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Inside

IN THIS ISSUE

This issue of the *Residents' Journal* features a variety of topics. Jennifer H. Harris, M.D., discusses prevention of posttraumatic stress disorder, with an overview of various responses to trauma. Ammar El Sara, M.B.Ch.B., presents a review of clinically applicable evidence-based interventions targeting obesity in schizophrenia patients. Stephanie Pope, M.D., examines antipsychotic-induced hyperprolactinemia, including variables affecting prolactin and clinical implications. Elizabeth S. Stevens, D.O., discusses several psychological, social, and spiritual developmental frameworks in a clinical case conference. Kamalika Roy, M.D., presents a case of priapism as a side effect of trazodone in a middle-aged patient. Lastly, Aaron J. Hauptman, M.D., offers his review of the book *Classifying Psychopathology: Mental Kinds and Natural Kinds.*

EDITORIAL

New Formats and New Opportunities: The Time to Get Involved is "Now"!

Rajiv Radhakrishnan, M.B.B.S., M.D., Editor-in-Chief

It is an exciting time at the *American Journal of Psychiatry Residents' Journal* Editorial Board as we plan ahead for the new academic year. This is also a special year, since it marks the 10th year of the journal since its inception in 2006.

We have seen remarkable growth in the journal over the past year, with approximately three-fold greater engagement by residents and fellows. Many of our Guest Section Editors and Editorial Board members of the past year have gone on to obtain fellowship positions and many academic awards at the national level. Hearty congratulations to them on their outstanding achievements and a heartfelt thank you for their contributions to the journal! The above facts speak to not only the educational content of the journal but also to the immense academic opportunities that participation with the journal provides.

At the beginning of the year, we asked ourselves, "What can we do to further improve the educational content of the journal and improve participation by medical students, residents, and fellows?" To answer these questions, we conducted a nation-wide survey of medical students, residents, and fellows and received an impressive number of responses. The majority of respondents indicated that they would like to see more articles on psychopharmacology, clinical treatment, neuroscience, and DSM-5.

In response to the survey, we are in the process of implementing some changes to the format of articles in the journal. In the coming months, medical students, residents, and fellows will have an opportunity to submit reviews of pharmacological medications in the new manuscript type called "Drug Review," as well as articles in another new manuscript type called "History of Psychiatry." These new formats, along with our existing formats on "Treatment in Psychiatry" and "Clinical Case Conference," will allow us to cover topics on psychopharmacology, clinical treatment, neuroscience, and DSM-5. Soon, each article will be accompanied by a table that lists "Key Points/Clinical Pearls" to enable readers to peruse key teaching points despite their busy schedules. Readers will also have an opportunity to answer quiz questions related to an issue. Additionally, authors will have an opportunity to create podcasts for their articles that would be featured on our Facebook page (https://www.facebook.com/AJPResidentsJournal). We also have a new landing page that lists the article titles of an issue. The titles are hot-linked and take the reader directly to the article when clicked. (Check it out here: http://ajp.psychiatryonline.org/residents_journal).

We have seen remarkable growth in the journal over the past year.

This is indeed an exciting time for the *American Journal of Psychiatry Residents' Journal* and a great time to get involved with the journal. Once you have successfully published in the journal, we offer additional opportunities to serve as a Guest Section Editor, receive a free book for writing a book review, and join the Editorial Board. This year, we had 14 applicants who competed to secure a place on the Editorial Board.

I'm very happy to introduce our fabulous new Editorial Board for this academic year: Senior Deputy Editor, Katherine Pier, M.D., from the Icahn School of Medicine at Mount Sinai; Deputy Editor, Hun Millard, M.D., M.A., from Yale University School of Medicine; and Associate Editors, Rafik Sidaros, M.B.B.Ch., from SUNY Downstate Medical Center, and Janet Charoensook, M.D., from the University of California, Riverside. We look forward to working with you to transform the *Residents' Journal* into a leading educational resource for residents and fellows.

The time to get involved is now!

Dr. Radhakrishnan is a fourth-year psychiatry resident in the Department of Psychiatry, Yale University School of Medicine. He is the past Senior Deputy-Editor (2014–2015), and new Editor-in-Chief (2015–2016) of the *American Journal of Psychiatry Residents' Journal*.

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ARTICLE

Prevention of Posttraumatic Stress Disorder: Predicting Response to Trauma

Jennifer H. Harris, M.D.

> "Although the world is full of suffering, it is full also of the overcoming of it."
>
> —*Helen Keller*

Most people in their lifetime will experience a traumatic event. In one study, 69% of people experienced "a violent encounter marked by sudden or extreme force and involving an external agent of nature, technology, or humankind" (1). But what determines why people respond so differently to trauma? Why are some people able to maintain normal functioning, while others respond pathologically with posttraumatic stress disorder (PTSD)?

RESPONSES TO TRAUMA

Researchers continue to debate about the exact nature of *resilience*, but most definitions include a concept of healthy, adaptive, or integrated positive functioning over time in the aftermath of adversity (2). Resilience is not simply "lack of psychopathology." Resilience is one of several possible trajectories in response to traumatic events characterized by a brief period of disequilibrium after trauma followed by continued health (Figure 1) (2).

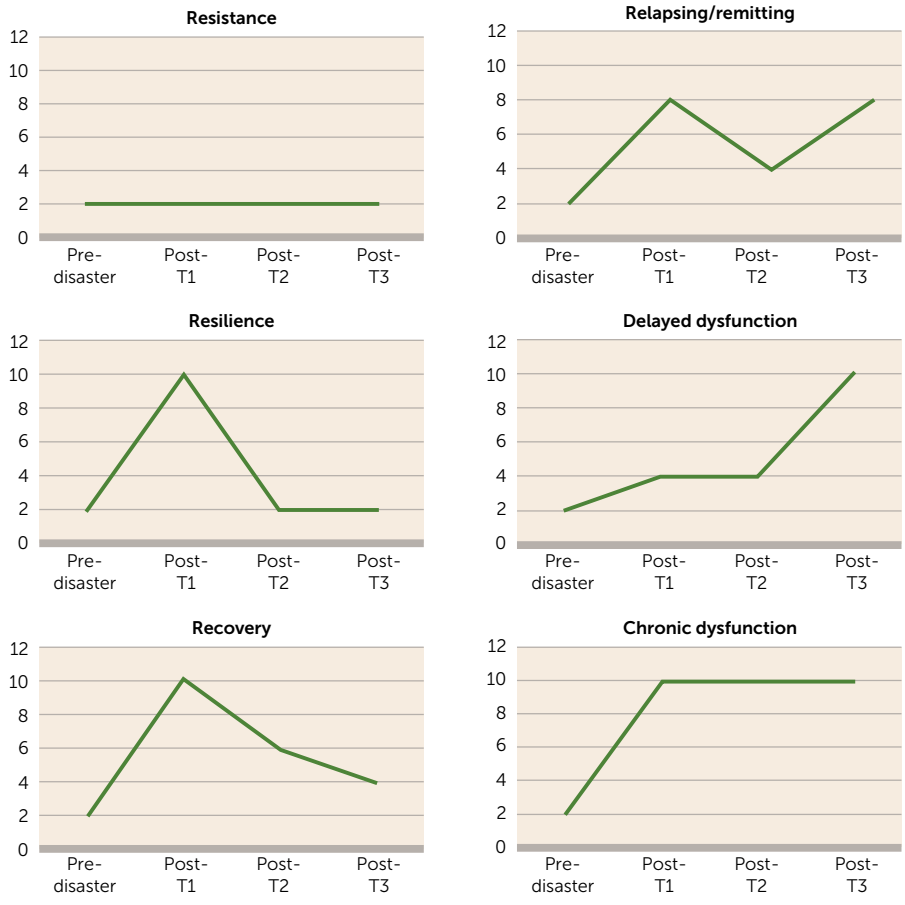
The ideal response to stress is *resistance*, a trajectory in which adaptive capacities have absorbed the impact of the stressor and there is no dysfunction at all (3). In reality, some level of distress is universal immediately after the trauma. A total of 96% of individuals directly exposed to the Oklahoma City bombing reported at least one posttraumatic symptom (4, 5). Immediately after trauma exposure, most individuals meet DSM criteria for intrusive (group B) symptoms and cognitive and mood (group D) symptoms (5). The presence of avoidance (group C) symptoms in particular has been strongly associated with meeting the full diagnostic criteria for PTSD (5). This suggests that intrusion and hyperarousal are normal responses to trauma and that avoidance may actually comprise the pathological part of PTSD.

