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Community Studies on Adolescent Substance Use, Abuse, or Dependence and Psychiatric Comorbidity

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A literature review on community studies of adolescent substance use, abuse, or dependence (SU/A/D) and psychiatric comorbidity yielded 22 articles from 15 studies with information on rates, specificity, timing, and differential patterns of comorbidity by gender, race/ethnicity, and other factors. Results revealed that 60% of youths with SU/A/D had a comorbid diagnosis, and conduct disorder (CD) and oppositional defiant disorder (not attention-deficit/hyperactivity disorder) were most commonly associated with SU/A/D, followed by depression. Child psychopathology (particularly CD) was associated with early onset of substance use and abuse in later adolescence. The authors suggest that available data relevant to SU/A/D and psychiatric comorbidity can be used to better address such questions.

The adult clinical and epidemiological literatures suggest that from 50% to over 80% of all types of substance abusers have also met criteria for at least one other psychiatric diagnosis (treating alcoholism and drug abuse as separate disorders) at some time in their lives (Helzer, 1988; Khantzian & Treece, 1985), with perhaps the highest rates of comorbidity being found in opiate abusers (Khantzian & Treece, 1985; Rounsaville, Weissman, Crits-Christoph, Wilber, & Kleber, 1982). The most common psychiatric comorbidities are antisocial personality disorder (ASPD; Cadoret, O'Gorman, Troughton, & Heywood, 1985; Cadoret, Troughton, & Widmer, 1984; Hesselbrock, Meyer, & Keener, 1985), depression (Schuckit, 1986; Stabenau & Hasselbrock, 1984; Weissman & Myers, 1980), and anxiety disorders (Mullaney & Trippett, 1979; Smail, Stockwell, Canter, & Hodgson, 1984; Stockwell & Bolderston, 1987).[1] According to retrospective evidence from the Epidemiologic Catchment Area (ECA) study of adults, the median age of onset of most of these disorders is before 20 (Christie et al., 1988), although the dating of the comorbidity is unclear. In this article, we review the state of published knowledge about psychiatric disorders in substance-using and -abusing youths from studies of children and adolescents.

Numerous studies have demonstrated associations between adolescent drug use and abuse and symptom scales on measures of low self-esteem, depression, antisocial behavior, rebelliousness, aggressiveness, crime, delinquency, truancy, and poor school performance (e.g., Jessor & Jessor, 1977; Johnston, O'Malley, & Eveland, 1978; Kandel, Kessler, & Margulies, 1978). In fact, some studies of antisocial behavior still include early drug use as one among a range of possible symptoms (e.g., Farrell & Taylor, 1994; Farrington, 1983), although it is treated in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) as an associated symptom. Children who later become problem drinkers have been found to have high rates of school drop out and poor achievement, rebelliousness, antisocial behavior, aggressive behavior, delinquency, and family problems (Greenbaum, Prange, Friedman, & Silver, 1991; Jessor et al., 1980; McCord & McCord, 1960; Robins, 1974; Zucker & Barron, 1973). In adolescence, an increased level of depressive symptomatology also appears to be associated with the use of substances including cigarettes and alcohol and then with progression to hard drugs (Kleinman, Wish, Deren, & Rainone, 1986; Neighbors, Kempton, & Forehand, 1992; Paton & Kandel, 1978).

The literature on adolescent diagnostic comorbidity is much smaller and mainly confined to clinical samples. It indicates that the high rates of alcohol use, drug abuse, and mental health comorbidity observed in adult populations are also found in adolescent treated samples (Bukstein, Glancy, & Kaminer, 1992; Hovens, Cantwell, & Kiriakos, 1994; Kaminer, Tarter, Bukstein, & Kabene, 1992). Other clinical studies have shown that attention-deficit/hyperactivity disorder (ADHD) is probably associated with an increased risk of substance abuse, but that association may be mediated through the association between conduct disorder (CD) and ADHD rather than made directly (Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Loney, 1988). Both depression and CD are strongly associated with parasuicide and suicide (Brent et al., 1986; Shaffer, 1982; Taylor & Stansfeld, 1984), and alcohol has been heavily implicated in adolescent suicide (Brent et al., 1986; Clayton, 1989).

It is, however, important to examine comorbidity in representative community samples as a check on the generalizability of the clinical literature, for the following reasons:

1. People with two illnesses are more likely to seek treatment than people with either one of those illnesses sep-

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[1] A more exhaustive list of citations may be obtained by contacting the authors.

arately (Berkson, 1946). This means that a clinic-based sample is likely to have a higher proportion of comorbid people in it than does the general population, so one cannot use them to estimate the size of the problem.

2. Some combinations of disorders may bring people into treatment settings more often than others. For example, youths with substance abuse disorder and CD might be referred to clinics in higher proportions than youths with substance abuse disorder and an anxiety disorder. This would give clinicians the impression that comorbidity with CD is very common and comorbidity with anxiety very rare. This may or may not be true, but the point cannot be established from clinic samples.

3. Clinic cases may or may not be more severely affected with either of the comorbid conditions than community cases. Alternatively, comorbid cases may seek clinical treatment even though they have less severe symptoms of one or the other disease than most community cases.

4. The temporal ordering of comorbidity may or may not be the same in clinical as in community cases.

5. Comorbidity seen in clinic cases may or may not be precipitated by the same risk factors as those that precipitate cases in the general population. These potential differences can be checked empirically for diseases where almost all cases get into treatment. However, in the case of drug abuse, there is very strong evidence that this is not so. We also know that many people with psychiatric disorders never receive treatment. For these reasons we cannot assume that the patterns of association and risk seen in clinic samples mirror those seen in the population.

6. What appear to be risk factors for one or the other disorder may in fact be predictors of treatment referral. For example, it might appear from a clinic study that poverty was associated with one or more disorders, where, in fact, stiff managed-care regulations restricted access to treatment for children from privately insured families, whereas Medicaid regulations were more generous. This could mean that only children on Medicaid had much chance of getting treatment, so poor children were overrepresented in the clinic sample.

For all these reasons, the scientific study of prevalence, incidence, development, and risk for comorbidity cannot rely solely on clinic-based samples. Enhanced interventions and public policy alike depend on a better understanding of the mechanisms implicated in the development of substance abuse and psychiatric comorbidity, and these are most accurately assessed in the general population.

This article reviews the published literature available on psychiatric comorbidity in community-based samples of children and adolescents who use or abuse substances. There are four basic questions about which researchers and service providers might turn to the descriptive epidemiologic literature for an answer:

1. How often do substance-using or -abusing youths have comorbid psychiatric disorders?

2. Is co-occurrence with some disorders more common than with others, and are some forms of substance abuse more likely than others to be comorbid with specific psychiatric disorders?

3. Does comorbid psychiatric disorder affect the probability or timing of transitions from substance use to abuse or dependence?

4. Are some groups of children (boys or girls, White or minority groups, younger or older children, poor or nonpoor, urban or rural) more at risk than others of co-occurring psychiatric disorders and drug abuse?

The answers to these questions are important not only scientifically but also as a basis for planning interventions. For example, understanding how a disorder that co-occurs with substance abuse in childhood or adolescence affects later risk of substance abuse can facilitate estimates of attributable risk or the proportion of later drug abuse that could be prevented by intervening with one risk factor (e.g., early depression) rather than another (e.g., early drug use; Rothman & Greenland, 1998). This kind of calculation is often useful to policy planners making cost- and efficiency-based choices among programs.

