

Effects of Adolescent Drug Use on Adult Mental Health: A Prospective Study of a Community Sample

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Mental health problems are often observed among drug abusers, although directional effects between drug use and psychopathology remain unclear. Latent-variable models were constructed with prospective data from a community sample of 487 participants who were assessed 4 times over 12 years, beginning when they were young adolescents. Drug use in adolescence and young adulthood included frequency of using alcohol, cigarettes, cannabis, cocaine, and hard drugs and reflected Polydrug Use constructs at both times. A strong stability effect was found for Polydrug Use, and the disturbance term captured the change in polydrug use over this time. Teenage Polydrug Use had few effects on adult mental health, whereas increased Polydrug Use exacerbated later Psychoticism, Suicide Ideation, and other indicators of mental distress. Specific drug use in adolescence and changes in these into young adulthood predicted later psychopathology.

Despite the common assumption that drug use causes emotional distress and psychopathology, there is little scientific evidence for this belief beyond anecdotal and correlational findings. For example, it has been noted that drugs are often used to improve mood and relieve emotional distress (Labouvie, 1986; Newcomb, Chou, Bentler, & Huba, 1988) and that psychiatric impairment is frequently observed in drug-abusing populations (e.g., Bukstein, Brent, & Kaminer, 1989; Ford, Hillard, Giesler, Lassen, & Thomas, 1989). Such associations lead many to conclude (perhaps prematurely) that drug use impairs mental health (e.g., Ross, Glaser, & Germanson, 1988; Rounsaville, Weissman, Chrits-Christoph, Wilber, & Kle-

ber, 1982). However, a clear resolution of whether drug abuse truly contributed to or simply co-occurs with psychopathology and emotional distress has evaded researchers (e.g., Kandel, Davies, Karus, & Yamaguchi, 1986). Research on the associations between drug use and mental health has emerged primarily from three sources: (a) studies of treatment and clinical samples, (b) cross-sectional examinations of more general samples, and (c) analyses of prospective data.

Treatment and Clinical Studies

Most evidence regarding the relationship between psychopathology and drug use comes from clinical samples (e.g., Hesselbrock, Meyer, & Keener, 1985; Ross et al., 1988). In general, these studies reveal that drug abuse is strongly associated with psychopathology before, during, and somewhat less after treatment (e.g., Dorus & Senay, 1980; Kosten, Rounsaville, & Kleber, 1988). However, several confounds typically preclude drawing causal inferences regarding drug abuse and psychopathology from these treatment data. These include the presence of many diverse psychiatric symptoms, inconsistent diagnostic practices, different patient population characteristics, and premorbid symptoms that may be misconstrued as sequelae. The most limiting problem, however, is

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that all patients have some type of severe problem or dysphoria. Thus, they are distressed individuals willing to seek help, whose pathology is manifest and a problem; the development of drug abuse and psychopathology has already occurred and the developmental process cannot be observed accurately. Results from such samples cannot be generalized to nonclinical, community samples.

Cross-Sectional Studies

Findings from cross-sectional studies reveal moderate to strong associations between drug use and psychopathology. For example, Gold, Washton, and Dackis (1985) screened a subset of cocaine abusers who called a national hotline and reported high levels of self-reported depression (83%), irritability (87%), paranoid feelings (65%), and suicide attempts (5%), with users reporting an average of 4 years of drug use. Based on their general population sample, Newcomb, Bentler, and Fahy (1987) reported several significant associations between cocaine use and psychopathology. For example, compared with male nonusers, male cocaine users reported higher levels of anxious mood, impaired cognitive functions, and impaired motivation. They also reported greater hostility, negative affect, a lack of purpose in life, and proneness toward psychotic thinking. Likewise, female cocaine users, when compared with their nonusing counterparts, reported greater levels of depressed and anxious mood, impaired cognitive functions, greater hostility, and greater proneness toward psychotic thinking, along with a host of other distressful symptoms. Kandel (1984) reported that heavier marijuana users were more likely to be hospitalized for mental health problems, were less happy about life, and were more likely to consult a mental health professional as compared with lower level users. However, all these analyses were cross-sectional, so that the causal directions remain obscure.

Prospective Studies

A few longitudinal studies have examined the mental health consequences of drug use in general community populations, and varied findings have emerged (e.g., Kandel et al., 1986; Newcomb & Bentler, 1988a, 1988b). Some have reported causal relationships between drug use and deteriorated

emotional health (e.g., Dackis & Gold, 1983; Newcomb & Bentler, 1988a, 1988b). Others have suggested that, though much accepted as common knowledge, evidence for causal relationships between early drug use and later deficits in emotional development is "hard to verify scientifically" (Newcomb & Bentler, 1988b, p. 64). For example, adequate controls for preexisting conditions and important confounds may not have been made. In addition, several studies have reported apparently positive effects from moderate alcohol use, including greater positive affect, stress reduction, and limited improvements in cognitive performance (Baum-Baicker, 1985; Kandel et al., 1986; Newcomb & Bentler, 1988a, 1988b; Newcomb, Bentler, & Collins, 1986).

Explanatory Models

Several theoretical models have been advanced to explain the association between drug use-abuse and psychopathology. These explanations range from biochemical to psychosocial processes. Several of these perspectives are summarized here.

Biochemical Mechanisms

Recent attention has been directed at the biological mechanisms of drugs on mood changes and the possible pharmacological treatments for drug and alcohol addiction (e.g., Gawin & Kleber, 1984, 1985; Gold et al., 1985; Kleber & Gawin, 1984). Several researchers have suggested that chronic drug use or abuse creates long-term changes in brain chemistry and synaptic transmission (e.g., Dackis & Gold, 1985; Wise, 1984).

Expectancies

A different literature documents the critical role of psychosocial expectations and learning on drug reactions and effects (e.g., Adesso, 1985; Newcomb et al., 1988; Sher & Levenson, 1982). Ethical and scientific restrictions limit these experiments to short-term outcomes and prevent controlled study of the long-term consequences of chronic drug use. With the exception of alcohol, much of this research has been limited to animal experiments, which cannot be generalized with accuracy to humans (for a review, see Adesso, 1980). Nevertheless, prospective studies have begun to address

these causal mechanisms in community samples (e.g., Newcomb et al., 1988; Stacy, Newcomb, & Bentler, 1991a). This leaves unresolved whether psychopharmacological, psychobehavioral, or lifestyle changes from chronic drug use create long-term mental health consequences.

Self-Medication and Biphasic Mechanisms

Alcohol and drug use can have paradoxical or biphasic psychological effects. Initially, alcohol and other drugs are used for their positive acute benefits to improve mood, reduce stress, and relieve emotional discomfort (Dackis & Gold, 1983; Newcomb et al., 1988; Sadava, Thistle, & Forsythe, 1978). These acute positive effects are often followed by emotional and cognitive distress and are exacerbated with continued, recurring, and chronic alcohol and drug consumption (e.g., Beckman, 1980; Bibbs & Chambless, 1986; Keykin, Levy, & Wells, 1987; Malow, West, Williams, & Sutker, 1989). Both learning and opponent process theories have been used to explain drug addiction mechanisms (e.g., Shipley, 1987).

Some hypothesize that drug use may be a form of self-medication: to relieve dysphoria and cope with stress (Khantzian & Treece, 1985; Kleber & Gawin, 1984; Wills, 1985). Barrett (1985) suggested that drug abusers increase their drug use to achieve an affective homeostasis, relying on previously learned psychological reactions and expectancies from ingestion. With increasing physiological and psychological tolerance, drug abusers require greater amounts of the drug to prevent emotional distress, which potentiates an abusive and addictive cycle (Barrett, 1985; Marlatt, Baer, Donovan, & Kivlahan, 1988; Washton & Gold, 1984). Moreover, a drug-abusing lifestyle may create economic, medical, relationship, and work problems that can lead to secondary emotional problems (Newcomb, 1988; Newcomb & Bentler, 1988a). Refuge from these accumulating psychological strains may be sought in continued or increased drug use, which then reinforces the addictive cycle.

Developmental Disruption

Adolescence is an essential developmental period during which rapid emotional, social, biological, and cognitive growth occur (e.g., Newcomb, 1987). It is a time to consolidate psychological,

emotional, cognitive, and problem-solving growth and maturity to assume adult roles and responsibilities (Havighurst, 1972). Disruption of this process through precocious development, persistent drug use, or other interference may impair the learning of adequate psychological adaptational skills necessary for adult roles and responsibilities and create or exacerbate psychopathology (Newcomb, 1987; Newcomb & Bentler, 1988a).

Some Problems in Longitudinal Research

Although prospective studies offer hope for disentangling causal sequences, there are several limitations with earlier research. Often, only a few measures of mental health were used. Kandel et al. (1986) only used scales of depression and psychosomaticism, whereas Newcomb and Bentler (1988a) examined a wider, but still restricted, array of mental health constructs. A more diverse set of mental health measures is necessary to capture precise and specific effects of drug use on psychological functioning.

The maturational course and development of psychological states unfold throughout life. To study these patterns requires long-term prospective data (e.g., Gollob & Reichardt, 1987; Newcomb et al., 1987). Most studies designed to assess influences of drug use on mental health have spanned relatively brief time periods, which prohibit investigation of major developmental transitions (e.g., Kandel et al., 1986; Newcomb & Bentler, 1988a). In their earlier longitudinal investigation, Newcomb and Bentler (1988a) found some limited support for negative effects from early drug use. It is quite possible that effects may be more pronounced over longer periods of time (e.g., Hartka et al., 1989). Certain developmental processes (i.e., including psychological disorders) may take many years to unfold, making it likely that more severe disturbances might only be identified over lengthy periods of time (e.g., Weissman et al., 1984).