Several studies have consistently shown that PTSD develops in about one-third of those exposed to trauma (1, 6, 7). Yet a resilient response is in fact more common (35%–65%), suggesting that the majority of the population possesses the capacities to adapt quickly to stressful events (8).

PATHOPHYSIOLOGY OF PTSD

Sensory input is appraised as stressful by the limbic brain structures, which include the amygdala, hippocampus, and

FIGURE 1. Hypothesized Trajectories of the Course of Stress Responses With Posttraumatic Stress Disorder Symptoms on the Y-Axis[a]



[a] Reprinted with permission from Norris FH, Tracy M, Galeo S: "Looking for Resilience: Understanding the Longitudinal Trajectories in Response to Stress." Soc Sci Med 2009; 68:2190–2198. Copyright © Social Science and Medicine 2009.

TABLE 1. Predictors of Response to Trauma

Risk Factors	Pathological Trajectory/ Posttraumatic Stress Disorder (PTSD)	Resilient Response Trajectory
Sympathetic nervous system hypothalamus-pituitary-adrenal axis	Increased activation after event	Decreased activation after event
Neuropeptide Y	Lower neuropeptide Y	Higher neuropeptide Y
Early life	Uncontrollable stress during infancy and childhood	Good maternal care; exposure to manageable stressors
Psychiatric comorbidities	Anxiety disorder; Illicit drug use; previous PTSD	
Trauma type	Assault; sexual trauma	Disaster (if previously resilient)
Gender	Female	Male
Age	Older age	Younger age
Education	Lower education	Higher education
Personality		High perceived control; trait self-enhancement; cognitive reappraisal
Social		Strong social/emotional support

prefrontal cortex. The stress response is chiefly mediated by the sympathetic nervous system and hypothalamus-pituitary-adrenal (HPA) axis. Resilient individuals can restrict the activation of the stress response to the stressful event; individuals with PTSD exhibit continued overactivation after the event has passed (9). This overactivation is mediated by corticosteroids, which reduce the branching and length of dendrites and the number of synaptic contacts, suppress cell proliferation, and increase cell death. Timing is crucial, as corticosteroids produce effects only when cells are concurrently excited by noradrenaline (10).

Genetic polymorphisms have been identified in the sympathetic nervous system, HPA axis, neuropeptide Y, and serotonin systems that predispose individuals toward pathological response to trauma. Variations in the corticotropin-releasing hormone type 1 receptor genes, as well as brain mineralcorticoid and glucocorticoid receptor genes, set the threshold and regulate the termination of the HPA response to stress and have been shown to predict the development of PTSD (10, 11).

Neuropeptide Y, a neurotransmitter in the brain and autonomic nervous system, counteracts the anxiogenic effects of corticotropin-releasing hormone in the limbic areas of the brain. Rodent studies have shown that administering neuropeptide Y both inhibits the development of fear conditioning and promotes the extinction of stress responses. These findings correlate with studies in humans showing that combat veterans without PTSD have higher levels of neuropeptide Y than those who have developed PTSD (11).

Brain-derived neurotrophic factor is an important nerve growth factor whose levels are affected by antidepressants. Chronic stress decreases brain-derived neurotrophic factor levels in the hippocampus but increases levels in the nucleus accumbens. Mice with an allele associated with decreased brain-derived neurotrophic factor function show increased anxiety and impaired hippocampus learning but are in fact more resilient to chronic stress (11).

Many of these neurotransmitter systems exert opposite and region-specific effects, suggesting that there are unlikely to be simple pharmacological therapies to prevent pathological stress responses.

EARLY-LIFE CONDITIONING

Early-life conditioning shapes the development and responsiveness of these systems, likely through epigenetic changes. Uncontrollable stress during the key developmental periods of infancy and childhood can lead to learned helplessness and chronically high levels of corticotropin releasing hormone and cause exaggerated responses to stress later on in life (9, 11). Good maternal care has been shown to protect against future stressors. Studies of rat pups found that those who experienced higher maternal care (as measured by licking and grooming) showed decreased stress activation and decreased anxiety behavior that persisted through adulthood compared with pups who received less maternal care (12).

EPIDEMIOLOGICAL RISK FACTORS

The presence of an anxiety disorder and illicit substance abuse predicts the occurrence of trauma (13). Preexisting anxiety disorder has been associated with an increased risk of trauma, apparently due to an increased tendency to report events as being horrific. On the other hand, presence of illicit drug use likely increases the risk of assaultive and sexual traumatic exposures.

After the traumatic event, the subsequent development of PTSD is most strongly predicted by female gender and assault or sexual trauma (Table 1) (5, 13). Resilient outcomes have been associated with male gender, older age, and higher levels of education (8). Previous development of PTSD in response to trauma also tends to predict future development of PTSD. However, previous resilient responses predict future resilience only in particular types of trauma, such as disaster (8).

PERSONALITY FACTORS

Whether a person considers a stressor to be a threat or a challenge to be mastered reflects beliefs about his or her own helplessness or agency. Individuals with traits of high perceived control, who utilize active rather than passive responses to threats, show lower glucocorticoid responses and are less likely to develop PTSD (8, 11).

One of the personality traits most strongly associated with resilient responses is trait self-enhancement. Trait self-enhancers are individuals who habitually engage in overly positive and self-serving biases, and although these traits may evoke negative reactions from other people, they also tend to enable resilient responses to trauma (8). These biases have been correlated with higher activation of the mesolimbic dopamine reward circuitry to facilitate fear extinction (11).

Another predictor of resilience is cognitive reappraisal, the ability to positively reframe and draw meaning, purpose, and strength from negative events. Cognitive reappraisal reflects the strength of the prefrontal cortex and its ability to regulate the limbic system (2, 9).

In addition, the presence of a strong social and emotional support system is associated with resilient responses to trauma. A strong social support system reflects an individual's psychosocial health and buffers responses to trauma (14). High levels of social support have been positively associated with active problem-focused coping, a sense of control and predictability in life, and dampened neuroendocrine and cardiovascular responses to stress (9).

INTERVENTIONS

Pharmacological Interventions

Pharmacologic treatment of PTSD after symptoms have become manifest has been extensively studied. Of recent interest is whether interventions prior to the development of symptoms can prevent psychopathology. Theoretically, several classes of drugs show promise for preventing PTSD, such as antidepressants and antiadrenergic agents (9, 15).

