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Mood Disorders and Substance Use Disorder: A Complex Comorbidity

Mood disorders, including depression and bipolar disorders, are the most common psychiatric comorbidities among patients with substance use disorders. Treating patients' co-occurring mood disorders may reduce their substance craving and taking and enhance their overall outcomes. A methodical, staged screening and assessment can ease the diagnostic challenge of distinguishing symptoms of affective disorders from manifestations of substance intoxication and withdrawal. Treatment should maximize the use of psychotherapeutic interventions and give first consideration to medications proven effective in the context of co-occurring substance abuse. Expanded communication and collaboration between substance abuse and mental health providers is crucial to improving outcomes for patients with these complex, difficult co-occurring disorders.

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Mood or affective disorders, as defined by the *Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition)*, are classified as depressive or bipolar (Table 1). During the past decade, research results and clinical experience have converged in the recognition that these psychiatric illnesses commonly co-occur with substance use disorders (SUDs) and that the combination has adverse clinical consequences. Mood and SUD comorbidity downgrades the clinical course, treatment outcome, and prognosis for each problem. Theoretically, the converse is also likely to be true: Successful alleviation of one condition should facilitate recovery from the other. Some evidence indicates that treating a comorbid affective disorder can decrease substance abuse and craving (Cornelius et al., 1997).

Researchers and clinicians have begun to develop treatment approaches that address both disorders simultaneously, with early indications of efficacy. This article explores the prevalence and relationship of co-occurring mood disorders and SUDs, describes a methodical approach to assessment, and reviews evidence-based psychotherapeutic and pharmacotherapeutic treatments.

EPIDEMIOLOGICAL RELATIONSHIPS

Two epidemiological studies have examined the prevalence of psychiatric and substance use disorders by conducting diagnostic interview surveys in representative community samples of adults: the National Institute of Mental Health Epidemiologic Catchment Area (ECA) Study (Regier et al., 1990) conducted in

the early 1980s and the National Comorbidity Survey (NCS) conducted in 1991 (Kessler et al., 1994). Both provided striking documentation that mood disorders increase the risk of SUD.

In one study, individuals with depression were twice as likely to have a substance abuse problem.

In the ECA Study, the lifetime prevalence rate for any non-SUD mental disorder was estimated to be 22.5 percent, compared with 13.5 percent for alcohol abuse/dependence and 6.1 percent for other drug abuse/dependence (Regier et al., 1990). Among individuals with a mood disorder, 32 percent had a co-occurring SUD. Of individuals with lifetime major depression, 16.5 percent had an alcohol use disorder and 18 percent had a drug use disorder. SUDs were particularly common among individuals with bipolar disorder—56 percent had a lifetime SUD.

In the NCS, the lifetime prevalence estimate for any mental disorder was 48 percent (Kessler et al., 1997). The estimate for alcohol dependence was 14.1 percent, and for drug dependence 7.5 percent. The lifetime prevalence rate for any mood disorder was 19.3 percent. Compared with individuals with no mood disorders, those with depression were approximately twice as likely, and those with bipolar disorder approximately seven times as likely, to have an SUD. (The ECA study also documented a high rate of co-occurrence of SUD, mood, and anxiety disorders; while anxiety disorders are clinically common and important, they are a separate category of illness and discussion of them is beyond the scope of this paper.)

Substance abuse sometimes 'unmasks' mood disorders by increasing symptom severity to clinically significant levels.

Studies of individuals seeking treatment have resulted in variable estimates of the comorbidity of mood disorders and SUDs. Among those seeking treatment for alcohol dependence, an estimated 20 to 67 percent had experienced depression and 6 to 8 percent had experienced a bipolar disorder at some time in their lives (Brady, Myrick, and Sonne, 1998). In samples of cocaine-dependent individuals, the corresponding estimates have ranged between 30 and 40 percent and between 10 and 30 percent. Rounsaville and colleagues (1991), after assessing 298 cocaine abusers seeking treatment, reported that 44.3 percent had a current mood disorder and 61 percent had a history of mood disorders; 30.5 percent had had at least one episode of major depression and 11.1 percent at least one episode of mania or hypomania. Bipolar disorder appears to be more prevalent among cocaine-dependent individuals than alcohol-dependent individuals (Sonne and Brady, 1999).