In some cases (e.g., Kandel et al., 1986), composite drug use measures were used. This practice obscures potential differential mental health effects related to specific drugs and prevents examination of across-time drug-specific effects (Newcomb, in press; Newcomb & Bentler, 1988a, 1988b).

Finally, most research depicts drug use at discrete points in time that reflect static "snapshots" of drug involvement and do not capture dynamic,

evolving patterns of use. Such static measures of drug use are certainly incomplete, because they do not reflect changes (increases or decreases) or stability of drug use behaviors as they evolve over time. A more comprehensive approach involves comparing early and later drug use measures to capture dynamic drug use transition. One important reason for making this distinction in drug use patterns is to understand whether drug use during adolescence alone affects later mental health or whether any subsequent changes (i.e., escalation, discontinuation) in drug use behaviors influence subsequent mental health. In other words, are teenagers doomed to suffer throughout life because of their adolescent drug use, or do subsequent changes in their drug use patterns alter the effects of earlier drug use?

Importance of This Research

Although research has documented the comorbidity of psychopathology and drug abuse in treatment samples, no casual priority between these disorders has been established, and little clarification has emerged from general community samples (Newcomb et al., 1987). It is unclear whether high rates of comorbidity in treatment populations also exist in community samples. Most important is the need to determine in a rigorous, empirical manner if and how teenage drug use and dynamic changes into adulthood are associated with or exacerbate mental health problems in later life.

This study was designed to answer the following research questions: (a) Does teenage drug use or do changes in subsequent drug use affect psychological functioning in adulthood? (b) Do static measures of adolescent polydrug and use of specific drugs better predict changes in psychological functioning than dynamic measures of the same drugs that capture changes in drug use patterns over an 8-year period reflected in residual variances? and (c) What specific types of mental health are most vulnerable to which type and pattern of adolescent drug use?

To answer these questions, we used latent-variable structural equation models (SEMs) to analyze four waves of data spanning a 12-year period. Numerous measures of drug use were gathered in early and late adolescence and young adulthood, and constructs of psychological functioning, emotional distress, traditional attitudes

(social conformity), and interpersonal contact (social support) from late adolescence were used to predict a wide range of mental health constructs in adulthood. The study spanned a 12-year period in its entirety, with early adolescent drug use measures obtained at baseline (1976) and follow-up measures obtained at Year 13 (1988), although causal inferences can only be made over the last 8 years (because Year 1 and Year 5 drug use data were combined).

This research cannot determine definitively whether types and extents of teenage drug use (and subsequent dynamic changes in these patterns of drug involvement) caused or created increments or decrements for various types of mental health functioning over time. Such precise and unequivocal conclusions can only be made with true experimental designs, including random assignment (e.g., Newcomb, 1990). Such strict control cannot ethically be imposed on long-term issues of drug use and psychopathology. Therefore, we chose the most powerful, nonexperimental method available to study these evolving relationships so that we can make the strongest conclusions regarding potential causal inferences and effects. This approach involves testing appropriate prospective data in complex SEMs and meeting as many causal criteria as possible (Newcomb, 1990, in press). We were able to conform with most criteria under which a causal inference may be made (Newcomb, 1987, 1990). The one critical criterion that this research and all other nonexperimental designs cannot meet is the need to control for all possible confounds and spurious influences. We controlled for the most likely "omitted third variable" problem by including numerous measures of social support and social conformity (e.g., Newcomb & Bentler, 1988a), but these clearly do not capture the universe of all potential confounding influences. Nevertheless, we believe that these conclusions will provide strong support for likely causal effects from teenage drug use and dynamic changes in their drug use patterns over time on various types of mental health functioning in adulthood.

Method

Sample

The sample was initially contacted as 7th-through 9th-grade students in 11 Los Angeles

Table1
Description of Sample

Variable	Men (<i>n</i> = 136)	Women (<i>n</i> = 351)	Total (<i>N</i> = 487)
Age (years)			
<i>M</i>	25.46	25.49	25.48
Range	24–28	24–27	24–28
Ethnicity (%)			
Black	8	15	13
Hispanic	10	11	11
White	71	62	64
Asian	10	11	11
Other	1	1	1
Education (years)			
<i>M</i>	14.65	14.03	14.21
Range	10–18	10–18	10–18
No. of children (%)			
None	78	64	68
One	15	21	19
Two	6	11	9
Three or more	1	4	4
Income for past year (%)			
Under \$5,000	7	19	16
\$5,000–\$15,000	28	26	26
\$15,000–\$30,000	44	46	46
Over \$30,000	21	9	12
Living situation(%)			
Alone	9	8	8
Parents	19	19	19
Spouse	41	41	41
Cohabitation	8	8	8
Dormitory	2	0	1
Roommates	14	13	13
Other	7	11	10
Current life activity (%)			
Military	3	0	2
Junior college	3	2	2
Four-year college	9	5	6
Part-time job	5	7	6
Full-time job	73	67	69
Child rearing/homemaker	1	15	11
Other	4	4	4

County schools and was followed for 12 years so that we could study adolescent and adult growth, development, and drug use. The sample for this study includes 487 participants who provided data in Year 1 (1976), Year 5 (1980), Year 9 (1984), and Year 13 (1988). A description of this sample as adults at Year 13 is given in Table 1. The sample is ethnically mixed (about one third non-White), has a mean age of about 25.5 years, and contains more women than men (which has been an unfortunate

feature from Year 1). Most are high school graduates with an average of 2 years of college, more than two thirds have full-time jobs, and about half are living with a mate. More complete descriptions of the sample and the scope of the research project are reported elsewhere (Newcomb, 1992; Newcomb & Bentler, 1988a, 1988b; Stacy, Newcomb, & Bentler, 1991a, 1991b).

Newcomb (1992) provided extensive attrition analyses across the entire 12 years of this study.

Overall, these analyses show that gender and ethnic composition have been only slightly altered by attrition. Likewise, there were only minor differences in Year 1 drug use prevalence rates between dropouts and retained subjects over the 12-year period. Moreover, participants who remained in the study were quite similar to dropouts on numerous drug and personality measures obtained at Year 1.

Measures

Adolescent drug use scales. In Year 1, frequency-of-use scales for 5 classes of drugs were generated: alcohol (beer, wine, and liquor), cigarettes (one item), marijuana (cannabis and hashish), cocaine (one item), and hard drugs (including downers, heroin, inhalants, psychedelics, and uppers). Year 1 drug use items were rated on 5-point anchored scales that ranged from *never* (1) to *regularly* (5). In Year 5, the same 5 drug use scales were created from identical substances as in Year 1 except for hard drugs, which in Year 5 were expanded to include 14 different substances. Year 5 drug items were rated on 7-point anchored scales, ranging from *never* (1) to *more than once a day* (7), for the past 6 months. All 10 drug use scales were standardized, and then the same scales from Year 1 and Year 5 were averaged to produce 5 composite drug use scales reflecting drug use during adolescence.

These five scales of adolescent drug use were used to reflect a Polydrug Use latent construct that captured the extent of using multiple substances during early and late adolescence. In addition, the residual variables from each of the five scales were used to capture the specific use of each separate drug and were tested as predictors. This procedure allowed us to separate the general effects of polydrug use, quite prevalent among adolescents (e.g., Clayton & Ritter, 1985), as a latent construct, from the effects of using specific types of drugs, as captured in the residual variables of the specific drug use scales (after prediction from the common factor: see Newcomb, in press). This method has been described by Newcomb (1990), with numerous examples elsewhere (e.g., Newcomb, 1988; Newcomb & Bentler, 1988a, 1988b). The one difficulty with this procedure, when used with first-order latent factor models, is that systematic and random errors are confounded in the residual variables (Newcomb, in press). Because of this

limitation, we primarily focus on large effects of these specific, residual variables (i.e., Stacy et al., 1991a, 1991b).

Increased drug use. Five young adult drug use scales (Year 9) consisted of the same items and rating scales as in Year 5 and were used to reflect a latent construct of Polydrug Use. For theoretical purposes, we wanted to use these drug use measures to construct variables that reflect change in drug use from adolescence to young adulthood. In other words, we wanted to compare drug use measures from adolescence to those in young adulthood to capture dynamic changes (increased, decreased, or remained the same) of evolving drug involvement after adolescence to young adulthood and determine whether these generated changes in psychological functioning and emotional distress into adulthood.

Dynamic changes in drug use can be captured in several ways from repeated assessments. Without a time-series design, differences can be represented as change scores. For example, $V_2 - V_1$ would represent the change in a variable V from Occasion 1 to Occasion 2. In earlier, preliminary work, we tested this typical method to capture these changes in drug use over time as difference scores (Newcomb & Bentler, 1989). This quite common method created several difficulties, such as floor and ceiling effects, and numerous paradoxical or uninterpretable results. Because of these difficulties, which are commonly encountered with change or difference scores (e.g., Cronbach & Furby, 1970; Woody & Costanzo, 1990), we used an alternative procedure in the present analyses. Furthermore, differences among observed scores do not adequately reflect changes in a latent variable, and even latent variables created from simple difference scores may not adequately reflect the stability of that latent construct (e.g., Nesselroade & Bartsch, 1977).

J. Cohen and Cohen (1983) suggested an hierarchical multiple regression approach as an alternative to difference scores with repeated measure data. However, this approach exaggerates the cross-sectional associations at the follow-up wave of data, which confounds change effects with contemporaneous correlations. This approach is also restricted to measured variables and is not extended to latent variables or unmeasured constructs.

