

Personality and Substance Use Disorders: A Prospective Study

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The personality systems of Cloninger (as measured by the Tridimensional Personality Questionnaire [TPQ]) and Eysenck (as measured by the Eysenck Personality Questionnaire [EPQ]) both have been linked to substance use and abuse. The current study examined the predictive utility of both systems for substance use disorder (SUD) diagnoses, both cross-sectionally and prospectively. Participants ($N = 489$ at baseline) completed the EPQ and TPQ and were assessed via structured diagnostic interview at baseline and 6 years later ($N = 457$ at follow-up). Both the EPQ and TPQ scales demonstrated bivariate cross-sectional and prospective associations with SUDs. Within each system, those dimensions marking a broad impulsive sensation-seeking or behavioral disinhibition trait were the best predictors prospectively, although the 2 systems were differentially sensitive to specific diagnoses. These relations remained significant even with autoregressivity, other concurrent SUD diagnoses, and multiple personality dimensions statistically controlled.

Personality traits continue to hold a central place in etiological theories of substance use disorders (SUDs; e.g., Caspi et al., 1997; Cloninger, 1987a; Galen, Henderson, & Whitman, 1997; Howard, Kivlahan, & Walker, 1997; Sher & Trull, 1994; Sher, Trull, Bartholow, & Vieth, 1999; Tarter, 1988; Wood, Vinson, & Sher, in press). However, relatively few systematic efforts have been made to predict clinically meaningful SUD diagnoses using multidimensional, validated systems of personality.

Over the past half century, a number of influential approaches have been developed for specifying the number and nature of domains of personality. From these, three dominant models have emerged: (a) the Big Five factor model (e.g., Costa & McCrae, 1992, 1995; Digman, 1990; Goldberg, 1982, 1990; John, 1990; Wiggins & Trapnell, 1997), (b) the Alternative Five factor model (e.g., Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993), and (c) the Big Three factor models (e.g., Buss & Plomin, 1984; Cloninger, 1987a, 1987b; H. J. Eysenck, 1947, 1967, 1981, 1990; H. J. Eysenck & Eysenck, 1975; S. B. G. Eysenck, Eysenck, & Barrett, 1985; Tellegen, 1985). All of these models have received empirical support and are considered to have strong potential for systematically organizing the findings on personality and sub-

stance abuse (e.g., see Martin & Sher, 1994; Sher & Trull, 1994). The current article focuses on two of the most prominent Big Three models—the personality systems of Cloninger (e.g., 1987a, 1987b) and Eysenck (e.g., H. J. Eysenck & Eysenck, 1975; S. B. G. Eysenck et al., 1985)—because both focus on the underlying neurobiological bases of personality that have implications for learning and psychopathology.

As it relates to substance abuse, Cloninger's model (e.g., Cloninger, 1987a, 1987b; Cloninger, Sigvardsson, Przybeck, & Svrakic, 1995; Cloninger, Svrakic, & Przybeck, 1993; Wills, Vaccaro, & McNamara, 1994) hypothesizes that brain systems of behavioral activation, behavioral inhibition, and behavioral maintenance will relate to heritable (i.e., genetic) dimensions of personality, labeled novelty seeking (NS), harm avoidance (HA), and reward dependence (RD). Table 1 displays the component traits related to these dimensions, which are assessed via the Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987c). In recent revisions to the theory (e.g., Cloninger et al., 1993), Cloninger proposed a fourth basic dimension labeled persistence (PS; originally subsumed under RD) and added three "character traits" assumed to develop in adulthood (see Cloninger & Svrakic, 1997). Cloninger proposed that specific patterns of extreme scores on the original three dimensions, coupled with environmental factors, relate to predictable brain-behavior relationships that predispose affected individuals toward alcohol dependence (see Cloninger, 1987a). A large number of studies have concluded that high NS consistently predicts alcohol and other substance abuse and problems (e.g., Battaglia, Przybeck, Bellodi, & Cloninger, 1996; Cloninger et al., 1995; Galen et al., 1997; Heath et al., 1997; Howard et al., 1997; Sher, Wood, Crews, & Vandiver, 1995; Wills et al., 1994). NS also is highly correlated with impulsive sensation seeking (e.g., Zuckerman & Cloninger, 1996), a measure that shows strong relationships with substance abuse (see Zuckerman, 1994). Some evidence also suggests a potentially important role for HA in predicting alcohol dependence (Sher et al., 1995).

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Table 1
*Personality Traits Included in Cloninger's and Eysenck's
 Big Three Personality Systems*

Trait	Description
Eysenck	
Neuroticism	Anxious, depressed, guilt feelings, tense irrational, shy, moody, emotional
Extraversion	Sociable, lively, active, assertive, sensation-seeking, carefree, dominant, surgent
Psychoticism	Aggressive, cold, egocentric, impersonal, impulsive, antisocial, creative, tough-minded
Cloninger	
Harm avoidance	Cautious, apprehensive, fatigable, inhibited, sensitive to punishment
Reward dependence	Ambitious, sympathetic, warm, industrious, sentimental, persistent, moody
Novelty seeking	Impulsive, excitable, exploratory, quick-tempered, fickle, extravagant, disinhibited

Eysenck's (e.g., H. J. Eysenck & Eysenck, 1975; S. B. G. Eysenck et al., 1985) model is composed of three broad dimensions, including introversion–extraversion (E), neuroticism (N), and psychoticism (P), assessed using the Eysenck Personality Questionnaire (EPQ; H. J. Eysenck & Eysenck, 1975) or the revised version (EPQ-R; H. J. Eysenck, 1988). Table 1 displays the traits related to these dimensions. In their study of personality dimensions, Zuckerman, Kuhlman, and Camac (1988) concluded that the EPQ was an excellent marker for the three-factor models they examined. Like Cloninger's (1987a) model, components of Eysenck's model also have been linked with substance abuse. Specifically, high scores on P and N have been associated with alcohol abuse (e.g., S. B. G. Eysenck & Eysenck, 1977; Heath et al., 1997; Kilbey, Downey, & Breslau, 1998), other substance use and abuse, or both (S. B. G. Eysenck & Eysenck, 1977; O'Boyle & Barratt, 1993; Rosenthal, Edwards, Ackerman, Knott, & Rosenthal, 1990; Zuckerman, 1993).

