An Open-Label Study of Naltrexone in the Treatment of Kleptomania

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Background: The present study was designed to test the short-term efficacy and safety of naltrexone in the treatment of kleptomania.

Method: 10 subjects (7 women, 3 men) who fulfilled DSM-IV criteria for kleptomania and were free from other Axis I diagnoses by the Structured Clinical Interview for DSM-IV screening participated in a 12-week naltrexone open-label trial. Kleptomania symptom change was assessed with the Clinical Global Impressions scale (both severity and improvement measures), Sheehan Disability Scale (SDS), Global Assessment of Functioning (GAF), and Kleptomania Symptom Assessment Scale. Side effects were monitored weekly, and liver function tests were administered every 2 weeks.

Results: Naltrexone reduced urges to steal and stealing behavior. Subjects showed significant improvement ($p < .005$) over the 11-week treatment period in all measures compared with measures taken at baseline. Seven subjects (70.0%) were very much improved and 2 (20.0%) were much improved at study end. Subjects also reported overall significant improvement in social and occupational functioning as determined by both the GAF and the SDS ($p < .000$). Men responded to naltrexone as well as women. The mean naltrexone dose required for effective symptom control was 145 mg/day. Nausea was common during the first week of treatment. Five subjects (50.0%) reported previous trials of medication and cognitive-behavioral therapy without any effect on kleptomania symptoms.

Conclusion: The present findings provide evidence that naltrexone may be effective in the treatment of kleptomania. The present report is preliminary. Further studies are needed to confirm these findings.

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Kleptomania, defined as the recurrent failure to resist the impulse to steal objects not needed for personal use or their monetary value, is a poorly understood disorder. With a possible prevalence of 0.6% of the general population,[1] kleptomania may account for a substantial proportion of the staggering 24 billion dollars in business losses attributed to shoplifting each year.[2] In fact, shoplifting is extremely common, and its prevalence appears to be increasing.[3] Although kleptomania differs from recurrent shoplifting in that kleptomaniacs steal for symptomatic relief instead of personal gain, approximately 4% to 24% of shoplifters may suffer from kleptomania.[4–6] In addition to the enormous social costs of this disorder, people with kleptomania suffer the pain and humiliation of repeated arrests, which in turn leads to feelings of guilt, depression, and even suicide.[4,7,8]

Kleptomania is currently classified by DSM-IV[9] as an impulse-control disorder. Kleptomania is defined by 3 essential features: (1) failure to resist an impulse to steal unneeded objects; (2) an increasing sense of tension or arousal before committing the theft; and (3) an experience of pleasure, gratification, or release at the time of committing the theft.

The cause, or pathophysiology, of kleptomania is not well understood. No genetic or biological studies of the disorder have been reported in the literature. Currently, only isolated case reports shed light on possible biological causes of kleptomania. The onset of kleptomania has been associated with dementia and decreased levels of biogenic amines,[10] presenile cortical atrophy,[11] a right parietal tumor,[12] and hypoglycemia secondary to an insulinoma.[13]

Although a number of therapeutic strategies have been proposed for the treatment of kleptomania, no formal drug studies for kleptomania are found in the literature. A small number of case reports cite improvement using tricyclic antidepressants,[14] selective serotonin reuptake inhibitors (SSRIs),[14–17] electroconvulsive therapy,[14] lithium,[18] or valproate.[19] One case series, however, found that SSRIs precipitated kleptomania.[20]

Like other impulse-control disorders, the core symptom of kleptomania is the urge to engage in unwanted behavior. Opioid antagonists have been effective in treating urge-driven disorders such as pathological gambling disorder,[21,22] alcoholism,[23,24] borderline personality disorder with self-injurious behavior,[25] cocaine abuse,[26,27] and

mental retardation with self-injurious behavior.[28] On the basis of the efficacy of opioid antagonists in treating disorders associated with urges, we designed an open-label study to evaluate the efficacy and safety of naltrexone in the treatment of kleptomania. In this study, we hypothesized that naltrexone would reduce both the urges to steal and stealing behavior.

METHOD

Subjects

The study subjects consisted of 13 consecutive outpatients who met inclusion and exclusion criteria for a 1-week single-blind placebo lead-in followed by an 11-week open-label naltrexone treatment. Subjects were recruited by media advertising (newspaper, radio, television). After complete description of the study to the subjects, written informed consent was obtained from all participants. The study and the consent were approved by the Institutional Review Board for the University of Minnesota. A Certificate of Confidentiality issued by the U.S. Department of Health and Human Services protected all study subjects' records from possible subpoena.

