic risk is already greater than that of the general population. These side effects are seen most often in atypical antipsychotics such as clozapine and olanzapine and less often in risperidone and quetiapine. Increased weight gain and its effect on overall cardiovascular health is nontrivial, especially in psychiatric disorders, since it may significantly account for the increased risk of premature death in patients with schizophrenia (2).

Evidence from the Clinical Antipsychotic Trials of Intervention Effectiveness demonstrated that 30% of patients taking olanzapine gained at least 7% of their pretrial body weight (on average, patients in the study gained about two additional pounds each month) (3). The mechanism of atypical antipsychotic-induced weight gain is not completely understood, although evidence suggests that effects at the H_1 and 5-HT_{2C} receptor subtypes correlate with weight gain, as do several susceptibility genes, including the *HTR2C* and leptin genes, among others (4). Abdominal obesity is a major risk factor for the development of metabolic syndrome, itself a risk factor for insulin resistance and diabetes (5). Metabolic syndrome criteria are summarized in Table 1.

Metformin is a first-line oral hypoglycemic medication, prescribed to patients with diabetes as well as metabolic syndrome, that works by suppressing hepatic gluconeogenesis; it also increases insulin-mediated glucose uptake in the peripheral tissues (6). Importantly, metformin is weight-neutral and is less likely to induce hypoglycemia compared with other medications used for the treatment of diabetes. It has also been shown to independently decrease hemoglobin $A1_C$ levels. Typically, metformin is a well-tolerated medication, although the most common side effect is gastrointestinal symptoms, while the most worrisome is lactic acidosis. Although incidence rates of lactic acidosis are extremely low (approximately 0.03 cases/1,000 patient-years), the Food and Drug Administration issued a black box warning of lactic acidosis due to its high fatality rate (50% of cases). As such, it is necessary to consider a patient's kidney function before starting a trial of metformin. Additionally, metformin should be avoided in patients who may have increased lactic acid levels (including those with sepsis), increased alcohol intake, dehydration, hepatic insufficiency, renal impairment, or unstable congestive heart failure, as well as those who are elderly (especially those >80 years old).

Importantly, in patients with impaired glucose tolerance, treatment with metformin has been shown to prevent or delay the onset of diabetes. This effect is even greater when metformin is used in combination with lifestyle modification, education about diet, and exercise in order to manage weight (7). As such, in patients taking atypical antipsychotics who have increased risk for metabolic syndrome and diabetes, metformin might be a promising adjuvant therapy to prevent the development of these risk factors and subsequent cardiovascular disease.

To our knowledge, there are only eight published studies in adults (8–15) and three in children (16–18) exploring the efficacy of metformin to prevent weight gain and risk for metabolic syndrome and diabetes in patients taking atypical antipsychotics. In most of the studies in adults, patients had been on an antipsychotic medication regimen prior to enrollment in the study. Three studies have investigated the use of metformin in young, antipsychotic-naive patients. The

TABLE 1. Metabolic Syndrome Criteria[a]

National Cholesterol Education Program Adult Treatment Panel III (three out of five criteria required)
Abdominal obesity: waist circumference for men >100 cm (40 inches) and for women >88 cm (35 inches)
Serum triglycerides: >150 mg/dL, or taking medication for elevated triglycerides
Serum HDL cholesterol: <40 mg/dL for men and <50 mg/dL for women, or taking medication for low HDLs
Blood pressure: ≥130/85 mmHg or taking medication for high blood pressure
Fasting blood glucose: ≥100 mg/dL, or taking medication for elevated blood glucose
International Diabetes Federation Criteria
Increased weight circumference plus two of the following:
Serum triglycerides: >150 mg/dL, or taking medication for elevated triglycerides
Serum HDL cholesterol: <40 mg/dL for men and <50 mg/dL for women, or taking medication for low HDLs
Systolic blood pressure >130, or diastolic blood pressure >85
Fasting blood glucose: ≥100 mg/dL, or taking medication for elevated blood glucose

[a] HDL=high-density lipoprotein.

majority of the above studies examined patients with a diagnosis of schizophrenia, although other patients taking atypical antipsychotics (for bipolar disorder, schizoaffective disorder, and other pediatric behavioral disorders) were included as well.