Comorbidity can be thought of as the probability of Disorder A in those with Disorder B or the probability of Disorder B in those with Disorder A; but the clinical and policy implications are often different. For example, the extent of psychiatric disorder in substance-abusing youths is a concern for substance abuse researchers, clinicians, and policy makers, who are likely only partially to overlap with the constituencies concerned about the degree of drug use or abuse in youths with psychiatric disorders. Here we have focused on psychiatric disorder in youths who use or abuse substances.

Method

Selection Method

Articles appropriate for this review were selected by several methods. First, literature searches were conducted in PsycINFO, Medline, and Web of Science using combinations of the keywords *adolescent, adolescence, drug or substance, use or abuse,* and *psychiatric comorbidity*. The search was limited to articles in the English language. Literature that focused solely on parental drug abuse as a predictor of adolescent drug abuse was excluded, as were manuscripts from Dissertation Abstracts International. Second, the bibliographies of these articles were examined for the purpose of yielding additional articles. Third, the in-house reference library of the Center for Developmental Epidemiology was searched. Finally, advisors to the Center were asked for references that might be appropriate for our purposes. One hundred sixty-five articles were found that contained all the keywords or were found through the other methods described. When we applied the additional criteria requiring that studies be based on community samples and using formal diagnostic procedures, 22 articles remained, some of them referring to the same 15 studies.

Analyses

We have presented the results in two forms: percentage of youths with substance use, abuse, or dependence who had a comorbid psychiatric

disorder and the odds ratio (OR). The OR is the ratio of the odds of a psychiatric disorder in the presence versus the absence of substance use/abuse. Thus, an OR of 2 indicates that a psychiatric diagnosis is twice as likely in the presence of substance use/abuse as in its absence, whereas an OR of 1 indicates that psychiatric disorder is equally likely with or without substance use/abuse, and an OR below 1 points to a lower probability of psychiatric disorder in the presence of substance use than in its absence. If the 95 % CI does not include 1, this can be taken as an indication that there is a significant difference between youths with and without substance use or abuse in the likelihood of psychopathology (Fleiss, 1981).

Caveats

Any review of the published data currently available to address the four questions about comorbidity needs to be read with three methodologic caveats in mind. First, although most studies now use standard criteria for psychiatric diagnoses, different studies have used very different criteria to estimate substance use and abuse and different terminology to describe them. As a general principle, those estimating *substance use* tend to include abuse and dependence in that category. Others refer only to *abuse*, or *abuse and/or dependence*. Still others discuss use and abuse/dependence separately. Some use *DSM–III* (3rd ed.; American Psychiatric Association, 1980), *DSM–III–R* (3rd ed., rev.; American Psychiatric Association, 1987), or *DSM–IV* (4th ed.; American Psychiatric Association, 1994) criteria for abuse and dependence, whereas others use their own decision rule. Even when the *DSM* term *substance use disorder* (SUD) is used, different groups use different criteria to make the diagnosis. Some studies concentrate on specific types of drug, such as alcohol, whereas others combine drugs in different ways. The different articles that discuss comorbidity have measured many levels of substance abuse severity, from occasional cigarette smoking to occasional alcohol use to intensive use of drugs that are illegal for adults (illicit drugs). Rather than either lumping or splitting the types of drug use and abuse any more than they already are in the different articles, we have tried to adopt the approach that Kandel et al. (1997) took by discussing comorbidity with each psychiatric disorder across the range of severity of drug use for which information is available. This variability, together with the small number of relevant studies, necessitates a careful descriptive approach to reviewing the literature as a precursor to formal meta-analysis. We presented the results of a meta-analytic approach to this problem in another publication (Armstrong, Erkanli, & Costello, 2001). Here, we describe the kind of studies that are available to answer questions about comorbidity and present ranges of estimates and medians.

Second, studies vary in their interpretation of *comorbidity*. Some treat conditions as comorbid only if they are diagnosed as being present at the same time (*concurrent comorbidity*), whereas others count two disorders in the same individual as comorbid even if they were present at different periods of time (*sequential comorbidity*). *Lifetime comorbidity* defines disorders as comorbid if both occurred at any time in a person's life, not necessarily together. In some cases, it is not clear which definition is being used. We try to preserve the distinctions in this review, where possible.

Third, the literature on comorbidity is less useful for our purposes than it might be because some researchers presented information about psychiatric disorder in youths who use/abuse substances, whereas others reported on substance use/abuse in youths with psychiatric disorders; few presented both. As this review is about psychiatric comorbidity in substance-abusing youths, we cannot present raw prevalence information from some studies that have the potential to address this issue but have not published their data this way. Of course, where ORs have been presented, these can be interpreted in either direction. This means that some studies whose prevalence data were not available have nevertheless contributed useful information in the form of ORs.

Finally, a word about abbreviations. We have tried to avoid them, except for the most familiar or word-saving. We retain ADHD, CD, ODD for oppositional defiant disorder, PTSD for posttraumatic stress disorder, and DBD for disruptive behavior disorders (CD, ODD, or ADHD). SUD refers to substance abuse or substance dependence. We have created two neologisms: SU/A/D for any substance use, abuse, or dependence, and OID (other illicit drugs) as shorthand for any use of amphetamines, stimulants, cocaine, heroin, opiates, nonprescribed barbiturates, glue, hallucinogens, psychedelics, ice, LSD, Ecstasy and other designer drugs, tranquilizers, sedatives, or other drugs that children should not be ingesting.

Results

Table 1 presents a summary of the 15 studies that provided published information that contributed to answering the four questions posed. Ten were based on samples from the United States, 2 from Canada, 2 from New Zealand, and 1 from Taiwan. Sample sizes varied widely, but many studies had at least 1,000 participants. A few provided only a single wave of data. Most were longitudinal studies with multiple waves of data collection. The distinction between concurrent and lifetime comorbidity referred to earlier cut across the single-stage/longitudinal distinction, because single-wave studies could use a lifetime framework for making a diagnosis, and longitudinal studies could present analyses of recent, concurrent comorbidity. Where the information was available, we have noted which type of analysis was used in which articles.

Question 1. How Often Do Drug Use and Abuse Co-Occur With Psychiatric Disorder?

Table 2 presents a summary of the information on psychiatric disorders seen in youths with SUD. It uses the numbering from Table 1 to designate the studies and letters to designate the diagnostic groups; thus, A refers to SU/A/D, B refers to depression, C to anxiety disorders, D to CD or ODD, E to ADHD, F to other diagnoses (one article on eating disorders, one on PTSD), and G to the presence of one or more psychiatric disorders of any kind. Only studies that contribute information on a particular comorbidity are included in each section of the table.

