Escitalopram Treatment of Kleptomania: An Open-Label Trial Followed by Double-Blind Discontinuation

Lorrin M. Koran, M.D.; Elias N. Aboujaoude, M.D.; and Nona N. Gamel, M.S.W.

Background: Kleptomania has no definitive treatment. Mixed reports of benefit from openly prescribed selective serotonin reuptake inhibitors led us to design a double-blind, placebo-controlled discontinuation trial of escitalopram.

Method: Between December 2002 and March 2005, we recruited 24 subjects aged ≥ 20 years with DSM-IV kleptomania of ≥ 1 year's duration. We excluded subjects with organic mental disorders, psychoses, substance or alcohol abuse, suicidal risk, bipolar I or II disorder, anorexia nervosa, or antisocial personality disorder and subjects requiring other psychotropic medications. Our primary outcome measure was theft episodes per week. A responder was defined as a subject having a > 50% decrease in theft episodes per week and a Clinical Global Impressions-Improvement scale score of "much improved" or "very much improved." Escitalopram was started at 10 mg/day and increased as necessary and tolerated after week 4 to 20 mg/day. At the end of week 7, responders were randomly assigned to a 1-week taper followed by 16 weeks of placebo or to continuation of treatment for 17 weeks at their week 7 escitalopram dose.

Results: Nineteen subjects (79%) were week 7 responders and 15 were randomly assigned. Five subjects (4 responders) withdrew early: 1 for unrelated illness, 1 for protocol deviation, 2 for side effects, and 1 for withdrawn consent. Three (43%) of 7 escitalopram subjects relapsed compared with 4 (50%) of 8 placebo subjects (Fisher exact test p = .38).

Conclusion: The high response rate during open-label escitalopram treatment was not better maintained by double-blind escitalopram than by placebo. Kleptomania may be a heterogeneous pathological behavior better treated with pharmacotherapy in some cases and psychologically or with combined treatment in others.

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Corresponding author and reprints: Lorrin M. Koran, M.D., OCD Clinic, Room 2363, 401 Quarry Rd., Stanford, CA 94305 (e-mail: lkoran@stanford.edu).

Kleptomania is an impulse control disorder with a long history of psychiatric attention[1] but no definitive treatment.[2] The Swiss physician Matthey described kleptomania (which he termed *klopemania* [stealing insanity]) in 1816,[3] and Esquirol introduced the term *kleptomania* in his 1838 psychiatric textbook, *Des Maladies Mentales*,[4] but the disorder was not officially recognized in U.S. diagnostic nomenclature until DSM-III[5] was published in 1980. The prevalence of kleptomania is unknown, but it has been estimated at 6 per 1000 persons,[6] which translates into about 1.2 million persons among the 200 million American adults. Kleptomania has been estimated to account for 4%[7] and 10%[8] of shoplifting, but the true proportion remains unknown. Each year, approximately 2 million Americans are charged with shoplifting, and for every person arrested, perhaps 35 more escape undetected.[6] Since shoplifting imposes an estimated $10 billion[9] to $50 billion[6] annual cost on the U.S. economy, kleptomania's cost to retailers, not to mention its psychic and legal system costs, is substantial.

Kleptomania is distinct from shoplifting by professional shoplifters (who steal for use or profit), teenagers (who steal for use or thrills), substance abusers or addicts (who steal to finance drug use), and the absent-minded or those with early dementia (who have no impulse to steal).[10] It is also distinct from shoplifting related to mania, conduct disorder, and antisocial personality disorder.[11]

Kleptomania begins by the age of 20 years in at least half of reported cases and is often chronic, although periods of exacerbation or remission may occur.[12–15] Kleptomania appears to be more common in women,[12,13,15,16] but this observation may reflect various selection biases, including courts' differential treatment of men and women arrested for stealing. An Italian study reported an equal gender distribution among 20 kleptomania outpatients.[14]