Eight studies have explored the efficacy of metformin in an adult psychiatric patient population. Of these, five found significant outcomes in the metformin group compared with the placebo group (8–12), while three found no difference between groups (13–15). Patient age varied between these studies (mean age range, 25 years–44.5 years), as did the length of time patients had been receiving atypical antipsychotics (treatment-naive to 86.5 months of treatment). Trials were between eight and 14 weeks long. In the trials with positive data, weight loss ranged from 2.03 kg to 6.3 kg. Positive results were also seen in reductions in insulin, insulin resistance index, blood sugar levels, reductions in glycated hemoglobin (HbA1c), reductions in triglycerides, and, in one study, an increase in high-density lipoprotein.

Because previous studies have shown metformin to be most effective in combination with lifestyle interventions (7), one group of investigators explored the effect of metformin as well as lifestyle interventions in the psychiatric patient population (11). One-hundred twenty-eight first-episode patients with psychosis, who were within 12 months of starting an atypical antipsychotic, were randomly assigned to a metformin group, a metformin plus lifestyle intervention, a lifestyle intervention alone, or a placebo group. All treatment groups exhibited greater weight loss and other associated outcome measures compared with the placebo group. Patients who were assigned to the metformin plus lifestyle intervention had significantly more weight loss (7.8 kg) and reductions in glucose-related measures compared with patients in the other intervention groups.

The pediatric/adolescent literature is limited, with three studies examining the effect of metformin in patients taking atypical antipsychotics (16–18). One of these studies was a 12-week open-label trial in 19 patients (mean age, 14 years [SD=2.5]) who had already gained at least 10% of their total body weight while taking atypical medications (16). There was a significant reduction in total body weight as well as body mass index when the patients were taking metformin. A randomized control trial in the pediatric population examined 39 patients (mean age, 13.1 years [data for standard deviation are not available]) and found similar results, with the metformin group exhibiting significantly more weight loss (17). Of note, this study also included nutritional counseling and individualized goals for all study patients. Finally, a randomized control trial of 49 first-episode patients (mean age, 11.3 years [data for standard deviation are not available]) found no difference between the metformin and placebo groups, although it should be noted that the only atypical antipsychotic used in this study was risperidone, which has been shown to be less likely to have the side effects of weight gain and insulin resistance (18).

Whether these findings could effect clinical practice, it is important to note that there is no consensus among the randomized controlled trials, in either the adult or pediatric literature. Varying dosages of metformin were used throughout these studies. The majority of these studies were not conducted in patients in the United States, where obesity and metabolic syndrome is of particular public health concern. To our knowledge, the role of lifestyle interventions in addition to metformin, which has been shown to be exceedingly effective (7), has only been investigated in one trial. None of the investigators in the above trials reported on the side effect profiles of metformin treatment in psychiatric groups, although it should be noted that metformin is not metabolized by hepatic CP450 enzymes and has not been shown to have drug-drug interactions with antipsychotics (19).

Despite these limitations, it is up to the psychiatrist or primary care physician to weigh the risks and benefits of adding metformin to atypical antipsychotic treatment. Perhaps an especially vulnerable population to consider is that of first-episode patients who are started on antipsychotics for the first time. Weight gain has been shown to occur most rapidly within the first 12 weeks of treatment. Since first-episode psychotic breaks tend to occur during late adolescence to early adulthood, rapid increased weight gain during these younger years may lead to increased stigma, psychological anguish, and worse outcomes. For these patients, early intervention may be beneficial to prevent longer-term metabolic and cardiovascular risks (20). For these and all patients receiving atypical antipsychotics, metformin should not be taken in place of but be included as part of a comprehensive cardiovascular health plan that incorporates a healthy diet, regular exercise, and smoking cessation.

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Editor's note: For further discussion of treatment of antipsychotic-induced weight gain using metformin, see the article by Wu et al. in the August 2012 issue of the American Journal of Psychiatry.

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Psychiatric Biomarkers: A Review of Recent Advances

Vivek Jayadeva, B.S.