One reason for the differences in reported preva-

lence rates is the complexity of diagnostic issues at the interface of mood disorders and SUDs. For example, because abstinence from drugs can temporarily depress mood, a patient who is evaluated while in withdrawal may be misdiagnosed as suffering from a mood disorder. Clinicians may reach different conclusions, depending on when they conduct assessments relative to the patient's entry into treatment.

WHY ARE THESE COMORBIDITIES SO COMMON?

The major mood disorders and their key clinical features can be found in Table 1. Several theories have been proposed to explain the high co-occurrence of substance abuse and mood disorders. In general, they fall into three categories.

Disorder Fostering Disorder

One theory proposes that the pathological effects of a mood disorder or SUD may increase risk for the other. For example, mood disorders may motivate individuals to resort to drugs and alcohol to cope with their negative affective states. Such an explanation would jibe with clinicians' everyday experience of individuals with SUDs saying they use drugs and alcohol to combat unwanted moods. The substances may initially minimize or moderate the mood symptoms, but withdrawal and chronic abuse typically exacerbate mood degradation, leading to increasing abuse and ultimately dependence.

The self-medication explanation implies that individuals will tend to select drugs that alleviate their specific psychiatric symptoms. For example, some psychologists suggest that people with uncontrollable feelings of rage and aggression may choose opiates for these drugs' mellowing effects, while people who are depressed may take cocaine because it exhilarates and energizes them. Studies showing such associations between abusers' drugs of choice and their psychiatric diagnoses or symptoms would strengthen the evidence for the self-medication model, but to date few have been published.

While the self-medication model suggests that mood disorders increase the risk of substance abuse, the converse is also possible. Chronic substance abuse sometimes "unmasks" bipolar or other mood disorders—that is, triggers an increase in symptom severity from a subclinical to a clinically significant level. This appears to occur because in genetically vulnera-

ble individuals, the drugs exacerbate pathophysiological changes in neurotransmitter systems or signaling pathways that already are abnormal and underlie the mood disorder (Markou, Kosten, and Koob, 1998).

Overlapping Neurobiological Pathways

Another proposed explanation for the high comorbidity rate of mood disorders with SUDs involves “kindling.” The term, usually associated with epilepsy, refers to the concept that repeated disruptions, such as occur during seizures, sensitize brain cells. The more sensitized the neurons become, the less it takes to disrupt them, which is why in untreated epilepsy, seizures tend to become more frequent and severe over time. Both alcohol and cocaine sensitize neurons, and this increased sensitivity may contribute to the typical progression from occasional to increasingly frequent and intense use of these substances. Mood disorders often follow a similar course of increasingly distressing symptomatic episodes separated by progressively shorter periods of remission, suggesting that they too may intensify via a kindling process (Post, Rubinow, and Ballenger, 1984). The kindling explanation for comorbidity, then, holds that in vulnerable individuals, an underlying neurobiological tendency to sensitization may promote both drug dependence and mood disorders.

Underlying Genetic Factors

Research has definitively shown that both substance abuse and mood disorders have genetic risk factors. In addition, families with substance abusers are more likely than those without to also have members with mood disorders, and vice versa. These facts raise the possibility that some gene variants may contribute to the risk for both types of illness. A person’s genes might:

- make him or her vulnerable to mood disorders, which he or she might then try to self-medicate, as discussed above;
- shape the brain so that it responds to initial drug exposures in ways that promote chronic substance abuse, with the drugs then wreaking changes that lead to mood disorders;
- cause the brain to develop in a way that directly fosters both types of disorder, for example through vulnerability to neuronal sensitization and kindling.

Diagnostic Confounding

Some portion of the reported high co-occurrence of

TABLE 1. DSM-IV MOOD DISORDER CATEGORIZATION

DISORDER CATEGORY	DISORDER TYPE	KEY FEATURES
Depressive Disorders (no history of manic,* mixed manic,** or hypomanic*** episodes)	Major depressive disorder	2-week duration, with depressed mood/loss of interest
	Dysthymic disorder	Chronic, less severe than MDD; 2-year duration
Bipolar Disorders (manic,* mixed,** or hypomanic*** episodes plus major depressive episodes)	Bipolar I	One or more manic/mixed and depressive episode
	Bipolar II	One or more depressive and at least one hypomanic episode
	Cyclothymia	Chronic/less severe form of bipolar disorder; 2-year duration, with multiple periods of hypomania/depression
Substance-Induced Mood Disorder		Prominent and persistent mood disturbance, direct physiologic consequence of substance use

* Manic episode: abnormally elevated/expansive/irritable mood for at least 1 week.
 ** Mixed episode: meets criteria for manic and depressed episode daily for 1 week.
 *** Hypomanic episode: elevated/expansive/irritable mood for at least 4 days—little functional impairment.