We took J. Cohen and Cohen's (1983) basic

approach and extended it to resolve both of these difficulties and unresolved issues. To deal with the follow-up confound problems, we needed at least three waves of data. Dwyer (1983) pointed out that to establish causal order between events, more than two measurement waves are necessary. We restricted our drug use follow-up measures (Year 9) to a data wave earlier than the dependent or consequence constructs (Year 13) but later than the independent variables or baseline measure (Years 1 and 5). Therefore, our change (residual) scores preceded our dependent measures by 4 years and did not capitalize on contemporaneous associations. Furthermore, we took J. Cohen and Cohen's approach and applied it to latent constructs by using the disturbance term of later Polydrug Use after prediction from earlier Polydrug Use as a change (residual) score to predict consequences at a later time. Similarly, we used measured-variable residual variables to simultaneously capture change in use of specific drugs.

These innovative procedures certainly deviate necessarily from the inadequate recommendations of J. Cohen and Cohen (1983). They reflect significant improvements in method (SEMs), confounds (three waves of data instead of two), and interpretation (change precedes effects). Our solution uses residualized latent- and measured-variable scores from young adulthood to predict changes in mental health measures into adulthood. That is, if $F2 = B * F1 + D2$, where $F1$ and $F2$ are the latent variables F measured at Occasions 1 and 2, and B is the usual optimal regression weight, $D2$ represents the part of $F2$ that is not predictable from $F1$ (Bentler, 1989). In our models, we evaluate the usual effects of $F1$ on various subsequent outcome latent variables (in adulthood), but, in addition, we permit $D2$ to be correlated with other factors at Time 1 (Year 9) and evaluate the "causal" effects of $D2$ on a variety of other factors measured at Time 3 (Year 13).

In other words, we use residual or disturbance terms as change variables. These residual variables reflect that portion of drug use that is not stable over time, capturing whether drug use increased, decreased, or remained the same from one assessment to the next. In our structural equation models, the residual variables reflect changes in each of the five types of drugs, as well as changes in polydrug use as captured by latent constructs. These residual variables are constructed so that

higher values reflect increased drug use and they are labeled accordingly.

Adolescent psychosocial scales. We included three other constructs from late adolescence (Year 5) to control for across-time stability and spurious confound effects on later psychopathology. A construct of Emotional Distress was reflected in three scales: self-derogation (seven items; Kaplan & Pokorny, 1969); (low) self-acceptance (four items; Stein, Newcomb, & Bentler, 1986); and depression (four items; Newcomb, Huba, & Bentler, 1986). This Emotional Distress construct served as a general distress measure to control for stability in the later mental health measures. Support for the use of a general distress construct is provided by both Scheier and Newcomb (1993) and Tanaka and Huba (1984).

Deviant or unconventional attitudes and behavior are strongly associated with drug use (McGee & Newcomb, 1992). To control for such possible confounds, we included a construct of Social Conformity from adolescence to reflect rejection or adherence to conventional values. This latent factor was reflected in three multiitem scales: law abidance, (low) liberalism, and religious commitment (Newcomb & Bentler, 1988a, 1988b; Stein et al., 1986).

Finally, a latent factor of Social Support during adolescence was included. This construct was captured by four four-item scales measuring the supportiveness of relationships with parents, family, adults, and peers (Newcomb & Bentler, 1988b). Social Support is an important protective factor against psychopathology and positive influence on emotional health (e.g., S. Cohen & Wills, 1985; Newcomb & Bentler, 1988b) that must be represented in the present analyses.

Adult Outcome Measures

Adult mental health status was captured by 27 measured variables used to reflect nine latent constructs: Emotional Distress, Self-Derogation, Psychoticism, Depression, Purpose in Life, Suicide Ideation, Anxiety, Hostility, and Disorganized Thinking. The confirmatory factor structure and more complete description of all items and scales are presented elsewhere (Scheier & Newcomb, 1993).

The adult Emotional Distress construct had two indicators. These were repeated assessments of

the same depression and (low) self-acceptance scales used in adolescence. The Self-Derogation scale was also repeated in adulthood but was not used as an indicator of the Emotional Distress construct as during adolescence. To gain greater sensitivity and specificity in adult affective functioning, we created a separate Self-Derogation construct with two indicators: one scale each of positive and negative self-image (Harlow, Newcomb, & Bentler, 1986).

The Magic Ideation (Magid) Scale (Eckblad & Chapman, 1983) was used to create indicators of a latent construct of Psychoticism. The 30 dichotomous items assess schizotypy and a predisposition to psychosis. These items were randomly parceled into three 10-item subscales (Magid 1, 2, and 3; Newcomb & Bentler, 1988a).

A Depression construct was reflected by four subscales of the 20-item Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977). The CES-D emphasizes the affective component of depression. The subscales were derived from previous factor analyses and included positive affect (the elevated mood items), negative affect, impaired motivation, and impaired relationships (Harlow et al., 1986).

A construct of Purpose in Life (PIL) was hypothesized to reflect three scales created from a slight modification of the 20-item Crumbaugh and Maholick (1964) measure. Previous exploratory and confirmatory factor analytic work verified the unidimensionality of the measure and the reliability of three scales of these 20 seven-point Likert-type items as indicators of a latent construct (PIL 1, 2, and 3; Harlow, Newcomb, & Bentler, 1986, 1987).

A Suicide Ideation construct was reflected by four items: two from the Petrie and Chamberlain (1983) Suicide Behavior Subscale ("I have been thinking about ways to kill myself" and "I have told someone I want to kill myself") and two additional items we created ("I have made attempts to kill myself" and "I imagine my life will end with suicide"; Harlow et al., 1986).

Separate constructs of Anxiety and Hostility were derived from a modified version of the Hopkins Symptom Checklist (Uhlenhuth, Balter, Mellinger, Cisin, & Clinthorne, 1983). Three items with the largest factor loadings from each scale were chosen as indicators for each construct.

Finally, three four-item scales reflected a construct of Disorganized Thinking: thought disorga-

nization, (less) deliberateness, and (less) diligence (Stein et al., 1986).

Results

Descriptive statistics for all adolescent, young adult, and adult variables are given in Table 2. The right-hand column presents point-biserial correlations of each variable with gender of the respondent and reveals several mean differences. The largest accounted for less than 4% of the variance between men and women. Only a few researchers have found sex differences in psychological consequences of alcohol and drug use (e.g., Robbins, 1989). The many variables needed in our model require a large sample for stable and reliable results. Separate analyses by sex would reduce our sample size to unacceptable numbers (e.g., Bentler & Chou, 1987; Marsh, Balla, & McDonald, 1988; Tanaka, 1987). Therefore, we had to combine our samples of men and women in our SEMs, even though this sacrifices our ability to examine gender-specific mental health consequences of drug use.

We now turn to our SEM analyses. Although a few of our variables may be considered quasi-continuous or border on categorical (i.e., drug use items, not the scales), categorical methods cannot currently be practically implemented with such a large model (Newcomb, 1990). More important, Hays and Huba (1988) demonstrated clearly that conclusions reached when drug use frequency items were analyzed by several different procedures all arrived at similar conclusions. Finally, the present analyses are based on presumed linear relationships among the variables and are not designed to test for potential curvilinear or other nonlinear patterns, as at least one other study has noted may exist between drug use and psychological variables (Shedler & Block, 1990).

Confirmatory Factor Analyses

Before developing our structural or path model, we conducted a confirmatory factor analysis (CFA) to assess how well the observed measures reflected the hypothesized latent constructs. This analysis also allowed us to examine the correlations among the unobserved latent factors. Change in Polydrug Use from adolescence to young adulthood was captured as a disturbance term (with higher values reflecting Increased Polydrug Use). We accom-

plished this by allowing the adolescent Polydrug Use construct to predict the young adult Polydrug Use construct, and the residual (or disturbance) variable of this adult latent factor represents Increased Polydrug Use. Cross-time correlated residuals were included a priori for repeatedly measured variables. In addition, we correlated residuals of cocaine and hard drugs at each time to capture their unique similarities as measures of extremely illicit or hard drugs.

The fit of the model was adequate according to several criteria, $\chi^2(932, N = 487) = 1,761.01, p < .001$, Comparative Fit Index (CFI; Bentler, 1990) = .92. The CFI is a sample-size adjusted analogue to the Normed Fit Index (Bentler & Bonett, 1980) and indicates the amount of covariation in the data accounted for by the hypothesized model. A rule of thumb is not to reject a model when the ratio of the model's chi-square to the degrees of freedom is less than 2.00 (Bentler, 1980; Bentler & Bonett, 1980). This ratio for the CFA model was 1.89, indicating an adequate model fit. Standardized factor loadings and residual variables (variances) of the observed variables for the CFA model are depicted in Figure 1.

As hypothesized, all factor loadings for the measured variables on the latent factors were sizable and significant ($p < .001$). The adolescent Polydrug Use construct largely reflected marijuana, which had the largest loading, followed in magnitude by alcohol and cigarette use and to a lesser extent by cocaine and hard drug use. Similarly, marijuana use had nearly the largest factor loading on the young adult Polydrug Use construct; however, in this case, cocaine use had a larger loading than alcohol and cigarette use.

Table 3 presents the intercorrelations among the 13 latent factors and Increased Polydrug Use residual variable. For the adolescent latent constructs, adolescent Polydrug Use was strongly and significantly associated with less Social Conformity and moderately correlated with less Social Support, more adolescent Emotional Distress, and more adult Psychoticism. The stability correlation of adolescent Emotional Distress with adult Emotional Distress was of moderate magnitude and larger than with the other adult constructs. Adolescent Emotional Distress was also significantly correlated with all but one of the other adult mental health constructs (excluding Psychoticism). This generally supports our use of the adolescent Emo-

tional Distress construct as a broad measure of adolescent psychological distress and control for stability effects of most adult mental health constructs.