A biomarker is a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacological responses to a therapeutic intervention (1). Biomarkers in psychiatry include 1) specific patterns of neuronal activity detected by imaging, such as MRI and positron emission tomography; 2) specific genetic sequences or single-nucleotide polymorphisms; and 3) specific endophenotypes (intermediate traits in the chain of causality between genes and diseases), such as biochemical, neurophysiological, or neuropsychological features. Different markers can be used to diagnose a condition, predict the natural outcome for an individual with that condition, predict whether the individual will benefit from a particular treatment and how aggressively to treat the individual, and assess an individual's response to the treatment (2). A number of studies have shown promise for the translation of biomarker research to the clinical setting.

Mood Disorders

Alexander et al. (3) targeted the p11 protein as a biomarker for major depressive disorder. P11 is an S100 family member recently identified as a serotonin 1B (5-HT_{1B}) and serotonin 4 (5-HT_4) receptor binding protein. P11 knockout mice have shown depression-like behaviors, suggesting that p11 may be a mediator of affective disorder pathophysiology. Using somatic gene transfer, the nucleus accumbens was identified as a key site of p11 action (3). Reduction of p11 with adeno-associated virus-mediated RNA interference in the nucleus accumbens, but not in the anterior cingulate, in normal adult mice resulted in depression-like behaviors nearly identical to those seen in p11 knockout mice. Restoration of p11 expression specifically in the nucleus accumbens of p11 knockout mice normalized depression-like behaviors. Human nucleus accumbens tissue showed a significant reduction of p11 protein in depressed patients compared with healthy matched comparison subjects. These results suggest that p11 loss in rodent and human nucleus accumbens may contribute to the pathophysiology of depression. Alexander et al. showed that normalization of p11 expression within this brain region with adeno-associated virus-mediated gene therapy may be of therapeutic value.

Cattaneo et al. (4) recently found that some inflammatory biomarkers may serve as predictors of antidepressant response, while other neuroplasticity biomarkers may indicate therapeutic targets for the depression treatment. The authors also found that the baseline levels of macrophage inhibiting factor, interleukin 1-b, and tumor necrosis factor alpha were "predictors" of antidepressant treatment response. They demonstrated that an enhancement of glucocorticoid receptor function and an improvement in neuroplasticity (increases in brain-derived neurotrophic factor, VGF, and p11) were needed to observe a response to antidepressant therapies, suggesting that future antidepressant strategies should specifically target these pathways.

In the May/June 2011 special issue of the *Harvard Review of Psychiatry*, Roffman (5) summarized some of the latest findings in psychiatric biomarker research available. One study he described focused on a functional polymorphism in the catechol-*O*-methyltransferase (*COMT*) gene. Patients who had the low-activity genetic variant of the *COMT* gene consistently demonstrated better cognitive improvement following treatment with dopamine antagonists, while preliminary studies in healthy individuals suggested that people with the high-activity variant might have better cognitive improvement with dopamine agonists. Roffman also described a review by D. Iosifescu examining how electroencephalographic measures have been used to predict the likelihood of antidepressant response within the first 1–2 weeks of treatment, well before substantial clinical effects are apparent. One prospective study in particular showed that for patients who exhibited a low likelihood of response based on their EEG data 1 week into treatment, switching immediately to a different antidepressant substantially improved their chances of treatment benefit, compared with a group of patients that did not change medications.

In a recent study, Hajek et al. (6) used structural MRI to identify a neural signature for a predisposition to bipolar disorder compared with long-standing illness and treatment. Five groups, including the unaffected and affected relatives of bipolar disorder probands from each study center, as well as participants early in the course of bipolar disorder, exhibited larger right inferior frontal gyrus volumes than comparison subjects ($p=0.001$, corrected). The right inferior frontal gyrus volume correlated negatively with illness duration ($p=0.01$, corrected) and, compared with comparison subjects, was smaller in participants with bipolar disorder with long-term illness burden and minimal lifetime lithium exposure ($p=0.001$, corrected). Li-treated subjects had normal right inferior frontal gyrus volumes despite substantial illness burden. These findings indicate that right inferior frontal gyrus volume could aid in the diagnosis of individuals at risk for bipolar disorder even before any behavioral manifestation.

