Gabapentin Treatment for Alcohol Dependence
A Randomized Clinical Trial

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IMPORTANCE Approved medications for alcohol dependence are prescribed for less than 9% of US alcoholics.

OBJECTIVE To determine if gabapentin, a widely prescribed generic calcium channel/γ-aminobutyric acid-modulating medication, increases rates of sustained abstinence and no heavy drinking and decreases alcohol-related insomnia, dysphoria, and craving, in a dose-dependent manner.

DESIGN, PARTICIPANTS AND SETTING A 12-week, double-blind, placebo-controlled, randomized dose-ranging trial of 150 men and women older than 18 years with current alcohol dependence, conducted from 2004 through 2010 at a single-site, outpatient clinical research facility adjoining a general medical hospital.

INTERVENTIONS Oral gabapentin (dosages of 0 [placebo], 900 mg, or 1800 mg/d) and concomitant manual-guided counseling.

MAIN OUTCOMES AND MEASURES Rates of complete abstinence and no heavy drinking (coprimary) and changes in mood, sleep, and craving (secondary) over the 12-week study.

RESULTS Gabapentin significantly improved the rates of abstinence and no heavy drinking. The abstinence rate was 4.1% (95% CI, 1.1%-13.7%) in the placebo group, 11.1% (95% CI, 5.2%-22.2%) in the 900-mg group, and 17.0% (95% CI, 8.9%-30.1%) in the 1800-mg group (P = .04 for linear dose effect; number needed to treat [NNT] = 8 for 1800 mg). The no heavy drinking rate was 22.5% (95% CI, 13.6%-37.2%) in the placebo group, 29.6% (95% CI, 19.1%-42.8%) in the 900-mg group, and 44.7% (95% CI, 31.4%-58.8%) in the 1800-mg group (P = .02 for linear dose effect; NNT = 5 for 1800 mg). Similar linear dose effects were obtained with measures of mood (F₂ = 7.37; P = .001), sleep (F₂ = 13.6; P < .001), and craving (F₂ = 3.56; P = .03). There were no serious drug-related adverse events, and terminations owing to adverse events (9 of 150 participants), time in the study (mean [SD], 91 [3.8] weeks), and rate of study completion (85 of 150 participants) did not differ among groups.

CONCLUSIONS AND RELEVANCE Gabapentin (particularly the 1800-mg dosage) was effective in treating alcohol dependence and relapse-related symptoms of insomnia, dysphoria, and craving, with a favorable safety profile. Increased implementation of pharmacological treatment of alcohol dependence in primary care may be a major benefit of gabapentin as a treatment option for alcohol dependence.

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Gabapentin Treatment for Alcohol Dependence

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n estimated 3.8% of all deaths and 4.6% of disability-adjusted life-years globally are attributable to patho-
logical alcohol use. Such alcohol-attributable costs exceed 1% of the gross national product of high- and
middle-income countries, making pathological alcohol use one of the largest avoidable risk factors for the worldwide
burden of disease. Alcohol use disorders are present across medical specialties, with alcohol-related deaths particularly
prevalent in the categories of injury, cancer, cardiovascular
disease, and liver cirrhosis. Nonetheless, implementation of
alcohol-specific medications remains limited across most
medical specialties. Of the estimated 8 450 000 Americans
with current alcohol dependence, only 720 000 prescriptions
were filled in 2007 for US Federal Food and Drug
Administration (FDA)-approved medications for alcohol
dependence; those prescriptions were provided primarily
by psychiatrists.

Alcohol dependence, also referred to as alcohol use
disorder, is a chronic, relapsing disorder marked by compulsive
alcohol use, an inability to stop drinking despite harmful
consequences, and the emergence of a withdrawal syndrome
on cessation of use. Early abstinence is associated with acti-
vation of brain stress systems in the extended amygdala. Clin-
ically, protracted abstinence involves symptoms of craving,
mood, and sleep disturbance, all of which have been identi-
fied as risk factors for relapse.