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SUD and mood disorders may represent confounding of mood disorders and transient symptoms related to acute abuse and withdrawal. Drug abuse symptoms can mimic symptoms of both depression and mania. Acute alcohol and stimulant intoxication can produce symptoms of mania or hypomania, and substance withdrawal often manifests as symptoms of dysphoria and depression. Chronic use of central nervous system (CNS) stimulants, such as cocaine and amphetamines, may produce symptoms that are typical of bipolar spectrum disorders, such as euphoria, increased energy, decreased appetite, grandiosity, and paranoia. Conversely, withdrawal from CNS stimulants (especially cocaine) can give rise to anhedonia (inability to feel pleasure), apathy, depressed mood, and suicidal

Some gene variants may contribute to risk for both substance abuse and mood disorders.

ideation. Chronic use of CNS depressants (e.g., alcohol, benzodiazepines, barbiturates, and opiates) can lead to depressive symptoms such as poor concentration, anhedonia, and problems sleeping, while withdrawal from these drugs can result in anxiety and agitation. The more subtle affective disorders such as dysthymia and cyclothymia are particularly difficult to differentiate from symptoms of SUD.

It remains unclear which, if any, of the models discussed explain the high comorbidity between mood disorders and SUDs. The relationship is complex and bidirectional, suggesting an ongoing interaction between these disorders; having one may affect the vulnerability to developing the second or change its clinical course.

ASSESSMENT AND DIAGNOSIS

A substance-abusing patient who exhibits symptoms of a mood disorder may be suffering from acute intoxication or withdrawal, substance-induced mood disorder, preexisting affective disorder, or a combination of these conditions. The potential for diagnostic uncertainty and confusion is high, but a methodical approach can point clinicians in the right direction.

One good strategy for busy treatment settings is to screen patients upon intake to identify those whose affective status warrants more careful assessment and followup. The Symptom Checklist (SCL-90) is a widely used instrument that has reasonable validity in detecting general distress as an indicator of psychiatric illness, but does not provide information about specific psychiatric diagnoses. The SCL-90 has high sensitivity and moderate specificity for anxiety and mood disorders in substance abuse patients, which are good characteristics for a screen: It will let few patients with these problems slip by, while false positives will be identified at the more thorough follow-up assessment. The Addiction Severity Index (ASI) is sometimes used in substance abuse treatment settings to guide treatment planning. This instrument has a psychiatric subscale score that detects general distress reasonably well. In one study, this subscale score correlated with SCL-90 scores in a group of substance-dependent women (Comfort et al., 1999). The Beck Depression Inventory (BDI) is another brief self-report instrument with demonstrated utility in detecting depression in substance abuse treatment settings.

Before attempting a definitive diagnostic assessment, it is best to wait until the patient has had a rea-

sonable period of abstinence. Doing so gives symptoms of acute intoxication and withdrawal time to subside. For example, a number of studies have found a 30 to 50 percent decrease in depression rating scores from the first day of abstinence to the end of the second week (Goldsmith and Ries, 2003). The appropriate minimum abstinence interval varies, depending on both the diagnosis under consideration and the substance of abuse. Long-acting drugs (e.g., diazepam, methadone) require longer abstinence prior to diagnosis than do shorter acting compounds (e.g., alcohol, cocaine). For individuals on methadone, a confident diagnosis can generally be made and treatment initiated if mood symptoms persist after 4 weeks on a stable maintenance dosage.

During abstinence pending diagnostic assessment, patients with symptoms indicating possible mood disorders generally need support and supervision. Some require observation in an inpatient setting, either due to symptoms of withdrawal that require treatment in a controlled environment or because of psychiatric symptoms such as suicidality or mania. When patients show severe symptoms of depression, mania, or hypomania, immediate treatment rather than continued waiting may be necessary to relieve suffering and facilitate treatment engagement.