Autism Spectrum Disorders

Schwarz et al. (7) demonstrated that male and female adults with Asperger's syndrome have different biomarker panels. Men with Asperger's syndrome exhibited altered levels of 24 biomarkers, including increased levels of cytokines and other inflammatory molecules. Multivariate statistical classification of men using this panel of 24 biomarkers revealed a marked distinction between those with Asperger's syndrome and comparison subjects, with a sensitivity of 0.86 and specificity of

0.88. Testing this same panel in women did not result in a separation between the Asperger's syndrome and comparison groups. In contrast, the women with Asperger's syndrome showed altered levels of 17 biomarkers, including growth factors and hormones such as androgens, growth hormone, and insulin-related molecules. Classification of women using this biomarker panel resulted in a marked difference between those with Asperger's syndrome and comparison subjects, with sensitivities and specificities of 0.96 and 0.83, respectively. Testing this same 17-biomarker panel in the male group did not result in a distinction between the patient and comparison groups. The authors concluded that sex stratification will be necessary to understand the etiology and development of autism spectrum disorders.

In their recent study, published in *Translational Psychiatry*, Momeni et al. (8) found biomarkers for autism involving C3 complement fragments. The authors used a proteomic approach consisting of surface-enhanced laser desorption/ionization time-of-flight mass spec and matrix-assisted laser desorption/ionization time-of-flight to compare peptide profiles of blood plasma from children with autism spectrum disorders and comparison subjects, with the aim of discovering novel peptide biomarkers with diagnostic utility and to understand the role of these in the pathophysiology of autism spectrum disorders. The study found differentially expressed peptides that correspond to fragments of the C3 complement protein. The first peptide corresponded to the peptide known as C3f of the complement protein C3. The second peptide corresponded to a peptide of 16 residues with the same sequence as C3f but lacked the C-terminal arginine and is known as C3f-desArg. The third peptide that appeared at higher concentration in the group of healthy comparison children had the same sequence as C3f but carried a modified arginine residue, corresponding to ornithine, in the C-terminus.

Schizophrenia

Light et al. (9) searched for endophenotypes for schizophrenia that 1) were associated with schizophrenia, 2) were stable over time, independent of state-related changes, and 3) were free of potential practice/maturation or differential attrition effects in schizophrenia patients and nonpsychiatric comparison subjects. The authors found that the following neurocognitive measures demonstrated significant deficits in patients with schizophrenia, and the deficits were stable after a 1-year follow-up evaluation: mismatch negativity, P3a, prepulse inhibition, and oculomotor antisaccade. Mismatch negativity is an indicator of brain response to aberrant auditory stimuli using EEG. P3a is an EEG potential associated with brain activity related to attention. Prepulse inhibition is when a weaker stimulus is presented before a stronger stimulus causing a reduction in the startle response. For this study, the stimulus utilized was an acoustic startle reflex. Oculomotor antisaccade is when an object appears in the periphery and the subject must suppress the reflexive urge to look at the target while instead looking in the opposite direction.

Conclusions

While the search for psychiatric biomarkers has long been focused on genes and protein products, research over the past few years has shown an increasing trend of expanding the repertoire of research tools, ranging from endophenotypes and EEG to MRI and immunological markers. In 2012 alone, studies have continuously revealed discovering reliable biomarkers for psychiatric disorders, and the near future holds much promise for the translation of this research to the clinic.

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Case Report

Gender Dysphoria in a 4-Year-Old Boy

Megan Mroczkowski, M.D.

"Matthew" is a 4-year-old Caucasian child with no psychiatric history who resides with his adoptive parents and fraternal twin brother. He was referred by his adoptive parents for expressing that he "is a girl" and that he desires to play and dress in a traditionally feminine manner. His parents requested guidance regarding the best way to support him.

The parents reported that for the last few months, the patient has been telling them that he is a girl. During this time, he has also preferred to associate with female rather than male peers. The parents stated that "he seems confused" about his gender. For example, he has made statements such as, "I am a girl" and "I want to be a girl." He has asked for reassurance from his mother that it is okay to pretend that he is a girl. Currently, she tells him that it is okay to pretend to be a girl, just like it is okay to pretend to be a doctor or firefighter, etc. At times, when playing with girls, the patient will tell his twin brother, "Go away, this game is just for girls." His brother does not have the same predilection for dressing and playing in traditionally feminine ways. Matthew recently began attending preschool, three times per week, and mostly enjoys the company of girls at the school and likes to dress up as a princess. He has shown no distress from his symptoms and has exhibited no change in his level of functioning. His parents stated that they are not concerned about his sexual orientation.