However, a 2014 Cochrane review of published studies found no evidence to support the efficacy of propranolol, escitalopram, temazepam, and gabapentin in preventing PTSD onset. Only hydrocortisone has demonstrated significant efficacy in preventing the development of PTSD (16). One theory is that a constant level of corticosteroid blocks the effect of the HPA axis activation and therefore reduces the emotional response and disrupts the over-consolidation of traumatic memories (10).

Psychological Interventions

Psychological interventions to prevent the development of PTSD have been studied more extensively than pharmacological interventions, but the results to date have been similarly disappointing. Initial efforts have focused on single-session early psychological debriefing. Results have consistently shown that single-session interventions are not helpful and may actually be harmful. In some studies, the intervention group reported more PTSD symptoms compared with the comparison group (8, 17).

Recent efforts have focused on multiple-session cognitive-behavioral therapy (CBT) for postexposure PTSD prevention. CBT generally falls under three subtypes: 1) exposure therapies, such as systematic desensitization and flooding, 2) anxiety management, which includes relaxation and self-distraction, and 3) cognitive therapy, which seeks to correct harmful cognitions.

Reviewing all the data on psychological interventions for prevention of PTSD up to 2007, a Cochrane analysis found no significant difference in the development of PTSD between psychological intervention and comparison groups (18). Treatment programs that involve a screening mechanism to identify individuals at high risk for developing PTSD or individuals manifesting acute stress disorder have shown better results (8, 18, 19).

CONCLUSIONS

Can resilience be learned or fostered? One difficulty with developing general PTSD prevention programs is that the known predictors of PTSD together account for, at best, only 27% of the variance in response to traumatic events (20). No single factor accounts for resilience; responses to stress are a result of subtle genetic polymorphisms and the accumulation of external events that produce vulnerable phenotypes (8, 10). However, as we continue to gain a better understanding of the human stress response, there is hope that we can develop a more effective multipronged strategy for preventing PTSD and promoting positive adaptation.

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ARTICLE

Weight Gain in Patients With Schizophrenia: A Recipe For Timely Intervention

Ammar El Sara, M.B.Ch.B.

Obesity is a key feature of the metabolic syndrome, with significant implications for long-term morbidity and mortality (1). Obesity is more prevalent among individuals with serious mental illness than in the general population (2).

Alarmingly, and despite improvements in health services worldwide, standardized mortality ratios in schizophrenia, especially cardiovascular mortality, appear to be increasing (3, 4). In addition, the long-term influence of second-generation antipsychotic drugs on adverse metabolic outcomes is a major reason for concern.

The aim of the present review is to propose an outline of clinically applicable evidence-based interventions that target obesity in individuals with schizophrenia, from both primary and secondary preventative points of view.

SCREENING

A problem cannot be remedied if it is not recognized. All patients should have their metabolic profiles routinely monitored, with more frequent monitoring for patients with significant weight gain or those taking specific antipsychotics (5). Despite being a low-cost/high-yield intervention, there is strong evidence that metabolic monitoring is alarmingly low in individuals prescribed antipsychotics (6).

LIFESTYLE INTERVENTIONS

Overview

In a systematic meta-analysis, Bonfioli et al. (7) examined 13 randomized controlled trials of nonpharmacologic interventions for weight management in patients with psychosis.

The interventions included cognitive-behavioral, psycho-educational, and nutritional- and physical activity-based interventions. Four studies evaluated individually delivered interventions, while the other nine evaluated group interventions. Interventions lasted for a mean of 18 weeks, with follow-up periods of up to 3 months. Participants were mostly outpatients, along with stable long-term inpatients and supported housing residents.

Overall, nonpharmacological interventions resulted in a 0.98-kg/m^2 reduction in body mass index (BMI) (or a loss of 3.12% of the initial weight), which is well below the 5%–10% proposed by the National Institutes of Health/National Heart, Lung, and Blood Institute. This could be explained in part by the short duration of intervention/follow-up. Interestingly, there was no difference in the dropout rates between experimental and control groups, suggesting that interventions were feasible and well accepted by patients.

The interventions with the highest impact were those of a preventive nature (as opposed to weight loss interventions), along with individual interventions that included diet and physical activity.

Addressing Diabetes

Designing and implementing a lifestyle intervention for individuals with schizophrenia becomes inherently more complicated when diabetes is a comorbidity.

Cimo et al. (8) systematically reviewed the literature on nonpharmacological interventions targeting the comorbidity of schizophrenia and type 2 diabetes. Despite an extensive literature search, they only identified four original studies (two randomized controlled trials, two retrospective trials). This highlights the significant gap in our knowledge regarding the customization of standard diabetes-management interventions for people with mental illness. Participants were inpatients in two studies, with ages ranging between 44 and 53. Both inpatient and outpatient interventions were built on diabetes self-management principles, with emphasis on simplification of instructions, gradual introduction of new concepts and skills, incorporating memory aids, and continuous motivational enhancement. In one intervention, patients were provided with exercise facilities. The length of interventions in these four studies was significantly longer than those in the nondiabetic population, ranging between 6 months and 1 year. Overall, both in- and outpatient interventions had a positive impact on weight, BMI, and blood glucose measurements.

"ACHIEVE"ing Weight Loss

The ACHIEVE trial (9) is a rigorously designed, large trial, in which 291 obese patients with severe mental illness attending a community outpatient psychiatric rehabilitation program were randomly assigned to an 18-month long behavioral weight-loss intervention or a control intervention. Ninety percent of these patients were taking an antipsychotic. The intervention consisted of individual and group weight-management sessions, as well as group exercise sessions. The educational component was tailored to the population's learning capabilities, with small incentives to encourage attendance. Patients in the intervention arm lost significantly and progressively more weight (average of 3.2 kg at 18 months). In addition, 37.8%

lost 5% or more of their body weight, compared with 22.7% in the control arm. This study represents strong evidence for the utility of long-term, intensive, tailored lifestyle interventions in overweight and obese adults with serious mental illness.

A "STRIDE" in The Right Direction

Another landmark study is a recently published randomized controlled trial of weight loss and lifestyle intervention for individuals taking antipsychotics (10). The STRIDE study recruited 200 overweight patients, 91% of whom received second-generation antipsychotics. The first 6 months of the year-long intervention consisted of weekly standalone group meetings, followed by 6 monthly group meetings focusing on weight maintenance and motivational enhancement.

The STRIDE intervention was based on the PREMIER lifestyle intervention with the DASH diet (11, 12) and on guidelines for obesity treatment for individuals at risk for cardiovascular disease (13). Components of the STRIDE intervention protocol are presented in Table 1.

At 12 months, patients in the intervention arm lost 2.6 kg more than the control group, along with a reduction in mean fasting glucose from 106.3 mg/dl to 100.4 mg/dl. Moreover, 6.7% of intervention patients compared with 18.8% of control subjects required medical hospitalization during that year.

In addition to being the first randomized controlled trial to show reductions in glucose levels and in the number of medical hospitalizations, the SRIDE study lends itself to implementation in most outpatient settings. The materials and instructions needed to run a STRIDE-based group (including a detailed "Facilitator Guide," a "Food and Fitness Diary," and a "Participant Workbook") are available online free of charge (www.kpchr.org/stridepublic).