Adolescent Polydrug Use significantly predicted young adult Polydrug Use ($\beta = .74$), leaving 45% of residual variance that we interpret as changes (increases or decreases) in Polydrug Use from adolescence to young adulthood. Increased Polydrug Use was correlated significantly with adolescent Social Conformity and only Psychoticism of the adult mental health constructs.

Structural Model Analyses

The same factor structure or measurement portion of the CFA model was used as the basis for our initial structural or path model. Correlations among all of the adolescent latent factors and Increased Polydrug Use variable (residual) were allowed to be estimated freely. We estimated unidirectional paths from each adolescent construct (and Increased Polydrug Use) to every adult construct, capturing possible causal relationships in a saturated fashion (Newcomb, in press). The disturbance terms of all dependent (adult) latent factors were allowed to intercorrelate freely. Each of the residual variables from the five specific types of adolescent drug use was allowed to predict its analogous measure in adulthood. The residuals of each of these five young adult drug use measures represent increased use of each drug. Finally, within-time correlated residuals for cocaine and hard drugs were retained, and residuals for repeatedly measured psychosocial variables were correlated across time.

As expected, this initial structural model had an identical fit to the CFA model. This initial structural model only tests standard across-time paths, those between latent constructs (Newcomb, in press). To capture more detailed, subtle, and comprehensive patterns in these data, we continued our development of this structural model by testing for specific or nonstandard paths across time (Newcomb, 1990, in press; Newcomb & Bentler, 1988a, 1988b). Specific nonstandard paths were added on the basis of empirical importance and substantive interpretations. Empirical guidance was obtained from stepwise Lagrangian multiplier tests (Chou & Bentler, 1990). In some cases, the sign of a parameter estimate was opposite to

Table 2
Summary of Descriptive Statistics

Latent construct/ measured variable	<i>M</i>	No. of items	Range	<i>SD</i>	Skew	Kurtosis	Mean sex difference ^a <i>r_{pb}</i>
Adolescence (Years 1 and 5)							
Adolescent Drug Use							
Alcohol frequency	—	6	–1.33–2.49	—	0.13	–0.91	–.03
Cigarette frequency	—	2	–0.85–2.22	—	0.95	0.07	.12**
Cannabis frequency	—	4	–0.60–3.25	—	1.73	2.67	.01
Cocaine frequency	—	2	–0.30–5.15	—	3.46	15.42	.06
Hard drug frequency	—	19	–0.33–8.74	—	4.16	33.31	.07
Emotional Distress							
Depression	7.66	4	4–18	3.25	0.85	–0.49	.03
Self-acceptance	15.89	4	4–20	3.12	–0.67	0.04	–.03
Self-derogation	2.41	7	0–10	2.79	0.87	–0.08	.09*
Social Conformity							
Liberalness	9.99	4	4–19	2.57	0.22	0.10	.03
Law abidance	13.15	4	4–20	3.96	–0.21	–0.70	.13**
Religious commitment	15.44	4	4–20	3.98	–0.71	–0.28	.14**
Social Support							
Relationships with:							
Parents	15.99	4	6–20	3.39	–0.78	–0.09	.03
Family	14.55	4	4–20	4.14	–0.57	–0.46	–.02
Adults	17.11	4	7–20	2.27	–0.70	0.40	.09*
Peers	16.74	4	6–20	2.74	–1.08	1.12	.04
Young adulthood (Year 9)							
Young Adult Drug Use							
Alcohol use	—	3	–1.62–2.17	—	–0.01	–1.04	–.09*
Cigarette use	—	2	–0.66–1.79	—	1.06	–0.69	–.07
Cannabis use	—	2	–0.65–3.63	—	1.46	1.02	.00
Cocaine use	—	2	–0.60–3.65	—	1.55	1.31	–.01
Hard drug use	—	14	–0.40–9.12	—	3.91	21.26	–.05
Adulthood (Year 13)							
Emotional Distress							
Depression	7.24	4	4–20	2.99	1.36	2.25	.02
Self-acceptance	16.06	4	4–20	2.91	–0.94	0.91	–.06
Self-Derogation							
Positive self-image	5.77	2	4–6	0.55	–2.26	3.95	–.11*
Negative self-image	10.97	5	10–15	1.28	1.24	0.70	.05
Purpose in Life (PIL)							
PIL 1	11.42	6	4.3–14	1.68	–0.97	0.87	–.06
PIL 2	10.84	7	3.6–14	1.71	–0.70	0.36	–.06
PIL 3	11.13	7	3.5–14	1.59	–0.65	0.78	.03
Psychoticism							
Magid 1	21.60	10	20–28	1.69	1.20	1.18	.07
Magid 2	16.22	10	14–21	1.32	0.90	0.54	.05
Magid 3	28.78	10	18–35	1.91	–0.50	5.68	.03
Suicide Ideation							
Told someone kill self	1.18	1	1–4	0.47	2.76	7.61	–.04
Think about killing self	1.25	1	1–4	0.54	2.31	5.56	–.05
Life end with suicide	1.10	1	1–4	0.39	4.35	21.01	–.06
Tried to kill self	1.09	1	1–4	0.38	5.05	29.45	.06

Table 2 (continued)

Latent construct/ measured variable	<i>M</i>	No. of items	Range	<i>SD</i>	Skew	Kurtosis	Mean sex difference ^a <i>r_{pb}</i>
Adulthood (Year 13) (continued)							
CES-D							
Positive affect	2.42	4	0.50–3	0.60	–1.00	0.32	–.12**
Negative affect	0.50	5	0–2.8	0.54	1.30	1.52	.16***
Impaired social relations	0.26	3	0–3	0.41	2.29	7.52	.00
Impaired cognitions	0.57	8	0–2.75	0.44	0.90	1.00	.10*
Hostility							
Hostility 1	2.47	1	1–5	0.86	0.25	–0.09	.19***
Hostility 2	2.36	1	1–5	0.80	0.34	0.20	.16***
Hostility 3	2.00	1	1–4	0.80	0.50	–0.21	.16***
Anxiety							
Anxiety 1	2.43	1	1–5	0.87	0.27	–0.27	.01
Anxiety 2	2.56	1	1–5	0.83	0.07	–0.29	.00
Anxiety 3	2.18	1	1–5	0.83	0.60	0.40	–.09*
Disorganized Thinking							
Deliberateness	13.46	1	4–20	3.04	–0.24	–0.10	–.06
Diligence	15.80	1	6–20	2.75	–0.60	0.32	.03
Thought disorganization	8.89	1	4–18	2.89	0.52	0.00	.06

Note. Dashes for adolescent drug use indicate standardized scale scores were used averaging over early and late adolescence, and dashes for young adult drug use indicate standardized scale scores were also used. Magid = magic ideation; CES-D = Center for Epidemiologic Studies–Depression Scale.

^aA positive correlation indicates that women had the larger value.

* $p \leq .05$. ** $p \leq .01$. *** $p \leq .001$.

the corresponding covariance; these parameters were regarded as suppressor effects and were not included. Specification searches were restricted to only three types of specific or nonstandard across-time structural paths and were considered in the following sequence: (a) adolescent latent factors and Increased Polydrug Use variable to measured variables in adulthood; (b) measured-variable residual terms in adolescence and those five residuals reflecting increases in specific types of drug use to adult latent factors; and (c) adolescent measured-variable residuals and five increased specific drug use residuals to adult measured variables. After paths were added with the Lagrangian test, we made final model adjustments by deleting nonsignificant parameters with the Wald test (Chou & Bentler, 1990). This procedure of additions and then deletions of model parameters has been found to be the best modification strategy to capture the “true” model (Chou & Bentler, 1990; MacCallum, 1986).

A final structural model was obtained with an adequate fit, $\chi^2(856, N = 487) = 1,215.31, p < .001$; CFI = .96. Figure 2 depicts the significant paths between latent factors in this final structural

model. Correlations among adult latent construct residuals were omitted from the figure for ease of depiction but are presented in Table 4.

Adolescent Polydrug Use did not significantly predict any of the mental health latent constructs in adulthood. On the other hand, Increased Polydrug Use was significantly associated with increased adult Psychoticism and Suicide Ideation. The effect on Psychoticism should be interpreted with caution, because no comparable baseline measure was available to control for its stability and adolescent Emotional Distress did not serve this function.

Several nondrug effects between adolescent Emotional Distress and adult latent constructs were also apparent. Adolescent Emotional Distress increased adult Emotional Distress, Self-Derogation, Suicide Ideation, Hostility, Disorganized Thinking, and decreased Purpose in Life. Adolescent Social Conformity and Social Support had no significant, direct effects on any of the adult latent constructs.