Although dimensions of both Cloninger's (e.g., 1987a, 1987b) and Eysenck's (e.g., H. J. Eysenck & Eysenck, 1975; S. B. G. Eysenck et al., 1985) systems have a hypothesized temperamental basis and both have been shown to relate to substance use and abuse, research indicates that the two systems are not simply alternative descriptions of the same dimensions of personality (Heath, Cloninger, & Martin, 1994; Sher et al., 1995; Zuckerman & Cloninger, 1996). Hence, it is useful to compare the two systems with respect to their correlations with SUDs.

Despite a multitude of studies of the personality correlates of alcohol and other substance use (see Sher et al., 1999), several shortcomings in this literature have left many unanswered questions. First, the majority of the studies in this area have investigated only concurrent (i.e., cross-sectional) substance involvement and have not examined prospective relations. Such study designs cannot resolve the issue of whether personality factors are antecedents to or consequences of problematic substance involvement (e.g., McGue, Slutske, & Iacono, 1999; Sher et al., 1999).

Prospective research designs allow the direct modeling of temporal relations. However, third-variable alternative explanations can still make the findings from many existing prospective studies difficult to interpret. For example, in some prospective studies (e.g., Krueger, Caspi, Moffitt, Silva, & McGee, 1996), researchers

have not modeled the influence of baseline SUD diagnoses on the relationship between personality measured at baseline and later SUDs. Failure to account for the influence of existing or previous SUDs (i.e., autoregressivity) can artificially inflate prospective relations between personality variables and SUDs (see Nathan, 1988). Accounting for the influence of previous substance abuse can provide stronger evidence of the etiologic relevance of specific personality variables.

On a related note, when modeling the relations between personality and specific substance abuse variables, it is important to examine the specificity of personality effects by statistically controlling for the influence of other concurrent substance abuse. That is, the personality correlates of alcohol abuse, for example, may be partially mediated by other drug or tobacco dependence. By statistically controlling for the effects of such other substance use and abuse, the relations between aspects of personality and specific substance abuse patterns become more clear (e.g., Chassin, Pitts, DeLucia, & Todd, 1999; McGue et al., 1999).

An additional limitation of most work to date has been the tendency to focus on a single SUD category, thus restricting the generalizability of findings. For example, in another recent prospective study, Caspi et al. (1997) examined the influence of personality on subsequent diagnosis of alcohol dependence but did not include other drugs and tobacco. This approach makes it difficult to distinguish whether correlates were specific to alcohol use disorders or reflected more generalized addictive propensities, a limitation noted by Caspi et al.

In addition, many studies in this literature (e.g., Galen et al., 1997; Rosenthal et al., 1990; Shedler & Block, 1990; Wills et al., 1994) have examined personality correlates of substance use or abuse, but not diagnoses of SUDs as assessed via structured diagnostic interview. Studies examining use or abuse may suggest dimensions of temperament that relate to the onset of alcohol or other drug involvement, but they do not directly speak to those aspects of personality that may foreshadow more serious and longer lasting problems with substance abuse. Moreover, among studies that have examined personality correlates of clinically relevant SUD diagnoses, many have used clinical samples (e.g., Battaglia et al., 1996; Schaefer, Sobieraj, & Hollyfield, 1987), some containing individuals with multiple diagnoses (e.g., Gallucci, 1997; Nixon & Parsons, 1990). As discussed elsewhere (Sher et al., 1999), the use of patients in treatment likely oversamples individuals with the most severe problems (i.e., those who have the most frequent or lengthy treatments; Cohen & Cohen, 1984) and often includes individuals with comorbid psychopathology. Furthermore, samples in residential treatment facilities may exhibit different personality characteristics (e.g., lower extraversion, higher neuroticism, or both) merely because of the treatment environment (H. J. Eysenck & Gudjonsson, 1989). When nonclinical samples are used, they frequently are based on convenience or are not systematically ascertained, thus limiting their generalizability.

A final limitation in this literature is that researchers have used a wide variety of constructs to measure personality or behavior tendencies, many of which do not represent broad descriptions of temperament dimensions. For example, researchers have used individual profiles from the MMPI (e.g., Gallucci, 1997; Jaffe & Archer, 1987), measures of sensation seeking and impulse expression (e.g., Ball, Carroll, Babor, & Rounsaville, 1995; also see

Brennan, Walfish, & AuBuchon, 1986), impulsivity (along with other dimensions) assessed via observer ratings of behavior (e.g., Harvey, Stokes, Lord, & Pogge, 1996; Mâsse & Tremblay, 1997), measures of passive-aggressive personality (e.g., Flett & Hewitt, 1995), measures of internalizing-externalizing behavior (e.g., Mezzich et al., 1993), measures of ego control and subjective distress (e.g., Shedler & Block, 1990), and measures of augmentation-reduction (e.g., see Ludwig, Caine, & Wikler, 1977), to name a few. Studies using measures based on multidimensional systems of personality may be better able to account for differences among individuals in that findings can be referenced to a well-mapped factor structure and related to the larger personality literature.