The following inclusion criteria were used: (1) age 15 to 75 years, (2) diagnosis of kleptomania by DSM-IV,[9] (3) no other current Axis I diagnosis by the Structured Clinical Interview for DSM-IV (SCID)[29] (comorbid simple phobia such as height phobia and nicotine dependence were accepted), (4) psychotropic drug–free period of at least 4 weeks, (5) 17-item Hamilton Rating Scale for Depression (HAM-D-17)[30] score of ≤ 16, (6) Hamilton Rating Scale for Anxiety (HAM-A)[31] score of ≤ 16, (7) normal liver function tests (LFTs), (8) negative pregnancy test in females of childbearing potential, (9) agreement from females of childbearing potential to use contraception during the course of the study, and (10) signed informed consent after complete description of the study.

Subjects were excluded from the study if any of the following criteria were met: (1) receiving group or individual therapy; (2) history of clinically significant cardiac, hepatic, renal, neurologic, or pulmonary disease; (3) prior naltrexone exposure or known hypersensitivity to naltrexone; (4) SCID diagnosis of drug or alcohol abuse within the past 3 months; (5) diagnosis of another DSM-V impulse-control disorder assessed by clinical interview; (6) severe personality disorder (e.g., borderline personality disorder, antisocial personality disorder) assessed by clinical interview; (7) analgesic use (due to a possible naltrexone and nonsteroidal analgesic interaction)[32]; and (8) pregnant or nursing mothers.

Seventy-nine subjects were screened by telephone. Thirty-nine subjects made appointments for interviews. Of those 39 potential subjects, 19 kept their appointments and were interviewed. Six subjects were excluded after the initial interview: 2 suffered from antisocial personality disorder, not kleptomania; 1 suffered from bipolar disorder, not kleptomania; 1 subject had comorbid major depressive disorder; 1 had comorbid obsessive-compulsive disorder; and 1 suffered from comorbid alcohol dependence. Thirteen subjects were enrolled in the study.

Assessments

Kleptomania was diagnosed using DSM-IV criteria.[9] We also administered a semistructured interview to elicit demographic data, lifetime comorbid psychiatric disorders, and information on the phenomenology, age at onset, course, associated features, treatment history, and response to treatment of the disorder. Because the SCID covers only certain DSM-IV disorders, a detailed interview assessing a history of impulse-control disorders (including impulse-control disorders not otherwise specified such as compulsive shopping, psychogenic excoriation, and sexual compulsions) was conducted.

The major outcome measures were the Clinical Global Impressions (CGI) scale[33] (both severity and improvement measures), Sheehan Disability Scale (SDS),[34] Global Assessment of Functioning (GAF),[35] and Kleptomania Symptom Assessment Scale (K-SAS). The CGI is a reliable and valid 7-point clinician-administered measure assessing both the severity of illness (1 = not ill at all, 7 = among the most extremely ill) and improvement over time (1 = very much improved, 7 = very much worse).[33] The CGI was limited to measuring kleptomania symptoms that had occurred during the previous week.

The SDS is a 3-item self-rated measure assessing disability in 3 domains, each on a 10-point scale: work, social life, and family life.[34] The SDS is a reliable and valid measure of change over time with effective treatment.[34,36] The 3 items may be summed into a single dimensional measure of overall functional impairment that ranges from 0 (unimpaired) to 30 (highly impaired). The SDS was limited to impairment secondary to symptoms of kleptomania for each week.

The GAF is a valid and reliable 100-point single-item rating scale used to indicate overall psychosocial functioning during the week before evaluation.[35] The GAF tracks the change over time in patients' psychological symptoms and level of social and occupational functioning.

The K-SAS is an 11-item self-rated scale designed to assess the change of kleptomania symptoms during treatment (Appendix 1). There are 11 items in the scale, and each item has a score of 0 to 4. Thus, a total score ranges from 0 to 44. All items ask for an average symptom based on the past 7 days. Items 1 through 4 ask for the average severity, frequency, duration, and control over urges (only urges to steal, excludes other urges); items 5 through 7 ask for the average frequency, duration, and control over thoughts associated with stealing (excludes other thoughts); items 8 and 9 ask for the degree of excitement felt before and during an act of stealing; item 10 asks for

the subjective distress caused by stealing; and item 11 asks for personal trouble (relationship, financial, legal, job, medical or health) caused by stealing. The K-SAS is a modification of the Gambling Symptom Assessment Scale (G-SAS), a 12-item self-rated scale that has demonstrated satisfactory psychometric properties in assessing change in the urges, thoughts, and behavior associated with pathological gambling.[21,22] Because the K-SAS is a new scale, we have examined its basic psychometric properties (see Results section).

Side effects were assessed by asking study subjects at each visit whether they had experienced any adverse physical symptoms while taking the study medication. Side effects were rated for severity, action taken, outcome, seriousness, and likely relationship to the study medication. Side effects judged as possibly, probably, or almost certainly related to the study medication are included in the reported rates.

Procedures

Initial screening visit. After a formal psychiatric interview and physical examination, the CGI (Severity), SDS, GAF, K-SAS, HAM-D, and HAM-A were administered. The SCID was administered by trained staff. Baseline LFTs, urine drug screen test, and a pregnancy test in females were obtained.