Gabapentin (Neurontin and multiple generic formulas-
tions) is FDA approved for the treatment of epileptic sei-
zures and neuropathic pain. It is believed to act by blocking
a specific α-2d subunit of the voltage-gated calcium channel
at selective presynaptic sites and, as a result, to indirectly
modulate γ-aminobutyric acid (GABA) neurotransmission.
Preclinical findings indicate that gabapentin normalizes the
stress-induced GABA activation in the amygdala that is
associated with alcohol dependence, and it provides an
excellent preclinical rationale for evaluating gabapentin as a
treatment for alcohol dependence. A human laboratory
study found that gabapentin reduced alcohol-cued craving
and sleep disturbance in alcohol-dependent participants, and
clinical studies of various disorders report that gaba-
pentin reduced craving and disturbances in sleep and mood.
Earlier studies of gabapentin in alcohol-dependent participants attempting to abstain following withdrawal support the safety and potential efficacy of gabapentin in alcohol-dependent patients, but definitive con-
clusions were limited by either small sample size, method-
ological issues, or dosing issues.

The present study was therefore designed to provide a more definitive evalua-
tion of the efficacy and safety of gabapentin at the highest
(1 800 mg/d) and lowest (900 mg/d) FDA-approved doses vs
placebo in a 3-arm, parallel-group, double-blind, random-
ized clinical trial involving recently abstinent outpatient
volunteers with alcohol dependence. We hypothesized that
gabapentin would be associated with significant linear
dose-related increases in rates of sustained abstinence and
no heavy drinking, and decreases in abstinence-related
symptoms involving sleep, mood, and craving, over the
12-week treatment course.

Methods

Setting and Participants

Our single-site outpatient study was conducted at The Scripps
Research Institute, La Jolla, California. Our study physicians also
practice internal and hospital medicine at the adjacent Scripps
Green Hospital and Clinics; these facilities provide a broad range
of medical services to the greater community of San Diego. The
study protocol was approved by the Scripps institutional re-
view board (IRB); written informed consent was obtained from
all participants.

Treatment-seeking volunteers with alcohol dependence
were recruited primarily via IRB-approved print and Internet
advertisements. The first participant was randomized in April
2004, and the last follow-up visit was completed February 2010.
To be eligible, men and women had to be older than 18 years;
meet the Diagnostic and Statistical Manual of Mental Disor-
ders (Fourth Edition) (DSM-IV) criteria for current alcohol
dependence; and be abstinent from alcohol at least 3 days prior
to randomization. Exclusion criteria were risk for significant
withdrawal based on a Clinical Institute Withdrawal Assess-
ment of Alcohol Scale, Revised (CIWA-Ar) score higher than
9; more than 1 month of abstinence; dependence on sub-
stances other than alcohol or nicotine; a urine drug screen
that was positive for benzodiazepines, cocaine, methamphet-
amine, tetrahydrocannabinol, methadone, or opiates; clini-
cally significant medical or psychiatric disorders; treatment
with medications that could affect study outcomes; and treat-
ment mandated by a legal authority.

Assessments

Medical clearance for randomization was provided by study
physicians (F.S., M.K., and A.B.) and included an electrocar-
diogram, pregnancy test, complete blood cell count with differen-
tial, urinalysis, blood chemistry testing, and physical exam-
ination. The Structured Clinical Interview for DSM IV
(SCID) was conducted by study clinicians to establish diag-
nostic admission criteria. Study visits took place weekly
throughout the 12-week, double-blind phase, at 13 and 24 weeks
posttreatment, and included standardized assessments of al-
cohol use, craving, mood, sleep, and safety evaluations.

Alcohol use was assessed with the daily record of standard
drinks obtained by the Timeline Followback Interview with a
drinking diary as a memory guide, and validated by weekly
breathalyzer determinations, monthly γ-glutamyltransferase
(GGT) values and collateral informant reports. A standard drink
was defined as 14 g of absolute ethanol content, which is equiva-
lent to 12 oz of beer, 1.5 oz of hard liquor, or 5 oz of wine.
A heavy drinking day was defined as 4 or more drinks per day for
women and 5 or more drinks per day for men. Drinking data
were collected by experienced research personnel.

Drinking urges were assessed by self-report using the Al-
cohol Craving Questionnaire—Short Form. Mood was evaluated
by self-report with the Beck Depression Inventory II. Multiple
components of sleep disturbance were assessed by self-
report using the Pittsburgh Sleep Quality Index, modi-

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Safety evaluations included weekly vital signs, the Systematic Assessment for Treatment Emergent Events–General Inquiry (SAFTEE-GI), and urine screening for drugs of abuse; specimens for blood chemistry testing and urinalysis were obtained monthly and analyzed by LabCorp.