Table 5 contains a summary of all specific or nonstandard effects reflecting paths from adolescent measured-variable residuals and latent fac-

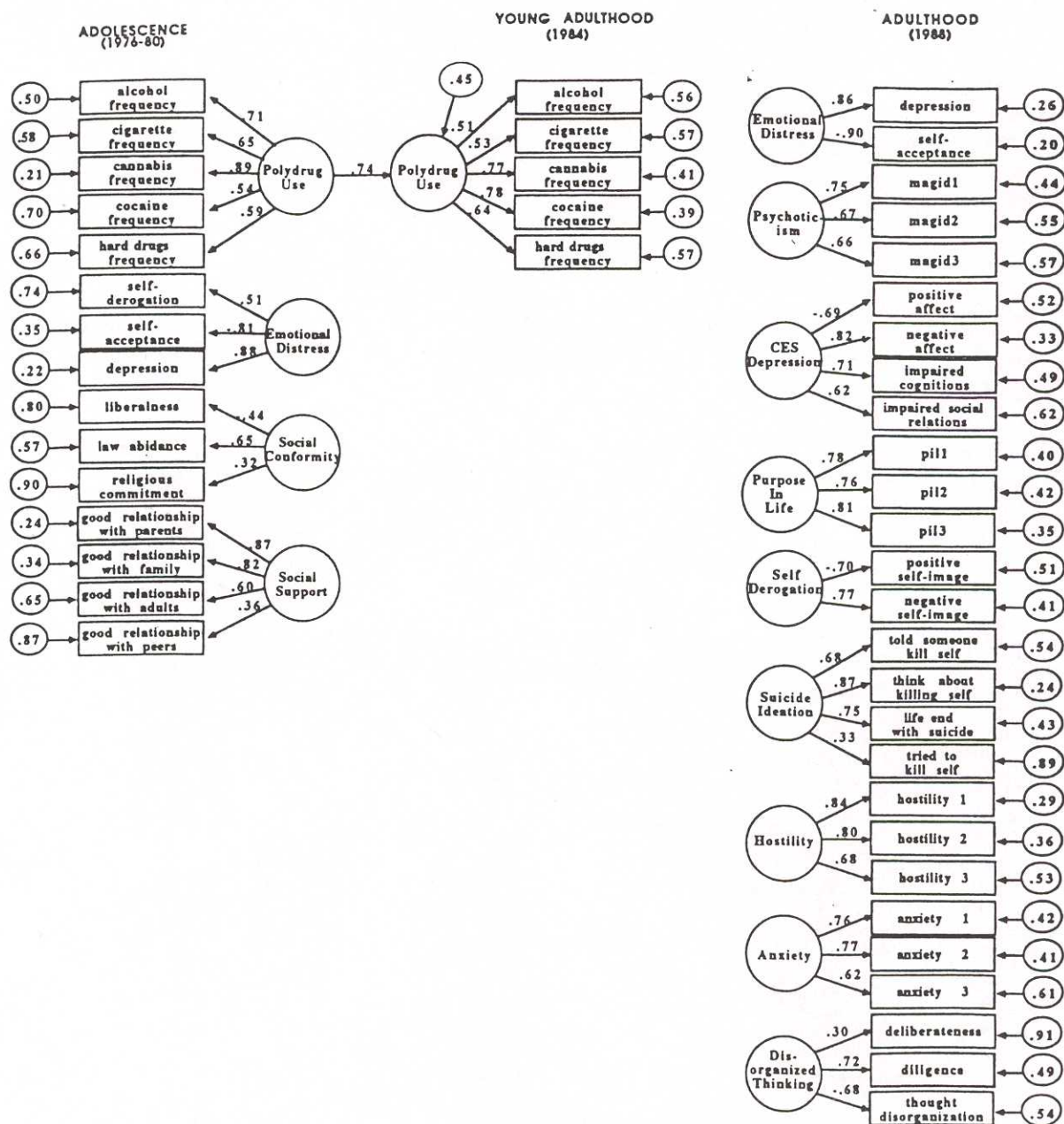


Figure 1. Final confirmatory factor analysis model. (Large circles represent latent constructs, rectangles are measured variables, and small circles with numbers are residual variables. Factor loadings are standardized and all are significant [$p < .001$]. Not depicted in the figure are two-headed arrows [i.e., correlations] joining each possible pair of factors. Estimates for these correlations are given in Table 3. CES = Center for Epidemiologic Studies; magid = magic ideation; pil = purpose in life.)

tors to the various adult mental health measures (both latent and observed). These paths are all from the final structural model and must be considered in concert with results from the same

model given in Figure 2 and Table 4. These specific or nonstandard paths provide a detailed picture of more specific influences of both drug and nondrug adolescent variables on adult mental health (see

Table 3
Factor Intercorrelations for the Confirmatory Factor Analysis Model

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Adolescence														
1. Adolescent Polydrug Use	—													
2. Increased Polydrug Use ^a	.00	—												
3. Emotional Distress	.14**	.01	—											
4. Social Conformity	-.70***	-.24**	-.38***	—										
5. Social Support	-.22***	-.12*	-.64***	.52***	—									
Adulthood														
6. Emotional Distress	.08	.04	.41***	-.20**	-.34***	—								
7. Psychoticism	.21***	.32***	.08	-.25***	-.10*	.19***	—							
8. CES-D	-.02	.06	.27***	-.09	-.24***	.74***	.31***	—						
9. Purpose in Life	-.10*	-.01	-.39***	.25***	.33***	-.86***	-.21***	-.68***	—					
10. Self-Derogation	.08	.01	.39***	-.22**	-.32***	.88***	.34***	.71***	-.75***	—				
11. Suicide Ideation	.03	.12*	.26***	-.12*	-.16**	.62***	.26***	.57***	-.63***	.62***	—			
12. Hostility	.05	-.01	.32***	-.12*	-.19***	.56***	.20***	.64***	-.57***	.63***	.42***	—		
13. Anxiety	.04	.08	.25***	-.05	-.09	.28***	.21***	.44***	-.35***	.37***	.23***	.55***	—	
14. Disorganized Thinking	.08	.01	.17**	-.24**	-.14*	.65***	.26***	.53***	-.61***	.57***	.32***	.46***	.28***	—

Note: Significance level is determined by a critical ratio of the unstandardized parameter estimate divided by its standard error. CES-D = Center for Epidemiologic Studies-Depression Scale.

^aThis measure reflects increased drug use from adolescence (Years 1-5) to young adulthood (Year 9).

* $p < .05$. ** $p < .01$. *** $p < .001$.

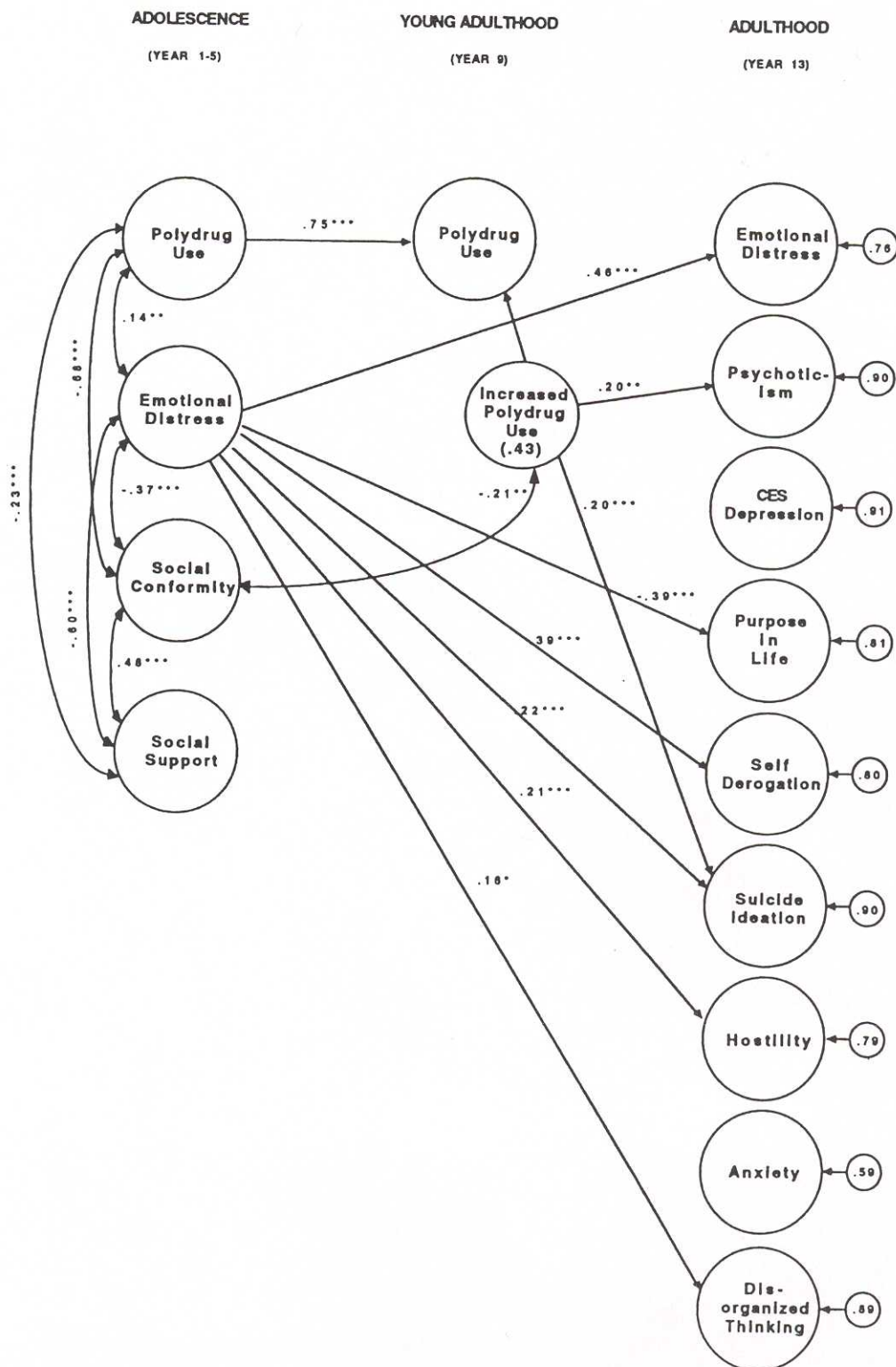


Table 4

Correlations Among the Factor Residuals in Adulthood From the Final Structural Model

Factor	1	2	3	4	5	6	7	8	9
1. Emotional Distress	—								
2. Psychoticism	.00 ^a	—							
3. CES-D	.89	.11*	—						
4. Purpose in Life	-.84	.00 ^a	-.78	—					
5. Self-Derogation	.86	.11*	.78	-.70	—				
6. Suicide Ideation	.62	.00 ^a	.61	-.63	.58	—			
7. Hostility	.60	.00 ^a	.55	-.60	.61	.39	—		
8. Anxiety	.48	.00 ^a	.24	-.51	.48	.22*	.45	—	
9. Disorganized Thinking	.78	.00 ^a	.43	-.69	.59	.31	.34	.00 ^a	—

Note. Unless otherwise noted, all residual correlations are significant at $p < .001$ level ($*p < .01$). CES-D = Center for Epidemiologic Studies–Depression Scale.