Eleven weekly visits. The CGI (Improvement and Severity) scales, SDS, GAF, K-SAS, HAM-D, and HAM-A were administered weekly after the screening visit to assess changes in kleptomania symptoms. Patients who improved 50% or more on the K-SAS at visit 2 (after 1 week on placebo) were excluded from the study as placebo responders. LFTs were carried out every other week. Adverse effects were assessed at baseline and at weekly intervals.

Drug administration. Subjects began the study with a 1-week single-blind placebo lead-in. At visit 2, if not judged to be placebo responders, subjects were started on naltrexone, 25 mg/day, for the first 2 days followed by 50 mg/day for the rest of the week. The naltrexone was dispensed in capsules (either with 25 mg or 50 mg of naltrexone per capsule) identical to the capsules used for the placebo. The dose was then gradually raised until an optimal outcome (clinical judgment) was obtained or the dose reached 200 mg/day, whichever came first. The rate of increase was limited to 50 mg/week. If the dose exceeded 50 mg/day, the dose was divided equally and given twice a day. If unpleasant side effects developed, the naltrexone dose was decreased until side effects were controlled. U.S. Food and Drug Administration approval was obtained for using doses of naltrexone greater than 50 mg/day.

No other psychotropic medications were allowed during the study. A capsule count was kept for each dose of medication taken. Psychotherapy of any form (including cognitive-behavioral therapy) was not initiated during the study.

Statistical Analysis

Baseline and subsequent scores on continuous study measures were compared with paired t tests (2-tailed). The Fisher exact test was used for comparisons of categorical variables. The Pearson correlation was used to assess the relationship between degree of change on various study measures. Mean values were accompanied by standard deviations. A decision was made before beginning the study to include in the intent-to-treat analyses only those subjects (N = 10) who returned for the first study visit after beginning naltrexone and took the medication for at least 1 week.

Statistical analyses used the last-observation-carried-forward data set, in which the last available efficacy data from patients dropping out of the study are carried to successive timepoints. Three subjects (2 men and 1 woman) were excluded from data analyses because of failure to take the medication for at least 1 week (2 did not return for visit 3, which corresponds to having taken the medication for 1 week; 1 was a placebo responder at visit 2 and was discontinued from the study).

The reliabilities of the K-SAS were measured by using the Pearson correlation for the 2 repeated test scores from each subject at visits 1 and 2 (placebo period). Factor analysis was used to examine the internal consistency of the 11 K-SAS items. Both 1- and 2-factor solutions were estimated (varimax rotation) to examine factor loading of each K-SAS item on each factor solution. The K-SAS, CGI, and GAF weekly data were used to test the validity of the K-SAS. The Pearson correlation analysis was used to find the correlation between the K-SAS versus CGI and K-SAS versus GAF.

RESULTS

Of the 13 subjects who entered the study, 10 completed at least through visit 3, which corresponds to taking naltrexone for at least 1 week. Seven of the subjects (4 women, 3 men) completed all study visits. One subject completed through visit 8 and withdrew due to depressive symptoms. Two subjects completed through visit 10 and withdrew due to inability to keep the study schedule. Only 1 (7.7%) of the 13 subjects was a placebo responder.

Baseline Data

Ten subjects (7 [70.0%] women, 3 [30.0%] men; mean age = 37.0 ± 11.4 years; range, 22–55 years) were included in an intent-to-treat analysis. The sample included 8 whites, 1 Puerto Rican female, and 1 Chinese American male. Nine (90.0%) of the subjects were married and 1 (10.0%) was single. Two (20.0%) had completed high school, 6 (60.0%) had completed a 4-year college degree program, and 2 (20.0%) had professional degrees. The mean annual income for the 10 subjects was $60,900 ± $56,177.

Table 1. Baseline and Terminal Visit Kleptomania Symptom Data in 10 Patients[a]

Outcome Measure	Baseline Mean	Baseline SD	Terminal Mean	Terminal SD	t Value	p Value
K-SAS						
Total score	21.75	7.78	9.85	5.33	3.807	.004
Kleptomania urge strength[b]	2.25	0.64	0.95	0.37	4.801	.001
Kleptomania urge frequency (urges/wk)[c]	2.40	0.70	1.60	0.84	2.449	.037
Kleptomania urge duration (h/wk)[d]	1.60	0.70	1.05	0.50	2.091	.066
Kleptomania thought frequency (thoughts/wk)[c]	2.30	0.68	1.50	0.71	2.449	.037
Kleptomania thought duration (h/wk)[c]	1.60	0.70	0.95	0.34	2.512	.033
Subjective distress[e]	2.20	1.23	0.80	0.63	3.096	.013
Stealing frequency/wk	2.30	1.57	0.10	0.32	4.125	.003
Global Assessment of Functioning score	53.60	6.54	86.60	7.15	–10.011	.000
Sheehan Disability Scale score	10.50	4.17	2.90	3.35	5.840	.000
Clinical Global Impressions-Severity of Illness scale score[f]	5.30	0.67	2.30	1.16	7.115	.000