Procedures
Simple randomization procedures were followed to randomly assign participants to double-blind treatment with oral gabapentin, 900 mg or 1800 mg (hereinafter, 900-mg and 1800-mg groups), or placebo, in a 1:1:1 ratio, using a computer-generated randomization code provided by our laboratory biostatistician (V.G.). The code was kept by the study pharmacist, who provided participants with weekly medication in a blister card package that was consecutively numbered for each participant and prepared according to the randomization schedule. For all groups, each package contained 2 identical capsules to be taken 3 times a day. For the gabapentin groups, a placebo capsule was replaced with an identical 300-mg capsule of gabapentin on the evening of day 1, the morning of day 2, the afternoon of day 3, and on a similar schedule each day until the assigned fixed dose of 900 mg was achieved on day 4 or 1800 mg was achieved on day 6 (eTable 1 in Supplement). Participants were maintained on the assigned dose until week 11, and then were titrated off active medication by substituting 1 placebo capsule for 1 capsule of active medication per day, in the reverse order of the initial dose titration, until all participants received only placebo by the end of week 12. Participants returned their blister cards at each weekly study visit for drug accountability and compliance review. Correct drug assignment was verified retrospectively by determining gabapentin concentration in plasma samples obtained at week 2 and frozen for poststudy analysis by gas chromatography and mass spectrometry.

Concurrent with study medication, study clinicians provided participants with 20 minutes of weekly manual-guided counseling designed to increase motivation, abstinence, and medication compliance. At study onset, participants were provided with schedules for local self-help groups and were encouraged to attend any self-help groups or psychosocial therapies they found beneficial; attendance was not further encouraged but was documented at each study visit.

Outcome Measures
Since the time our statistical plan was designed (2003), responder analyses based on definitions that predict clinical benefit have been proposed by the FDA as preferable to analyses of group means. The FDA's rationale for this change is that mean differences are difficult to interpret with regard to clinical relevance. Thus, we modified our original analysis of mean abstinence duration to be a responder analysis based on the rate of complete abstinence over the 12-week study. We also included the rate of no heavy drinking over the 12-week trial as a coprimary outcome. We also reported change (number of heavy drinking days per week) over the 12-week study period, as supportive primary outcomes. We also report change in GGT, a widely accepted and validated biomarker of drinking reduction, as a supportive primary outcome.

Prespecified secondary outcomes were standardized measures of alcohol craving, sleep, and mood over the 12-week study period.

Figure 1. Flow of Participants Through the Trial

Pl indicates principal investigator.
Baseline demographic and clinical characteristics were compared by $\chi^2$ and analysis of variance as appropriate. Outcome analyses were intention-to-treat and involved all participants who were randomly assigned ($n = 150$). All tests were 2-tailed, and an $\alpha < .05$ was considered statistically significant. Linear dose effects for rates of complete abstinence and no heavy drinking over the 12-week study were assessed using the extended Mantel-Haenszel $\chi^2$ test for linear association. Linear dose effects for rates of complete abstinence and no heavy drinking over the 12-week study were assessed using the extended Mantel-Haenszel $\chi^2$ test for linear association.