Comorbidity with depression. Among specific subtypes of psychiatric disorder, depressive comorbidity has been studied more widely than any other type. Kandel et al. (1997; Table 1, Study 4) showed that the prevalence of depression increased from around 5.0% in abstaining youths to 23.8% in youths who used alcohol at least weekly and to 24.1% in youths who reported using illicit drugs at least once a year. Similar rates of depression were reported in 7 other studies (Table 1, Study 2; Fergusson, Lynskey, & Horwood, 1996; Study 3; Feehan, McGee, Raja, & Williams, 1994; Study 5 [concurrent comorbidity]; Kandel et al., 1999; Study 6; Windle & Davies, 1999; Study 9; Rohde, Lewinsohn, & Seeley, 1991; Study 10; Beitchman et al., 1999; and Study 12; Deykin, Levy, & Wells, 1987). Interestingly, Kandel et al. (1997) found very similar rates of mood disorder, from 20.0% to 30.0%, comorbid with a wide range in severity of substance use and abuse, from recent cigarette use to occasional illicit drug use. One study that evaluated lifetime comorbidity found rates close to 50.0% (Rohde et al., 1991), but a second (Table 1, Study 12; Deykin et al., 1987) reported a prevalence of 22.8% for lifetime comorbidity with alcohol abuse, and 20.0% for lifetime comorbidity with SUD. Across the studies that estimated concurrent comorbidity between SUD and depression, the estimates ranged from 11.1% to 32.0%, with a median of 18.8%.

An exception to the finding of depression in at least 20.0% of substance-using adolescents was Chong, Chan, and Cheng (1999; Table 1, Study 11), who found that in Taiwan only 11.1% of youths with SUD, compared with 4.8% of other adolescents, had a *DSM–III–R*-defined depressive disorder.

Concurrent ORs varied eightfold, from 1.1 for depression comorbid with cannabis use in Ontario (Table 1, Study 8) to 8.03 for concurrent SUD comorbidity in Oregon (Table 1, Study 9; Rohde et al., 1991). However, the majority of studies reported ORs in the 1.5–2.5 range, and the median was 2.2; these figures are significantly different from 1, but not by a great deal. ORs for lifetime comorbidity were somewhat higher, with a range from 1.5 to 4.5 and a median of 3.3. There was little indication of a gradient in the size of ORs with increasing severity of drug problems.

Comorbidity with anxiety. Anxiety is the disorder that showed the smallest and most inconsistent difference in prevalence between adolescents with SUD and the rest of the populations sampled. Comorbidity ranged from 7.0% (Table 1, Study 1; Brook, Cohen, & Brook, 1998) to 40.4% in the Dunedin longitudinal sample taken at age 18 (Feehan et al., 1994). However, the latter degree of comorbidity with anxiety was an outlier, and the median level was between 16.2% (Table 1, Study 9; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993) and 18.2% (Fergusson et al., 1996).

Eight of 15 studies reported that ORs were not significantly different from 1 (i.e., the likelihood of an anxiety disorder was not significantly higher in youths with SUD than in other youths). The highest OR was 3.9 (Table 1, Study 4; Kandel et al., 1997), and the median value was only 1.3.

Comorbidity with DBD. With very few exceptions, comorbidity with DBDs was high, with a median prevalence of 46.0% and only two reports below 25.0%. In the absence of any substance use, reported rates of DBDs were between 0.0% and 12.0%, with a median between 7.0% and 8.0%. Even the Taiwan study (Chong et al., 1999), which generally found low levels of comorbidity, reported that 59.3% of adolescents with SUD also had a DBD; most frequently CD (44.0%).

ORs were also high, ranging from 1 to 32, with a median of 4, indicating a fourfold increase of risk for DBD in substance-using or -abusing youths. However, there was no clear indication of any gradient of comorbidity with increasing severity of substance use. This may reflect the wide range of developmental stages reached by the participants of the different studies, many of whom, however delinquent, had not yet been exposed to hard drugs.

Comorbidity with ADHD. There was little information available about comorbidity with ADHD, perhaps because ADHD is rare in adolescence, and substance use/abuse is rare in childhood. The only prevalence rate reported came from Chong et al. (1999), who found that 12.3% of their youths with SUD had ADHD compared with 1.8% of the control group.

Only about half of the ORs were significantly different from 1, and the median value fell between 1.2 and 1.5. This suggests that the developmental relationship between SUD and ADHD is markedly different from that between SUD and other DBDs.

Comorbidity with other disorders. A couple of other reports are added for completeness. One showed a high and significant lifetime relationship between substance abuse and PTSD, with an OR close to 4 (Table 1, Study 14; Giaconia et al., 2000). However, it is not clear which way the direction of association may have run, as both were measured as lifetime variables. The other (Table 1, Study 15; Zaider, Johnson, & Cockell, 2000) showed a small and statistically nonsignificant risk of eating disorders in youths with SUD, alcohol dependence, or OID dependence.

Comorbidity with any psychiatric disorder. In the 3 studies that reported on the prevalence of any psychiatric disorder in youths with SUD, estimates of psychiatric disorder in substance users/abusers were consistently high across a range of definitions of SUD, suggesting that about 2 of 3 of these adolescents had at least one diagnosable psychiatric disorder. In their study of high school students in Oregon, Lewinsohn et al. (1993) found a lifetime (sequential) comorbidity rate of over 60.0% for 14- to 18-year-olds with SUD, twice that of youths with no SUD, whereas in New York, Kandel et al. (1999; Table 1, Study 5) found similar rates of disorder in adolescents who were heavy alcohol and/or illicit drug users compared with rates of 16.0%–18.0% psychopathology in youths who did not use alcohol or drugs. Although the likelihood of having two or more comorbid psychiatric disorders was low, the Taiwanese study showed a much higher likelihood of multiple comorbidity in youths with SUD than in others (Chong et al., 1999).

The OR estimates of overall comorbidity with psychiatric disorder varied widely, from 3.5 to 18.6, with a median of 4.7.

Question 2. What Patterns of Co-Occurrence Were Most Common?

This review of the prevalence of psychiatric disorder in youths who use/abuse drugs supports the evidence from clinical studies in showing that DBDs, especially CD, were the most frequently diagnosed comorbid conditions, with prevalence of 25.0% to 50.0%, and a median OR of 4, followed by depression, with a prevalence of 20.0% to 30.0% and a median OR above 2. Anxiety disorders and ADHD had median ORs close to 1, and many of the reported ORs were not significantly different from 1. Individual studies showed a strong likelihood of PTSD in youths with SUD but not an increase in the risk of eating disorders.

With one exception, the literature reviewed here did not support a strong argument that some types of SUD were distinctively comorbid with some psychiatric disorders in children or adolescents. The exception was cannabis. The data in Table 2 show that whereas there was a clear association between cannabis use and DBDs, there was a lack of association between cannabis use and anxiety or depression. In no case were any of the ORs significant, unlike the ORs between tobacco, alcohol, and OID use and depression and anxiety. (There was no association between cannabis use and ADHD, but neither was there a clear association between ADHD and any other specific type of substance.) Apart from this, there was no evidence for substance-specific comorbidity, nor was there much evidence for a gradient suggesting greater comorbidity with more severe types of SUD. The ORs were not markedly greater for hard drugs than they were for nicotine or alcohol. However, this lack of findings could result from lack of power or from too gross an amalgamation across diagnoses within broad groups. For example, Kaplow, Curran, Angold, and Costello (2001) found that children with separation anxiety disorder had a

(*text continues on page 1234*)

Table 1
Descriptions of Community Studies on Adolescent Substance Use, Abuse, or Dependence and Psychiatric Comorbidity