^aParameter was nonsignificant and constrained at zero in the final structural model.

Newcomb, in press). Although many of these effects are relatively small, they represent partial regression coefficients, indicating unique contributions beyond stability and baseline controls. Table 5 is organized into three sections on the basis of the content of the predictor variables: adolescent drug effects, increased drug use effects, and non-drug effects. These effects are numerous, and only selected ones are discussed here (all are presented in Table 5).

By far the largest nonstandard regression effect involving drug use was from adolescent Polydrug Use to one indicator of Psychoticism (Magid 1). Among the various adolescent drug use predictor variables, specific use of alcohol (residual) had five significant across-time effects: higher alcohol use in adolescence decreased later CES-D, impaired cognitions (apparent beneficial effects), and PIL 3 and increased one indicator of Suicide Ideation (“think about killing self”) and one indicator of Anxiety. Adolescent cigarette use increased adult impaired cognitions, Magid 3, and thought disorganization and decreased PIL 2, positive self-image, and deliberateness. Adolescent cannabis fre-

quency had four significant nonstandard effects: increased CES-D and Magid 3 and decreased positive self-image and “tried to kill self.” Only one effect was noted for adolescent cocaine use: It increased one indicator of Psychoticism. Adolescent hard drug use increased negative self-image, “told someone kill self,” and Magid 2 and decreased PIL 2.

Increases in Polydrug Use and specific types of drug use had a variety of effects on mental health in adulthood and are presented in the second section of Table 5. Increased Polydrug Use exacerbated impaired relationships and Anxiety 3 and reduced positive affect. Increased consumption of alcohol into young adulthood had 3 specific effects: improved self-image, reduced impaired relations (apparent beneficial effects), and decreased PIL 1. Increased cigarette use decreased Purpose in Life and deliberateness and heightened thoughts about killing self. Increased cannabis use had 10 specific effects on adult mental health functioning: greater Psychoticism (and Magid 1), negative affect, Hostility 1, thought disorganization, Emotional Distress, and Self-Derogation. Increased cannabis involve-

Figure 2 (opposite). Final structural model of the across-time effects between latent constructs. (Large circles represent latent factors, and small circles with numbers reflect residual variances. Path coefficients are standardized, and significance levels were determined by critical ratios on unstandardized coefficients [$*p < .05$; $**p < .01$; $***p < .001$]. Other nonstandard regression effects from this same final model that do not relate latent constructs to one another are given in Table 5. Correlations among the residuals of adult latent factors are given in Table 4. CES = Center for Epidemiologic Studies.)

Table 5
Nonstandard Effects Not Depicted in Figure 2

Predictors		Consequences		Standard- ized parameter estimate
Observed	Latent	Observed	Latent	
		Adolescent drug effects		
	Polydrug Use	Magid 1		.25**
Alcohol freq.			CES-D	-.12**
Alcohol freq.		Impaired cognitions		-.07*
Alcohol freq.		PIL 3		-.08**
Alcohol freq.		Think about killing self		.07*
Alcohol freq.		Anxiety 3		.12**
Cigarette freq.		Magid 3		.10*
Cigarette freq.		Impaired cognitions		.07*
Cigarette freq.		PIL 2		-.09**
Cigarette freq.		Positive self-image		-.08*
Cigarette freq.		Deliberateness		-.11*
Cigarette freq.		Thought disorganization		.07*
Cannabis freq.			CES-D	.17**
Cannabis freq.		Positive self-image		-.12*
Cannabis freq.		Tried to kill self		-.10*
Cannabis freq.		Magid 3		.11*
Cocaine freq.		Magid 3		.10*
Hard drug freq.		Magid 2		.09*
Hard drug freq.		PIL 2		-.06*
Hard drug freq.		Negative self-image		.13***
Hard drug freq.		Told someone kill self		.07*
		Increased drug use effects		
	Increased Polydrug Use	Positive affect		-.13**
	Increased Polydrug Use	Impaired relations		.18***
	Increased Polydrug Use	Anxiety 3		.15**
Increased alcohol use		Impaired relations		-.14***
Increased alcohol use		PIL 1		-.06**
Increased alcohol use		Positive self-image		.09**
Increased cigarette use		Think about killing self		.05*
Increased cigarette use		Deliberateness		-.15**
Increased cigarette use			Purpose in Life	-.06*
Increased cannabis use			Psychoticism	.11**
Increased cannabis use		Magid 1		.11*
Increased cannabis use		Positive affect		-.17***
Increased cannabis use		Negative affect		.09**
Increased cannabis use		Tried to kill self		-.15**
Increased cannabis use		Hostility 1		.09**
Increased cannabis use		Thought disorganization		.10**
Increased cannabis use			Emotional Distress	.14***
Increased cannabis use			Purpose in Life	-.14***
Increased cannabis use			Self-Derogation	.16**
Increased cocaine use		Impaired relations		.14**
Increased cocaine use		Negative self-image		.06**
Increased cocaine use		Tried to kill self		.14**
Increased hard drug use		Negative affect		-.10**
Increased hard drug use		Impaired relations		.08*
Increased hard drug use		Hostility 1		-.07*
Increased hard drug use		Anxiety 2		.07*
Increased hard drug use			Psychoticism	.14**
Increased hard drug use			Disorganized Thinking	.12*

Table 5 (continued)

Predictors		Consequences		Standardized parameter estimate
Observed	Latent	Observed	Latent	
		Nondrug effects		
	Emotional Distress	Impaired relations		.13**
	Emotional Distress	Positive affect		-.25***
	Emotional Distress	PIL 2		-.08*
	Emotional Distress	Told someone kill self		.08*
	Emotional Distress	Anxiety 1		.07*
	Emotional Distress	Diligence		.15*
Self-derogation		Negative affect		.09**
Self-derogation		Impaired cognitions		.14***
Self-derogation		Impaired relations		.12**
Self-derogation		Hostility 2		.07*
Self-derogation		Hostility 3		.11**
Self-derogation			Psychoticism	.12*
Self-derogation			Self-Derogation	.07*
Self-derogation			Anxiety	.13*
Self-derogation			Disorganized Thinking	.09*
Self-acceptance		Magid 3		.08*
Self-acceptance			CES-D	-.13*
Self-acceptance		Negative affect		-.14**
Self-acceptance		Impaired cognitions		-.18***
Self-acceptance		Negative self-image		.10**
Self-acceptance		Think about killing self		-.07*
Self-acceptance			Hostility	-.20***
Self-acceptance		Hostility 3		.06*
Self-acceptance			Anxiety	-.31***
Self-acceptance			Disorganized Thinking	.13*
Self-acceptance		Thought disorganization		-.15***
Depression		Magid 2		.17***
Depression		Magid 3		.11**
Depression			CES-D	.15*
Depression		Negative affect		.21**
Depression		Impaired cognitions		.31***
Depression		Impaired relations		.14**
Depression		Negative self-image		.12**
Depression			Purpose in Life	-.13**
Depression		PIL 1		.13**
Depression			Hostility	.35***
Depression			Anxiety	.53***
Depression		Anxiety 2		.21***
Depression		Anxiety 3		.14**
Depression			Disorganized Thinking	.21**
Depression		Deliberateness		-.15**
Depression		Thought disorganization		.24***
Depression			Suicide Ideation	.10*
	Social Conformity	Hostility 2		-.07*
	Social Conformity	Diligence		.10*
Liberalness			Purpose in Life	-.07*
Liberalness		Deliberateness		-.08*
Law abidance			Self-Derogation	.11**
Law abidance		Tried to kill self		.09*

(table continues)

Table 5 (continued)

Predictors		Consequences		Standard- ized parameter estimate
Observed	Latent	Observed	Latent	
Nondrug effects (<i>continued</i>)				
Law abidance		Diligence		.10*
Religious commitment		Positive self-image		.07*
Religious commitment		Negative self-image		-.06*
Religious commitment		PIL 2		.07*
Religious commitment		Tried to kill self		.11**
	Social Support	Depression		-.05*
	Social Support	Negative affect		-.15***
	Social Support	Impaired cognitions		-.08*
	Social Support	Impaired relations		-.11*
Good relations with parents			Psychoticism	.12*
Good relations with parents			Purpose in Life	.07*
Good relations with parents		Impaired relations		-.12*
Good relations with parents		Life end with suicide		-.14**
Good relations with parents		Tried to kill self		-.16*
Good relations with parents		Deliberateness		.19**
Good relations with parents		Thought disorganization		-.18***
Good relations with family		Positive self-image		.08*
Good relations with family		Think about killing self		-.10**
Good relations with family		Life end with suicide		-.14**
Good relations with family		Tried to kill self		-.15**
Good relations with family		Diligence		-.10*
Good relations with adults		Positive self-image		.09*
Good relations with adults		Hostility 3		-.12**
Good relations with adults			Anxiety	-.09*
Good relations with peers		Magid 2		-.08*
Good relations with peers			Anxiety	-.08*
Good relations with peers		Think about killing self		-.07*
Good relations with peers		Life end with suicide		-.08*

Note. All observed predictors are from the residual of that variable. Magid = magic ideation; CES-D = Center for Epidemiologic Studies-Depression Scale; PIL = Purpose in Life.