The symptoms and history should be weighed carefully together in the diagnostic assessment. Guiding principles are:

- If a patient's symptoms seem typical of intoxication or withdrawal from a drug the patient has been abusing, especially with no previous psychiatric history, that is probably all they are. Expect them to remit soon, generally within days.
- Patients who have substance-induced mood disorders exhibit symptoms that result directly from the lingering physiological effects of the substance of abuse. Their affective difficulties commence within 4 weeks of last exposure to the substance, but are more severe and long-lasting than those normally associated with intoxication or withdrawal.
- Patients with underlying preexisting mood disorders may experience symptoms of varying type and intensity at any time during withdrawal or treatment. A strong family history of mood disorder makes diagnosis of a primary mood disorder more likely, as do a patient history showing onset of symptoms prior to substance abuse, severity of symptoms exceeding that normally seen in intoxication and

*A good
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screen
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tion warrants
full
assessment
later on.*

withdrawal, and sustained mood symptoms following lengthy abstinence.

CLINICAL COURSE AND TREATMENT

Studies indicate that individuals with SUD and a mood disorder have a more severe clinical course and worse outcomes than individuals who have only one or the other. Alcohol and drug abuse are associated with mood destabilization in individuals with affective disorders (Markou, Kosten, and Koob, 1998). Keller and colleagues (1986) found that among a group of bipolar patients, those with alcohol dependence recovered more slowly than those without. In comparing the histories of individuals with bipolar disorder, Sonne and colleagues (1994) found that those who also were substance abusers had an earlier age of onset of bipolar episodes, more frequent hospitalizations, and more comorbid psychiatric disorders. Hasin and colleagues (2002) studied the timing of depressive episodes relative to remission and relapse to substance abuse. They found that current substance-induced major depressive disorder (MDD), a history of MDD prior to the onset of substance dependence, or a depressive episode experienced during a 4-week initial abstinence reduced the odds that the patient would achieve 6 months of continuous abstinence.

A tragic association between SUDs, mood disorders, and suicide has long been recognized. The disinhibition and despair often associated with intoxication likely set the stage for impulsive and self-destructive acts. In one study, two-thirds of individuals who committed suicide had an SUD (Rich, Fowler, and Young, 1989). In comparing a number of psychiatric diagnostic groups, Young and colleagues (1994) found that individuals with MDD co-occurring with alcohol and drug dependence were at the highest risk for suicide.

In sum, substance abuse appears to have an adverse impact on the course and prognosis of mood disorders, leading to more frequent hospitalizations and treatment-resistant symptoms. On the other hand, some data indicate that alleviation of mood symptoms can improve substance-abuse-related outcomes.

In general, treatment efforts addressing mood disorders and SUDs have developed in parallel. Integration of services and effective treatment strategies from both fields can optimize outcomes when the two disorders converge.

Psychotherapeutic Treatment

Maximum use of behavioral approaches is the first principle of treatment for patients with SUD and a concurrent mood disorder. First, learning and gaining self-confidence in one's ability to self-regulate subjective states can be extremely helpful in recovery from both disorders. Second, learning strategies to self-regulate mood symptoms may help patients to break out of the mindset of using external agents to combat intolerable subjective states.

The behavioral strategies used for patients with co-occurring disorders should include elements of proven efficacy for each. Cognitive-behavioral therapies (CBTs) are among the most effective psychosocial treatments for affective disorders and also have demonstrated efficacy in the treatment of SUDs. Several recently published pilot studies have validated psychotherapeutic strategies specifically designed for individuals with co-occurring disorders. In a study of depressed alcoholics who received standard alcohol treatment plus either CBT for depression or relaxation training (control group), the CBT group showed greater improvements in depressive symptoms during treatment and also had better drinking outcomes at 3- and 6-month followup visits (Brown et al., 1997). Weiss and colleagues (2000) compared a manual-based CBT group therapy with treatment as usual for patients with SUD and bipolar disorder, and found that CBT produced significantly better outcomes in a number of domains. A large treatment-matching study of alcohol-dependent individuals compared three psychosocial interventions: 12-step facilitation, CBT, and brief motivational therapy. The treatments were equally effective overall; however, patients with high psychopathology as assessed by the ASI had better outcomes with CBT than with 12-step facilitation (Project MATCH Research Group, 1997).

Active participation in Alcoholics Anonymous (AA) or Narcotics Anonymous (NA) can greatly enhance recovery. Clinicians should encourage patients with co-occurring disorders to affiliate with 12-step groups that give clear, positive messages about the use of prescribed psychotropic medications. Recognition of the prevalence of co-occurring disorders has led to the establishment of a number of self-help groups for dual-diagnosis patients modeled on AA and NA, such as Double-Trouble and Dual Recovery Anonymous. While this development holds promise, the efficacy of these groups needs to be systematically explored.