Study	*N* and age at time of analyses, and description of the sample	Comorbidity: concurrent, lifetime, or predictive	Diagnostic instruments, informants, and taxonomy	Types of psychiatric disorders and other types of comorbidities	Measurement of substance use/abuse, informants, and how SU/A/D is assessed	Types of substance use/abuse reported
1. Children in the Community Study (Brook et al., 1998; Cohen et al., 1993; Velez, Johnson, & Cohen, 1989)	698, 16–22 years; families were randomly selected from two counties in upstate NY	Predictive, from mid- to late-adolescence	DISC-1; parent and adolescent; *DSM–III–R*	Diagnostic groups: DBD (CD, ODD, ADHD), ASPD in adults, anxiety disorders (separation anxiety, overanxious disorder, and social phobia), depressive disorders (MDD and dysthymia) *not* SD and SA Only internalizing and externalizing comorbidities with psychosocial variables	1. DISC-1 2. Paper-and-pencil measure, written responses to paper-and-pencil assessment preceded interview; parent and adolescent	From 1: Substance A/D From 2: Alcohol (5-point scale), cannabis (7-point scale), and OID (6-point scale)
2. Christchurch Health and Development Study (Fergusson, Horwood, & Lynskey, 1994; Fergusson, Horwood, & Lynskey, 1993)	1,265, 15 years	Concurrent	DISC-1, DIS, and SRED; parent and adolescent; *DSM–III–R*	Anxiety disorders (overanxious, separation anxiety, simple phobia, social phobia); mood disorders (major depressive, current and past year, and dysthymia); GAD, ODD, ADHD, and CD comorbidities among psychiatric behaviors comorbidities available between all variables	1. Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989) 2. Study-specific scales; combined/joint information based on child (survey questions and RAPI) and parent (perceptions and knowledge of child's use) reports, "custom written survey items"; parent and adolescent	Nicotine dependence, alcohol abuse, other (mainly cannabis) From 1 and 2: Abusive or hazardous drinking From 2: Daily smoking; any cannabis use
(Fergusson et al., 1996)	927, 14–16 years; birth cohort or children born in Christchurch urban region during mid-1977	Predictive, from cannabis use at 15 years to problems at 16 years				
3. Dunedin Birth Cohort (Anderson, Williams, McGee, & Silva, 1987; Feehan et al., 1994; Krueger, Caspi, Moffitt, & Silva, 1998)	930, 18 years; "unselected" representative sample of New Zealand children born 4/1972–3/1973 in the only obstetric hospital in the Dunedin metropolitan area	Concurrent	DISC and DYS; adolescent; *DSM–III–R*	ADD, CD, ODD, MDD, dysthymia, separation anxiety, overanxious, avoidant, simple, and social phobia, panic disorder, OCD, psychosis, GAD, agoraphobia, MDE—current and past year; comorbidity rates available; rates of internalizing, externalizing, and mixed disorders by psychosocial variables also available	DISC Diagnostic Interview	Alcohol dependence and cannabis dependence

Table 1 (*continued*)

Study	*N* and age at time of analyses, and description of the sample	Comorbidity: concurrent, lifetime, or predictive	Diagnostic instruments, informants, and taxonomy	Types of psychiatric disorders and other types of comorbidities	Measurement of substance use/abuse, informants, and how SU/A/D is assessed	Types of substance use/abuse reported
4. Methods for the Epidemiology of Child and Adolescent Mental Disorders (MECA; Goodman et al., 1998; Kandel et al., 1997)	1,258, 9–18 years; probability samples from CT, GA, NY, and Puerto Rico	Concurrent during past year	DISC 2.3; parent and child; *DSM–III–R*	Anxiety disorders (separation anxiety, avoidant disorder, OCD, overanxious, simple and social phobia, agoraphobia, panic disorder, GAD) DBDs: ADHD, CD, and ODD; mood disorders (dysthymia, hypomania, MDD, mania and miscellaneous: eating disorders, elimination disorders, anorexia nervosa, tic disorders); psychosis screening Comorbidities of other psychiatric disorders with alcohol, nicotine, and illicit substance use	DISC 2.3 questionnaire (Likert-type scale); parent and child	Alcohol use on 5-point scale (*never, not in past year, 1–5 times in past year, 6 to 25 times in past year, weekly in past year*) Nicotine use on 4-point scale (*never, not in past 6 months, less than daily use in past 6 months, daily use in past 6 months*) OID use on 3-point scale (*never, 1 to 2 times in past year, 3 or more times in past year*)
5. MECA same as 4 (Kandel et al., 1999)	401, 14–17 years, Puerto Ricans excluded from these analyses	1. Concurrent in past 6 months 2. Lifetime	DISC 2.3, parent and adolescent	Comorbidities only with SUD	DISC 2.3 Structured Interview, parent and adolescent	SUD: alcohol A/D, cannabis A/D, and OID A/D
6. Middle Adolescent Vulnerability Study (Windle & Davies, 1999)	975, 15–17 years; high school sophomores and juniors recruited from 3 homogeneous suburban high schools in western NY	Predictive	CES-D, RETROPROB, (Windle, 1993); parent and adolescent	Depressive symptoms scale scores for ADHD, ODD, and CD; avoidant PD comorbidities between depressive and external, avoidance, and temperament variables	QFI questionnaires	Heavy drinking (> 45 drinks in past 30 days)
7. Minnesota Twin Family Study (Disney et al., 1999; McGue, 1994)	626 twin pairs (reared together) 17 years; Population-based twin ascertainment from public records	Lifetime	DICA-R; parent and adolescent; *DSM–III–R*	Definite subthreshold cases of ADHD and CD; comorbidities only with SUD	UM–CIDI (SA module); parent and adolescent; *DSM–III–R* plus computer administered SU and A questionnaire (self-administered in a sound-dampened room)	Current use of tobacco, alcohol, and cannabis; nicotine dependence, alcohol A/D, cannabis A/D, and any SUD
8. Ontario Child Health Survey (Boyle & Offord, 1991; Offord et al., 1987)	1,265, 12–16 years; Stratified, random, and cluster sampling from 1981 Canada Census file; from female head of household and youths	Concurrent	Questionnaires; parent and adolescent; *DSM–III*	CD, hyperactivity, and emotional disorders; no other comorbidities available	Questionnaires sealed in an envelope; parents and adolescent	Regular use of tobacco measured from adolescents only (every day for at least 30 days); regular use of alcohol (at least once a week for 4 weeks), any use of cannabis, any use of OID

(*table continued*)

Table 1 (*continued*)