A major goal of this work was to examine the role of personality in predicting SUDs, using multidimensional personality systems and standardized clinically relevant diagnostic criteria. Another goal of the study was to model these relationships both cross-sectionally and prospectively (over 7 years) to test the long-term predictive utility of personality constructs. This approach is useful in that variables that predict both current (i.e., cross-sectional) and subsequent (i.e., prospective) substance use disorders arguably can be considered the most important or diagnostic personality predictors. Further, our prospective models were constructed to account for the influence of Year 1 (baseline) SUD diagnoses and sex in order to statistically control autoregressivity and gender differences. In addition, to test the specificity of personality effects on SUDs, we controlled for other concurrent SUD diagnoses in both our cross-sectional and prospective models.

Our review of the literature indicates that traits related to impulsivity-behavioral disinhibition are most strongly and consistently associated with substance use and abuse problems (e.g., Battaglia et al., 1996; Cloninger et al., 1995; Galen et al., 1997; Heath et al., 1997; Howard et al., 1997; Kilbey et al., 1998; O'Boyle & Barratt, 1993; Sher et al., 1995; Tarter, 1988; Wills et al., 1994; Zuckerman, 1993). As such, we anticipated that TPQ-NS and EPQ-P would emerge as the most consistent predictors of SUD diagnoses, both cross-sectionally and prospectively. The literature linking neuroticism-negative emotionality and substance abuse is somewhat less compelling (e.g., Sher & Trull, 1994; Sher et al., 1999), but still suggests a positive relationship. Therefore, we expected both TPQ-HA and EPQ-N to be positively related to SUDs. Extraversion-sociability has been less consistently related to substance use and abuse (see Sher et al., 1999). Hence, no specific hypotheses were made concerning the utility of EPQ-E in predicting SUDs. Finally, Cloninger proposed that depending on alcoholism typology (Cloninger, 1987a), reward dependence may exhibit either a positive or a negative association with substance abuse. Because of the young age of our sample (i.e., most relevant for early onset of problems), we anticipated that RD would be negatively related to SUDs.

Method

Participants and Procedure

Baseline Screening

An extended description of participant ascertainment and recruitment is provided in Sher, Walitzer, Wood, and Brent (1991) and is briefly reviewed here. All incoming, first-time freshman ($N = 3,944$) at a large Midwestern

university were contacted as potential participants in a research study. Approximately 80% ($N = 3,156$) agreed to take part, and those students were screened for the presence of alcoholism in biological parents using versions of the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975) adapted for assessing alcoholism in biological fathers (F-SMAST) and biological mothers (M-SMAST; Crews & Sher, 1992). Approximately 26% ($n = 808$) of those screened were tentatively classified as either family history positive (FH+) or family history negative (FH-) on the basis of their adapted SMAST scores (the remainder had SMAST scores that did not clearly identify them as either FH+ or FH-, and they were not assessed further). Attempts were then made to administer portions of the Family History-Research Diagnostic Criteria interview (FH-RDC; Endicott, Andreasen, & Spitzer, 1978) to all potential FH+s ($n = 373$); interviews were completed with 97% of them ($n = 362$). A random sample of FH-s also were targeted for FH-RDC interviews ($n = 435$), and interviews were completed with 95% of them ($n = 413$). Participants whose biological fathers met both F-SMAST and FH-RDC criteria for alcoholism were classified as FH+s, and participants whose first-degree relatives did not meet either F-SMAST or FH-RDC for alcoholism, drug abuse, or antisocial personality disorder, and whose second-degree relatives did not meet FH-RDC criteria for alcohol or drug abuse were classified as FH-s. Participants whose biological mothers but not fathers were alcoholic were not retained for further study ($n = 20$), because of a very low base rate. Participants also were excluded because of inconsistency between adapted SMAST scores and FH-RDC interviews ($n = 154$) and because of concern for possible SUD and antisocial personality disorder in control relatives of our FH- participants ($n = 33$). The sample targeted for further study ($n = 489$) was composed of roughly equal numbers of male and female FH+ and FH- participants (n s ranging from 113 to 134). The mean age of this sample (at screening) was 18.2 years, and 94% of participants were White.

Present Study Sample

Participants were assessed at baseline (Year 1), at three subsequent yearly intervals (Years 2, 3, and 4), and again 3 years later at Year 7. (At baseline, a neuropsychological test battery also was administered.) At each assessment, a trained interviewer who was unaware of participants' family history status administered several sections of the Diagnostic Interview Schedule (DIS). DIS Version III-A (DIS-III-A; Robins, Helzer, Croughan, Williams, & Spitzer, 1985) was used for assessment at baseline and Year 2, and the DIS-III-R (Robins, Helzer, Cottler, & Goldring, 1989) was used at Years 3, 4, and 7. All interviews were cross-edited by a second independent interviewer (also unaware of participants' family history classification) and then reviewed by the interview supervisor. Participants whose interview data were deemed incomplete or unclear during the editing process were recontacted by telephone for further information. In addition to the DIS at each year, participants completed a questionnaire battery containing measures of personality traits, and alcohol, tobacco, and drug consumption patterns, among other measures. For each annual assessment in which they took part, participants received either course credit (if enrolled in introductory psychology), or were paid \$25 (at Years 1-4) or \$75 (at Year 7), plus additional stipends for travel to the testing location. The mean age of the sample at Year 7 was 24.5 years.