We are unaware of any controlled trials of pharmacotherapy for kleptomania. Open-label cases and series report successful treatment with selective serotonin reuptake inhibitors (SSRIs): 2 of 10 patients treated with fluoxetine, with remissions lasting 3 and 11 months[12]; a fluvoxamine case, with a 9-month remission[17]; 4 fluoxetine cases, with remissions of 7, 12, 18, and 20 months[18]; and 1 paroxetine case, with remission of 3 months.[19] In all of these cases, kleptomania improvement or remission occurred within 2 to 4 weeks of starting medication. In addition, successful treatment has been reported with fluoxetine plus lithium,[20] fluvoxamine plus buspirone,[21] fluoxetine plus imipramine,[12] and paroxetine plus naltrexone[22] and with drugs other than SSRIs—trazodone,[12,23] lithium,[12] nortriptyline,[12] naltrexone,[24,25] valproate,[12] and topiramate.[26] On the other hand, Grant and Kim[13] reported a discouraging response in 19 prior SSRI treatment trials aimed at comorbid anxiety or depressive symptoms (kleptomania had remained undisclosed) in 15 subjects volunteering for a kleptomania medication (naltrexone) trial: only 2 of 8 fluoxetine trials had been moderately helpful and none of 4 sertraline, 3 citalopram, 2 paroxetine, and 2 fluvoxamine trials proved helpful.

As a result of mixed reports of therapeutic effects of various SSRIs openly prescribed, we designed a double-blind, placebo-controlled discontinuation trial. We hypothesized that the SSRI escitalopram would markedly diminish or abolish kleptomania behavior under open-label conditions and, under double-blind conditions, would be superior to placebo in preventing relapse among responders. Escitalopram was chosen for its good tolerability and relative lack of drug interactions.[27]

METHOD

Between December 2002 and March 2005, we recruited, by means of radio and print advertisements and notification of local courts and lawyers in the San Francisco Bay Area, subjects aged 20 years and older with kleptomania of ≥ 1 year's duration meeting DSM-IV diagnostic criteria. The DSM-IV criterion of stealing "objects that are not needed for personal use or for their monetary value" was particularly heavily weighted in judging study eligibility. Subjects' kleptomania had to have resulted in appearance before a judge for sentencing or occurred at least weekly by self-report. Comorbid conditions were determined by psychiatric interview using the Mini-International Neuropsychiatric Interview, version 4.4[28,29] and the Minnesota Impulsive Disorders Interview-Self Report,[30] reviewed with the subject. We excluded subjects with organic mental disorders, psychotic mental disorders, mental retardation or developmental disabilities, substance or alcohol abuse, mood disorders with current suicidal risk, a history of bipolar I or II disorder, factitious disorders, dissociative disorders, anorexia nervosa, or antisocial personality disorder or other personality disorders sufficiently severe to interfere with cooperation with the study and those requiring other psychotropic medications or taking medications that may interact with escitalopram. We obtained informed consent from all participants, and an institutional review board, the Stanford University Administrative Panel on Human Subjects, approved the project.

To increase subjects' willingness to report truthfully their theft episodes, subjects were informed that, to help protect their privacy, the investigators had obtained a confidentiality certificate from the U.S. Department of Health and Human Services to prevent the investigators from being forced (for example, by court subpoena) to disclose information that would identify the subject in any federal, state, or local civil, criminal, administrative, legislative, or other proceedings. Subjects were further informed that even without this certificate, a physician aware of a misdemeanor crime (for example, stealing) is not obligated under California law to report that crime.

Our primary outcome measure was the number of theft episodes per week as recorded and reported by the subject. Since no validated scale exists for measuring the severity of kleptomania, we adapted as a secondary outcome measure the Yale-Brown Obsessive Compulsive Scale[31,32] (YBOCS-K), which has been previously adapted to measure outcome in other impulse control disorders (pathological gambling, skin picking, trichotillomania). Other secondary outcome measures included the following: the subject's rating of the average intensity of stealing impulses (on a 0–4 scale: 0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = irresistible); arrest rates (as reported by subjects); Clinical Global Impressions-Severity scale (CGI-S) and -Improvement scale (CGI-I) scores; and the Patient Global Impressions-Improvement (PGI-I) score.[33] Depressive symptoms were rated by means of the Montgomery-Asberg Depression Rating Scale (MADRS).[34]

A responder was prospectively defined as a subject experiencing a > 50% decrease from the baseline rate of theft episodes per week and a CGI-I score of "much" or "very much improved." The 50% criterion was chosen as a clinically meaningful but not unduly demanding indicator of symptom improvement. Relapse was defined as returning to a weekly theft episode rate greater than 50% of the baseline rate and having a CGI-I score of "minimally improved" or less. A depression responder was defined as

Table 1. Baseline Characteristics of 24 Kleptomania Subjects Treated With Escitalopram