Study	*N* and age at time of analyses, and description of the sample	Comorbidity: concurrent, lifetime, or predictive	Diagnostic instruments, informants, and taxonomy	Types of psychiatric disorders and other types of comorbidities	Measurement of substance use/abuse, informants, and how SU/A/D is assessed	Types of substance use/abuse reported
9. Oregon Adolescent Depression Project (Lewinsohn et al., 1993; Lewinsohn et al., 1995; Lewinsohn, Rohde, Seeley, & Hops, 1991; Rohde et al., 1991; Rohde et al., 1996)	1,710, 14–18 years; total enrollment of 2 urban and 3 rural high schools in west central Oregon	Concurrent, lifetime community sample	K-SADS, LIFE, and CES-D; adolescent; BDI and *DSM–III–R*	Affective disorders: unipolar (MDD & dysthymia) and bipolar; anxiety disorders (panic agoraphobia, social and simple phobia, OCD, separation anxiety, overanxious, and DBD); ADHD, CD, ODD; eating disorders (anorexia nervosa and bulimia nervosa); adjustment disorders and others Comorbidities with depression and other mental disorders	K-SADS; adolescent; *DSM–III–R* and Semistructured Diagnostic Interview	1. SUD (any abuse or dependence) 2. Alcohol A/D, cannabis A/D, cocaine A/D, amphetamine A/D, 4-point alcohol scale (0 = *never*, 1 = *experimenter*, 2 = *social*, 3 = *problem*, 4 = *A/D*)
10. Ottawa Speech and Language Study (Beitchman et al., 1999)	315 control subjects, 19 years; 1 in 3 random sample of kindergarteners in Ontario	Concurrent	UM–CIDI; adolescent; *DSM–III–R*	Psychiatric disorders: affective, anxiety disorders, SUD, A and SD, eating disorders, antisocial personality disorders; comorbidities only with drug abuse (by speech and language impairment)	UM-CIDI Adolescent *DSM–III–R* Structured Interview	SUD, alcohol use disorder, cannabis use disorder, and OID use disorder
11. Project on Adolescent SUDs in Taiwan (Chong et al., 1999)	774, 14–16 years; students (9th grade) from schools randomized by region (urban, suburban, and rural) and promotion rates	Concurrent	K-SADS–E; adolescent; *DSM–III–R*	DBDs (CD, ADHD, and ODD), depressive (MDD and dysthymic disorder), anxiety (GAD, phobia, panic, and OCD), adjustment (psychoses and other); no comorbidity estimates between other psychiatric disorders	K-SADS–E; adolescent; *DSM–III–R* and Clinical Interview	SUD
12. Boston College Students Study (Deykin et al., 1987)	424, 16–19 years; convenience sample—letter sent to all college students from 2 Boston-area colleges	Lifetime	DIS; adolescent; *DSM–III*	MDD, phobic disorders, and OCD (tested for other psychiatric disorders, i.e., bipolar, dysthymia, panic, and schizophrenia); comorbidities only with drug abuse	DIS; adolescent; *DSM–III* and Structured Interview	Alcohol A/D: pathologic use or impairment, substance A/D: pathologic use or impairment, substance A/D: pathologic use and impairment for at least 1 month

Table 1 (*continued*)

Study	*N* and age at time of analyses, and description of the sample	Comorbidity: concurrent, lifetime, or predictive	Diagnostic instruments, informants, and taxonomy	Types of psychiatric disorders and other types of comorbidities	Measurement of substance use/abuse, informants, and how SU/A/D is assessed	Types of substance use/abuse reported
13. Pittsburgh Youth Study (Loeber, Farrington, Stouthamer-Loeber, Moffitt, & Caspi, 1998; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998)	1,517; 3 cohorts: (a) 7–8 years, (b) 10–11 years, (c) 13–14 years; random samples of 1st, 4th, and 7th grade boys from Pittsburgh public schools	Lifetime and concurrent	DISC-1 and MFQ; parent and child; *DSM–III–R*	Lifetime delinquency, ADHD score, conduct problems symptom score, lifetime physical aggression, covert behaviors (previous year), depressed mood (previous 2 weeks), shy/withdrawn behavior (previous year); ORs between all psychiatric disorders available	DISC-1; parent and child; Self-Report Questionnaire	5-point scale: 0 (*no use*), 1 (*ever had beer or wine*), 2 (*ever smoked*), 3 (*ever had hard liquor*), 4 (*ever used OID*) Substance use = (a) 0 vs. rest, (b) 0 or 1 vs. rest, (c) 0–2 vs. rest
14. Boston Longitudinal Study (Giaconia et al., 2000)	384; single-age cohort of kindergartners from 1 public school system in working-class community; 1990 sample of seniors in high school	Lifetime	DIS–III–R; adolescents; *DSM–III–R*	PTSD, only looked at associations between SUD, PTSD, internalizing, externalizing behavior, and psychosocial variables	DIS–III–R; adolescents; *DSM–III–R* and Structured Clinical Interview in person	Alcohol A/D and drug A/D
15. Eating Disorders Study (Zaider et al., 2000)	403, 15–18 years; selected from consecutive admissions to adolescent clinics in NY and high school nurse's office in NJ for medical issues (except 5%)	Concurrent	PRIME-MD	Depressive (MDD and dysthymia), anxious (GAD, panic, and all personality disorders); comorbidity of eating disorders with all other disorders reported	PRIME-MD, Structured Clinical Interview by phone	SUD, alcohol dependence, and OID dependence

Note. SU/A/D = substance use, abuse, and/or dependence; DISC-1 = Diagnostic Interview Schedule for Children (Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982; DISC-2; Shaffer et al., 1996); *DSM-III-R = Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); DBD = disruptive behavior disorder; CD = conduct disorder; ODD = oppositional–defiant disorder; ADHD = attention-deficit/hyperactivity disorder; ASPD = antisocial personality disorder; MDD = major depressive disorder; SD = substance dependence; SA = substance abuse; OID = other illicit drugs (amphetamines, stimulants, cocaine, heroin, opiates, barbiturates, hallucinogens, psychedelics, LSD, quaaludes, tranquilizers, and sedatives); DIS = Diagnostic Interview Schedule (Robins, Helzer, Cottler, & Goldring, 1989; Robins, Helzer, Croughan, & Ratcliff, 1981); SRED = Self-Report Early Delinquency (Moffitt & Silva, 1988); GAD = generalized anxiety disorder; DYS = Denver Youth Survey Youth Interview Schedule (Huizinga, 1989); ADD = attention deficit disorder; OCD = obsessive-compulsive disorder; MDE = major depressive episode; SUD = substance use disorder; CES-D = Center for the Epidemiologic Studies of Depression (Radloff, 1977); RETROPROB = Retrospective Childhood Problems measure; PD = personality disorder; QFI = Quantity-Frequency Index (Armor & Polich, 1982); DICA–R = Diagnostic Interview for Children and Adolescents—Revised; UM–CIDI = University of Michigan Composite International Diagnostic Interview (Kessler et al., 1994); K-SADS = Schedule for Affective Disorders and Schizophrenia (Chambers et al., 1985); LIFE = Longitudinal Interval Follow-Up Evaluation (Shapiro & Keller, 1981); BDI = Beck Depression Inventory (Beck, 1967; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); K-SADS–E = Schedule for Affective Disorders and Schizophrenia, Epidemiologic version (Orvaschel, Puig-Antich, Chambers, Tabrizi, & Johnson, 1982); MFQ = Mood and Feelings Questionnaire; ORs = odds ratios; DIS–III–R = Diagnostic Interview Schedule (3rd ed., rev.; Robins et al., 1989); PTSD = posttraumatic stress disorder; PRIME-MD = Primary Care Evaluation of Mental Disorders (Spitzer et al., 1995).