TABLE 1. STRIDE Core Intervention Components

Component
Increasing awareness through monitoring: diet, physical activity, and sleep
Creating personalized diet and physical activity plans
Reducing calories
Reducing portion sizes, identifying and choosing alternative foods, modifying meals
Increasing consumption of fruits, vegetables, fiber, and low-fat dairy products
Increasing physical activity
Developing action plans for high-risk eating situations
Graphing progress and making adjustments
Addressing mental health effects on lifestyle-change efforts

PHARMACOLOGICAL INTERVENTIONS

Switching Antipsychotics

Weight gain has long been considered an intrinsic side effect of antipsychotic medication, with suggested mechanisms including 5-HT_{2c} antagonism, H_1 antagonism, peripheral M_3 muscarinic receptor antagonism, hyperprolactinemia, and leptin desensitization (14).

Antipsychotics differ significantly in their capacity to induce weight gain (15). Antipsychotics most associated with weight gain are clozapine and olanzapine, while chlorpormazine, iloperidone, quetiapine, and risperidone fall under the moderate weight gain category. The antipsychotics associated with the least weight gain include ziprazidone, haloperidole, aripiprazole, asenapine, and lurasidone.

When clinically feasible, carefully switching one antipsychotic to another with lesser potential for causing weight gain could be an effective strategy (16).

Add-On Medications

In the most recent and extensive systematic meta-analysis of pharmacological interventions for antipsychotic-induced weight gain, Mizuno et al. (17) have identified and analyzed data from 40 randomized controlled trials between 1950 and November 2013. Conclusions from this meta-analysis are discussed below.

With change in weight as the primary outcome, data on 19 unique interventions were pooled, including: amantadine, aripiprazole, atomoxetine, D-fenfluramine (nonselective 5-HT agonist), dextroamphetamine, famotidine, fluoxetine, intranasal insulin, metformin, nizatidine (H_2 competitive inhibitor), orlistat (reversible inhibitor of gastric and pancreatic lipases), phenylpropanolamine (alpha$_1$ agonist), reboxetine (selective norepinephrine reuptake inhibitor), reboxetine-betahistine combination (betahistine is an H_1 agonist, H_3 antagonist), rosiglitazone, sibutramine (norepinephrine and 5-HT reuptake inhibitor), metformin-sibutramine combination, topiramate, and zonisamide (exact mechanism of action is not known).

It is important to note that some of these medications are no longer available in U.S. markets, including D-fenfluramine (withdrawn in 1997 after reports of heart valve disease) and sibutramine (withdrawn in 2010 after being associated with a higher risk of nonfatal myocardial infarction and nonfatal stroke). While widely available, amantadine may (theoretically at least) exacerbate psychosis, and the use of orlistat while not adhering to a low-fat diet will result in fatty diarrhea and malabsorption of orally administered medications.

Metformin was the most extensively studied medication, with a –3.17-kg mean difference in body weight. Dosage range was 500 mg/day–2,550 mg/day. Despite the common side effects of diarrhea, nausea, and flatulence, metformin was generally well tolerated. It should not be prescribed for individuals with impaired creatinine clearance.

Patients who received both adjunctive metformin and lifestyle changes did better in terms of weight loss and overall metabolic profiles than those receiving lifestyle changes plus placebo or placebo alone (18).

Topiramate, sibutramine, aripiprazole, and reboxetine were also separated from placebo in the meta-analysis. Aripiprazole was associated with modest

but significant effects on weight gain. These results, however, should be interpreted in the context of the risks and benefits of antipsychotic polypharmacy.

When secondary outcomes were analyzed (including fasting glucose, HbA1c, fasting insulin, insulin resistance, cholesterol, and triglycerides), metformin and rosiglitazone improved insulin resistance, while aripiprazole, metformin, and sibutramine decreased blood lipids.

First-episode patients seemed to derive the most benefit from all effective interventions, highlighting the role of metformin in primary (starting metformin and antipsychotics concurrently), as well as secondary, prevention of antipsychotic-related weight gain.

SUMMARY

Prescribers should be cognizant of the long-term morbidity and mortality implications of weight gain on individuals with schizophrenia. All such patients should have their metabolic profiles routinely screened.

The body of evidence supporting the utility of early, long-term, tailored, multipronged lifestyle interventions is constantly growing. Such interventions need to be widely disseminated and integrated with other modalities of interventions.

When lifestyle interventions alone are insufficient, and switching antipsychotics to relatively weight-neutral agents is not possible, adjuvant metformin is the first choice among multiple possible pharmacological interventions. A combination of adjuvant metformin and a lifestyle intervention is feasible and effective.

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For additional information on the topic of weight gain and schizophrenia, see the seminal study on metformin for treatment of antipsychotic-induced weight gain by Ren-Rong Wu, M.D., Ph.D., et al., in the *American Journal of Psychiatry*.

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ARTICLE

Hyperprolactinemia and Antipsychotics: Update for the Training Psychiatrist

Stephanie Pope, M.D.

Antipsychotic-induced hyperprolactinemia has become a larger concern in psychiatry given the growing number of antipsychotics, larger focus on the short- and long-term impacts of such medications, and recent litigation. One study found that in patients treated with antipsychotics for severe mental illness, 18% of men and 47% of women were asymptomatic but had elevated serum prolactin levels (1). In another study examining those with schizophrenia treated with antipsychotics, 42% of men and 75% of women suffered hyperprolactinemia (2).

Recent research has expanded our understanding of antipsychotic-induced hyperprolactinemia beyond blockade in the tuberoinfundibular tract. Other variables affecting serum prolactin levels, along with dopamine antagonism, include D_2 receptor polymorphisms, endogenous and exogenous estrogens, and smoking status. In patients treated with antipsychotics, recent literature suggests regularly monitoring serum prolactin and assessing for hyperprolactinemia symptoms, as well as making pharmacological changes to a less offending agent if symptoms or levels become problematic.

VARIABLES AFFECTING PROLACTIN

Tuberoinfundibular Pathway and Physiologic Prolactin

Because antipsychotics cause dopamine blockade in the mesocortical and mesolimbic dopaminergic pathways, they also cause blockage in the tuberoinfundibular pathway. This pathway, as it projects from the hypothalamus to the anterior pituitary, regulates the release of prolactin. Prolactin is released and responsible for galactorrhea and amenorrhea either in a physiological functional way, such as in the case for postpartum women, or as a side effect of antipsychotic medications. With the presence of antipsychotics blocking dopamine receptors in this pathway, prolactin levels increase and can cause galactorrhea, amenorrhea, infertility issues, demineralization of bone, sexual dysfunction, and weight gain (3). Dopamine blockade is an important component of antipsychotic-induced hyperprolactinemia.

Dopamine Blockade

The more dopamine blockade an antipsychotic offers, the stronger the association with hyperprolactinemia. Agents such as first-generation antipsychotics and risperidone are known offenders, but any antipsychotic medication with dopaminergic blockage could theoretically cause hyperprolactinemia (4). In a study using the data set from the Clinical Antipsychotic Trials of Intervention Effectiveness, the relationship between hyperprolactinemia and the D_2 occupancy of risperidone, olanzapine, and ziprasidone was demonstrated. In that study, the threshold of D_2 occupants was slightly below the therapeutic window for these antipsychotics. This suggests that therapeutic effect encompasses D_2 blockade. Therefore, when using an antipsychotic for therapeutic effect, consideration should be made for prolactin elevation (5).