Characteristic	Value
Age, mean ± SD (range), y	49.5 ± 10.7 (27–69)
Age at onset, mean ± SD (range), y	17.3 ± 13.4 (5–56)
Gender, N (%)	
Women	17 (71)
Men	7 (29)
Ethnicity, N (%)	
White	17 (71)
African American	4 (17)
Asian/Pacific Islander	1 (4)
Hispanic	2 (8)
Marital status, N (%)	
Single	5 (21)
Married	13 (54)
Divorced	6 (25)
Employment status, N (%)	
Full time	14 (58)
Part time	3 (13)
Not seeking	2 (8)
Unemployed	2 (8)
Student	1 (4)
Retired	1 (4)
Disabled	1 (4)
Comorbid conditions, N (%)	
Major depression	3 (13)
Panic disorder	3 (13)
Social phobia	2 (8)
Bulimia	2 (8)
Generalized anxiety disorder	1 (4)
Compulsive shopping disorder	4 (17)
Trichotillomania	1 (4)
Skin picking	1 (4)
Intermittent explosive disorder	1 (4)

having a baseline MADRS of ≥ 16 and an endpoint MADRS of < 16, with a ≥ 50% decrease from baseline.

Subjects were seen for 12 visits across 26 weeks: at screening and at baseline (the day medication started), then again at the end of weeks 1, 2, 4, 6, 7, 8, 12, 16, 20, and 24. At each visit we monitored vital signs, obtained the primary and secondary outcome measures, and monitored spontaneously reported medication side effects.

Escitalopram was started at 10 mg/day and increased at the end of week 4 to 20 mg/day if the therapeutic response was incomplete and troubling side effects were absent.

At the end of week 7, responders were randomly assigned to either a 1-week taper followed by 16 weeks of placebo or continuation treatment for 17 additional weeks at their week 7 escitalopram dose. During these 17 weeks no dose adjustments were allowed.

We utilized 2-sided t tests to examine changes in continuous variables for statistical significance, with $p \leq .05$. We looked for relationships between outcome measures using Spearman (nonparametric) correlation analyses. In the double-blind phase, we compared the treatment groups using t tests for continuous variables and Fisher exact test for categorical variables, again with $p \leq .05$.

RESULTS

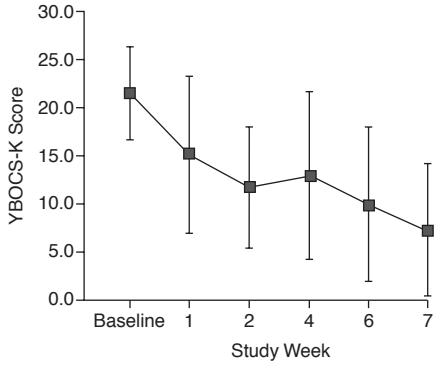
We interviewed 78 callers and screened 26 potential subjects in person before 24 subjects were enrolled. Of the 32 callers who did not have kleptomania, 11 had remorseless stealing more consistent with antisocial personality disorder, 3 had bipolar affective disorder and stole only while in a manic or hypomanic phase, 3 stole to support a drug habit, and the remaining 15 called to learn more about the disorder but did not themselves suffer from it. Four callers (1 of whom was screened in person) were not currently stealing but had suffered from kleptomania and were willing to participate. The other 18 callers were not eligible because they lived too far away (N = 6), were receiving treatment for kleptomania (N = 10), or suffered from comorbid depression that appeared to require treatment not constrained by our study (N = 2).

The 24 subjects (17 women, 7 men) had a mean age of 49.5 years and a mean age at onset of kleptomania of 17.3 years (Table 1). No subject was court-referred, but 6 were referred by their lawyers or probation officers. The subjects' mean number of kleptomania-related arrests was 3.6 (range, 0–10). Ten had been treated with an SSRI, although not always specifically for kleptomania. We were unable to verify the adequacy and effectiveness of many of these trials. Twelve subjects had received individual psychotherapy, and 3 subjects had received group psychotherapy, all without substantial effect on kleptomania. At baseline, 9 subjects had 1 active comorbid condition, 3 subjects had 2 conditions, and 1 subject had 3 conditions (Table 1).