Although efforts were made to assess all participants from the initial baseline sample ($N = 489$) at each year of the study, not all participants were retained. By Year 7, individuals who refused further participation ($n = 29$), whom we were unable to locate ($n = 2$), or who were deceased ($n = 1$) were no longer in the data set. The remaining sample size at Year 7, therefore, was 457 (93% of participants targeted for follow-up). Although attempts were made to complete all assessments in person, it was not possible to do so in all cases, primarily because of participants' relocation out of the area. These participants were mailed the interview package and completed the interview by telephone. By Year 7, we assessed 27% of participants in this way.

Measures

Personality

Tridimensional Personality Questionnaire (TPQ). The 98-item TPQ was developed by Cloninger (1987c) to measure three basic personality dimensions—novelty seeking (TPQ-NS), harm avoidance (TPQ-HA), and reward dependence (TPQ-RD)—hypothesized to be related to alcoholism and personality disorders. For the current analyses, the TPQ was partitioned into four scales: TPQ-NS, TPQ-HA, persistence (TPQ-PS; originally Subscale 2 of TPQ-RD), and social sensitivity (TPQ-SS; composed of the original TPQ-RD subscales except PS). Previous studies in which the factor structure of the TPQ has been discussed (e.g., Cloninger, Przybeck, & Svrakic, 1991; Heath et al., 1994; Sher et al., 1995) indicate that the persistence subscale of the Reward Dependence scale does not load on any of the factors associated with the other TPQ subscales and, as such, should be considered a separate factor. Coefficient alphas for the TPQ scales were .85 for TPQ-HA, .80 for TPQ-NS, .75 for TPQ-SS, and .60 for TPQ-PS. The TPQ was administered only at baseline.

Eysenck Personality Questionnaire (EPQ). The EPQ (H. J. Eysenck & Eysenck, 1975) consists of 90 items designed to assess the personality traits of extraversion (EPQ-E), neuroticism (EPQ-N), and psychoticism (EPQ-P). A Lie scale also is included in the instrument to measure dissimulation. In previous research, the temporal stability of the EPQ over 1 month has been good, with reliability coefficients ranging from .83 to .90. The EPQ was administered at each wave of data collection, but the current report focuses only on EPQ data collected at baseline.

Responses to one item from the EPQ-P scale ("Would you take drugs which may have strange or dangerous effects?") were not considered during scoring because this question inquires directly about drug use. Its inclusion in the scoring could artificially inflate the magnitude of the relationship between the EPQ-P scale score and drug and alcohol diagnoses due to criterion contamination (e.g., see Darkes, Greenbaum, & Goldman, 1998). In the current sample, coefficient alphas for the EPQ scales were .83 for EPQ-E, .63 for EPQ-P, and .85 for EPQ-N, consistent with previous work (e.g., H. J. Eysenck & Eysenck, 1975).

Substance Use Disorder Diagnoses

Diagnostic measures of alcohol-, drug- and tobacco-related difficulties were collected during the interview appointments at each year using the DIS. In order to maintain consistency across all years of data collection, *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM-III*; American Psychiatric Association, 1980) diagnostic criteria were used throughout.¹ For the purposes of the present analyses, three broad diagnostic categories of specific SUDs were examined at both baseline and Year 7 (scored for occurrence in the past 12 months): *DSM-III* alcohol use disorder (AUD; alcohol abuse or dependence), *DSM-III* drug use disorder (DUD; drug abuse or dependence), and *DSM-III* tobacco dependence (TD). Alcohol use disorder was partitioned into both broad-band diagnoses (AUD) and narrow-band diagnoses (alcohol dependence, or AD). In addition, a superordinate diagnosis of substance use disorder (SUD-any) was defined as the presence of an AUD, DUD, or TD. Table 2 shows the numbers of participants with or without each SUD diagnosis at Years 1 and 7 and includes stability coefficients (calculated as product-moment correlations).

Results

We present the results of our cross-sectional analyses first, followed by our prospective analyses. Each table in which the results of the hierarchical logistic regression analyses are displayed includes values of *c* for corresponding steps. The *c* index (which ranges from .5 to 1.0) assesses the relationship between actual

Table 2
Numbers of Participants Diagnosed at Year 1 and Year 7 as a Function Type of Substance Use Disorder

Temporal pattern	Diagnoses				
	SUD-any	AUD	AD	DUD	TD
Not diagnosed at either year	268 (58%)	322 (70%)	410 (90%)	407 (90%)	369 (81%)
Diagnosed at Year 1 only	61 (13%)	62 (14%)	20 (4%)	26 (5%)	17 (4%)
Diagnosed at Year 7 only	46 (10%)	27 (6%)	18 (4%)	17 (4%)	42 (9%)
Diagnosed at both Years 1 and 7	82 (18%)	46 (10%)	9 (2%)	7 (1%)	29 (6%)
Stability coefficient	$r = .44$	$r = .40$	$r = .28$	$r = .20$	$r = .44$

Note. Each column represents the total Year 7 sample ($N = 457$). Numbers in parentheses represent percentages of the total within columns. Diagnoses were made according to Diagnostic Interview Schedule and *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980) 12-month criteria. SUD-any = any substance use disorder; AUD = alcohol use disorder; AD = alcohol dependence; DUD = drug use disorder; TD = tobacco dependence.

diagnosis and predicted probability of diagnosis and represents an index of fit in logistic regression.² Although a number of other methods for assessing model fit are available, we present the *c* statistic because it may be thought of as corresponding to the area under a receiver operating characteristic (ROC) curve (Hanley & McNeil, 1982), which has been described as a useful tool for assessing diagnostic performance (e.g., Hsiao, Bartko, & Potter, 1989; Murphy et al., 1987; also see Trull & Sher, 1994).