### Table. Pretreatment Demographic and Clinical Characteristics by Treatment Group

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Placebo (n = 49)</th>
<th>Gabapentin, 900 mg/d (n = 54)</th>
<th>Gabapentin, 1800 mg/d (n = 47)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic Characteristic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>46.8 (11.3)</td>
<td>41.9 (10.1)</td>
<td>45.2 (11.3)</td>
</tr>
<tr>
<td>Sex, No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>28 (57.1)</td>
<td>21 (38.9)</td>
<td>16 (34.0)</td>
</tr>
<tr>
<td>Male</td>
<td>21 (42.9)</td>
<td>33 (61.1)</td>
<td>31 (66.0)</td>
</tr>
<tr>
<td>White, non-Hispanic,a,b No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>42 (85.7)</td>
<td>40 (74.1)</td>
<td>40 (85.1)</td>
</tr>
<tr>
<td>Full-time employment, No. (%)</td>
<td>23 (46.9)</td>
<td>30 (55.6)</td>
<td>17 (36.2)</td>
</tr>
<tr>
<td><strong>Clinical Characteristic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of DSM-IV criteria met for alcohol dependence, 3 of 7 criteria required for diagnosis</td>
<td>5.8 (1.2)</td>
<td>6.1 (1.0)</td>
<td>5.5 (1.3)</td>
</tr>
<tr>
<td>Alcoholism Clinical Global Impression, No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very mild</td>
<td>1 (2.0)</td>
<td>2 (3.7)</td>
<td>0</td>
</tr>
<tr>
<td>Mild</td>
<td>9 (18.4)</td>
<td>10 (18.25)</td>
<td>12 (25.5)</td>
</tr>
<tr>
<td>Moderate</td>
<td>26 (53.1)</td>
<td>32 (59.3)</td>
<td>28 (59.6)</td>
</tr>
<tr>
<td>Marked</td>
<td>10 (20.4)</td>
<td>8 (14.8)</td>
<td>5 (10.6)</td>
</tr>
<tr>
<td>Severe</td>
<td>3 (6.1)</td>
<td>2 (3.7)</td>
<td>1 (2.1)</td>
</tr>
<tr>
<td>Very severe</td>
<td>0</td>
<td>0</td>
<td>1 (2.1)</td>
</tr>
<tr>
<td>No. of drinks per weekc</td>
<td>47.3 (28.7)</td>
<td>40.5 (25.0)</td>
<td>40.9 (23.2)</td>
</tr>
<tr>
<td>Drinking days per weekc</td>
<td>5.2 (2.0)</td>
<td>5.4 (3.1)</td>
<td>5.3 (1.8)</td>
</tr>
<tr>
<td>Years of heavy drinking</td>
<td>15.0 (10.4)</td>
<td>14.3 (9.7)</td>
<td>14.0 (9.6)</td>
</tr>
<tr>
<td>Parental alcoholism, No. (%)</td>
<td>18 (36.7)</td>
<td>26 (49.1)</td>
<td>21 (44.7)</td>
</tr>
<tr>
<td>No prior alcoholism treatment, No. (%)</td>
<td>34 (70.1)</td>
<td>32 (60.4)</td>
<td>33 (71.7)</td>
</tr>
<tr>
<td>Consecutive days abstinent prior to randomization</td>
<td>3.2 (4.1)</td>
<td>3.2 (4.0)</td>
<td>2.7 (3.0)</td>
</tr>
<tr>
<td>γ-glutamyl transferase level $&gt;$ ULN, No. (%)</td>
<td>7 (14.3)</td>
<td>13 (24.1)</td>
<td>7 (14.9)</td>
</tr>
<tr>
<td>Pittsburgh Sleep Quality Index scoreb (range, 0-30)</td>
<td>4.9 (2.7)</td>
<td>3.9 (2.3)</td>
<td>3.5 (2.6)</td>
</tr>
<tr>
<td>Beck Depression Inventory II scoreb (range 0-63)</td>
<td>8.6 (6.7)</td>
<td>9.5 (8.0)</td>
<td>8.3 (6.9)</td>
</tr>
<tr>
<td>Alcohol Craving Questionnaire scoreb (range, 7-84)</td>
<td>42.5 (13.6)</td>
<td>42.5 (12.0)</td>
<td>42.5 (10.6)</td>
</tr>
</tbody>
</table>

Abbreviations: DSM-IV, Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition); ULN, upper level of normal range.

a Data are given as means (SDs) unless otherwise indicated.

b Treatment groups did not differ significantly on any pretreatment variable.

Race and ethnicity were self-reported by the participants.

Mean values are derived from the 90-day period prior to intake.

d Higher scores indicate worse condition.

### Results

#### Participants

Following recruitment, 185 evaluations yielded the desired sample size of 150 randomized participants (Figure 1). Treatment groups did not differ on pretreatment demographic and clinical variables, as shown in the Table.

The mean (SD) time in study (9.1 [3.8] weeks; $P = .52$) and rate of study completion (85 of 150 participants; $P = .46$) did not differ.
differ among treatment groups, nor did the reasons for termination (Figure 1) \((P = .83)\). The mean rate of medication compliance, defined as number of pills taken divided by number prescribed during study participation, was 96.2% and did not differ among groups \((P = .79)\). Groups were similar in their ability to correctly guess the identity of their medication when asked to do so on study completion (59% for participants receiving gabapentin and 45% for participants receiving placebo; \(P = .21\)).