Age

Along with the dopamine blockade properties of a pharmacologic treatment, age is also an important component of changes in serum prolactin. Younger patients seem to be more susceptible to antipsychotic-induced prolactin changes (4). In adults, it seems that the elevation of prolactin from risperidone remains for at least 1 year (2). The association between age and antipsychotic-induced hyperprolactinemia is also compounded by the patient's sex, since estrogen is a risk factor.

Estrogen

It is now known that endogenous and exogenous estrogens are associated with a higher increase in prolactin from antipsychotics. One study examined serum prolactin levels in healthy men and women who were given haloperidol, reserpine, aripiprazole, or placebo. In the haloperidol and reserpine groups, serum prolactin levels were elevated in contrast to the aripiprazole and placebo groups. Meanwhile, the increase in the haloperidol and reserpine groups was significantly higher in women compared with men. Lastly, women taking hormonal contraception had a higher increase than women not taking hormonal contraception. This study also supports findings from the literature that aripiprazole, a partial dopamine agonist, is a protective against antipsychotic-induced hyperprolactinemia (6). Use of aripiprazole in antipsychotic-induced hyperprolactinemia is an important concept that may warrant further investigation.

Addition of Aripiprazole

Considering the idea that aripiprazole maintains prolactin levels, a study assayed the prolactin serum levels of female patients with schizophrenia with risperidone-induced hyperprolactinemia who were then given aripiprazole. The dosages were increased every 2 to 4 weeks, from 3 mg/day to 12 mg/day. Serum prolactin levels were obtained at baseline and then every 2 to 4 weeks. Serum prolactin levels after receiving

aripiprazole were lower than levels at baseline but without significant change in serum concentration or reduction ratios among the 6, 9, and 12 mg/day dosages. This study suggests that adjunctive use of aripiprazole reduces prolactin in risperidone-induced hyperprolactinemia (7).

CLINICAL IMPLICATIONS

Clinical Manifestations

Clinical sequelae of hyperprolactinemia are either from distortions in the hypothalamic-pituitary axis or direct effects on specific tissues. Symptoms of hyperprolactinemia can include amenorrhea, breast enlargement or engorgement, galactorrhea, decreased libido or erectile dysfunction, or disruptions in pubertal changes. Those with hyperprolactinemia are at higher risk of benign breast tumors and disruption in bone density. In adolescents, this bone loss can persist for up to 2 years after antipsychotics have been discontinued. Premenopausal women with schizophrenia treated with antipsychotics known to increase prolactin have higher rates of osteoporosis compared with those treated with olanzapine (8).

Monitoring Serum Prolactin

Laboratory serum testing for prolactin levels is an appropriate monitoring tool and should be considered annually (9). It has been suggested that a baseline prolactin level be considered for all pediatric patients starting risperidone therapy, especially in those who have reached sexual maturity. Serum prolactin levels should be checked every 2 months after initiation, along with screening questions for symptoms of hyperprolactinemia. If any level is above 100 ng/mL, imaging is recommended to evaluate for a tumor (10). It is important to note that the estimated prevalence of asymptomatic pituitary tumors is common, and any lesions found could be misinterpreted as being related to an antipsychotic treatment (11).

Others suggest that if the serum level is elevated without reported symptoms, it is recommended to repeat the level in another 2 months. If the patient is benefiting from risperidone therapy and experiencing mild hyperprolactinemia symptoms, it is reasonable to continue treatment for 6–12 months. This recommendation is based on noted evidence that serum levels peak 1–2 months after starting risperidone but then return close to normal within 3–5 months (10). Another study found a similar increase at around 2 months but an eventual return close to normal near 22 months (12). Other studies have found that elevations of serum prolactin can be detected at 6 months, and patients have been symptomatic within weeks of increases in antipsychotic dosage (13). Once hyperprolactinemia is suspected, the differential diagnosis should be considered.

Differential Diagnosis

Hyperprolactinemia can come from a variety of endocrine or nonendocrine pathologies. Endocrine pathologies include craniopharyngoima, sellar or parasellar masses, granulomatous infiltration of the hypothalamus, head trauma, and pituitary adenomas. Nonendocrine pathologies associated with increased serum prolactin levels include chronic renal failure, chronic hepatic failure, and pregnancy polycystic ovarian syndrome. Interestingly, hypothyroidism is known to elevate serum prolactin in some cases, but the mechanism is unclear. Other nonpathologic causes of elevated prolactin include pregnancy, exercise, eating, stress, sexual intercourse, and chest wall stimulation. Medications, other than antipsychotics, known to elevate prolactin levels include antidepressants (such as tricyclic antidepressants, monoamine oxidase inhibitors, and selective serotonin reuptake inhibitors), opiates, antihypertensives, metoclopramine, and domperidone, as well as the stimulant cocaine. Medications thought to possibly increase serum prolactin include H_2 blockers, protease inhibitors, and estrogens. Lastly, idiopathic hyperprolactinemia should also be considered in the differential for hyperprolactinemia (9). If hyperprolactinemia is due to an antipsychotic and does not remit with time, a different pharmacologic treatment regimen should be considered.

Pharmacologic Treatment Options

The question about which antipsychotics are less associated with hyperprolactinemia has been studied in various ways. If a patient is experiencing hyperprolactinemia symptoms with noted serum elevation, it is recommended to discontinue the medication for 3 days or substitute another medication and recheck serum prolactin. If the medication cannot be stopped, it is recommended to obtain imaging to differentiate between a pituitary or hypothalamic mass and antipsychotic-induced etiology (9).

A meta-analysis of the efficacy and tolerability of antipsychotics in schizophrenia found that aripiprazole was the least associated with an increase in prolactin levels, while paliperidone was the worst offender (14). A recent literature review addressed such concerns in relationship to the newest antipsychotics (15). This review suggests that amisulpride, risperidone, and paliperidone have the strongest association, while aripiprazole and quetiapine have the lowest. This review also suggests that asenapine and iloperidone are comparable to clozapine, while lurasidone is comparable to ziprasidone and olanzapine (15).

Overall, these associations are dose-dependent, and the profiles are similar for children and adults (15). Antipsychotics known to be less offending than risperidone include, in no specific order, aripiprazole (16), ziprasidone (17), quetiapine (18), and paliperidone (19). Switching from risperidone to aripiprazole in adolescents with autism spectrum disorder requiring pharmacotherapy has been shown to be well tolerated (20).

CONCLUSIONS

Hyperprolactinemia associated with antipsychotics, specifically risperidone and paliperidone, should be considered. Baseline and periodic serum prolactin levels should be obtained, along with monitoring for symptoms associated with hyperprolactinemia. If such a concern arises, and it does not improve with time or is too severe, a switch to another

antipsychotic, such as aripiprazole, is warranted.

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2. Answers should be complete and include detailed explanations with references from pertinent peer-reviewed journals, textbooks, or reference manuals.

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CASE REPORT

A Clinical Case Conference on Spiritual Growth and Healing

Elizabeth S. Stevens, D.O.

Spiritual growth, along with cognitive and psychosocial development, often occurs during developmental transitions. The onus for spiritual growth may be life crises, disruptions, intrapsychic conflict, or environmental changes. Developmental crises can either shake one's worldview or mature one's faith to become a coping mechanism during life's transitions.