* $p < .05$. ** $p < .01$. *** $p < .001$.

ment also reduced Purpose in Life, positive affect, and attempts to kill self. Increased cocaine use exacerbated impaired relations, negative self-image, and attempts to kill self. Finally, increased hard drug use had 6 significant nonstandard effects related to heightened Psychoticism, Disorganized Thinking, impaired relations, and Anxiety 2, and it also reduced negative affect and Hostility 1.

Other specific or nonstandard effects from adolescent predictors to adult mental health functioning were quite numerous and are presented in the third section of Table 5. For instance, Emotional Distress increased impaired relations, Anxiety 1, diligence, and "told someone kill self" and decreased positive affect and PIL 2. These results

further confirm our use of a general Emotional Distress construct to control for adolescent levels of subsequent mental health measures (Scheier & Newcomb, 1993). The fewest nondrug, specific effects of adolescent variables were for Social Conformity and its indicators. Both adolescent Emotional Distress and Social Support, in conjunction with their specific indicators, had numerous significant effects on adult mental health functioning. Among some of the larger effects was adolescent depression increasing both Hostility and Anxiety in adulthood. Social Support and various good relations with family and friends as a teenager ameliorated or reduced numerous types of mental health problems as an adult.

Discussion

Mental health and psychological functioning evolve over time in response to various internal and external forces. To test whether drug use affects mental health and psychological development, one must account for the effects of known or likely influences of these outcomes. In our final path model, changes in mental health due to drug use could not be attributed to spurious influences related to conventionality or traditionalism (Social Conformity); parental, peer, or family support (Social Support); or prior levels of Emotional Distress. All of these likely factors were included as baseline controls. If drug use and emotional distress are generated by the same underlying cause or vulnerability to psychopathology, this research design would reveal no unique, direct effects from earlier drug use to later mental health problems (e.g., Garber & Hollon, 1991; Newcomb & Bentler, 1988a). Although comorbidity between mental disorders and alcohol and drug abuse is quite high in both community and institutional samples (e.g., Regier et al., 1990), we found that drug use may precede or exacerbate mental health problems.

Drug Use Consequences

The Adolescent and Increased Polydrug Use constructs had unequal influences on adult mental health. Adolescent Polydrug Use had no significant effects on any adult constructs and only one specific effect on a measured variable in adulthood (Magid 1). In contrast, Increased Polydrug Use had five significant long-term negative effects on adult mental health. Those who increased their polydrug use from adolescence to young adulthood expressed greater Psychoticism and Suicide Ideation, as general latent constructs, and specifically more impaired relations, Anxiety 3, and less positive affect than when they were teenagers. At least in terms of Polydrug Use (reflecting a high level of drug involvement akin to drug abuse—Newcomb, 1992; Newcomb & Bentler, 1988b), substance use as a teenager had few long-term detrimental effects on mental health functioning. This is in contrast to the more numerous mental health adversities associated with increased drug involvement from adolescence into adulthood.

Use of specific substances also adversely influenced several aspects of mental health, although

many of these effects were small in magnitude. These effects were more prevalent for increases in specific substances but also included several specific adolescent drug use measures. In our discussion, we focus on effects of .10 or higher and give less attention to smaller, though significant, influences.

Cannabis use had the greatest number of unique effects on the outcome measures. Chronic marijuana consumption has been associated with an "amotivational syndrome," based on the belief that excessive drug consumption leads to emotional disenfranchisement (Baumrind & Moselle, 1985), perceived parental detachment (Brook, Gordon, Brook, & Brook, 1989), impairments in cognitive functioning (Ferraro, 1980), and deficits in academic performance (Mellinger, Somers, Davidson, & Manheimer, 1976). Others have failed to confirm that marijuana precipitates declines in mental health or intellectual functioning (e.g., Ginsberg & Greeley, 1978; Kandel, 1978). Kandel reviewed relevant research and found little evidence that teenage marijuana use preceded the problems associated with amotivation and concluded that many of these problems actually preceded marijuana use. Our data indicate that adolescent and, particularly, increased cannabis use had several long-term negative mental health consequences. Thus, the mental health consequences of cannabis use may be more pronounced subsequent to adolescence rather than during the teenage years.

Adolescent cigarette use was related to increased Magid 3 and decreased deliberateness and had four other smaller effects. Increased cigarette use was also related to decreased deliberateness and had two smaller adverse effects. Previously (Newcomb & Bentler, 1988b), we reported that cigarette use increased emotional distress and also had damaging effects on other areas of life functioning. This earlier study spanned 4 years (in contrast to the present 12-year study) and suggests that the negative psychosocial consequences of cigarette use occur over a relatively short period of time and may not persist over a greater length of time. The long-term major adversity of cigarette smoking may be largely physical.

Cocaine use has been associated with emotional deterioration and psychosocial problems in clinical (e.g., Chitwood, 1985; Kosten et al., 1988) and survey populations (Newcomb & Bentler, 1986,

1987, 1988a, 1988b). Kosten et al. reported more disturbance on a range of psychosocial outcomes among addicts who increased their cocaine use over a 2.5-year period while in treatment for opioid dependence. On the other hand, Newcomb and Bentler (1986) found no serious short-term consequences from cocaine use in their general population study, although teenagers were followed for only 1 year and the extent of their cocaine consumption was limited. Discussing their findings, Newcomb and Bentler (1986) reported, "cocaine use in this more or less normal adolescent sample, generated no significant increases or decreases in eight measures of emotional distress" (p. 272). Negative effects may take longer to emerge (e.g., Newcomb & Bentler, 1988a), as is typical with psychiatric disturbances (e.g., Lewinsohn, Duncan, Stanton, & Hautzinger, 1986). Contrary to these earlier findings from adolescence, we found four specific negative effects in adulthood associated with adolescent and increased cocaine use into young adulthood. Higher cocaine use during adolescence increased one indicator of Psychoticism in adulthood, whereas increased cocaine use exacerbated impaired relations, negative self-image, and attempts to kill self. Petronis, Samuels, Moscicki, and Anthony (1990) also found that cocaine use was associated with risk of making a suicide attempt.

Specific use of hard drugs during adolescence was associated with increased later negative self-image, one indicator of Psychoticism (Magid 2), thoughts about suicide, and reduced PIL 2; these generally reflect exacerbated personal alienation, self-dislike, and bizarre beliefs. The extreme legal sanctions, psychoactive potency, and social awkwardness associated with using hard drugs (their use is generally not condoned even by peer groups) may contribute to these adverse effects. Equally alarming, increased hard drug use was moderately associated with increased Psychoticism and Disorganized Thinking and had small, but significant, influences on increasing Anxiety 2 and impaired relationships and reducing hostility (only one indicator) and negative affect. We found similar effects of teenage hard drug use on suicidal ideation over a 4-year period (Newcomb & Bentler, 1988a), and Ward and Schuckit (1980) reported that polydrug users (including hard drugs) had self-destructive ideation. The various potent sympathomimetic, sedating, anesthetic, or euphoriant properties of

these drugs (i.e., hypnotics, stimulants, inhalants, and narcotics) may provide immediate, but temporary relief from psychic distress; however, when use is excessive or escalates over prolonged periods of time, hard drugs create several types of adverse mental health consequences.

There were both adverse and beneficial effects on adult mental health from specific early alcohol use and increased alcohol use, when other drug effects were controlled. The adverse consequences were related to indicators of Purpose in Life (lower), Anxiety (higher), and Suicide Ideation (higher).

Positive effects from alcohol use may be attributed to the fact that alcohol, for most of society, is legal and does not carry the social, moral, and legal ramifications associated with illicit drugs. Current laws prohibit alcohol use during adolescence; however, in general, use of alcohol receives fewer negative social sanctions and may be considered by many as a necessary social link in making the transition between adolescence and adulthood (e.g., Jessor, 1986). Also, alcohol is generally consumed in social situations that may reinforce positive expectancies in ways unlike other drugs, whose effects are more personal. Therefore, it is not too surprising that alcohol has some limited beneficial effects generally related to affect (reduced CES-D), social relations (reduced impaired relations), and self-perception (increased positive self-image). Similar beneficial effects of alcohol have been noted in our earlier research (Newcomb & Bentler, 1988a, 1988b) and other studies (e.g., Kandel et al., 1986).

Nondrug Relationships

Numerous developmental patterns unrelated to drugs were evident in the final SEM. Most of those effects related to adolescent Emotional Distress and its specific measured-variable indicators and reflect synergistic influences over time among various types of psychopathology or mental health. They also demonstrate how dysphoric affect and poor self-regard (Emotional Distress in adolescence) can have a generalized disruptive influence on many aspects of adult mental health (emotions, attitudes, and perceptions) and social (impaired relations) functioning. Emotional Distress also increased diligence, a finding that was unexpected. One possible interpretation is that heightened

diligence may be an attempt to cope with or control the chaos and helplessness of inner turmoil. In other words, more diligence may be one way to impose structure, order, and predictability on a life plagued with dissatisfaction and distress.