Open-Label Results

Mean ± SD thefts per week decreased significantly from 4.0 ± 3.3 (range, 1–17) to 0.9 ± 1.2 (range, 0–4) ($t = 5.7$, $p < .0001$). The mean ± SD score on the YBOCS-K decreased significantly from 21.5 ± 4.9 (range, 14–31) at baseline to 7.3 ± 6.9 (range, 0–23) at the end of week 7 (Figure 1) ($t = 10.1$, $p \leq .0001$). Mean strength of urges to steal decreased significantly from 2.6 to 0.9 ($t = 9.9$, $p \leq .0001$). Nineteen subjects (79%) were responders, and 15 were randomly assigned to double-blind treatment. Five subjects (4 responders) withdrew early: 1 for an unrelated illness, 1 for protocol deviation, 2 for side effect (insomnia), and 1 for the decision to withdraw consent. Mean ± SD MADRS scores decreased from 9.1 ± 8.8 (range, 0–34) to 4.6 ± 5.2 (range, 0–18). Percent changes from baseline to week 7 in YBOCS-K and MADRS scores were significantly correlated (Spearman $r = 0.61$, $p = .006$). Of the 3 subjects with major depression at baseline, 1 withdrew because of an unrelated physical illness, 1 was a kleptomania but not a depression responder (baseline MADRS = 16, week 7 MADRS = 13), and 1 experienced response for both major depression and kleptomania. Five (83%) of the 6 subjects referred by lawyers or probation officers were responders.

Figure 1. Mean ± SD YBOCS-K Score (LOCF) During 7 Weeks of Open-Label Escitalopram Treatment (N = 24)



Abbreviations: LOCF = last observation carried forward, YBOCS-K = Yale-Brown Obsessive Compulsive Scale modified for kleptomania.

Adverse events were generally mild to moderate; those affecting 10% or more of subjects were nausea (29%), insomnia (21%), somnolence (17%), and diarrhea (13%).

Double-Blind Results

Randomization assigned 7 subjects to continue escitalopram treatment and 8 subjects to discontinue escitalopram treatment and begin taking placebo. The 2 treatment groups did not differ at study baseline or at randomization in gender distribution, ethnicity (white versus non-white), age at kleptomania onset, number of years of stealing, weekly theft frequency, number of previous arrests, or YBOCS-K or MADRS scores. Further, they did not differ significantly in the mean percent decrease from baseline to week 7 in YBOCS-K score (escitalopram: 74% and placebo: 81%). However, the proportion of married subjects was higher in the escitalopram group (86%) than in the placebo group (25%) (Fisher exact p = .04, 2 sided). Mean ± SD medication doses were escitalopram, 18.6 ± 3.8 mg/day and placebo 17.5 ± 4.6 mg/day. Two placebo subjects withdrew early: 1 for depression and 1 because she was jailed for a pre-study offense. One escitalopram subject withdrew early by moving away.

Three (43%) of 7 subjects randomly assigned to escitalopram relapsed compared with 4 (50%) of 8 subjects randomly assigned to placebo (Fisher exact test p = .38). Relapses occurred somewhat earlier in the escitalopram group (1 each in weeks 8, 10, and 18) than in the placebo group (1 each in weeks 10, 22, 23, and 24). Only 1 subject (in the placebo group) was arrested during the double-blind phase, at week 24. The probability of relapse in both treatment groups was unrelated to kleptomania severity at baseline or at randomization, as measured by YBOCS-K score. Percent change in YBOCS-K and MADRS scores from randomization to endpoint were not significantly correlated (Spearman r = 0.08, p = .90). Both subjects with baseline comorbid major depression had recovered at week 24 (MADRS = 0) even though the subject randomly assigned to placebo had had mild mood symptoms (MADRS = 13) at randomization. Both remained kleptomania responders at week 24.

The outcome for the 5 subjects referred by lawyers or probation officers did not differ from that of the other subjects: 1 of 2 subjects randomly assigned to continue escitalopram treatment relapsed (in week 8), and 1 of 3 subjects randomly assigned to placebo relapsed (at week 23).

Only 2 subjects, 1 in each treatment group, had side effects at the time of randomization, and in both cases these side effects continued unchanged until endpoint.

DISCUSSION

Limitations of this study include the recruitment methods; the small sample size, which severely limited the power to identify potential treatment-responsive subgroups of subjects; and failure to evaluate the integrity of the blind (a common shortcoming).[35] The blind was not, however, significantly compromised by changes in side effects after randomization, since these were present in only 2 subjects and remained unchanged. The study is also limited by reliance on self-reported rates of stealing. Although we removed legal jeopardy motivations to lie about stealing, we cannot be certain that subjects did not underreport stealing or lie to us for other reasons. The failure of active drug to outperform placebo in the double-blind discontinuation phase indicates, however, that any underreporting that may have occurred did not bias the study toward a favorable conclusion regarding drug treatment.