In the present clinical case conference involving psychiatry faculty and residents examining the development of spiritual growth and healing, several developmental theories illuminate the discussion: James Fowler's work on stages of faith, which utilizes Jean Piaget's theory on cognitive development and Erik Erikson's theory on psychosocial development. Fowler posits that faith develops through a series of predetermined stages, leading to either successful or impoverished relationships and self-perception (1). Key points of the three theories of Fowler, Piaget, and Erikson are presented in Table 1. Faith interacts with cognitive and emotional regulatory systems at each developmental transition. In Stages of Faith, Fowler asserts that "faith affects the shaping of our initiatives and responses, as well as our relationships and aspirations in life by enabling us to see them against the backdrop of a comprehensive image of what constitutes true power, true value, and the meaning of life" (2). In what Fowler describes as initial stage, "undifferentiated faith," infants develop a rudimentary foundational faith with a basic trust and hope built upon secure attachments. In "intuitive-projective faith," one's faith is magical, illogical, and fantastical as one acquires language, moral emotions, and a sense of the sacred. Representations of God correlate with one's experiences of attachment figures. "Mythic-literal faith" is a logical, literal, and realistic faith perspective. "Synthetic-conventional faith" is an emotional investment in one's faith without critical, independent evaluation of it. Relational stressors can produce lasting negative effects on one's spirituality, value, and intimacy with God (2). In "individuative-reflective faith," one claims an autonomy and responsibility for one's faith without relying on an external authority. In "conjunctive faith," one embraces and integrates polarities of life, beyond the explicit ideological system. In "universalizing faith," one's faith overcomes division, oppression, and violence (2).

This case report illustrates key points of Fowler's theory, using a psychological, social, and spiritual developmental framework (3).

CASE

A 40-year-old divorced, Caucasian man with no previous psychiatric history presented to an emergency department with depressed mood and neurovegetative symptoms of depression, reporting feelings of "failure as a father and as a Christian."

The patient reported 2 months of insomnia, depressed mood, decreased interest, decreased energy, and worsened concentration. He reported psychosocial and spiritual stressors, including his 14-year-old daughter's suicidal behavior. This same daughter had stated to him, "You are dead to me." He perseverated on his divorce, his wife's abortion, spiritual decisions he had made that adversely affected his faith, and separation from his daughters. Over the last 2 months, he was prescribed clon-

TABLE 1. Developmental Models on Spiritual Growth and Healing From James Fowler, Jean Piaget, and Erik Erikson

	Theoretical Model		
Life Stage	Erikson	Piaget	Fowler
Infancy (ages 0–2)	Basic trust versus mistrust (hope)	Sensorimotor	Primal faith
Early childhood (ages 2–6)	Autonomy versus shame and doubt (will)	Preoperational or intuitive	Intuitive-projective faith
	Initiative versus guilt (purpose)	Preoperational or intuitive	Intuitive-projective faith
Childhood (ages 7–12)	Industry versus inferiority (competence)	Concrete operational	Mythic-literal faith
Adolescence (ages 13–21)	Identity versus role confusion (fidelity)	Formal operational	Synthetic-conventional faith
Young adulthood (ages 21–35)	Intimacy versus isolation (love)	Formal operational	Individuative-reflective faith
Adulthood (ages 35–60)	Generativity versus stagnation (care)	Formal operational	Conjunctive faith
Maturity (ages 60 and older)	Integrity versus despair (wisdom)	Formal operational	Universalizing faith

azepam and citalopram for anxiety and depressed mood and zolpidem for insomnia. He reported that his medications were ineffective. He endorsed suicidal ideation with a plan to overdose on his medications the night prior to his emergency department visit. He denied having medical problems, alcohol or illicit substance misuse, or any other prominent psychiatric symptoms. He was medically cleared and admitted to the inpatient psychiatric unit.

The patient never knew his biological father, and his mother was rarely present because of her own "depression" and busy schedule as a single mother. His step-father forced him and his mother out of their home and told him, "I don't love your mother anymore." During childhood, he endorsed low self-esteem and difficulty relating emotionally to others, which resulted in few long-term girlfriends. His lack of belonging, purpose, and fear that he would be compelled to marry his girlfriend and remain trapped in the same town led him to join the military.

Regarding his spiritual growth, he reported a lack of faith or relationship to a higher power. At his first military base assignment, he met Christian men who modeled lifestyles consistent with their beliefs. He met weekly with these men, and he began to heal in the sense that his depression, shame, and low self-worth were replaced by acceptance, love, and significance.

At the same time, he became sexually intimate with his new girlfriend, which he felt was inappropriate for his new moral standards. Despite this spiritual conflict, they married. He chose to desert his male friends and God. After 12 years, little religious fellowship, two daughters, and an abortion, the marriage ended in divorce.

The patient discontinued sertraline, reporting that this medication caused him to feel "out of it." He was started on trazodone for insomnia. His hospital stay was 3 days, and he was discharged to an intensive outpatient program. He met consistently with the chaplain to discuss his excessive shame and guilt and the inconsistencies with his faith of forgiveness and redemption. He then requested therapy, specifically to address his spiritual and psychosocial stressors.

Initially, the patient reported that he felt "stuck in the adolescence of Christianity." He was haunted by feelings of "abandonment of God." Therapy focused on the patient's ambivalence and indecision regarding his faith and relationships, which produced his perceived stagnation as a father, Christian, and friend. Additional foci included incongruence of his Christian beliefs and self-worth, specifically treating his perceived disintegration of his faith and its impact on his identity. His mistrust in himself and others led to a fear of judgment as a "hypocrite," which perpetuated his isolation and impoverished sense of spiritual community. Through therapy, he began to make healthier decisions in his relationships. He reconnected with his spiritual community and became more involved in his daughters' lives.

DISCUSSION

This case conference raised questions regarding what precludes residents from talking about spirituality with patients and provided insight into how to help formulate aspects of spiritual development and cognitive and psychosocial development. This case conference also showed how understanding our own spiritual development is valuable in understanding our patients' current functioning and in identifying the focus of therapy.

This patient's presentation is consistent with Erikson's generativity versus stagnation stage (4), with his initial presentation being one of idleness and "failure" as a father and Christian. His struggle to make independent decisions demonstrates an immature cognitive ego and thus is partially situated in Piaget's formal operational stage (2).

His faith development is situated in Fowler's synthetic-conventional stage. He was unable to critically evaluate his faith. Shame and guilt from his "abandonment" of God during this stage left a lasting feeling of inadequacy.

CONCLUSIONS

This case presentation highlighted the importance of empathy, which may require encouragement for our patients to grow in their faith in God. In a Baylor College of Medicine clinical case conference on sacred experiences in psychotherapy, Lomax et al. (5) agreed that spiritual integration, or a therapeutic rapport built on affirmation, respect, and interest in the patient's spirituality, can allow for growth and strengthen the patient's trust and commitment to the therapist and healing. In the above case, the patient realized how to use his faith to break self-destructive patterns. After assessing a patient's spiritual and psychosocial development, the therapeutic goal is to establish an empathetic relationship that cultivates trust and motivates decisions that strengthen the patient's relationships. The patient can then revisit incomplete developmental stages to begin healing psychological wounds.

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CASE REPORT

Priapism: A Rare but Serious Side Effect of Trazodone

Kamalika Roy, M.D.