[a]Abbreviation: K-SAS = Kleptomania Symptom Assessment Scale.
[b]0 = no urges, 1 = mild, 2 = moderate, 3 = severe, 4 = extreme urges.
[c]0 = none, 1 = once, 2 = 2 to 4 times, 3 = several to many, 4 = constant or near constant urges.
[d]0 = none, 1 = 1 h or less, 2 = 1 to 4 h, 3 = 4 to 10 h, 4 = over 10 h/wk.
[e]0 = none, 1 = minimal, 2 = moderate, 3 = much, 4 = very much.
[f]1 = normal, 2 = borderline mentally ill, 3 = mildly ill, 4 = moderately ill, 5 = markedly ill, 6 = severely ill, 7 = among the most extremely ill.

The mean ± SD baseline measures are presented in Table 1. The mean HAM-A score at baseline was 4.10 ± 3.78, and the mean HAM-D score was 3.70 ± 3.40. Baseline severity measures did not differ significantly between male and female subjects.

Clinical Characteristics

The reported mean age at onset of stealing behavior was 10.2 ± 3.1 years (range, 6–15 years). The mean duration of kleptomania symptoms prior to entrance into the study was 26.8 ± 12.1 years. On average, the subjects reported stealing 2.3 ± 1.6 times per week. Eight subjects stole exclusively from stores, while 2 subjects stole from friends, work, and stores. No subject had ever gone longer than 3 weeks without stealing during the course of his or her illness.

Various triggers were reported as provoking the urge to steal. Five subjects (50.0%) reported that the sights and sounds inside stores, not particular objects, prompted the urge to steal. Four subjects (40.0%) reported spontaneous urges to steal upon waking in the morning without any specific item in mind. One subject (10.0%) reported that boredom prompted the urge to steal. All 10 subjects reported that urges to steal were eliminated by the act of stealing, but were then replaced by feelings of shame and guilt.

In addition to the guilt secondary to stealing, subjects reported numerous other problems that resulted from their stealing: 9 (90.0%) reported lying to family or friends about their behavior, 6 subjects (60.0%) reported marital problems secondary to stealing, and 4 subjects (40.0%) reported work-related difficulties due to stealing (usually inability to concentrate). Only 2 subjects (20.0%) had ever been arrested for stealing (1 arrest each), and in both cases, the arrests had occurred greater than 2 years prior to entrance into the study.

Although no subject had a current Axis I disorder, 4 subjects (40.0%) reported a history of depressive symptoms. In all 4 cases, these depressive symptoms had occurred at greater than 2 years prior to entrance into the study. Three of the 4 subjects had been treated with an antidepressant (2 with fluoxetine, 80 mg/day; 1 with paroxetine, 60 mg/day). Although all 3 subjects reported relief from depressive symptoms with medications, none of these subjects reported a remission in kleptomania symptoms with these medication trials. One subject (10.0%) had a history of alcohol dependence, which had been in full, sustained remission for 8 years. None of the subjects with psychiatric histories had ever been hospitalized for those problems. No one had a history of an anxiety disorder, obsessive-compulsive disorder, other impulse-control disorder, or bipolar disorder.

Two subjects (20.0%) reported receiving psychotherapy for their kleptomania symptoms. Both subjects had undergone cognitive-behavioral therapy weekly for 16 and 20 weeks, respectively. Neither subject reported a reduction in kleptomania urges due to the therapy. One subject (10.0%) had attended Shoplifters Anonymous weekly for a period of 12 weeks, but reported no reduction in kleptomania symptoms. No subject had received either outpatient or inpatient psychiatric treatment specifically for symptoms of kleptomania.

Outcome Data

Subjects showed significant improvement over the 11-week treatment period in all measures compared with baseline (see Table 1). Seven (70.0%) were very much improved, 2 (20.0%) were much improved, and 1 (10.0%) was minimally improved by the end visit using the CGI-Improvement scale.

With respect to stealing urges, at completion of the study, 2 subjects (20.0%) reported that their urges to steal were in remission, while 8 (80.0%) reported urges to steal were significantly reduced and therefore were easier to resist. Of those 2 subjects with remitted symptoms, the mean length of time without kleptomania symptoms at time of study completion was 5.0 ± 1.4 weeks. None of the subjects who stopped stealing or had remission of the urges to steal developed other impulsive or addictive behavior during the study period.

Compared with baseline when all subjects were actively stealing multiple times per week, by study end, 9 subjects

reported no stealing behavior for a mean of 4.3 ± 1.3 weeks. Only 1 subject reported continued stealing behavior, although it was reduced in frequency.