Table 2
Comorbidity of Diagnostic Groups

Study number and notes[a]	A	% B in those with A	% B in those without A	OR	95% CI
1. % prevalence of heavy use. OR for ASPD associated with 1-point increase in substance use rating scale	Nicotine use			1.19*	1.02–1.4
	Alcohol use			1.14	0.91–1.4
	Cannabis use			1.13	0.95–1.3
	OID use			1.16*	1.03–1.3
2. Concurrent association at age 15	SUD			4.4*	2.4–8.4
Prediction from 15 to 16 years, adjusted for confounders	Cannabis use	20.5	8.3	1.4	0.7–2.7
3. Concurrent	SUD	28.2			
4. Concurrent	Alcohol use	23.8	5.0	2.1*	
	Nicotine use	23.9	5.2	1.4	
	OID use	24.1	6.0	2.8*	
5. Concurrent	SUD	32.0	11.2	3.7*	1.4–10.1
		% C in those with A	% C in those without A		
6. Predictive over 1 year	Alcohol (heavy drinking)	Girls, 27–33 Boys, 18–20			
8. Concurrent emotional disorder	Tobacco use			2.30*	
	Alcohol use			1.85*	
	Cannabis use			1.10	
	OID use			2.15*	
9. Concurrent	SUD	17.5		8.03*	3.36–19.16
Lifetime	SUD	49.3	17.7	4.5*	3.2–6.4
10. Concurrent	SUD	18.8	7.1		
11. Concurrent	SUD	11.1	4.8	2.5	0.8–7.7
12. Lifetime	Alcohol abuse	22.8	6.9	3.6*	1.7–5.4
	SUD	20.0	7.0	3.3*	1.4–7.5
13. Psychiatric disorder in past 6 months	Lifetime SUD: Cohort (a)			2.3*	
	Cohort (b)			1.7*	
	Cohort (c)			1.5	
		% C in those with A	% C in those without A		
1. % prevalence of heavy use. OR for ASPD associated with 1-point increase in substance use rating scale	Nicotine use			1.21*	1.05–1.39
	Alcohol use			1.30*	1.07–1.57
	Cannabis use			1.15	0.99–1.34
	OID use			1.14*	1.02–1.28
2. Concurrent association at age 15	SUD			2.4*	1.4–4.2
Prediction from 15 to 16 years, adjusted for confounders	Cannabis use	18.2%	7.5%	1.2	0.5–2.8
3. Concurrent	SUD	40.4			
4. Concurrent	Alcohol use	21.4	11.6	1.1	
	Nicotine use	39.1	12.2	3.9*	
	OID use	31.0	12.0	2.0	
5. Concurrent	SUD	20.0	15.7	1.5	0.5–4.4
9. Lifetime	SUD	16.2	8.1	2.2*	1.4–3.6
10. Concurrent	SUD	33.3			
11. Concurrent	SUD	7.4	3.0	3.2	0.7–16.1
13. Psychiatric disorder in past 6 months	Lifetime SUD: Cohort (a)			1.3	
	Cohort (b)			1.3	
	Cohort (c)			1.5	
		% D in those with A	% D in those without A		
1. OR for ASPD associated with 1-point increase in substance use rating scale	Nicotine use	50	12	1.37	1.14–1.64
	Alcohol use	54	12	1.36	1.08–1.71
	Cannabis use	65	10	1.44	1.20–1.73
	OID use	12	3	1.32	1.16–1.52
2. Conduct disorder					
Concurrent association at age 15	SUD			11.4*	6.8–19.2
Prediction from 15 to 16 years, adjusted for confounders	Cannabis use	38.2	8.1	1.0	0.5–2.1

Table 2 (*continued*)

Study number and notes[a]	A	% D in those with A	% D in those without A	OR	95% CI
3. Concurrent conduct disorder	SUD	51.5			
4. Concurrent DBD	Alcohol use or nicotine use	45.2	7.4	5.2*	
	OID use	47.8	7.7	5.7*	
	SUD	51.7	8.6	3.4*	
5. Concurrent		68.0	10.1	20.3*	7.1–57.8
7. Lifetime conduct disorder	Nicotine D			5.59*	3.82–8.18
	Alcohol A/D			6.23*	4.34–8.97
	Cannabis A/D			6.93*	4.10–11.72
	SUD			5.20*	3.78–7.14
8. Concurrent conduct disorder	Tobacco use			4.38*	
	Alcohol use			3.51*	
	Cannabis use			3.95*	
	OID use			6.48*	
9. Lifetime DBD	SUD	25.4	5.7	5.6*	3.7–8.7
10. Concurrent ASPD	SUD	43.8			
11. Concurrent, any DBD, conduct disorder, and oppositional–defiant disorder	SUD	59.3	3.0	32.6*	11.9–112
		44.0	0.0		
		2.5	1.2	2.1	0.3–17
13. Conduct disorder in past 6 months:	Lifetime SUD: Cohort (a)			1.5	
	Cohort (b)			2.1*	
	Cohort (c)			1.6*	
		% E in those with A	% E in those without A		
2. Concurrent association at age 15	SUD			7.0*	3.6–13.7
7. Lifetime	Nicotine D			2.01*	1.22–3.31
	Alcohol A/D			1.13	0.68–1.91
	Cannabis A/D			1.07	0.51–2.23
	SUD			1.49	0.94–2.34
8. Concurrent	Tobacco use			0.93	
	Alcohol use			0.61	
	Cannabis use			1.23	
	OID use			0.45	
11. Concurrent	SUD	12.3	1.8	5.4*	1.3–22
13. ADHD in past 6 months:	Lifetime SUD: Cohort (a)			1.6*	
	Cohort (b)			2.2*	
	Cohort (c)			1.7*	
		% F in those with A	% F in those without A		
14. Lifetime PTSD	Lifetime alcohol or drug A/D	10.9	3.9	3.83*	1.6–8.9
15. Concurrent eating disorder	Any SUD	5.3		1.97	0.41–9.35
	Alcohol D	9.5		3.91	0.80–19.14
	OID D	4.3		1.52	0.19–12.35
		% G in those with A	% G in those without A		
4. Concurrent	Alcohol use	66.7	15.7	3.8*	
	Nicotine use	73.9	17.0	4.7*	
	OID use	69.0	17.8	3.5*	
5. Concurrent	SUD	76.0	24.5	8.2*	3.0–22.2
9. Lifetime	SUD	66.2	31.3	4.3*	3.0–6.2
11. Concurrent	SUD				
1 disorder		46.9	12.6	8.4*	4.1–17.1
2 or more disorders		14.8	1.2	18.6*	5.6–26.2

Note. A = substance use, abuse (A), and/or dependence (D); B = depression; C = anxiety disorders; D = disruptive behavior disorder (DBD), conduct disorder, or oppositional-defiant disorder; E = attention-deficit/hyperactivity disorder (ADHD); F = other disorders; G = any psychiatric disorder; OR = odds ratio; CI = confidence interval; ASPD = antisocial personality disorder; OID = other illicit drugs (amphetamines, stimulants, cocaine, heroin, opiates, barbiturates, hallucinogens, psychedelics, LSD, quaaludes, tranquilizers, and sedatives); SUD = substance use disorder; PTSD = posttraumatic stress disorder.

[a] Study numbers are listed in Table 1.

* $p < .05$.

lower probability of and delayed onset for alcohol use, whereas children with generalized anxiety disorder used alcohol more frequently and earlier than their peers. Consequently, when anxiety disorders were treated as a single category, no effect of anxiety on alcohol use was found.

Question 3. Does Psychiatric Disorder Affect the Transition From Use to Abuse/Dependence?