A tragic association between substance abuse, mood disorders, and suicide has long been recognized.

TABLE 2. MEDICATIONS USED FOR THE TREATMENT OF MOOD DISORDERS IN SUBSTANCE ABUSERS

MOOD DISORDER	AGENTS USED	AGENTS TO AVOID
Depression	<p>Serotonin Reuptake Inhibitors Fluoxetine (Prozac) Sertraline (Zoloft) Paroxetine (Paxil) Citalopram (Celexa) Escitalopram (Lexapro)</p> <p>Tricyclic Antidepressants Imipramine (Tofranil) Nortriptyline (Pamelor)</p> <p>Other Venlafaxine (Effexor) Bupropion (Wellbutrin)</p>	<p>Monoamine Oxidase Inhibitors Tranylcypromine (Parnate) Phenelzine (Nardil)</p>
Bipolar Disorder	<p>Mood Stabilizers Lithium (Eskalith) Valproate (Depakote) Lamotrigine (Lamictal)</p> <p>Atypical Antipsychotics Olanzapine (Zyprexa) Risperidone (Risperdal)</p> <p>Typical Antipsychotics Haloperidol (Haldol)</p> <p>Benzodiazepines* Clonazepam (Klonopin)</p>	<p>Benzodiazepines Diazepam (Valium) Alprazolam (Xanax)</p> <p>Stimulants Methylphenidate (Ritalin)</p>

* Long-term use of benzodiazepines should be avoided.

Pharmacotherapy

The past 10 years have seen an explosion of new psychotherapeutic medications with enhanced efficacy and tolerability. Treatment options have increased for individuals with SUDs and those with mood disorders, yet few studies have evaluated the new agents in individuals who have both concurrently.

Even when community programs diagnose or suspect mood disorders, they often lack clinicians who can prescribe medications on site. In one recent study, McLellan and colleagues (2003) found that fewer than half of the Nation's drug and alcohol treatment programs had a full-time physician or nurse. Considering

the prevalence of co-occurring disorders, developing strong collaborative relationships between addiction treatment professionals and local physicians and mental health centers is critical for optimizing patient outcomes.

Medicating Depression

As discussed, prudence usually dictates waiting to start a patient with SUD on a medication for depressed mood until after detoxification. Doing so avoids unnecessarily exposing the patient to the expense and risk of medications when his or her symptoms may resolve of themselves in days or weeks. As well, it removes the possibility of confounding symptoms of withdrawal with side effects of the antidepressant medication. The most common symptoms of substance withdrawal—*anxiety, agitation, sedation, nausea, and headache*—are also potential side effects of the agents commonly used to treat depression, such as the serotonin reuptake inhibitors (SRIs; e.g., sertraline), venlafaxine, and bupropion. Nevertheless, if depression is severe and shows little sign of remitting within the first few days of abstinence, and if risk factors are present to suggest that a mood disorder may underlie it (e.g., a positive family history of MDD), early pharmacotherapeutic intervention may be justified. Table 2 lists the medications most frequently used to treat mood disorders, as well as medications that individuals with SUDs should avoid.

Recently, Nuñez and Levin (2004) presented a systematic review and meta-analysis of studies of antidepressant medications in the treatment of co-occurring depression and SUDs. While over 300 studies have explored this issue, only 14 were placebo-controlled trials that included subjects meeting recognized diagnostic criteria for depression and SUD. Of the 14, five used tricyclic antidepressants (TCAs), seven used SRIs, and two used other types of antidepressant agents. The results varied widely, with eight studies reporting significant or trend antidepressant effects and the rest finding none. Overall, the authors concluded that antidepressant medications demonstrated a modest beneficial effect for patients with combined depression and SUD. The finding of efficacy was more robust for studies of alcohol-dependent individuals than of drug-dependent individuals. Among the studies that demonstrated a decrease in depression, medication also had a favorable effect on substance abuse.

Nuñez and Levin also found that studies using TCAs yielded more positive results than those using SRIs, although they noted that this finding must be interpreted with caution since:

- the negative SRI studies had high placebo response rates, and
- the SRI studies that were positive demonstrated substantial medication effects on both depression and substance abuse.

In their final analysis, the authors concluded that SRIs should be the first-line medications for depression when SUD is also present based on their favorable tolerability and low toxicity compared to the TCAs. Other new classes of antidepressant drugs, like venlafaxine and bupropion, have shown promise in pilot studies with individuals with comorbid depression and SUDs and warrant further investigation.