In addition to the specific improvement in kleptomania symptoms, analysis also showed overall significant improvement in social and occupational functioning as determined by both the GAF and the SDS (see Table 1).

The mean ± SD naltrexone dose at the end of the study was 145.0 ± 49.7 mg/day.

Side Effects

Nausea (50%), drowsiness (40%), dry mouth (30%), and vivid dreams (30%) were common. Nausea was mild in 4 of the 5 subjects reporting that symptom, and moderate in 1 subject. No subject reported nausea for longer than 14 days. Drowsiness and dry mouth were mild in all subjects and lasted less than 1 week. No subject had elevated liver transaminases at anytime during the study.

K-SAS Psychometrics

The K-SAS test-retest reliability showed a fair correlation: N = 12, r = 0.572 (test-retest period = 1 week; p = .051). For the internal consistency, Cronbach's $\alpha = 0.903$ and 1-factor model K-SAS item loading ranged from 0.623 to 0.888. The 2-factor model showed that urge symptom items 1 and 2 (severity and frequency measures) cohere with kleptomania-related subjective distress. The K-SAS showed a good convergent validity when compared with the CGI at visit 3 (N = 10, r = 0.854, p = .002) (the first visit in which changes in symptoms were recorded), and at visits 4 to 12, r ranged from 0.634 to 0.870, $p < .03$ (2-tailed) in all visits. When compared with GAF scores, K-SAS showed good convergent validity beginning at visit 4 (N = 10, r = –0.806, p = .005), and at visits 5 to 12, r ranged from 0.620 to 0.883, $p < .040$ (2-tailed) in all visits.

DISCUSSION

The present study results suggest that naltrexone may be effective in treating kleptomania. Most patients had moderate to severe kleptomania symptoms at baseline, and 5 of the 10 subjects had failed to respond to prior pharmacologic interventions or cognitive-behavioral therapy. At the end of the study, 90% of subjects were much or very much improved on the CGI. Most patients had stopped stealing and reported significantly less personal distress and improved overall functioning as shown in the subjective distress scale score (of the K-SAS), CGI, SDS, and GAF.

As the naltrexone treatment progressed, many patients expressed that their struggle to resist the urges to steal and thoughts associated with stealing were reduced or abolished altogether. In addition to reducing urges, naltrexone seemed to reduce the subjective experience of pleasure if they engaged in stealing. Remission of symptoms, however, does not necessarily imply a correlation with a response to naltrexone. Although the course of kleptomania is unclear, for some patients the symptoms fluctuate over time with remissions and exacerbations.[37] The remission seen during this study period may simply reflect the course of the illness.

Although previous pharmacologic studies of impulse-control disorders have found high rates of placebo responders,[22,38,39] there was only a single placebo responder in this study. Most subjects required approximately 2 weeks at a particular dose before they reported a decrease in their kleptomania symptoms. In fact, because most subjects had been stealing for approximately 28 years before entering the study, they remained incredulous concerning their improvement until approximately 3 or 4 weeks of continued improvement.

In this study, the number of female subjects (N = 7) was substantially greater than the number of male subjects (N = 3). This gender difference is in keeping with the literature on kleptomania.[2,4,14] In other addiction studies, there is evidence that females have a more favorable clinical and biological response to naltrexone.[40,41] In this study, however, there was no evidence that female kleptomaniacs responded more favorably to naltrexone than the male kleptomaniacs. Thus, the larger number of women in this study does not appear to have contributed significantly to the effectiveness of naltrexone on kleptomania symptoms.

Naltrexone may be effective in treating kleptomania behavior because of its ability to reduce urges. Preclinical and clinical studies demonstrate that the underlying biological mechanism of urge-based disorders may involve the processing of incoming reward inputs by the ventral tegmental area–nucleus accumbens–orbital frontal cortex (VTA-NA-OFC) circuit.[42–46] This circuit then influences behavior by modulating animal and human motivation (e.g., urges, cravings). Dopamine may also play a major role in the regulation of this region's functioning.[42,46–48]

Urges linked to the experiencing of reward and pleasure represent clinical targets in impulse-control disorders. Studies of naltrexone in the treatment of pathological gambling disorder have also demonstrated efficacy in reducing urges.[21,22] The primary pharmacologic action of naltrexone within the central nervous system is the antagonism of the μ-opioid receptor, the site at which β-endorphins, morphine, and heroin act as endogenous and exogenous agonists. The μ-opioid system is involved in the processing of reward, pleasure, and pain. The effects of naltrexone across these diagnostic categories may be due to the drug's modulation of dopamine function within the VTA-NA-OFC via the antagonism of opioid receptors in the VTA.[49–51]