At Year 1, two participants did not provide complete EPQ data, and two others did not provide complete TPQ data. As such, sample sizes for the baseline cross-sectional analyses ranged from 485 to 489, and sample sizes for prospective analyses ranged from 451 to 457.

Cross-Sectional Analyses at Baseline

Bivariate Associations

To examine the comparability of the two systems of personality description, we correlated the scales of the EPQ with those of the TPQ (see Table 3). The strongest associations appear to represent similar assessment of two global dimensions. First, consistent with existing data suggesting that novelty seeking and psychoticism are indicators of a broad impulsivity-disinhibition factor (e.g., Zuckerman & Cloninger, 1996), TPQ-NS and EPQ-P show a moderate, positive association. Second, TPQ-HA and EPQ-N are strongly

¹ Note that the *DSM-III* criteria for alcohol dependence requires evidence of physical dependence and are thus more stringent than the criteria used in the most recent versions of the manual (*DSM-III-R*, American Psychiatric Association, 1987; *DSM-IV*, American Psychiatric Association, 1994). In unpublished analyses using data from Years 3, 4, and 7, we have found that most cases of *DSM-III-R* dependence would be classified as abuse using *DIS-DSM-III* diagnoses.

² All logistic regression coefficients were produced using SAS Proc Logistic (SAS Institute, 1990), and all independent variables were standardized prior to analyses (Aiken & West, 1991).

Table 3
Cross-Sectional Bivariate Associations Between
EPQ and TPQ Scales

Scale	TPQ Scale				EPQ Scale		
	HA	NS	SS	PS	E	N	P
TPQ							
NS	-.15**	—					
SS	-.02	.07	—				
PS	-.13*	-.26**	.07	—			
EPQ							
E	-.49**	.30**	.25**	.15**	—		
N	.52**	.14*	-.06	.02	-.13*	—	
P	-.08	.34**	-.31**	-.11*	.00	.17**	—

Note. *N*s for correlations range from 487 to 489. TPQ = Tridimensional Personality Questionnaire; EPQ = Eysenck Personality Questionnaire. For TPQ scales, HA = Harm Avoidance, NS = Novelty Seeking, SS = Social Sensitivity, PS = Persistence (see text). For EPQ Scales, E = Extraversion, N = Neuroticism, P = Psychoticism.
* $p < .01$. ** $p < .001$.

associated, indicating that both may represent negative emotionality. However, the significant association between TPQ-HA and EPQ-E indicates that TPQ-HA is factorially complex when viewed from the perspective of the EPQ. Examination of other correlations in the matrix reveals several small to moderate associations between EPQ and TPQ scales indicating varying degrees of construct overlap.

Bivariate product-moment correlations between EPQ and TPQ scales and SUD diagnoses are presented in Table 4. As predicted, those dimensions most clearly related to impulsivity–disinhibition (i.e., EPQ-P and TPQ-NS) showed the strongest and most consistent associations with the diagnoses we examined. In addition, EPQ-N was consistently and positively related to all SUD diagnoses. It is interesting to note that although TPQ-HA and EPQ-N both appear to represent negative emotionality, and although the two scales were strongly associated in this sample (see Table 3), only EPQ-N showed any association with SUD diagnoses in this analysis. Also consistent with our hypotheses based on Cloninger's (1987a) early onset alcoholism typology, TPQ-PS and TPQ-SS were both negatively associated with SUD diagnoses. EPQ-E showed only a small but reliable association with AUD.

Regression Models Relating Personality Scales and SUD Diagnoses

In order to examine the unique effects of each scale in predicting each disorder, we constructed several hierarchical logistic regression models. Although we were not interested in sex as a predictor of SUDs, sex was included in the first step of each model as a covariate to control for its effects.³

Table 5 presents the results of two logistic regression analyses, in which SUD diagnoses were predicted from EPQ and TPQ scale main effects.⁴ For both systems, the addition of the personality constructs resulted in a significant increment in model fit ($p < .05$) for each of the SUDs examined. Further, EPQ-P and EPQ-N were positive cross-sectional predictors of all SUD diagnoses. In addition, EPQ-E was significantly related to AUD but not the narrow-band AD diagnosis. As shown in the bottom section of Table 5,

TPQ-NS emerged as a consistent, significant associate of each disorder. In addition, TPQ-HA was significantly related to the narrow-band AD diagnosis but not AUD. Also, the negative relationship between TPQ-SS and SUD-any suggests that individuals with a particularly low sensitivity for social approval may be more likely to obtain an SUD diagnosis.

As a more conservative strategy, we examined the specificity of personality trait effects on AUD, AD, DUD, and TD by including other concurrent SUD diagnoses as covariates (along with sex) in the first step of logistic regression analyses similar to those presented in Table 5.⁵ These analyses demonstrate the unique effects of EPQ and TPQ scale scores on specific SUD diagnoses over the effects of other SUD diagnoses. In predicting AUD, the effects of all three EPQ scales remained significant (standard estimates ranging from .25–.30, $ps < .05$), as did TPQ-NS (standard estimate = .40, $p < .05$), when controlling for the effects of DUD and TD. Similarly for AD, the effects of EPQ-P, EPQ-N, and TPQ-NS remained significant (standard estimates = .22, .25, and .26, respectively, $ps < .05$) while controlling for the effects of DUD and TD. On the other hand, in predicting DUD, the effects of EPQ-P, EPQ-N, and TPQ-NS all were reduced to nonsignificance (standard estimates = .13, .15, and .17, respectively) when the effects of AUD and TD were modeled. For TD, the effects of

³ In addition, no specific hypotheses involving family history of alcoholism were made in this study. Previous analyses using much of the same sample reported here (Sher et al., 1991, 1995) indicated that variations in adult temperament may mediate the effects of family history on substance use and abuse. Thus, family history was not included in our primary models so that the size of any personality–SUD relations could be better estimated. However, it is also possible that personality traits may moderate family history effects (Rogosch, Chassin, & Sher, 1990). Furthermore, family history was an important component of the sampling framework for this study, and as such its exclusion could have implications for our results. Therefore, we included family history main effects and interactions with personality scales in a separate set of regression models identical to those we report. The nature of our results was unchanged in these analyses. That is, we found no evidence of moderation by family history, and the personality–SUD relations in these analyses were essentially the same as those we report.