Although the child has no psychiatric history, his medical history is notable for recurrent ear infections that required tympanostomy tubes to be inserted at age 3. He and his twin brother were adopted at 4 days old. They were born at 31 weeks gestation, and Matthew weighed 4.5 pounds. Matthew was delayed in meeting several developmental milestones: he sat up at 8 months, cruised at 10.5 months, walked at 14 months, and talked at 14.5 months. He received early intervention services in speech, language, and occupational therapy. The family history is notable for the biological mother having a history of bipolar affective disorder and polysubstance dependence. The health history of the biological father is unknown.

On mental status examination, the child appears to be slightly younger than his stated age. His speech is notable for a slight lisp but otherwise normal in rate, rhythm, volume, and tone. His mood is "happy," assessed as euthymic with full range of affect. He does not appear to have hallucinations, delusions, obsessions, compulsions, or phobias. A sample of his speech is as follows: "I want to be a girl when I grow up."

Discussion

For this patient, education on his diagnosis, gender dysphoria, was provided to his parents. More specifically, the team educated the parents that statistically, the child is not more likely to have gender identity disorder as an adult but may identify as homosexual. In addition to education, the parents were provided with resources to continue with outpatient psychotherapy focusing on coping mechanisms should the child's symptoms cause distress or interfere with functioning.

DSM-IV-TR classifies gender identity disorder as cross-gender identification that can manifest in children or adults. In children, the diagnosis is made when four of the following five descriptors are met: the child states repeatedly that he or she is the opposite sex; the child prefers wearing clothing typical of the other sex; the child's play is more typical of the other sex or make-believe play involves taking on roles of the other sex; the child desires to participate in games or pastimes typical of the other sex; and the child prefers playmates of the opposite sex (1). Furthermore, in children the disorder consists of the following descriptors: the child will be uncomfortable with his or her sex, such that boys might state that their penises are undesirable or wish that they did not have a penis or reject stereotypical male play and toys, while girls might urinate standing up or indicate that they wish that they could grow a penis. Additionally, the disorder is not associated with an intersex condition and must cause distress or impair functioning in some way (1). In the above case, the child repeatedly stated that he was a girl, and he preferred clothing, play, and games typical of girls, along with the company of female playmates at school. While he did not discuss his sexual organs or desire their removal, he did eschew male-typical play and typical toys for boys. He did not have an intersex condition. Given that he did not express distress from his symptoms and his functioning was not impaired, a full diagnosis of gender identity disorder was not warranted; rather, he was diagnosed with gender dysphoria. Gender dysphoria is defined as the sense of discomfort resulting from incongruence between gender identity and assigned sex (2).

Intervention for gender dysphoria in children may not be necessary, since most children will not go on to have gender identity disorder in adolescence. One prospective study of children with gender dysphoria found that 15.8% of subjects had persistent gender dysphoria in adolescence (3). Adolescents with gender dysphoria identified the years between ages 10 and 13 as being crucial in determining whether they continued to have gender dysphoria. Another study found that out of 54 children with gender dysphoria, 27% remained gender dysphoric in adulthood (4). In this study, nearly all of the men and women with continued gender dysphoria identified with a homosexual or bisexual orientation; one-half of the men and none of the women without continued gender dysphoria identified as homosexual or bisexual in adolescence. These findings are limited due to small sample sizes. If a child develops signs of gender identity disorder in adoles-

cence, a psychiatric evaluation to confirm a diagnosis of gender identity disorder should be considered, as well as an endocrine evaluation to consider whether hormonal therapy with gonadotropin-releasing hormone is optional. Hormonal therapy will suppress pubertal hormones and, if used, should be started when the physical changes of puberty are evident. Pubertal development of the opposite sex should be initiated at around age 16 using a gradually increasing dosing regimen of the cross-sex steroids (5).