Other opioid models to explain stealing, however, have also been suggested.[2,37] It has been hypothesized that patients with kleptomania steal as a means of stimulating

the opioid system. The opioid release "soothes" the patients, treats their sadness, or reduces their anxiety. Thus, stealing is a mechanism to relieve oneself from a chronic state of hyperarousal, perhaps produced by prior stressful or traumatic events, and thereby modulate affective states.[2,37]

The use of naltrexone, however, has been limited to 50 mg/day in part because of a fear that it may elevate liver enzymes. Naltrexone causes hepatic enzyme elevations dose dependently. In physically healthy individuals, however, doses up to 150 mg/day rarely result in enzyme elevations.[52] In obese patients, however, naltrexone doses of 300 mg/day caused a significantly higher rate of enzyme elevations than the standard 50 mg/day.[53] Recently, we noticed an increased risk of liver enzyme elevation when naltrexone is used concurrently with analgesics.[32] Presently, we use naltrexone only for those patients who do not need nonsteroidal analgesics. Elevated enzymes return to normal levels if the drug is discontinued.[54] There were, however, no instances of elevated enzymes in this study. In fact, although some subjects experienced mild nausea with naltrexone, most patients tolerated the medication without difficulty.

Limitations

This study has several limitations. First, our sample of kleptomaniacs may not reflect the larger population of patients who suffer from kleptomania. Because subjects were excluded from the study if they had comorbid Axis I disorders, and because comorbidity with affective or anxiety disorders appears to be common in kleptomania,[2,4,14] our subjects with kleptomania may not be characteristic of patients with kleptomania in general. Although no subject was diagnosed with a current comorbid Axis I disorder, and HAM-D and HAM-A scores reflected mild or no depressive or anxiety symptoms, 4 of our subjects reported lifetime major depressive disorder. This finding of lifetime comorbidity is consistent with the literature.[2] Also, while the exclusion of only 3 subjects due to current psychiatric comorbidity may raise issues of how representative our sample is of patients with kleptomania, it may also provide useful data concerning the phenomenology of the "typical" presentation of this poorly understood disorder.

A second limitation of this study is that no structured interview of possible Axis II pathology was performed. Some researchers have suggested that most patients with kleptomania do not come forward for treatment.[37] The fact that the subjects in this study voluntarily sought treatment may raise the question of whether these patients are "true" kleptomaniacs or are suffering from personality disorders. An unstructured interview did exclude severe personality disorders (borderline personality disorder, antisocial personality disorder), but the lack of a standardized Axis II interview may have resulted in the underdiagnosis of some personality disorders or traits.

A third limitation of this study is the sample size. Any conclusions about the efficacy of naltrexone in kleptomania must be made cautiously given the extremely small study sample. The small sample size may also underestimate or overestimate the psychometric properties of the K-SAS. Although the psychometric properties of the K-SAS are satisfactory, this measure is still investigational, and the validity and reliability still need to be vigorously assessed.

Until further study data are available, the results of this study should be viewed as preliminary. The small study number makes it difficult to weigh the validity of the present data. Double-blind studies will provide more reliable information in this area. Because of the flexible dosing design of the study, one also cannot determine with certainty whether improvement was due to a longer duration on at least 50 mg/day of naltrexone or to the higher doses given at later times.

In conclusion, the present findings provide evidence that naltrexone may be effective in the treatment of kleptomania. Further controlled trials are needed to confirm these findings.

Drug names: fluoxetine (Prozac and others), naltrexone (ReVia and others), paroxetine (Paxil).

REFERENCES

1. Goldman MJ. Kleptomania: an overview. Psychiatr Ann 1992;22:68–71
2. Goldman MJ. Kleptomania: making sense of the nonsensical. Am J Psychiatry 1991;148:986–996
3. Cupchik W, Atcheson JD. Shoplifting: an occasional crime of the moral majority. Bull Am Acad Psychiatry Law 1993;11:343–352
4. McElroy SL, Hudson JI, Pope HG, et al. Kleptomania: clinical characteristics and associated psychopathology. Psychol Med 1991;21:93–108
5. Bradford J, Balmaceda R. Shoplifting: is there a specific psychiatric syndrome? Can J Psychiatry 1983;28:248–254
6. Schlueter GR, O'Neal FC, Hickey J. Rational versus nonrational shoplifting types: the implications for loss prevention strategies. Int J Off Ther Comp Criminol 1989;33:227–239
7. Sarasalo E, Bergman B, Toth J. Personality traits and psychiatric and somatic morbidity among kleptomaniacs. Acta Psychiatr Scand 1996;94:358–364
8. McElroy SL, Keck PE Jr, Phillips KA. Kleptomania, compulsive buying, and binge-eating disorder. J Clin Psychiatry 1995;56(suppl 4):14–27
9. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
10. Moak GS, Zimmer B, Stein EM. Clinical perspectives on elderly first-offender shoplifters. Hosp Community Psychiatry 1988;39:648–651
11. Khan K, Martin IC. Kleptomania as a presenting feature of cortical atrophy. Acta Psychiatr Scand 1977;56:168–172
12. Chiswick D. Shoplifting, depression and an unusual intracranial lesion. Med Sci Law 1976;16:266–268
13. Segal M. Shoplifting [letter]. Br Med J 1976;1:960
14. McElroy SL, Pope HG, Hudson JI, et al. Kleptomania: a report of 20 cases. Am J Psychiatry 1991;148:652–657
15. Chong SA, Low BL. Treatment of kleptomania with fluvoxamine. Acta Psychiatr Scand 1996;93:314–315
16. McElroy SL, Keck PE Jr, Pope HG Jr, et al. Pharmacological treatment of kleptomania and bulimia nervosa. J Clin Psychopharmacol 1989;9:358–360
17. Kraus JE. Treatment of kleptomania with paroxetine [letter]. J Clin Psychiatry 1999;60:793
18. Burstein A. Fluoxetine-lithium treatment for kleptomania [letter]. J Clin