⁴ In both models presented in Table 5, a third step was included in which interactions of scales were considered. However, including interaction terms in the present data set did not lead to a significant change in chi-square for any of the diagnoses for either the EPQ or the TPQ analysis, indicating that the majority of the unique variance in diagnoses was accounted for by the main effect terms. As such, no interactions are presented in the table. The same is true for the analogous prospective analyses presented in Table 6. In all cases where interactions among scale scores were examined, main effect terms were centered prior to construction of cross-products (Aiken & West, 1991). In addition, quadratic cross-product terms were entered into all models containing interaction terms to control for potentially spurious moderator effects (Lubinski & Humphreys, 1990). Although a three-way interaction involving RD, HA, and NS related to substance use was reported by Wills et al. (1994) and may be implied in Cloninger's (1987b) theory, no such relationship was apparent in our data for any of the diagnoses we examined.

⁵ In examining the specificity of personality trait effects on DUD and TD, we covaried the broadband AUD diagnosis and not the narrow-band AD diagnosis because the former encompasses the latter. Similarly, in examining specific prediction of AUD, we did not control for AD, and vice versa.

Table 4

Bivariate Associations Between TPQ and EPQ Scale Scores and Substance Use Disorder Diagnoses, Cross-Sectionally and Prospectively

Year 1 personality	Year 1 diagnosis					Year 7 diagnosis				
	SUD-any	AUD	AD	DUD	TD	SUD-any	AUD	AD	DUD	TD
TPQ										
HA	.05	.01	.08	.02	.07	.05	.03	-.02	.02	.08
NS	.31**	.30**	.13**	.15**	.21**	.28*	.14*	.10*	.14*	.21*
SS	-.18**	-.15**	-.06	-.05	-.04	-.10*	-.10*	-.08	-.08	-.06
PS	-.19**	-.14**	-.06	-.08	-.13**	-.14*	-.07	-.07	-.10*	-.14*
EPQ										
N	.24**	.22**	.17**	.16**	.15**	.17*	.13*	.12*	.10*	.14*
E	.07	.12**	-.02	-.03	.05	.01	.05	.00	-.01	-.02
P	.31**	.34**	.19**	.17**	.16**	.25*	.23*	.25*	.16*	.06

Note. *N*s for cross-sectional correlations range from 487 to 489; *N*s for prospective correlations range from 451 to 457. Reported associations are point biserial coefficients. All diagnoses were made according to Diagnostic Interview Schedule and *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980) 12-month criteria. Tridimensional Personality Questionnaire (TPQ) scales: HA = Harm Avoidance; NS = Novelty Seeking; SS = Social Sensitivity; PS = Persistence. Eysenck Personality Questionnaire (EPQ) scales: N = Neuroticism; E = Extraversion; P = Psychoticism. Diagnoses: AUD = alcohol abuse, dependence, or both; AD = alcohol dependence; DUD = drug abuse, dependence, or both; TD = tobacco dependence; SUD-any = any substance use disorder.

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

EPQ-P and TPQ-NS remained significant (standard estimates = .16 and .30, respectively, $ps < .05$) when controlling for AUD and DUD, and although the size of the EPQ-N effect was comparable with that of EPQ-P, it did not reach statistical significance (standard estimate = .16, $p < .07$). Thus, in general it appears that EPQ-P, EPQ-N, and TPQ-NS are robust cross-sectional correlates of most SUD diagnoses even when controlling for potential comorbidity with other concurrent SUDs. However, associations with DUD were reduced when the effects of TD and AUD were simultaneously modeled.

Prospective Analyses: Baseline to Year 7

Bivariate Associations

Table 4 presents product-moment correlations between baseline EPQ and TPQ scales and Year 7 SUD diagnoses. As shown in the table, the pattern of significant prospective associations is highly similar to that found with the cross-sectional analyses at Year 1. In general, TPQ-NS, EPQ-P, and EPQ-N were all consistent and positive correlates of later SUD diagnoses, whereas TPQ-SS and TPQ-PS were negatively associated with diagnoses.

Predicting Year 7 SUD Diagnoses From Year 1 Personality

Next, we constructed a series of prospective, hierarchical regression models analogous to the cross-sectional models presented in Table 5. To control for autoregressivity in our outcome variables (see stability coefficients in Table 2), Year 1 diagnosis was entered into each model in the first step as a covariate, along with sex. The results of these analyses are presented in Table 6. As expected, receiving an SUD diagnosis at baseline consistently predicted diagnosing at Year 7. In addition, and consistent with the findings of nationally based epidemiological studies (e.g., Harford & Grant, 1994; Warner, Kessler, Hughes, Anthony, & Nelson, 1995), men

were significantly more likely to receive each diagnosis at Year 7 than were women.