Clinical studies of the temporal ordering of SUD and psychiatric disorders have generally found that onset of other psychiatric disorders preceded that of problem drug use (Ellickson & Hays, 1991; Elliott, Huizinga, & Menard, 1988; Gittelman et al., 1985). Community studies, however, have been inconsistent, some agreeing with the clinical literature (Boyle et al., 1992; Gittelman et al., 1985; Kellam & Anthony, 1998; Rohde, Lewinsohn, & Seeley, 1996; Van Kammen & Loeber, 1994) and others disagreeing (Brook et al., 1998; Feehan et al., 1994; Fergusson et al., 1996; Henry et al., 1993). Co-occurring psychiatric disorders could affect the transition from first substance use to later abuse or dependence in two ways: They could make the transition more or less likely or they could affect the timing of the transition.

Does psychiatric disorder affect the likelihood of a transition from use to abuse/dependence? In the most detailed of the predictive analyses, Brook et al. (1998) found that, controlling for earlier psychiatric disorder, levels of tobacco, alcohol, cannabis, and OID use all predicted higher rates of ASPD in young adulthood. There were also predictions from tobacco and OID use to later depressive disorders and from tobacco, alcohol, and OID use to anxiety disorders. Even the predictions that missed statistical significance were in the same direction. Their analyses support Robins's (Robins & Przybeck, 1993) earlier work showing that adolescent substance abuse predicted adult depression. There was considerable consensus that psychiatric disorder, in particular CD, increased the risk of SUD, especially alcohol abuse/dependence (Deykin et al., 1987; Disney, Elkins, McGue, & Iacono, 1999; Lewinsohn et al., 1993). The evidence linking psychiatric disorder to higher rates of substance use, however, was more equivocal. Fergusson et al. (1996) found that the link between CD at age 15 and later cannabis use disappeared when a range of confounders (risk factors associated independently with both CD and cannabis use) were included in the model. The Dunedin longitudinal study found no prediction from CD to substance use (Henry et al., 1993).

Some of the confusion is the result of different definitions of onset and substance abuse. Age at first use has been shown to precede the onset of substance abuse or dependence by about 5 years in those who developed substance abuse and/or dependence during adolescence (Anthony & Petronis, 1995; Beitchman et al., 1999). In Anthony and Petronis's (1995) retrospective analysis of the ECA data, there was about a 4-year interval between first use and abuse, irrespective of age at onset of use. The study did not, however, factor in the effect of psychiatric comorbidity. This progression was tracked by Brook et al. (1998) from drug use, to CD, to drug abuse. Costello, Erkanli, Federman, and Angold (1999) found that the first symptoms of most psychiatric disorders (except depression) preceded the onset of substance use, which was followed by a full-blown psychiatric diagnosis, and then by SUD.

Does psychiatric disorder affect the timing of the transition from use to abuse/dependence? Costello et al. (1999) have provided the most detailed information on this question from their sample of 1,420 youths followed to age 16. Compared with youths who had no psychiatric disorder by age 16, those with disorders started using substances earlier in 26 of 36 comparisons; significantly so in 6 of 36 comparisons. For example, depression was associated with significantly earlier alcohol use in girls and with earlier cannabis and OID use in boys, and DBDs in girls were associated with earlier drug use in general, and smoking in particular. In another article, Kaplow et al. (2001) found that whereas generalized anxiety symptoms were associated with earlier onset of alcohol use, separation anxiety symptoms were linked to significantly later than usual onset. This is consistent with Windle's (1993) thesis that avoidant characteristics in childhood were protective against certain behavioral problems, including adolescent substance abuse.

Costello et al. (1999) also noted that, in most cases, the onsets of first use of any substance, alcohol without permission, cannabis, and OID were all earlier in youths who would move on from use to abuse or dependence by the age of 16. However, the differences were significant only for boys. Indeed, girls who would go on to abuse/dependence started smoking significantly later than other girls (at age 14.3 compared with 12.5).

Question 4. Are Some Groups at Greater Risk for Substance Use and Psychiatric Comorbidity?

Here we review the evidence for similarities and differences in patterns of comorbidity as a function of gender, race/ethnicity, and other factors on which data have been reported.

Gender. Although some studies have reported gender differences in patterns of comorbidity (Boyle & Offord, 1991; Deykin et al., 1987; Disney et al., 1999), the similarities between the sexes have been more remarkable than the differences. Thus, of the 12 studies in Table 1 that reported on gender effects, 6 reported no gender differences, and the rest reported a mixed picture. Kandel et al. (1997) found that there was more psychiatric comorbidity associated with SUD (mainly alcohol abuse) in boys than in girls, as did the Minnesota Twin study (Disney et al., 1999) and the Oregon study (Lewinsohn, Rohde, & Seeley, 1995). Other studies have reported greater comorbidity in girls. The Minnesota Twin study found greater comorbidity between CD and tobacco or cannabis use in girls, and Boyle and Offord (1991) reported higher comorbidity between CD and SUD in girls in the Ontario study. Beitchman et al. (1999), Deykin et al. (1987), Lewinsohn et al. (1995), and Windle and Davies (1999), on the other hand, found an increased risk of emotional rather than behavioral disorders comorbid with SU/A/D in girls: anxiety disorders in the first case, depression in the second and third, and both in the fourth. The mixed pattern found in the Great Smoky Mountains Study for gender effects on the timing of SU/A/D was noted in the previous section.

It is important to bear in mind that the relative paucity of gender differences may be in part an effect of lack of power to detect such interactions.

Race/ethnicity. The effects of race/ethnicity on comorbidity were rarely reported. The Methods for the Epidemiology of Child and Adolescent Mental Disorder (MECA; Kandel et al., 1997) study found that Hispanic ethnicity was associated with lower rates of psychiatric comorbidity, whereas African American ethnicity was associated with higher rates of psychiatric comorbidity. In the Great Smoky Study, American Indian girls reporting tobacco use exhibited higher rates of behavioral disorders compared with White girls reporting tobacco use. Chong et al.'s (1999) report of results from a Taiwanese sample generally showed lower rates of comorbidity, but it is not clear how much this reflected methodologic rather than substantive differences.

Other risk factors. Fergusson et al. (1996), in their analyses of the Christchurch birth cohort data, have always been careful to warn of the importance of including in any explanatory model those risk factors that may act as confounders (Rothman & Greenland, 1998); that is, they may be associated with both the causal variable of interest and with the outcome variable. Thus, their analysis of the link between CD and cannabis use showed that it disappeared when confounders were modeled.

Other factors that need to be included in models of comorbidity are all the usual suspects long known to be associated with both adolescent SU/A/D and with several psychiatric disorders: family structure and functioning, family psychiatric and substance abuse history, poverty, rural versus urban residence, traumatic events, peer relations and peer group characteristics, and so on. With rare exceptions (Fergusson et al., 1996), such models are still lacking. Additionally, given the presence of both substance use and psychiatric disorder in the general adolescent population, it is important to address effect modification in future models.

Discussion

The first conclusion we drew from this review of the published epidemiologic literature on psychiatric comorbidity in youths with SU/A/D was that very few published articles addressed this issue. Of some 160 articles reviewed, only 22 combined the requisite properties of representative community sampling, measurement at the level of *DSM* or International Classification of Diseases diagnoses, and analyses that examined psychiatric disorders in substance-using youths. Given the public health importance of the issue, this is remarkable.