Trazodone is a multifunctional drug with variable receptor efficacy at different dosages. It is one of the most commonly used off-label drug for insomnia when used in a range of 25 mg–200 mg daily. Potent 5HT-$_{2a}$ blocking effect and additional alpha adrenergic action and H-$_1$ antihistamine action are prominent in low-dose range, which makes trazodone an effective hypnotic medicine with no dependence potential and a short half-life (1). It is usually well tolerated with minor side effects such as dizziness, morning drowsiness, headache, and fatigue. However, there are reported cases of prolonged, painful penile erection or priapism lasting for more than 4 hours and resulting in urological emergency. The term "priapism" is originated from the Greek word "Priapus," the Greek God with an oversized penis and protector of garden and livestock. Priapism is documented as a rare but dramatic and potentially harmful side effect affecting 0.01%–0.1% of the population on trazodone (2). Most of the cases manifest within 1 month of starting therapy. However, there is one reported case of priapism with one single dose of trazodone (100 mg) (3).

The present case is of a patient with priapism that resulted after adding trazodone; however, permanent functional impairment was avoided by patient education and prompt treatment.

CASE

"Mr. B" is a 52-year-old male patient with a history of sleep apnea and hypertension who was initially prescribed paroxetine for symptoms of persistent depressive disorder. The dose was titrated to 40 mg daily. The patient tolerated the daily dose well, without any reported side effects. He was on paroxetine for more than 1 year. He did not have any history of using alcohol, cocaine, marijuana, or any other elicit substances in the past 10 years. His other medications were hydrochlorothiazide and aspirin. During his outpatient follow-up, he complained of persistent sleep difficulty despite using his continuous positive airway pressure machine with proper settings. Trazodone (200 mg at night) was added for his insomnia. Possible rare instances of priapism were explained to him. He was instructed to report to the emergency department if he experienced an erection lasting longer than usual. After 2 months, he had one episode of prolonged penile erection for 4 hours, which resolved spontaneously. Although it was not reported, he stopped taking trazodone for the next 2 months. However, he resumed taking it because it helped him with insomnia. At that time, he reported that using trazodone at a dosage of 400 mg at night had a more sedative effect. He did not consult before increasing the dose by himself. This time, after 4 hours of taking one 400-mg dose, he developed a prolonged, painful erection, sustained for 8 hours. He visited the emergency department, and urology was consulted immediately. He was not using any medications for erectile dysfunction at that time or in the past year. He was treated with intracavernosal instillation of a 300-microgram phenylephrine injection to achieve detumescence. Subsequently, the patient was followed up by urology, and permanent erectile dysfunction was avoided.

DISCUSSION

Physiology of Erection

The erection or tumescence and flaccidity of the penis are controlled by an intricate balance between parasympathetic and sympathetic control of both trabecular smooth muscle and blood vessels. This mechanism is described in Figure 1.

Intrinsic smooth muscles of arterioles and adrenergic tone maintain the high resistance to blood flow in flaccid state. Alpha adrenergic blockers cause smooth muscle relaxation and minimal resistance to blood flow during the erection phase.

Another mechanism is through inhibition of cyclic guanosine monophosphate degradation, which results in smooth muscle relaxation (vasodilatation) of arterioles. Phosphodiesterase inhibitors produce erection by this pathway, which is not dependent on sympathetic control. Release of vasoactive intestinal peptide (4) has also been proposed as a mechanism of erection.

Types of Priapism

Low-flow (ischemic) priapism leads to cavernosal fibrosis and impotence in 49%–50% of cases. Associated factors are sickle cell disease, leukemia, coagulopathy, fat emboli, drugs with alpha-1 blocking effect, and 5HT-$_{1b}$ stimulation effect.

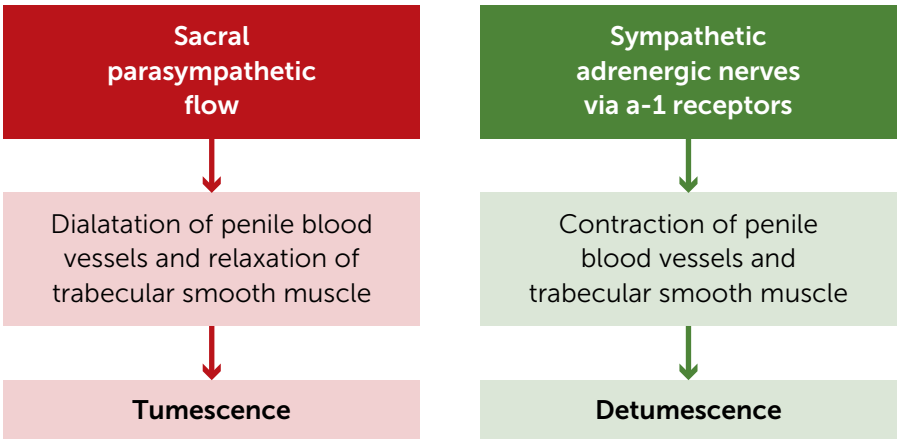
High-flow (nonischemic) priapism does not lead to permanent damage. The underlying mechanism is arterial inflow, which is more than venous outflow (e.g., in the straddle injury of groin).

Drugs Causing Priapism

The overall incidence of priapism is 1.5/100,000 person-years (5). The most common cause is documented to be idiopathic, which accounts for almost 50% of cases. Drug-induced priapism is the second most common cause, accounting for almost 30% of cases.

Almost all typical antipsychotics are known to cause priapism. Chlorpromazine and thioridazine have the greatest alpha-1 adrenergic affinity, and thus

FIGURE 1. Tumescence: Erection, Detumescence, and Flaccidity



these are most frequently reported to be associated with priapism. Among the atypical antipsychotics, risperidone has the highest alpha-1 adrenergic affinity and is most frequently associated with priapism. Clozapine (6), olanzapine (6), and quetiapine (6) are also reported to be associated with priapism, probably as a result of their potent 5HT-$_{2a}$ inhibitory property (1). Although trazodone is the antidepressant most frequently associated with priapism, other selective serotonin reuptake inhibitors, such as fluoxetine, citalopram, paroxetine, sertraline, venlafaxine, bupropion, and buspirone, are also reported to be related to it. Drugs with a phosphodiesterase inhibition property, used for erectile dysfunction, can result in prolonged erection. Prostaglandin E_1 agonists, such as alprostadil, cause vasodilatation and can result in priapism if used in excess. Some antihypertensive (7) drugs have strong alpha-1-adrenergic action. Prazosin, terazosin, and labetolol can result in priapism through this mechanism. Hydralazine has also been reported to be associated with priapism by its direct vasodialatation (7) action. Lastly, alcohol and illicit substances, such as cocaine and ecstasy (8), have also been reported to be associated with priapism.

Proposed Mechanisms of Priapism With Trazodone

Proposed mechanisms of priapism with trazodone include alpha adrenergic blocking effect, stimulation of central 5HT-$_{1b}$ receptor, potent inhibition of 5HT-$_{2a}$, and synergistic 5HT-$_{1b}$ inhibition by active metabolite meta-chloro-phenyl-piperazine.

One study conducted in a small sample size found the relation of trazodone's effect on penile erection activity and sleep phases. Trazodone was shown to prolong the nocturnal tumescence in nonrapid-eye-movement sleep phase in this double-blind cross-over placebo-controlled study. It was also shown that the interval from the REM sleep phase to penile detumescence was prolonged with 200 mg of trazodone (9).

In the above case, inhibition of cytochrome P450 2D6 and 3A4 by paroxetine could also play a role in increasing the level of trazodone and its metabolite, which, in turn, might have played a role in increasing the chance of priapism.