Of greater interest in Table 6 is the prospective prediction of diagnoses by baseline EPQ and TPQ scales. Similar to the cross-sectional analyses, EPQ-P and TPQ-NS emerged as the most important scales in predicting later substance abuse problems. Specifically, high baseline scores on EPQ-P were predictive of later alcohol dependence, whereas high baseline scores on TPQ-NS predicted later drug use disorder and tobacco dependence. No other personality scales emerged as significant prospective predictors of SUD diagnoses in these analyses where baseline diagnosis was statistically controlled.⁶

Next, as with our cross-sectional models, we examined the specificity of personality predictors of AUD, AD, DUD, and TD by including other concurrent SUD diagnoses as covariates (along with sex and baseline diagnoses) in the first step of logistic regression analyses similar to those presented in Table 6. EPQ-P remained a significant predictor of AD (standard estimate = .30, $p < .05$) when controlling for the effects of DUD and TD. In addition, TPQ-NS significantly predicted DUD (standard estimate = .29, $p < .05$) when controlling for AUD and TD, and it significantly predicted TD (standard estimate = .21, $p < .05$) when controlling for AUD and DUD. Hence, controlling for other concurrent SUD diagnoses had little impact on the prospective prediction of specific SUD diagnoses by EPQ-P and TPQ-NS, indicating that these scales are fairly robust in predicting later problems with alcohol, and other drugs and tobacco, respectively.

⁶ As a less stringent test of prospective prediction, we constructed an additional set of models in which baseline diagnoses were not included as covariates. In these models, EPQ-P, EPQ-N, and TPQ-NS all emerged as strong predictors of each diagnosis, and TPQ-HA was important in predicting TD and AUD. Hence, although neuroticism and harm avoidance do not appear to be important prospective predictors in our other models, researchers may wish to consider their influence as potentially important.

Table 5
 Baseline Cross-Sectional Logistic Regression Analyses Predicting Substance Use Disorder Diagnoses From EPQ and TPQ Scales Separately

Year 1 predictor	Year 1 diagnosis														
	SUD-any			AUD			AD			DUD			TD		
	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.
Model using EPQ scales															
Step 1: Covariate	18.3*	.60	-.21*	22.9*	.63	-.27*	.23	.52	.02	.10	.51	.03	.90	.54	.14
Sex															
Step 2: EPQ Scales	66.0*	.76	.30*	71.1*	.80	.33*	24.9*	.73	.29*	20.0*	.69	.24*	22.3*	.68	.24*
Psychoticism			.14*			.24*			-.02			-.04			.10
Extraversion			.31*			.32*			.31*			.26*			.22*
Neuroticism															
Model using TPQ scales															
Step 1: Covariate	18.3*	.60	-.21*	22.9*	.63	-.29*	.23	.52	-.03	.10	.51	.03	.90	.54	.12
Sex															
Step 2: TPQ Scales	72.9*	.76	.15*	57.1*	.77	.44*	16.2*	.72	.23*	20.4*	.70	.33*	31.7*	.73	.16
Harm avoidance			.43*			.44*			.35*			.33*			.39*
Novelty seeking			-.18*			-.12			-.14			-.14			-.14
Social sensitivity			-.11			-.06			.00			-.06			-.12
Persistence															

Note. All coefficients are taken from the second step in each analysis. The c statistic relates to model fit in logistic regression (see text). For EPQ analyses, Step 2 $\Delta\chi^2 df = 3$; for TPQ analyses, Step 2 $\Delta\chi^2 df = 4$. For both analyses, Step 1 $\chi^2 df = 1$. All diagnoses were made according to Diagnostic Interview Schedule and Diagnostic and Statistical Manual of Mental Disorders (3rd ed.; American Psychiatric Association, 1980) 12-month criteria. Sex was coded 1 = female, 0 = male. EPQ = Eysenck Personality Questionnaire; TPQ = Tridimensional Personality Questionnaire; AUD = alcohol abuse, dependence, or both; AD = alcohol dependence; DUD = drug use disorder; TD = tobacco dependence; SUD-any = any substance use disorder. Std. est. = standardized logistic regression coefficient.

* $p < .05$.

Table 6
Prospective Analyses Predicting Substance Use Disorder Diagnoses From EPQ and TPQ Scales

Year 1 predictor	Year 7 diagnosis															
	SUD-any			AUD			AD			DUD			TD			
	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	$\Delta\chi^2$	c	Std. est.	
Model using EPQ scales																
Step 1: Covariates	93.2*	.76		75.5*	.77		27.4*	.73		21.7*	.74		66.6*	.73		
Baseline diagnosis			.45*			.41*			.27*			.24*			.46*	
Sex			-.17*			-.28*			-.29*			-.40*			-.18*	
Step 2: EPQ Scales	8.0	.78		5.4	.79		15.0*	.84		5.7	.80		4.8	.75		
Psychoticism			.13*			.12			.33*			.15			-.07	
Extraversion			.00			.07			.09			.05			-.06	
Neuroticism			.10			.10			.14			.18			.14	
Model using TPQ scales																
Step 1: Covariates	93.2*	.76		75.5*	.77		27.4*	.73		21.7*	.74		66.6*	.73		
Baseline diagnosis			.42*			.44*			.31*			.20*			.42*	
Sex			-.22*			-.31*			-.30*			-.43*			-.17	
Step 2: TPQ Scales	19.9*	.79		2.9	.78		4.7	.76		13.6*	.81		14.2*	.79		
Harm avoidance			.10			.08			-.09			.14			.15	
Novelty seeking			.28*			.10			.15			.35*			.25*	
Social sensitivity			-.01			.01			-.08			-.07			-.01	
Persistence			-.05			-.03			-.15			-.16			-.09	

