

Black-Market Value of Antipsychotics, Antidepressants, and Hypnotics in Las Vegas, Nevada

TO THE EDITOR: To our knowledge, this letter represents the first effort to document the monetary value of several antipsychotic and antidepressant medications outside of a custody setting on the black-market in a major U.S. city. The monetary value of benzodiazepines and narcotics in Canada has been previously published (1), and we include a few of these medications in this letter as well. Stimulants are not included in this letter because of insufficient reports of street prices in our cohort.

Several reports have documented the potential misuse of medications, not traditionally considered to be addictive, for recreational purposes, primarily in custody settings. These include quetiapine (2, 3), anticholinergics (4), and tricyclics (5).

When publicly funded outpatient clinic staff members were asked if they had any suspicions about misuse of these medications, many said that they had heard of patients selling their medications on the street, and several suspected that some patients feigned psychosis to obtain medications to sell.

Between Oct. and Dec. of 2005, a variety of health professionals were asked if they had heard the street prices of medications. The results from sixty-one buyer and seller reports are summarized in Table 1.

TABLE 1. Results From Sixty-One Buyer and Seller Reports

Medication	Strength	Price Per Bottle (25–30 Doses)	Single Dose Price
Antipsychotics			
Olanzapine	10 mg	\$90–\$150	\$5–\$12
Quetiapine	25 mg	\$40–\$50	\$3–\$8
Antidepressants			
Mirtazapine	15 mg	\$30–\$43	\$3–\$5
Citalopram	10 mg	\$20–\$30	
Fluoxetine	20 mg	\$22–\$29	
Hypnotics			
Clonazepam	1 mg	\$50–\$80	\$5–\$11
Diazepam	5 mg	\$35–\$60	\$5–\$7
Zolpidem	5 mg	\$20–\$28	\$3–\$6
Alprazolam	0.5 mg	\$12–\$22	

Sellers reported that it was relatively easy to sell the medications they had received free of charge from the clinics and convert them into money for rent, utilities, food, illicit drugs, or alcohol. Buyers reported that they use these medications for their sedative effect as a sleep aid, to “zone-out” or to “take the edge off.” Buyers included individuals attempting to self-medicate, not having a third-party method of payment.

More detailed studies should be performed to elucidate the abuse potential of these and other medications as well as the epidemiology of their misuse. The health effects of black-market use are as yet unstudied, and physicians should be aware that patients might be covertly taking psychotropic medicines. Diversion of these medications may represent a significant expense for public and private agencies, and suspected malingering or other requests for early refills should alert agencies to the possibility of black-market activity.

References

1. Sajan A, Corneil T, Grzybowski S: The street value of prescription drugs. *CMAJ* 1998; 159:139–142
2. Pierre JM, Shnyder I, Wirshing DA, Wirshing WC: Intranasal quetiapine abuse (letter). *Am J Psychiatry* 2004; 161:1718
3. Hussain MZ, Waheed W, Hussain S: Intravenous quetiapine abuse (letter). *Am J Psychiatry* 2005; 162:1755–1756
4. Buhrich N, Weller A, Kevans P: Misuse of anticholinergic drugs by people with serious mental illness. *Psychiatr Serv* 2000; 51: 928–929
5. Hepburn S, Harden J, Grieve JHK, Hiscox J: Deliberate misuse of tricyclic antidepressants by intravenous drug users: case studies and report. *Scott Med J* 2005; 50:131–133

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Physical Exercise as a Treatment for Non-Suicidal Self-Injury: Evidence From a Single-Case Study

TO THE EDITOR: Nonsuicidal self-injury refers to deliberate damage to one's own bodily tissue without suicidal intent. Nonsuicidal self-injury is a pervasive behavior problem, yet effective treatments are lacking. Research has found that people engage in nonsuicidal self-injury for several different reasons, including tension relief and the induction of pleasurable affective states (1). Preliminary evidence suggests that the release of endogenous opioids may be central to this process of emotional regulation (2). We hypothesized that aerobic physical exercise, which has been shown to regulate mood (3) and stimulate the release of beta-endorphin (4), would decrease the frequency of nonsuicidal self-injury. We report on the results of a single-case study demonstrating the effectiveness of physical exercise as a treatment for nonsuicidal self-injury.

“Ms. A” was an overweight 26-year-old woman with a 13-year history of ongoing psychological and pharmacological treatment for persistent nonsuicidal self-injury, including one inpatient hospitalization for nonsuicidal self-injury within the past year. Ms. A was receiving twice-weekly outpatient psychotherapy for the duration of this study. In an initial baseline assessment, she reported 2.25 episodes of nonsuicidal self-injury per week over the previous month, including self-hitting and head-banging.

We provided Ms. A with a 60-minute workout video, instructing her to exercise three times per week and to exercise in response to nonsuicidal self-injury urges at any time. We also provided a daily assessment form in which she recorded mood and self-injurious urges (both on 0–9 scales) and behaviors.

The frequency of Ms. A's nonsuicidal self-injury decreased immediately after the introduction of exercise to 0.37 times per week during a five-week experimental phase. She then independently discontinued exercise. During this quasi-experimental return to baseline, nonsuicidal self-injury increased to 2.33 times per week. When exercise was re-introduced, nonsuicidal self-injury decreased to 0.00 times per week for the remainder of the study.