Epidemiology reveals that the mean age of presentation to a pediatric clinic for gender identity disorder symptoms is 14.8 years [SD=3.4] (6). Studies have explored genes and hormones as possible etiologies of gender identity disorder, although individuals with the disorder have no proven genetic, hormonal, or anatomic abnormalities (6). One study found that monozygotic twins were more likely to both have gender identity disorder than dizygotic twins (7). While this may point to possible genetic influences, specific genes have not been located as yet. Furthermore, one aspect of temperament, activity level, may be a possible predisposing biological factor of gender identity disorder. Activity level is usually higher in men than in women, but it is usually higher in girls than in boys with the disorder. One can posit that genes and prenatal hormones may also be factors in the development of the disorder (8).

Psychological factors implicated in gender identity disorder have been investigated. One study described fathers of individuals with the disorder, particularly of male subjects, as more rejecting and hostile than fathers of comparison subjects (9). Mothers of individuals with the disorder were described as more intrusive than mothers of comparison subjects (9). Additionally, women with gender identity disorder exhibit fewer symptoms of mental distress and more stable relationships than men with the disorder (9). In a study of psychiatric comorbidity in adolescents with the disorder, 67.6% of participants had no concomitant psychiatric disorder, 21% had anxiety disorders, 12.4% had mood disorders, and 11.4% had disruptive disorders (10).

Conclusions

Gender identity disorder usually presents in adolescence and may be comorbid with anxiety disorders. Etiology of the disorder is unclear. Children with gender dysphoria are not more likely to develop gender identity disorder as adults but are more likely to identify as homosexual or bisexual. Treatment for children with gender identity disorder may not be necessary, since most children will not continue to have symptoms in adolescence. However, referral to outpatient psychiatry and endocrinology may be considered.

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Transcending the Glass Ceiling: Advancement of Women in Academic Psychiatry

Misty C. Richards, M.D., M.S.

"What exactly does *glass ceiling* mean?" Everyone turned to the medical student sitting sheepishly at the back of the room, who was mortified that her question had drawn so much attention. The speaker at this well-attended psychiatric conference looked surprised and awkwardly dismissed the question. Unfortunately, the metaphor describing the invisible barriers that make it difficult for women to attain the highest positions applies to academic psychiatry as much as anywhere else. Women are underrepresented in psychiatry departments throughout the United States, particularly in the senior faculty rank (1). Specifically, only 38% of psychiatric faculty are women, with only 8% serving in full professor positions (1). The statistics are striking and may be alarming to the ambitious early-career female psychiatrist.

Women face a unique set of challenges. Pregnancy and motherhood, as well as sexism and gender stereotypes, can hinder success in an organizational culture. The structure of the promotion and tenure system rewards hard work and sacrifice in the early years and allows a less rigorous schedule once established. Mothers might prefer the opposite timing, allowing for more flexibility while both their careers and children are young. Feminine stereotypes, such as emotional expressivity and sensitivity may also be considered mutually exclusive to the competitive, hierarchical world of academia. Motivated women will have to work extra hard to overcome these disadvantages.

Several key recommendations that can help women who want to become successful academic psychiatrists are summarized below.

Mentorship. Seek the guidance of several senior mentors who specialize in the areas of psychiatry that interest you. Find a mentor whose work you admire and whose career trajectory you wish to emulate. Establish contact, and introduce yourself and your goals. Engage in this new relationship, and ask to join a project (2).

Networking/leadership. Join national organizations, and pursue leadership roles. Introduce yourself to people with similar interests; your paths will likely cross in the small world of psychiatric subspecialties. Work toward developing a broad-based support system (this may be particularly helpful when extramural letters are needed for promotion). Take the lead in a platform or cause, and the visibility may catapult you past others who may be too timid to do so (3).

Publish. This is a humble way of "blowing your own horn." Strive to feature your work in peer-reviewed journals, although any forum is an accomplishment. The more quality publications you have, the more likely you are to get a promotion or grant (4).

Challenge yourself. This may be most important. Take calculated risks. Serve as a mentor to others, forcing yourself to create a professional life for which you are accountable (5). The higher you set the bar, the higher you will have to rise.

Someday, perhaps all medical students will be clueless about the meaning of a glass ceiling because it will have become an archaic term formerly used to describe a reality that will no longer exist.