Using the data available, we asked four questions. First, what is the evidence for psychiatric comorbidity in the general population? The review of the current literature revealed a high level of comorbidity; about 60.0% of youths with SU/A/D had some form of comorbid diagnosis. Evidence that the degree of comorbidity increased with the severity of the substance abuse was equivocal for all but DBDs (Kandel et al., 1999; Rohde et al., 1996). This may be a developmental issue, as we discuss later. In general, however, it highlights the point that youths with SU/A/D are not, on the whole, just normal adolescents who happen to be experimenting with substances but, in many cases, are youths with other, and remediable, problems.

The second question explored the category of psychiatric disorder most often comorbid with SU/A/D. CD, and where it was assessed, ODD, proved to be most commonly associated with SU/A/D. ADHD, the third of the group included as a DBD, showed only a weak association with SU/A/D. It is possible that even the associations reported were in fact attributable to the high degree of comorbidity between ADHD and ODD or CD at some developmental stages. More work is needed on this, but the data as they stand should allay the fear that the high level of medication of ADHD might encourage children to become dependent on drugs for control of their moods and behavior, as some have feared. Depression was also significantly associated with SU/A/D, but at a lower rate than CD. Few found a significant association with anxiety (Lewinsohn et al., 1993), and most did not control for comorbidity between other psychiatric disorders and anxiety.

The third question addressed the effect of comorbidity on transitions from substance use to abuse/dependence. Most studies concluded that childhood psychopathology was associated with earlier onset of substance use and substance abuse in later adolescence. CD, in particular, was implicated in this process. The MECA study indicated that a current or lifetime SUD diagnosis placed adolescents at risk for psychiatric comorbidity into adulthood. Such findings suggest the long-term debilitating consequences of adolescent SUD; however, finer-grained analyses are needed to determine whether this trend is specific to age of onset, abuse of specific substances, or development of specific types of symptomatology.

The fourth topic addressed the effect of gender and other risk factors and confounders on the association between SU/A/D and psychiatric disorder. Few studies have conformed to Fergusson et al.'s (1996) warning of the importance of controlling for confounding when examining the effect of potential risk factors. For example, it is well known that DBDs are more common in boys, whereas depression is more common in girls after puberty (Costello et al., 1999). The mixed findings on gender effects may, in part, be results of differences in base rates of the relevant psychiatric disorders in different samples. This in turn may be affected by the age of the samples when interviewed.

With regard to ethnicity, very few studies had sufficiently diverse samples to detect group differences. Although Kandel et al. (1997) reported that Hispanics were less likely to develop psychiatric comorbidity and African Americans were more likely to develop psychiatric comorbidity compared with Whites, none of the current studies controlled for poverty status in analyses linking ethnicity to psychiatric comorbidity. Also, the lower rates of psychiatric comorbidity in Chong et al.'s (1999) Taiwanese sample suggest that the differences may be somewhat attributable to cultural influences such as perceptions of drug use and attitudes toward symptoms of anxiety and depression.

There were several limitations to what could be done on the basis of the published data. Our inability to explore a range of possible risk factors has been discussed. In addition, it was not possible to control for comorbidities among the comorbid psychiatric disorders. A meta-analytic model that attempted this failed to converge, because so few studies reported the necessary information. For the same reason, it was not possible to examine different patterns of comorbidity in different age groups.

Fortunately, there are a number of well-designed studies currently that hold much promise for making progress toward clarifying the nature and significance of psychiatric comorbidity among adolescents with SU/A/D. As part of the process of conducting this review, we sent a questionnaire to every research group that we could identify, from personal knowledge or from a

review of the federal database of currently and previously funded grants, to ask whether they had data addressing the question of psychiatric comorbidity with SU/A/D. Investigators were very generous in sharing this information. We found 15 studies (funded by agencies including the National Institute on Alcoholism and Alcohol Abuse [NIAAA], the National Institute on Drug Abuse [NIDA], and the National Institute of Mental Health [NIMH]), containing over 100,000 person-years of data on over 13,000 participants, including 2,000 African American and 450 American Indian participants (nearly 6,000 and 2,000 person-observations, respectively). Although only those cited above had published data in the form needed for addressing the question of psychiatric comorbidity with SU/A/D, all the investigators answered that they would be willing to do so, if they had the necessary resources for data analysis. This suggests that although much more research is needed to answer the question of comorbidity definitively, the data may already have been collected that could produce the answers needed. One particularly feasible and cost-efficient option is for funding agencies to contract with the study investigators to collaborate on parallel studies of existing data, with one site providing the meta-analytic expertise to combine the results (Armstrong, Erkanli, & Costello, 2001). Given the clinical and public health significance of the problem, this is encouraging.

In terms of future research directions, the studies featured in this literature review can be used to address the important issue of temporal associations between SU/A/D and psychiatric disorder. Many studies up to this point have implemented measurement and design issues that have obscured these relationships (e.g., the use of only one informant, study of age groups in which substance use has already commenced). However, with the wealth of data currently available, researchers can more definitively determine temporal relationships, the mechanisms of the progression from substance use to SU/A/D, the impact of demographic differences (e.g., gender, race, class), type of substance, age of onset, genetic predisposition, even personality traits of the adolescent. In turn, these findings can be replicated in future studies.

The breadth of data sources make the conclusions of these analyses all the more viable for the purposes of informing clinical interventions as well as public policy. Despite the current dearth of treatment studies focused on SUDs and comorbid psychiatric disorders, more data are emerging on evidenced-based treatments for the more common psychiatric disorders (see Compton, Egger, Burns, & Robertson, 2002; Farmer, Compton, Burns, & Robertson, 2002). Moreover, the current state of the literature on adolescent SUD treatment suggests that multisystemic therapy and family therapy are effective among populations of substance-abusing juvenile delinquents (e.g., Bukstein et al., 1997). Empirically supported treatments for SU/A/D and specific comorbid psychiatric disorders can then be developed for implementation, with adequate attention paid to the unique developmental considerations of the adolescent. Given the gap that commonly exists between science and practice regarding effective treatments for SA/D, ongoing meaningful communication between researchers and clinicians can contribute toward course corrections in the development of research designs, specific treatments, and data models that can demonstrate real-world efficacy while preserving scientific fidelity.

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Correction to Speer and Greenbaum (1995)

In the article "Five Methods for Computing Significant Individual Client Change and Improvement Rates: Support for an Individual Growth Curve Approach," by David C. Speer and Paul E. Greenbaum (*Journal of Consulting and Clinical Psychology*, 1995, Vol. 63, No. 6, pp. 1044–1048), the values reported for hierarchical linear modeling (HLM) in Table 2 (p. 1046) were incorrect. The correct values for *SE* are 3.08–4.10, the improvement rate is 32.9%, and the percentage unchanged is 67.1%. These corrections change the results and conclusions of the Speer and Greenbaum study. Instead of producing the highest rate of reliable change, the HLM approach had the scond lowest rate among the five methods investigated. The primary conclusion of the study, that the HLM approach was best for detecting client change, was not supported. Subsequently, among the five methods examined, we now recommend using the Jacobsen-Truax method because of its simple computation and wide acceptance among clinical researchers (Speer, 1999).

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