Recommendations for Clinical Practice

Trazodone is reported to be associated with almost 80% of cases of drug-induced priapism. It is a rare side effect, but often patients would not logically associate prolonged erection with their medications. They may not report it in all cases, as exemplified in the above case. Considering the rarity of the side effect, it is often used as off-label, at times without any specific mention of the gravity of the side effect. Before prescribing any antidepressant, consideration of its side effects and proper patient education will be very helpful to prevent any adverse outcome.

Often, a barrier of embarrassment and privacy concerns is present in both the physician's and patient's minds. Educating patients about any sexual side effects of drugs is important, not only to avoid adverse events but also to ensure adherence.

Drug interaction should be seriously considered in elderly male patients who are already on alpha-1 blockers for prostatic hyperplasia and/or drugs for erectile dysfunction. Interaction between trazodone and other cytochrome P450 enzyme inhibitors should also be considered, especially in higher dose.

Medical attention should be sought for any erection lasting more than 4 hours to avoid serious permanent dysfunction.

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BOOK FORUM

Classifying Psychopathology: Mental Kinds and Natural Kinds

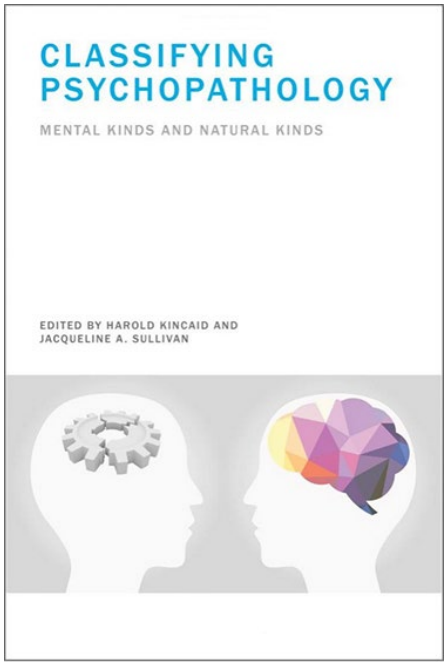
Reviewed by Aaron J. Hauptman, M.D.

This volume is a 13-chapter collection of papers on such disparate topics as major depression, pathological gambling, and oppositional defiant disorder, each of which explores aspects of a single, core philosophical question: Are mental illnesses natural kinds? Natural kinds are categories that result from splitting reality apart at its seams:

> "To say that a kind is natural is to say that it corresponds to a grouping or ordering that does not depend on humans .... The existence of these real and independent kinds of things is held to justify our scientific inferences and practices." (1)

In terms of psychiatry, the question is this: Is the category of "a mental illness" a natural one, which can be justifiably used in scientific inference and practice, or is it a gerrymandered collection of mental states and behaviors that we have deemed pathological? This book addresses a wide range of fascinating questions surrounding issues that scrutinize the understructure of psychiatry.

The question you might ask is this: Why do I, a psychiatry resident, care about how philosophers of psychiatry pick apart our diagnoses? The clinician always asks, "How will this affect my clinical practice?" In a sense, the content of this book will have no impact on what the clinician does in his or her day-to-day treatment of patients, charting of documentation, and after-hours considerations of the struggles of his or her day. In another sense, though, the ideas discussed in this book challenge much that is taken for granted and that underlies every aspect of psychiatric practice and care.



Edited by Harold Kincaid and Jacqueline A. Sullivan. Cambridge, Mass., MIT Press, 2014, pp. 296, $40.00 (hardcover).

Here is an example. The text addresses a central issue of concern to clinicians: the use of DSM criteria. Some writers think that conditions like depression are natural kinds but that the DSM doesn't delineate this entity. On this view, there is a natural kind, depression, that exists independently of the DSM's conceptualization of it. The category called major depressive disorder in the DSM, however, is not that kind—it is, rather, a disunified collection of symptoms. The book argues that because our diagnostic terms don't pick out a natural kind, we see a great deal of inconsistency when it comes to etiology, pathophysiology, and outcome. We conflate a bunch of things and leave some other things out. This is not to say that people are not depressed; this is rather to say that what we call major depressive disorder isn't an adequate characterization of the psychopathology. The book argues that if we are using terms that don't pick out natural kinds, much of our research into major depressive disorder is called into question.

So, no, this won't change how you prescribe sertraline next week. But it may affect how you conceptualize psychiatry and rock some of the fundamental assumptions that you have had about our field and its scientific underpinnings. Such a shaking of the ground can only be a good thing; though uncomfortable, it brings about change and, ultimately, forces us all to build stronger foundations.

Dr. Hauptman is a fourth-year resident in the Department of Child and Adolescent Psychiatry at New York University.

REFERENCE

1. Bird A, Tobin E: Natural kinds, in the Stanford Encyclopedia of Philosophy, Winter, 2012. (http://plato.stanford.edu/archives/win2012/entries/natural-kinds/>)

Residents' Resources

Here we highlight upcoming national opportunities for medical students and trainees to be recognized for their hard work, dedication, and scholarship.

**To contribute to the Residents' Resources feature, contact Hun Millard, M.D., M.A., Deputy Editor (hun.millard@yale.edu).*

AUGUST DEADLINES

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
Academy of Psychosomatic Medicine (APM) Trainee Travel Awards **Deadline: August 1, 2015**	APM	To encourage psychosomatic medicine fellows, residents, and medical students to join APM, attend the annual meeting, and eventually become new leaders of the Academy; a limited number of monetary awards are given to help offset the cost of attending the annual meeting.	Psychosomatic medicine fellows, residents, and medical students.	N/A	http://www.apm.org/awards/trainee-travel.shtml#how_to_apply
Webb Fellowship Program **Deadline: August 1, 2015**	APM	One-year appointment that fosters the career development and leadership potential of advanced psychiatry residents and psychosomatic medicine fellows via mentorship, networking, sustained involvement, and affirmation and consolidation of professional identity.	PGY-3 or 4; Psychosomatic medicine fellow; Recent graduates of psychosomatic medicine fellowship (within 1 year of graduation).	N/A	http://www.apm.org/awards/webb-fship.shtml
The PRITE (Psychiatry Resident In-Training Examination) and Child PRITE Fellowships **Deadline: August 15, 2015**	The American College of Psychiatrists	The PRITE Fellowship Selection Committee chooses PRITE Fellows to serve at least 1 year on the PRITE Editorial Board. They provide a resident perspective to the exam as they participate in the question writing process, developing an assigned number of questions, and then editing and referencing exam items.	PRITE: PGY-2 or 3; Child PRITE: First-year child fellow	Kathryn@acpsych.org	http://www.acpsych.org/resident-fellowships/the-prite-fellowship-program

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.

Race/Ethnicity and Psychiatry

If you have a submission related to this theme, contact the Section Editors Jacqueline Landess, M.D., J.D. (jacqueline.landess@ucdenver.edu) and Aparna Atluru, M.D. (aparna.atluru@phhs.org)

Psychiatry and the Humanities

If you have a submission related to this theme, contact the Section Editor Vivek Datta, M.D. (vdatta@uw.edu)

Integrated Care/ Mental Health Care Delivery

If you have a submission related to this theme, contact the Section Editor Connie Lee, M.D. (Connie.Lee@ucsf.edu)

*If you are interested in serving as a **Guest Section Editor** for the *Residents' Journal*, please send your CV, and include your ideas for topics, to Rajiv Radhakrishnan, M.B.B.S., M.D., Editor-in-Chief (rajiv.radhakrishnan@yale.edu).