Outcomes

Gabapentin had a significant linear dose effect in increasing the rates of complete abstinence \((\chi^2 = 4.19; P = .04)\) and no heavy drinking \((\chi^2 = 5.39; P = .02)\) over the 12-week course of treatment, relative to placebo (Figure 2). The rate of sustained 12-week abstinence was 4.1% (95% CI, 1.1%-13.7%) in the placebo group, 11.1% (95% CI, 5.2%-22.2%) in the 900-mg group, and 17.0% (95% CI, 8.9%-30.1%) in the 1800-mg group. Gabapentin, 1800 mg, had the greatest treatment effect, with an NNT of 8 (95% CI, 6 to \(\infty\)) and an OR = 4.8 (95% CI, 0.9-35.0), indicating a large effect size for abstinence. The rate of no heavy drinking was 22.5% (95% CI, 13.6%-37.2%) in the placebo group, 29.6% (95% CI, 19.1%-42.8%) in the 900-mg group, and 44.7% (95% CI, 31.4%-58.8%) in the 1800-mg group. The 1800-mg group had an NNT of 5 (95% CI, 3-78) and OR = 2.8 (95% CI, 1.1-7.5), indicating a medium effect size for no heavy drinking. Compared with placebo, gabapentin also showed significant linear decreases in the average number of days of heavy drinking per week \((t = -13.12; P < .001 [Figure 3A])\); 900-mg group: -1.8 [95% CI, -2.2 to -1.3]; 1800-mg group: -2.0 [95% CI, -2.5 to -1.5]; \(t = -8.14; P < .001\) and the number of drinks consumed per week \((t = -5.32; P < .001 [Figure 3B])\); 900-mg group: -2.2 [95% CI, -5.3 to 1.0]; 1800-mg group: -4.7 [95% CI, -7.8 to -1.5].
Gabapentin showed significant linear dose effects on craving, mood, and sleep (Figure 4A-C). Over the course of treatment, significant dose-dependent reductions were obtained on the Alcohol Craving Questionnaire ($F_2 = 3.56; P = .03$; gabapentin, 1800 mg, vs placebo: $-6.8$ [95% CI, $-12.1$ to $-1.5$]; $t = -2.52; P = .01$) the Beck Depression Inventory II ($F_2 = 7.37; P = .001$; gabapentin, 1800 mg, vs placebo: $-1.1$ [95% CI, $-2.0$ to $-0.3$]; $t = -2.57; P = .01$), and the Pittsburgh Sleep Quality Index total score ($F_2 = 136; P < .001$; gabapentin, 1800 mg, vs placebo: $-1.5$ [95% CI, $-2.1$ to $-0.8$]; $t = -4.46; P < .001$).

Safety, Tolerability, and Concomitant Therapy
Gabapentin was well tolerated, with no deaths and no serious drug-related adverse events. Nine participants discontinued the study owing to adverse events. Of these, 5 were rated as drug related by blinded study physicians: 2 complaints of headache (900 mg), 2 complaints of fatigue (1 in the 900-mg group and 1 in the 1800-mg group), and 1 complaint of euphoria and feeling “on speed” (placebo group). No differences were found among groups in type of adverse events (eTable 2 in Supplement), with 10% or more of the sample complaining of fatigue (23%), insomnia (18%), and headache (14%). Groups also were similar in the number (mean [SD], 1.98 [2.14]; $P = .53$) and severity (1.72 [1.14]; $1 =$ mild, $2 =$ moderate; $P = .63$) of adverse events reported. Groups did not differ in body weight, vital signs, or in measures from urinalysis and blood chemistry testing that took place over the course of treatment. No evidence was found of drug diversion or substitution; of the 1242 urine drug screen samples collected in our study, 27 (2%) tested positive for other drugs of abuse, primarily marijuana and prescription drugs. Five participants attended individual therapy, and 9 attended Alcoholics Anonymous (AA) meetings during the course of the study. Attendance was not associated with drug group or primary outcome measures, with 1 exception: participants who were completely abstinent attended fewer AA meetings than those who were not abstinent (41 vs 89 meetings; $P = .01$). All drug-related adverse events resolved within 1 week of drug discontinuation. There was no evidence of rebound in alcohol use, craving, insomnia, or dysphoria when gabapentin was tapered.