Various indicators of adolescent Social Conformity influenced several aspects of adult psychological functioning. Adherence to conventionality had both positive and negative effects on adult mental health status and may reflect the mixed blessing of following social norms (e.g., Shedler & Block, 1990). For instance, law abidance increased Self-Derogation and attempts to kill self (as did religious commitment). On the other hand, more adolescent Social Conformity and law abidance increased later diligence, and religious commitment enhanced self-image.

Finally, Social Support and many of the indicators of the Social Support construct enhanced emotional health and may be protective forces. These variables decreased Anxiety and various indicators of Suicide Ideation, reduced negative affect, and improved social relations. Like Brook et al. (1989), we speculate that early disenfranchisement from traditional socialization influences and supportive interpersonal connections may exacerbate feelings of alienation and poor self-regard during this critical developmental period and may result in both increases in drug use and increased dysphoria. The latter finding is reinforced by the higher levels of depression in adulthood for those youth reporting low Social Support during adolescence. The one inconsistent finding to this pattern was that good relations with parents in adolescence increased adult Psychoticism. It is possible that overinvolvement with parents during adolescence may be antithetical to the development tasks of this age, which are to separate and individuate from parents and establish one's own intimate support network (e.g., Havighurst, 1972). As many family theorists indicate (e.g., Bowen), overinvolvement with parents may lead to psychotic conditions, as noted in our findings.

Theoretical Implications

Our findings are especially important when we consider that adolescence is a period of life during which critical emotional, social, and psychological skills and competencies are acquired, tested, and refined (e.g., Erikson, 1968). These involve acquisi-

tion of adult social roles, leaving home, attending college, beginning independent living, employment, marriage, and parenthood, to name just a few. Failure to acquire and master these competencies can result in serious emotional deficits and psychopathology (Havighurst, 1972). Conceivably, drug-abusing or frequent drug-using teenagers may have insufficient or inadequate socialization experiences, fail to cope successfully with various age-related stressors and transitions, and may be alienated from institutions from which many coping skills can be acquired and learned (i.e., family and schools). These drug-using, disaffected youth may become depressed and blame themselves for their inadequacies, and some may believe that increased tension arising from self-doubt and persistent thoughts of failure and negative self-image can be assuaged through continued or increased drug involvement, in the hope of alleviating this emotional distress. Our findings suggest that disruption to developmental mechanisms through precocious, continued, or escalated drug use may cause long-term emotional harm.

Several theoretical explanations have been suggested to explain why drug-abusing or heavily drug-involved youth lack adequate coping skills. Baumrind and Moselle (1985) suggested that adolescent drug use creates an hiatus from normal developmental processes, leaving youth without the necessary skills and cognitive abilities to adequately meet life's challenges. Alternatively, Newcomb (1987; Newcomb & Bentler, 1988a, 1988b) suggested that drug-involved youth accelerate their development and pass over important developmental experiences needed to acquire critical life skills. One example from our research includes abandonment of socially valued educational pursuits (Weng, Newcomb, & Bentler, 1988). Our present data do not directly test these hypotheses, although they emphasize the importance of the later transitional years between adolescence and adulthood, such that drug abuse during this critical developmental period may disrupt normal development and create serious mental health consequences.

Several researchers have suggested that drug abuse does not cause mental illness, but rather, that drug abuse causes unconventional behavior (e.g., Kaplan, Johnson, & Bailey, 1988) that lead to detachment and alienation from important social institutions (i.e., school and family). Over time, such experiences may reduce feelings of purpose in

life, create social and interpersonal deficits, heighten anxiety and hostility, or even lead to thoughts about suicide. These resulting cognitions, behavior, and affect reflect deficits in emotional functioning (e.g., B. P. Dohrenwend, Shrout, Egri, & Mendelsohn, 1980). This hypothesis is consistent with D. P. Dohrenwend's (1961) drift theory, which suggests that mental illness is a cause of disorganized social living, ultimately promulgating a downward socioeconomic and functional spiral of the mentally ill. It is known from studies of clinical populations that drug-abusing lifestyles can incur severe economic hardships, medical complications, and often serious emotional difficulties that may further reinforce a downward drift (Khantzian & Schneider, 1986; Weiss, Mirin, Griffin, Michael, & Sollogub, 1986). As more treatment alternatives become available to these individuals, including pharmacologic modalities, it becomes increasingly important to refine and sharpen clinical distinctions between mental health problems that are primary and secondary substance abuse disorders (e.g., Meyer, 1986).

One must also pay attention to problems associated with dual diagnosis of substance abuse and other disorders (Ford et al., 1989; Khantzian & Treece, 1985; Regier et al., 1990; Rounsaville & Kleber, 1985). Intervention with drug abusers requires difficult decisions to be made regarding choice of treatment and problems associated with complicated and interrelated presenting symptoms. It is possible that the developmental trajectory of psychopathology—when not accompanied with drug abuse—departs from a similar emotional dysfunction with substance abuse (e.g., Khantzian & Schneider, 1986). In addition, poor social and cognitive skills of long-term drug abusers may hinder treatment and recovery and require direct remediation over a lengthy period. If early initiation and continued use of drugs impair mental and emotional functioning, as we have shown, this heightens the importance of prevention efforts to delay drug use onset and to provide alternative affective coping and social skills (e.g., Hawkins, Lishner, & Catalano, 1985).

Limitations

There are several limitations to this study. First, the data were based on self-report measures and their possible biases (e.g., Stacy, Widaman, Hays,

& DiMatteo, 1985). Others have noted that self-report measures of drug use are valid and have provided empirical tests to underscore these claims (e.g., Needle, Jou, & Su, 1989). Notwithstanding, error in measurement could be minimized through use of collateral and independent reports of drug use, although this information was not available for these data.

Second, although youth may confront a number of important developmental tasks during the transition from adolescence to young adulthood, negotiation of other life periods may also produce stress and disequilibrium that could motivate individuals to use or abuse drugs. Even though this study focused on one developmental period that represents a considerably long span of the life course, manifestation of the negative effects of drug abuse may not surface until even later in life, into older adulthood. Certainly, it would be valuable to reassess these adults further along their developmental course to determine whether the patterns we observed continue unabated into their later adult lives.

Third, and corollary to the previous limitation, the time lag in this study may underestimate the causal relationships between adolescent measures and subsequent adult psychological functioning, if such processes occur over a shorter period of time (e.g., Gollob & Reichardt, 1987). On the other hand, viewed from a life span perspective, our time span may have limited our ability to make generalizations regarding the effects of drug use on developmental processes to later and more extended periods of development.

Fourth, we did not assess psychiatric impairment with standard clinical diagnostic criteria (i.e., the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd. ed, rev.; American Psychiatric Association, 1987). Rather, we used standard and proven epidemiological assessment tools that have demonstrated validity and reliability in general community studies. Although standard diagnostic assessment is most appropriate with clinical populations (e.g. Ford et al., 1989), these categorical decision criteria are less appropriate and useful with community samples. They are impractical, have little utility with low base-rate pathology, and are cost-prohibitive. Most important, however, is that they are dichotomous and therefore are restricted to only one or a few diagnostic "hits" (dual or triple diagnoses at most) and do not capture the

true range of psychopathology that is subclinical in extent. Accordingly, the measures used in this study provided a broad, but still limited, assessment of psychopathology, as well as capturing the full range of disturbance in each type of psychopathology. A wider selection of assessments along with a broader definition of psychopathology may strengthen the effects observed between drug use and psychopathology.

Fifth, our assessments of drug use represented frequency of consumption and did not determine drug abuse, alcoholism, or other criteria of drug dependencies. The associations between use and abuse of drugs appeared to vary on the basis of the particular substance (Newcomb, 1992), and our present findings were restricted to frequency of drug use.

Sixth, because of the necessarily complex nature of our analyses, requiring inclusion and consideration of numerous influences to make the strongest causal inferences possible, we chose not to provide simple descriptive statistics regarding how much greater risk for a particular type of psychopathology is associated with a specific increment in drug use. Although these figures could be provided, they would be erroneous and misleading, because they could not reflect important confounds, other controls, and shared variance of critical constructs. Therefore, the public health significance of our various findings must be inferred from the size and significance of the standardized path estimates. In general, the effects were modest to moderate in magnitude.

Seventh, data for both men and women were analyzed together. As with other studies of survey data (e.g., Johnston, O'Malley, & Bachman, 1988), patterns of drug use may differ between sexes, although we did not find substantial differences in drug use patterns. Previous analyses on this sample conducted separately by sex have revealed few substantial gender differences in substantive, prospective results (Newcomb et al., 1988; Stein, Newcomb, & Bentler, 1987). Conducting analyses separately by sex would have produced too small of samples and compromised the robustness of our statistical techniques (e.g., Tanaka, 1987). Notwithstanding, collapsing data across sexes (and other demographic characteristics) may have masked potential gender differences in linkages between adolescent drug use and adult psychological functioning.

Finally, several other explanations for declines in psychological functioning due to drug use could not be directly explored. For example, drug use may alter neurophysiology and thereby impair neuropsychological functioning (e.g., Becker & Kaplan, 1986). Structural brain changes induced through chronic drug use and concomitant neuropsychological impairment may be synergistic and difficult to separate. Quite possibly, acute cognitive impairment resulting from early drug use may motivate heightened drug use to relieve distress or avoid the deficits. This may mask further cognitive impairment or facilitate misattribution of decrements in functioning to drug-induced behavioral changes. Various biochemical models of addiction need to be considered (e.g., cocaine and dopamine depletion), which suggest that drug abuse may precipitate neurochemical changes that alone may contribute to both acute and chronic neuropsychological impairment.

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