Dr. Richards is a first-year psychiatry intern at the Semel Institute for Neuroscience and Human Behavior, University of Los Angeles.

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Perspective

In the Eye of the Storm: A Resident's Perspective on Hurricane Sandy

Carmen Casasnovas, M.D.

What difference can a psychiatry resident make in the middle of a storm? We certainly are not in the "life and death business." We help alleviate difficult situations, providing support and/or medications to temper moods, behavior, and psychosis. We do not pump stomachs or perform cardiac surgery. One would think that during a storm like Hurricane Sandy, few would want to brave the elements and seek mental health care. So when my chief resident called that morning, the last thing on my mind was that he would be asking residents to stay overnight at the hospital. I thought we would be asked to manage ailing patients and those who used mental illness as a reason to obtain shelter. I did not want to leave my apartment unattended or stay at the hospital not knowing where I would be sleeping or taking a shower. Yet I begrudgingly accepted.

Upon arrival at Lincoln Hospital, one of the busiest hospitals in the northeast area, things were not as expected. It was a reality check to find that psychiatrists-in-training did have a role to play during crisis, as even through the raging storm patients needed refills of prescriptions, heard voices, felt depressed, and could not find meaning to life. It did not matter that we were not pulling people from under a pile of bricks or actively breathing life back into our patients. Our patients continued to need us. Our stretchers were full. Life, as defined by our patients, did not pause for Hurricane Sandy.

As the storm gained momentum, so did our psychiatric emergency department. We were immediately called to action. Transferring patients from the crowded emergency department to other hospitals became a priority in view of limited inpatient beds in our facility. Safely discharging as many as possible before the brunt of the storm hit was our mission. Residents became temporary social workers, calling hospitals to set up transfers. Others accompanied patients to their homes in the mobile crisis van. A few continued to evaluate new arrivals to the psychiatric emergency department. The complex wheels of the emergency department became a well-oiled machine.

We "camped" in offices of the outpatient clinic; we scavenged for extra showers to use. Attendings and residents bonded over pizza—fun times until we realized the extent of the devastation. Hot food, running water, and electricity taken for granted by us were not available to many of those who had their lives upended. Families were separated, and hospitals were evacuated. The next day, residents who had not stayed overnight still came to work, regardless of personal issues, and patient care continued.

Hurricane Sandy remains a testament to how an inner city public hospital rose up to the challenge. It was an opportunity to learn about dedication, selfless service, and teamwork. There is continued need for mental health services during times of disaster. Now it is time to help pick up the pieces and start rebuilding the city. We as psychiatrists-in-training need to brace ourselves to manage the emotional and mental aftermath of a natural disaster.

Dr. Casasnovas is a third-year resident in the Department of Psychiatry, Lincoln Medical and Mental Health Center, Bronx, New York.

CALL FOR PAPERS

Submissions for *Psychiatric News* Sought

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.

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.

Book Review

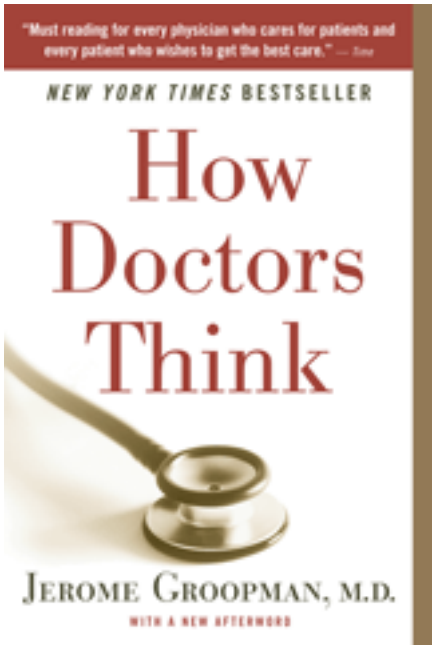
How Doctors Think

by Jerome Groopman, M.D. Boston, Mariner Books, 2008, 336 pp., $15.95.

Reviewed by David Hsu, M.D.