Psychiatry 1992;53:28–29
19. Kmetz GF, McElroy SL, Collins DJ. Response of kleptomania and mixed mania to valproate. Am J Psychiatry 1997;154:580–581
20. Kindler S, Dannon PN, Iancu I, et al. Emergence of kleptomania during treatment for depression with serotonin selective reuptake inhibitors. Clin Neuropharmacol 1997;20:126–129
21. Kim SW, Grant JE. An open naltrexone treatment study in pathological gambling disorder. Int Clin Psychopharmacol 2001;16:285–289
22. Kim SW, Grant JE, Adson D, et al. Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. Biol Psychiatry 2001;49:914–921
23. Croop RS, Faulkner EB, Labriola DF, for the Naltrexone Usage Study Group. The safety profile of naltrexone in the treatment of alcoholism. Arch Gen Psychiatry 1997;54:1130–1135
24. Volpicelli JR, Watson NT, King AC, et al. Effect of naltrexone on alcohol "high" in alcoholics. Am J Psychiatry 1995;152:613–615
25. Sonne S, Rubey R, Brady K. Naltrexone treatment of self-injurious thoughts and behaviors. J Nerv Ment Dis 1996;184:192–195
26. Corrigall WA, Coen KM. Opiate antagonists reduce cocaine but not nicotine self-administration. Psychopharmacology (Berl) 1991;104:167–170
27. Meyer RE. New pharmacotherapies for cocaine dependence revisited. Arch Gen Psychiatry 1992;49:900–904
28. Winchel RM, Stanley M. Self-injurious behavior: a review of the behavior and biology of self-mutilation. Am J Psychiatry 1991;148:306–317
29. First MB, Spitzer RL, Gibbon M, et al. Structured Clinical Interview for DSM-IV Axis I Disorders-Patient Edition (SCID-I/P, version 2.0). New York, NY: Biometric Research, New York State Psychiatric Institute; 1995
30. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry 1960;23:56–62
31. Hamilton M. The assessment of anxiety states by rating. Br J Med Psychol 1959;32:50–55
32. Kim SW, Grant JE, Adson DE, et al. A preliminary report on possible naltrexone and nonsteroidal analgesics interactions [letter]. J Clin Psychopharmacol 2001;21:632–634
33. Guy W. ECDEU Assessment Manual for Psychopharmacology. US Dept Health, Education, and Welfare publication (ADM) 76-338. Rockville, Md: National Institute of Mental Health; 1976:218–222
34. Sheehan DV. The Anxiety Disease. New York, NY: Scribner's; 1983
35. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994:32
36. Leon AC, Olfson M, Portera L, et al. Assessing impairment in primary care: a psychometric analysis of the Sheehan Disability Scale. Int J Psychiatry Med 1997;27:93–105
37. Goldman MJ. Kleptomania: The Compulsion to Steal—What Can Be Done? Far Hills, NJ: New Horizon Press; 1998
38. Hollander E, DeCaria CM, Mari E, et al. Short-term single-blind fluvoxamine treatment of pathological gambling. Am J Psychiatry 1998;155: 1781–1783
39. Ninan PT, McElroy SL, Kane CP, et al. Placebo-controlled study of fluvoxamine in the treatment of patients with compulsive buying. J Clin Psychopharmacol 2000;20:362–366
40. Covey LS, Glassman AH, Stetner F. Naltrexone effects on short-term and long-term smoking cessation. J Addict Dis 1999;18:31–40
41. Kreek MJ, Schluger J, Borg L, et al. Dynorphin A_{1-13} causes elevation of serum levels of prolactin through an opioid receptor mechanism in humans: gender differences and implications for modulation of dopaminergic tone in the treatment of addictions. J Pharmacol Exp Ther 1999;288: 260–269
42. Koob GF, Bloom FE. Cellular and molecular mechanisms of drug dependence. Science 1988;242:715–723
43. Mogenson GJ, Jones DJ, Yim CY. From motivation to action: functional interface between the limbic system and motor system. Prog Neurobiol 1980;14:69–97
44. Berridge KC. Food reward: brain substrates of wanting and liking. Neurosci Biobehav Rev 1996;20:1–25
45. Hyman SE. Molecular and cell biology of addiction. Curr Opin Neurol Neurosurg 1993;6:609–613
46. Koob GF. Drugs of abuse: anatomy, pharmacology and function of reward pathways. Trends Pharmacol Sci 1992;13:177–184
47. Kuhar MJ, Ritz MC, Boja JW. The dopamine hypothesis of the reinforcing properties of cocaine. Trends Neurosci 1991;14:299–302
48. Self DW, Barnhart WJ, Lehman DA, et al. Opposite modulation of cocaine-seeking behavior by D_1- and D_2-like dopamine receptor agonists. Science 1996;271:1586–1598
49. Stewart J. Reinstatement of heroin and cocaine self-administration behavior in the rat by intracerebral application of morphine in the ventral tegmental area. Pharmacol Biochem Behav 1984;20:917–923
50. Matthews RT, German DC. Electrophysiological evidence for excitation of rat ventral tegmental area and dopamine neurons by morphine. Neuroscience 1984;11:617–625
51. Broekkamp CL, Phillips AG. Facilitation of self-stimulation behavior following intracerebral microinjections of opioids into the ventral tegmental area. Pharmacol Biochem Behav 1979;11:289–295
52. Verebey KG, Mule SJ. Naltrexone (Trexan): a review of hepatotoxicity issues. NIDA Res Monogr 1986;67:73–81
53. Mitchell JE, Morley JE, Levine AS, et al. High-dose naltrexone therapy and dietary counseling for obesity. Biol Psychiatry 1987;22:35–42
54. Pfohl DN, Allen JI, Atkinson RL, et al. Naltrexone hydrochloride (Trexan): a review of serum transaminase elevations at high dosage. NIDA Res Monogr 1986;67:66–72