While antidepressant medications may be useful, interventions to promote recovery from addictions and stabilize the environment are essential parts of successful treatment. A recent study starkly demonstrated this point: In a controlled trial with depressed individuals in methadone maintenance for opioid addiction, sertraline demonstrated significant effects in ameliorating depression and decreasing drug use only in those subjects who had more positive environments—i.e., more social and familial contact, better employment and monetary resources, and fewer legal problems and interpersonal conflicts (Carpenter et al., 2004).

Medicating Bipolar Disorder

While individuals with bipolar I disorder often receive treatment in mental health rather than addiction settings, the more subtle forms of bipolar disorder (bipolar spectrum disorders: bipolar II, cyclothymia) are more common than previously appreciated (Hirschfeld et al., 2003). Compared with bipolar I disorder, bipolar spectrum disorders are more difficult to distinguish from substance-induced mood symptoms and may be more likely to present in the addiction treatment setting. As with depression, the most accurate diagnostic assessment can be made after a period of abstinence. For bipolar patients in particular, a close working relationship with mental health care providers and/or psychiatric consultants will be essential to providing optimal care.

Despite the relatively frequent co-occurrence of SUD and bipolar disorder, few studies have focused

on pharmacological therapy for patients with this dual diagnosis. Several small pilot studies have shown that patients with bipolar-SUD comorbidity benefit from valproate and tolerate it well (Albanese, Clodfelter, and Khantzian, 2000; Brady et al., 1995; Hertzman, 2000). In one such study, this population tolerated and adhered to valproate better than to lithium (Weiss et al., 1998). A recent double-blind placebo-controlled trial examining actively drinking bipolar individuals treated with valproate versus placebo, with both groups also receiving lithium and psychosocial intervention, documented lower alcohol consumption with valproate (LeFauve et al., 2004). While valproate did not produce superior mood outcomes, there was a trend for individuals receiving it to remit from mania sooner. A recent open-label trial by the same investigators found that valproate plus naltrexone outperformed valproate alone in reducing substance abuse (0 percent versus 75 percent relapse rate) as well as mood symptoms (Salloum, Cornelius, and Chakravorthy, 2003). Additionally, carbamazepine has been shown in one study to reduce cocaine abuse in persons with cocaine dependence and affective disorder (Brady et al., 2002).

Individuals who have bipolar disorder complicated by SUD are more likely to experience mixed episodes of depression and mania and rapid cycling between the two (Sonne and Brady, 1999). In patients with uncomplicated bipolar disorder, both characteristics predict a better response to anticonvulsant mood-stabilizing agents (e.g., valproate, carbamazepine, lamotrigine) than to lithium (Swann et al., 1999, 2002). Accordingly, some specialists have hypothesized that substance-abusing bipolar patients should receive an anticonvulsant rather than lithium as a first-line therapy. This may be a reasonable approach, but lithium can also be effective: A small placebo-controlled trial with 25 adolescents with bipolar disorder and SUDs yielded significant associations between lithium therapy, reduced drug abuse, and improved mood symptoms (Geller et al., 1998).

With respect to other medications used to treat bipolar disorder, no controlled studies to date have evaluated the use of olanzapine in patients with bipolar-SUD comorbidity. In one reported case involving three patients, olanzapine reduced substance abuse, cravings, and anxiety (Sattar et al., 2003). In a recently conducted open-label trial, lamotrigine treatment brought improvement in bipolar symptoms and

In studies where medication decreased depression, there was also a favorable effect on co-occurring substance abuse.

decreased cocaine craving, but did not have a significant effect on drug abuse (Brown et al., 2003). Although the pharmacotherapeutic treatment of comorbid bipolar disorder and SUDs clearly needs further elucidation, research has opened a number of promising avenues for development.

CONCLUSIONS

Co-occurrence of SUD and affective disorders is common and has an impact on prognosis and treatment. Diagnosis and assessment of these comorbid disorders are difficult because of the substantial overlap in the symptoms of substance intoxication, withdrawal, and mood disorders. Using screening instruments to identify individuals with possible mood disorders and following up after a period of abstinence may be a particularly useful approach for busy clinicians.

Several treatment approaches have been developed specifically for patients with SUD and depression or bipolar disorder, and manuals for these approaches are available. While the use of complex manual-guided therapies may not be practical in some community treatment settings, these studies and others now under way should establish the critical components and techniques of effective treatment for these patients. The best approach to implementing these treatments in practice is an area that requires further exploration.