As psychiatrists, we often point out cognitive errors in our patients, but we rarely discuss the cognitive errors that can happen in ourselves. *How Doctors Think* informs physicians and lay readers about the most common biases that can occur in clinical medicine. Since the Institute of Medicine report on medical errors in 2000, much has been written on how physicians make assessments in the face of uncertainty, and this book has been well received by both clinicians and the public alike. The theme for Groopman is that cognition cannot be separated from emotion, and his straightforward writing style makes this wisdom accessible to the lay reader.

Although Groopman discusses several patients who have psychiatric conditions, he writes, in the only footnote in the book, that assessing "how psychiatrists think was beyond my abilities" (p. 7). He admits, "I do not delve into psychiatry in this book" (p. 7). Groopman devotes so much time to the influence of emotions on the reasoning of physicians that perhaps the book could have been titled *How Doctors Feel*. Alcohol dependence, eating disorders, psychotic disorders—many of these conditions can have challenging transference issues if the clinician is not prepared. "You are filled with a sense of disgust" (p. 45), states one of the doctors



interviewed in the book, but Groopman is quick to remind the reader to be mindful of that emotion and to reflect on how emotion will translate into care for the patient. "Once you remove yourself from the patient's story, you no longer are truly a doctor. How a doctor thinks can first be discerned by how he speaks and how he listens" (p. 17).

In response to Groopman's footnote, two psychiatrists published an article titled "How Psychiatrists Think" (1); in which, they stated, "The cognitive style of psychiatrists is surely not so esoteric as to be un-understandable." They argue that psychiatrists are just like any other doctors.

"No doctor is right all the time. Every physician, even the most brilliant, makes a misdiagnosis or chooses the wrong therapy. The doctors didn't stumble because of their ignorance of clinical facts; rather, they missed diagnoses because they fell into cognitive traps. As many as 15 percent of all diagnoses are inaccurate" (p. 24). But Groopman reminds us that in spite of these intellectual challenges, the clinician can still be an astute observer. He concludes, "Machines cannot replace the doctor's mind, his thinking about what he sees and what he does not see" (p. 202). Groopman's stories are infused with humanism and inspiration. *How Doctors Think* should be read by all psychiatrists who seek to improve their clinical acumen in working with complex patients.

Dr. Hsu is a fifth-year, Chief Resident in the Departments of Internal Medicine and Psychiatry, University of California Davis Medical Center, Sacramento, Calif.

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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 Kunal Tank, M.D., a third-year psychiatry resident at the University of Kansas School of Medicine, Kansas City, Kan.

Question #1

Which one of the following medications reversibly inhibits monoamine oxidase enzyme?

A) Tranylcypromine
B) Isocarboxazid
C) Phenelzine
D) Moclobemide

Question #2

Which one of the following medications is a more potent monoamine oxidase uptake blocker?

A) Tranylcypromine
B) Isocarboxazid
C) Phenelzine
D) Selegiline

ANSWERS TO FEBRUARY QUESTIONS

Question #1

Answer: C. Alpha adrenergic antagonist

Trazodone is an alpha adrenergic antagonist and has been implicated in nearly 80% of all drug-induced cases of priapism (1, 2). Selective serotonin reuptake inhibitors and serotonin norepinephrine reuptake inhibitors are not associated with priapism but can cause difficulty initiating or maintaining an erection, difficulty becoming aroused, and delayed orgasm. Dopamine antagonists are associated with impaired orgasm and ejaculation due to increased prolactin levels.

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Question #2

Answer: B. Pseudoephedrine

Pseudoephedrine is an alpha-agonist agent that exerts a constriction effect on smooth muscle of corpora cavernosum that in turn facilitates venous outflow (1). Acetaminophen, sertraline, and lidocaine are not agents used in treatment of priapism.

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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. Vahabzadeh: arshya.vahabzadeh@emory.edu.**

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 Issue Themes

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

May 2013

Section Theme: DSM-5
Editor-in-Chief: Monifa Seawell, M.D.
mseawell@med.wayne.edu

June 2013

Section Theme: Psychiatry and Social Justice
Guest Section Editor: Megan Testa, M.D.
Megan.testa@mh.ohio.gov

July 2013

Section Theme: Open
Editor-in-Chief: Monifa Seawell, M.D.
mseawell@med.wayne.edu

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