Appendix 1 appears on page 356.

Appendix 1. Kleptomania Symptom Assessment Scale (K-SAS)[a]

The following questionnaire is aimed at evaluating kleptomania symptoms. Please read the questions carefully before you answer.

1) If you had urges to steal during the past WEEK, on average, how strong were your urges? Please circle the most appropriate number.

None	Mild	Moderate	Severe	Extreme
0	1	2	3	4

2) During the past WEEK, how many times did you experience urges to steal? Please circle the most appropriate answer.
   0) None
   1) Once
   2) Two or 3 times
   3) Several to many times
   4) Constant or near constant urges

3) During the past WEEK, how many hours (add up hours) were you preoccupied with your urges to steal? Please circle the most appropriate number.

None	1 hr or less	1 to 4 hr	4 to 10 hr	Over 10 hr
0	1	2	3	4

4) During the past WEEK, how much were you able to control your urges? Please circle the most appropriate number.

None	Minimal	Moderate	Much	Very much
0	1	2	3	4

5) During the past WEEK, how often did thoughts about stealing come up? Please circle the most appropriate answer.
   0) None
   1) Once
   2) Two to 4 times
   3) Several to many times
   4) Constantly or nearly constantly

6) During the past WEEK, approximately how many hours (add up hours) did you spend thinking about stealing? Please circle the most appropriate number.

None	1 hr or less	1 to 4 hr	4 to 10 hr	Over 10 hr
0	1	2	3	4

7) During the past WEEK, how much were you able to control your thoughts of stealing? Please circle the most appropriate number.

None	Minimal	Moderate	Much	Very much
0	1	2	3	4

8) During the past WEEK, on average, how much excitement did you have shortly before you committed a theft? If you did not actually steal anything, please estimate how much anticipatory excitement you believe you would have experienced if you had committed a theft. Please circle the most appropriate number.

None	Minimal	Moderate	Much	Very much
0	1	2	3	4

9) During the past WEEK, on average, how much excitement and pleasure did you feel when you successfully committed a theft? If you did not actually steal, please estimate how much excitement and pleasure you believe you would have experienced if you had committed a theft. Please circle the most appropriate number.

None	Minimal	Moderate	Much	Very much
0	1	2	3	4

10) During the past WEEK, how much emotional distress (mental pain or anguish, shame, guilt, embarrassment) has your stealing caused you? Please circle the most appropriate number.

None	Minimal	Moderate	Much	Very much
0	1	2	3	4

11) During the past WEEK, how much personal trouble (relationships, financial, legal, job, medical or health) has your stealing caused you? Please circle the most appropriate number.

None	Minimal	Moderate	Much	Very much
0	1	2	3	4

[a]The K-SAS assesses kleptomania symptom severity. This scale was designed so that modified versions could be developed for other impulse-control disorders. Maximum score = 44, severe = 31–44, moderate = 21–30, mild = 8–20.