Recent advances in the pharmacotherapy of mood disorders benefit the population with co-occurring disorders because the newer agents have less toxicity, fewer side effects, and fewer interactions with substances of abuse. While few studies have specifically

evaluated pharmacotherapy in dual-diagnosis patients, the results to date indicate that similar medications are effective for mood disorders, whether or not SUD also is present. Moreover, treatment of mood disorders may be associated with decreased substance abuse. Clearly, specific considerations in choosing a pharmacologic agent for use in patients with SUD include safety, toxicity, and abuse liability.

While recent advances have identified specific therapeutic options for individuals with co-occurring SUDs and mood disorders, circumstances in both the mental health and addiction treatment systems make the delivery of optimal care difficult. In a recent study of the national addiction treatment infrastructure, McLellan and colleagues (2003) found that staff turnover was high, fewer than one-half of programs had a full-time nurse or physician, and very few programs had a social worker or psychologist. Given these constraints, expanding communication and collaboration between agencies and health care providers is vital to providing optimal care for patients with co-occurring disorders.

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RESPONSE: THE PATH TO DOUBLE RECOVERY

William Haning, III, M.D., Edward Nuñez, M.D., and Patricia E. Penn, Ph.D.

Screening

Clinicians
should
monitor for
mood
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all times.

Edward Nuñez: The authors' recommendation that programs screen patients for potential mental health problems on intake is sensible. I'm convinced, as they are, that line clinicians in substance abuse programs can develop the clinical sophistication necessary to recognize mood disorders and make appropriate referrals.

Patricia Penn: I agree. We also need to keep in mind that symptoms often emerge during the treatment process. Clinicians should monitor the client for symptoms at all times, not just during the initial screening.

Nuñez: The SCL-90 symptom checklist is a good instrument for making the initial assessment, although it only assesses symptom levels—it doesn't point to diagnoses.

Penn: There is a real need for more clinician-friendly screening tools. In one of our programs, the counselors have combined the most effective and efficient parts of several instruments into our own hybrid screener. We also have tried CAAPE, the Comprehensive Addictions and Psychological Evaluation developed by Dr. Norman Hoffman, which is designed specifically to diagnose co-occurring disorders, and it looks promising. Dr. Hoffman's screens, which also include one for adolescents, are available at www.evinceassessment.com.

William Haning, III: In my hospital, we screen patients with the SCID [Structured Clinical Interview for DSM-IV-TR] at the very outset, then follow up with some of the faster, cheaper screeners. In the community facility where I work, we use a variety of cheap, fast screens. Interestingly, the results in terms of patient profile turn out to be similar in both places. The more comprehensive SCID does not lead to a greater number of diagnoses. I wonder sometimes if its results warrant the extra effort it requires. The computerized ver-

sion of the SCID is quicker, and we're satisfied that personnel at a bachelor's level or even below can manage it nicely.

Nuñez: I would also advocate for the CAAPE or the Hopkins Symptom Checklist.

The makers of SCID, Drs. Bob Spitzer, Janet Williams, and Michael Frist, have also developed a couple of simplified versions for use in primary care. One is called PRIME-MD, which is clinician-administered and brief. The other is called PHQ, Patient's Health Questionnaire, which the patient fills out. A colleague here at Columbia, Dr. Carlos Blanco, is adding modules on attention deficit disorder and gambling, concerns which aren't present in the primary care versions but are important to substance abuse providers.

Diagnosis

Haning: Unfortunately, a fair number of people are still convinced that all mood disorders in substance abusers derive from the substance use and/or some character pathology that will resolve after a few years of progressive work. It isn't so. There is a considerable comorbidity that really does need to get treated.

Penn: Some patients feel relieved when they receive a mood disorder diagnosis, because it helps explain why they have felt bad for so long. Others, of course, find the diagnosis difficult to accept and resist it.

Haning: Some understand that having a mood disorder is going to complicate their prognosis and give them a harder time in life. They will contest the diagnosis with you.

Nuñez: Patients also resist because plenty of stigma still surrounds mood disorders. A line I hear occasionally is, "I'm not crazy, I'm a drug addict. I don't need to see any shrink."

Haning: The biggest risk in discussing a potential mood disorder diagnosis with a patient is that for some, it provides a rationale for not acknowledging drug dependence. That's not a reason to withhold the information, but it does mean we need to educate the patient about the implications of having two interactive illnesses simultaneously.