Overall, nonsuicidal self-injury frequency was significantly lower during exercise phases ($M=0.29$, $SD=0.49$) relative to nonexercise phases ($[M=2.20$, $SD=0.45]$ $t=6.93$, $df=10$, $p<0.001$). Moreover, analysis of mood ratings showed an increase from before exercise ($M=2.23$, $SD=$

0.86) to after ($M=4.77$, $SD=1.48$) exercise ($t=7.56$, $df=50$, $p<0.001$). When Ms. A exercised in direct response to self-injurious thoughts, exercise acutely reduced her urge to self-injure, from before ($M=3.00$, $SD=1.87$) to after ($M=0.15$, $SD=0.38$) exercise in every single instance ($t=5.38$, $df=24$, $p<0.001$). An 8-week follow-up interview revealed sustained improvement in Ms. A's mental and physical well-being and a decrease in body weight by 20 pounds.

These initial results are promising, and future research is needed to further investigate the effectiveness of exercise as a treatment for nonsuicidal self-injury.

References

1. Nock MK, Prinstein MJ: Contextual features and behavioral functions of self-mutilation among adolescents. *J Abnorm Psychol* 2005; 114:140–146
2. Roth AS, Ostroff RB, Hoffman RE: Naltrexone as a treatment for repetitive self-injurious behavior: an open-label trial. *J Clin Psychiatry* 1996; 57:233–237
3. Yeung RR: The acute effects of exercise on mood state. *J Psychosom Res* 1996; 40:123–141
4. Carr DB, Bullen BA, Skrinar GS, Arnold MA, Rosenblatt M, Beitins IZ, Martin JB, McArthur JW: Physical conditioning facilitates the exercise-induced secretion of beta-endorphin and beta-lipotropin in women. *N Engl J Med* 1981; 305:560–563

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A Case of Mania Following the Use of Pramipexole

TO THE EDITOR: Pramipexole, a dopamine receptor agonist, is currently indicated for treatment of idiopathic Parkinson's disease. There is also preliminary evidence suggesting that it may have a role in the treatment of bipolar depression (1–3). Pramipexole is generally well tolerated, but there are occasional reports of induction of hypomania. Singh et al. (4) described a case of mania in a patient with Parkinson's disease with no personal or family history of bipolar disorder. We report the case of a woman with bipolar II disorder who developed a manic episode following treatment of depression with pramipexole. Written informed consent was obtained from the patient after the procedure had been fully explained.

"Ms. B" was a 41-year-old woman with a history of bipolar II disorder that began with an episode of depression at the age of 16. During the early illness course, she received

several serotonin reuptake inhibitors alone or in combination with lithium, valproic acid, and carbamazepine that led to the development of hypomania and rapid cycling. She had also experienced spontaneous hypomanic spells over the past 3 years. Her usual duration of hypomania was 2 days (range 1–7 days).

Because of the refractory nature of depression over the previous couple of years that had failed to respond to adequate trials of medications, including lamotrigine, oxcarbazepine, and modafinil, we decided to add pramipexole to valproic acid 750 mg b.i.d. (serum level 110 µg/ml). The starting dose of 0.125 mg was escalated over a 3-week period to 1.5 mg a day, to which there was a partial response. As the dosage was increased to 2 mg, Ms. B developed mania characterized by symptoms of euphoria, irritability, paranoia, decreased requirement of sleep, poor judgment, increased libido, and excessive spending of money. The manic symptoms lasted 2 months, but subsided within a week of discontinuation of pramipexole.

The temporal association of mania with the use of pramipexole and the absence of prior manic or mixed episodes in this patient who had a well-documented history of bipolar II disorder would strongly suggest a causal role for pramipexole in the induction of mania. Close monitoring of patients with bipolar depression is required during treatment with pramipexole. Studies are needed to assess the effect of pramipexole on the long-term course of bipolar disorder.

References

1. Goldberg JF, Burdick KE, Endick CJ: Preliminary randomized, double-blind, placebo-controlled trial of pramipexole added to mood stabilizers for treatment-resistant bipolar depression. *Am J Psychiatry* 2004; 161:564–566
2. Zarate CA Jr, Payne JL, Singh J, Quiroz JA, Luckenbaugh DA, Denicoff KD, Charney DS, Manji HK: Pramipexole for bipolar II depression: a placebo-controlled proof of concept study. *Biol Psychiatry* 2004; 56:54–60
3. Sporn J, Ghaemi SN, Sambur MR, Rankin MA, Recht J, Sachs GS, Rosenbaum JF, Fava M: Pramipexole augmentation in the treatment of unipolar and bipolar depression: a retrospective chart review. *Ann Clin Psychiatry* 2000; 12:137–140
4. Singh A, Althoff R, Martineau RJ, Jacobson J: Pramipexole, ropinirole, and mania in Parkinson's disease. *Am J Psychiatry* 2005; 162:814–815

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The authors report no competing interests.

Reprints are not available; however, Letters to the Editor can be downloaded at <http://ajp.psychiatryonline.org>.

Corrections

In a letter to the editor titled "Bradycardia at Low Doses of Risperidone" (*Am J Psychiatry* 2004; 161:2325–2326), the second author should have been listed as Peter Golden, M.D.

The page in each issue in which the *Journal* publishes the APA Chairpersons of Councils, Commissions, Committees, and Task Forces ran incorrectly from August through December. There was an incorrect listing for the Chair of the Audit Committee. This committee is always chaired by the APA Secretary-Treasurer, who for 2006–2008 is Donna M. Norris, M.D.