A high response rate occurred in the 7-week, open-label escitalopram treatment phase (79%). This high response rate may reflect in part the subjects' high hopes for what we presented as a promising new drug treatment. In addition, elements of supportive psychotherapy, such as providing information, support, and caring, may have been therapeutic, although subjects' previous psychotherapy trials did not give evidence that these elements, ordinarily present in psychotherapy experiences, had been particularly beneficial. It is difficult to credit improvement to a decision to seek treatment, since many subjects had many times unsuccessfully resolved to stop stealing.

The similar relapse rates in the escitalopram and placebo groups during the 17-week double-blind discontinuation phase suggest that the open-label response was largely or even wholly a placebo response. One could argue that the relapses in the escitalopram continuation group were largely due to drug tolerance or to an unfortunate increase in stress or some other kleptomania inducer and that some placebo subjects did not relapse because they had learned during the open-label phase how to control their stealing urges. This explanation, however, fails

the test of Occam's razor, which counsels against multiplying assumptions. Moreover, the relapses in the escitalopram continuation group tended to occur earlier than those in the placebo group and well before the end of the remission periods reported in the SSRI case report literature cited earlier.[12,17–19] Thus, a placebo response underlying most or all of the open-label escitalopram response remains the most likely explanation. Still, the small number of subjects and the high rate of attrition (33% over the course of the study) leave room for other explanations.

Perhaps, as a historical overview suggests,[1] kleptomania, like major depression and other psychiatric disorders of unknown etiology, is not a homogeneous disorder, but rather a pathological behavior with a number of possible causes, some best conceptualized in biological terms (e.g., neurotransmitter dysfunction) and some in psychological terms (e.g., impaired risk/reward assessment). Such heterogeneity would explain the persistence of drug effect in some subjects and its disappearance in others.

Consistent with this possibility, and despite our every effort to screen out individuals with sociopathic tendencies, some subjects challenged the notion that their behavior was purely impulsive. Upstanding citizens in other regards and clearly not meeting criteria for antisocial personality disorder, they still "planned" their stealing: they felt an urge to steal an hour or more before reaching the store, and sometimes awakened with the urge. They went with the intention of obtaining relief, often choosing retailers with loose security (e.g., no camera surveillance) and, in 2 cases, wearing oversized clothes with big pockets to better hide the stolen items. Consistent with the DSM-IV criteria, the items were largely unneeded and easily affordable, and our subjects wished they could stop stealing (1 subject was arrested returning a stolen item). However, they often rationalized their stealing, attributing it to deprived childhoods or other stressors, and wished to stop more out of fear of apprehension and anticipated shame than from deep feelings of guilt.

Kleptomania did not generally appear to be a symptom of a formal mood or anxiety disorder; most patients had neither. Moreover, we observed no consistent relationship between change in mood symptoms as measured by the MADRS and change in severity of kleptomania as measured by the YBOCS-K.

In view of our results, there remains no pharmacotherapeutic (or psychotherapeutic) treatment of kleptomania with effectiveness demonstrated under controlled conditions. Clinicians who encounter patients with this disorder could reasonably explore treatment with medications for which the greatest number of positive reports exist (e.g., naltrexone and the SSRIs) and the form of psychotherapy with the greatest number of positive case reports (cognitive-behavioral therapy [reviewed elsewhere[2,10]]). Although interesting speculation regarding the pathophysiology underlying kleptomania has been put forward,[24,36] no empirical evidence regarding abnormal neural functioning is available to guide treatment decisions.

CONCLUSION

The high response rate during open-label escitalopram treatment was not better maintained by double-blind escitalopram than by placebo. Nonetheless, a true drug effect of escitalopram, removing or markedly reducing the urge to steal in some individuals, cannot be ruled out by this small study's results. In our present state of knowledge, kleptomania can be regarded as a pathological behavior that may respond to various medications, to psychotherapy, or to their combination; but definitive studies to guide these choices are needed.

Drug names: buspirone (BuSpar and others), citalopram (Celexa and others), escitalopram (Lexapro and others), fluoxetine (Prozac and others), imipramine (Tofranil and others), lithium (Eskalith, Lithobid, and others), naltrexone (Vivitrol, Revia, and others), nortriptyline (Pamelor, Aventyl, and others), paroxetine (Paxil, Pexeva, and others), sertraline (Zoloft and others), topiramate (Topamax and others).

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