Theoretical models and treatment

Haning: I was taken with the authors' discussion of the kindling model, because it could explain a pattern we observe clinically. When methamphetamine users begin treatment, they typically are psychotic or hypomanic and subsequently become depressed. They feel low for a long time, but after somewhere between 10 and 14 months, they perk up, their affect comes alive, and they begin to have less difficulty paying attention and concentrating. The kindling theory, along with imaging studies by Drs. Linda Chang and Nora Volkow [Chang et al., 2002; Volkow et al., 2001], suggest to me that the temporal pattern of recovery might reflect the gradual resolution of methamphetamine-induced brain inflammation.

Penn: Our patients abuse an average of three and a half drugs each, including amphetamines. We see the same pattern: a long struggle, and then recovery becomes self-reinforcing after a year to 14 months.

Haning: Patients don't need to be completely abstinent the whole year to achieve this watershed, but almost.

Penn: The authors' discussion of the theories relating to mood and substance abuse disorders is absorbing, and solving that puzzle will lead to better interventions. In clinical settings, however, we should beware of getting caught up in chicken-and-egg debates about which is primary. They can be a distraction from treatment. I like to ask clients, "What do you think is causing this?" or "How does your substance use affect your mood, and vice versa?" The client's impression gives you an idea how to proceed in treatment.

Nuñez: I also ask patients what their experience with the drug has been, and look very hard for evidence that the substance abuse makes the mood syndrome worse. Often it does, not when the patient is taking the drug, but afterwards.

I work in a motivational framework, so I try to steer patients toward connecting their drug abuse and mental health problems for themselves. Some find it difficult. I'm thinking, in particular, about a patient who has bipolar illness and alcoholism. Medication controls his affective disorder reasonably well when he's not drinking, but when he starts to drink, he deteriorates. We have to constantly remind him that he has been down this path before—we know what the outcome will be. If we can get him to see that connection, it'll make a big difference.

*"I'm not crazy,
I'm a drug
addict."*

Haning: The trick for therapists is to get somebody who has lived from moment to moment to look a year into the future, have confidence that things will get better, and just hang in there until they do. In my experience, a major rationale for giving medications such as antidepressants and anticonvulsants is to keep patients coming back for their appointments—whether the meds really suppress symptoms or have only a placebo effect.

Penn: At La Frontera, we have noticed that many people come into our intensive program for a month or two or three, go away for a while, come back, go away, and so on. They seem to need that time to test out and practice what they learn in therapy and let it sink in. We try to maintain an open-door policy so clients can come back as needed.

Haning: We compel patients to rewrite their relapse prevention program each time they come back. We also get into long arguments with insurers about what they see as a revolving door and we see as a progressive acclimatization to recovery.

Penn: I was glad the authors mentioned a group CBT model for treating co-occurring disorders. We also need a model that can accommodate rolling enrollment. With nonrolling enrollment models, we must ask people to wait for weeks just to get into group support. This doesn't work—people don't come back.

Mix or match?

Haning: Some treatment programs mix all their patients together; others provide separate groups for patients with mood disorders. I think separation is best. These patients need a safe environment to discuss their medications and the elements of their treat-

ment that are specific to their co-occurring disorders. In mixed groups, they can get the worst of both worlds: not taking full responsibility for their bad decision-making around drug abuse, and not getting their symptoms taken seriously when there is a need to modify the medication regimen or enhance cognitive-behavioral strategies. Also, referring too often to ‘my bipolar disorder’ or ‘my manic depression’ is an invitation to be ostracized.

Penn: This issue also comes up in relation to outside support groups. For example, even though a 12-step pamphlet encourages people to take their medications as prescribed, a lot of individual groups don’t subscribe to that. These groups often don’t know how to deal with people who have a mental illness. Our patients, especially those with serious mental illness, have lots of problems and generally have trouble finding sponsors. They have had happier experiences with

Smart Recovery [www.smartrecovery.org], which uses cognitive-behavioral treatment methods that apply to both mood and substance abuse disorders. They like the program because it uses a trained facilitator to keep the meetings contained and ensure that everyone is respected.

Haning: I agree. The key to success in these programs is having somebody who is quasi-professional, either on the periphery of the meeting or actually facilitating it to ensure the fundamental needs of the attending population are met.

Penn: We also need more good modules for training counselors in co-occurring disorders. Online sources would be especially helpful. They could reach people everywhere, including rural areas, where there is a big problem finding people who are competent in treating co-occurring disorders. &

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