Discussion
Beneficial effects of gabapentin for the treatment of alcohol dependence were found in the intention-to-treat population over the 12-week course of treatment on (1) the rates of complete abstinence and no heavy drinking; (2) the number of heavy drinking days and the number of drinks consumed per week; and (3) severity of craving, insomnia, and dysphoria. Results followed a linear dose-effect, with greatest efficacy achieved at the 1800-mg dose. Laboratory measures of GGT
provided validation of gabapentin’s effects on self-reported drinking outcomes. Significant effects were found to persist posttreatment in study completers who participated in the week-24 follow-up assessment.

Gabapentin had a favorable safety profile, and there were no unexpected or serious drug-related adverse events or differences in study discontinuation rates owing to adverse events. Of note, somnolence has been a commonly reported adverse event in gabapentin pain and epilepsy trials, but it was not a common complaint among our alcohol-dependent participants. Conversely, prior to treatment our participants reported experiencing sleep disturbance and related daytime dysfunction that significantly improved with gabapentin relative to placebo. No evidence of drug substitution or misuse of gabapentin was detected.

This study has several limitations to consider. First, the dropout rate is significant, as is often the case in clinical trials in substance dependence. However, to put our results in context, the treatment completion rates reported in a meta-analysis of randomized controlled trials involving 6111 outpatients with alcohol dependence were 52.7% for placebo and 57.8% for acamprosate, which is directly comparable with our treatment completion rate of 56%. Furthermore, our mean duration of study participation was 9.1 weeks of a 12-week study, which is a clinically relevant period of drug exposure for assessing treatment effects. Concerns about potential bias introduced by dropouts are mitigated by a lack of differential dropout between groups and by consistency across outcomes that include the assumption of missing at-random and response variables derived from data collected on study without assumption for 96.7% of participants. The validity of results is supported by preclinical and human laboratory studies of gabapentin effects on models of protracted abstinence and by clinical proof-of-concept studies from different groups.

Another limitation is that results from a single-site study may not generalize to all treatment settings and alcohol-dependent populations. Nevertheless, generalizability is supported by the absence of associations between demographic variables with any outcome variable, the high rate of randomized to evaluated volunteers (150:185), and the broad range of alcoholism severity included in our sample. However, none of our community-dwelling volunteers required detoxification. Indeed, our participants typically drank 5 days per week and were able to achieve the required 3 days of abstinence prior to randomization simply with monitoring and advice to taper drinking to further reduce risk.

Rates of alcohol dependence exceed those of all illicit drug dependence disorders combined, and there is a great unmet need for medications to treat alcohol dependence, per se. Thus, co-occurring illicit substance dependence disorders were excluded from the present study. Future studies are warranted to assess gabapentin efficacy in substance use disorders, alone and in combination, that have protracted abstinence symptoms involving craving, mood, and sleep. Indeed, a recent randomized controlled trial of gabapentin in cannabis dependence, the most prevalent illicit drug dependence disorder, found significant reductions in marijuana use, craving, mood, and sleep disturbance with gabapentin relative to placebo. Of note, gabapentin is not appreciably metabolized in the liver, an advantage for patients with alcohol-related liver dysfunction, and is not known to interfere with the metabolism of commonly used illicit or prescribed drugs.

In summary, gabapentin (particularly the 1800-mg dose) effectively treated alcohol dependence and relapse-associated symptoms involving craving, mood, and sleep, and had a favorable safety profile. A sustained posttreatment effect on drinking outcomes was found in those who responded well to gabapentin in the study. Larger studies in more diverse populations of patients with alcohol dependence are needed to replicate and extend these findings. Gabapentin has been used ubiquitously by primary care physicians for many other indications, resulting in familiarity with its pharmacology, pharmacokinetics, and adverse effects. Thus, unlike other approved treatments for alcohol dependence that are prescribed by a small number of specialists, gabapentin may be more readily utilized by primary care physicians. Increased implementation of effective pharmacological treatment for alcohol dependence in primary care may be a major benefit of gabapentin as a treatment option for alcohol dependence.