Note. All coefficients are taken from the second step in each analysis. The c statistic relates to model fit in logistic regression (see text). For EPQ analyses, Step 2 $\Delta\chi^2 df = 3$; for TPQ analyses, Step 2 $\Delta\chi^2 df = 4$. For both analyses, Step 1 $\chi^2 df = 2$. All diagnoses were made according to Diagnostic Interview Schedule and Diagnostic and Statistical Manual of Mental Disorders (3rd ed.; American Psychiatric Association, 1980) 12-month criteria. Sex was coded 1 = female, 2 = male. EPQ = Eysenck Personality Questionnaire; TPQ = Tridimensional Personality Questionnaire; AUD = alcohol abuse, dependence, or both; AD = alcohol dependence; DUD = drug use disorder; TD = tobacco dependence; SUD-any = any substance use disorder. Std. est. = standardized logistic regression coefficient.
* $p < .05$.

Discussion

The primary goal advanced for this study was to examine the nature of cross-sectional and prospective relations among well-defined systems of personality and interview-derived SUD diagnoses. Several important findings related to this goal emerged in our analyses. First, within each personality system, traits that relate most clearly to disinhibition or behavioral undercontrol (i.e., TPQ-NS and EPQ-P) were the most consistent predictors of SUDs, both cross-sectionally and prospectively. This finding is generally consistent with previous work linking antisociality–disinhibition with alcohol involvement, drug involvement, or both (e.g., Bates & Labouvie, 1995; Caspi et al., 1997; McGue et al., 1999; Schuckit, 1998; Sher et al., 1995). In the cross-sectional analyses using multiple predictors, both TPQ-NS and EPQ-P provided very robust prediction of all of the SUDs we examined. However, using a conservative approach, prospective prediction was very limited with both personality systems when baseline diagnoses were modeled. Nevertheless, individuals with high baseline scores on either TPQ-NS or EPQ-P were more likely than their lower scoring peers to later receive an SUD diagnosis. Even when the effects of other concurrent SUD diagnoses were statistically controlled, EPQ-P and TPQ-NS showed significant cross-sectional relations to all SUD diagnoses other than DUD. Furthermore, prospective models controlling for other concurrent SUDs similarly showed that EPQ-P remained a significant factor in predicting later AD, and TPQ-NS reliably predicted later problems with other drugs and tobacco.

In addition, traits related to negative emotionality were reliable correlates of SUD diagnoses cross-sectionally. Specifically, EPQ-N demonstrated significant small to moderate correlations as well as moderate logistic regression coefficients with each of the outcomes we examined. Controlling for the effects of other concurrent SUDs did not eliminate these effects for AUD and AD, a finding similar to those of McGue et al. (1999). TPQ-HA was a less important predictor overall and was primarily related to AD. Prospectively, scales assessing negative emotionality did not demonstrate robust prediction of SUD diagnoses when autoregressivity was controlled. However, TPQ-HA and EPQ-N did emerge as significant prospective predictors using less conservative models (see Footnote 5). Because controlling for autoregression eliminated prospective prediction from traits related to negative emotionality, the interpretation of these data is ambiguous. More specifically, our findings are consistent both with the perspective that negative emotionality is a consequence of SUDs and that negative emotionality is causally related to SUDs (but long-term effects are mediated by autoregression of diagnosis). More extensive statistical modeling of the association between alcohol use disorders and anxiety disorders (Kushner, Sher, & Erickson, 1998) also provides evidence consistent with both perspectives.

Extraversion, also identified in the literature as a potentially important correlate of SUDs (e.g., see Sher et al., 1999), was a reliable cross-sectional predictor of AUD and a weak but reliable predictor of the superordinate SUD-any category. However, it did not relate to any SUD diagnoses prospectively. Thus, we must conclude that the support for an Extraversion–SUD link is weak at best and most implicated with respect to (broadband) alcohol use disorders. Presumably, highly sociable individuals might be at high risk for developing drinking problems primarily because they

seek out situations where alcohol consumption is embedded in the social context. Perhaps one reason why no prospective effect of EPQ-E was found in this study is because the social context of drinking changes dramatically between the freshman year in college and six years later (see Sher, Bartholow, & Nanda, *in press*).

The EPQ and TPQ appeared to be differentially sensitive to specific diagnoses in our data. In all of the prospective models we reported, the personality system assessed by the EPQ, and specifically the dimension of personality measured by the P scale, added significantly to the prediction of a diagnosis of alcohol dependence even when the variance from several other factors (i.e., baseline diagnosis, concurrent SUD diagnosis, sex) was modeled. However, Eysenck's (e.g., H. J. Eysenck & Eysenck, 1975; S. B. G. Eysenck et al., 1985) system does not appear to make any unique contribution in prospectively predicting other diagnoses, such as tobacco and drug abuse or dependence.

On the other hand, the findings from all of our prospective models indicate that Cloninger's (1987a, 1987b) system of personality contains unique and important predictors of both tobacco and drug abuse and dependence, but does not appear to reliably predict alcohol abuse or dependence over 7 years. Specifically, it appears that the dimension of personality tapped by the NS scale should be considered an extremely important personality factor in determining which late adolescents or young adults may be at risk for developing problems with tobacco and other drugs by the time they reach their mid-20s. This finding is entirely consistent with prior behavioral genetic research (Heath, Madden, Slutske, & Martin, 1995), which shows that novelty seeking, but not psychoticism, is an important personality predictor of smoking behavior among Australian twins.

These patterns of findings may speak to the issue of where the two systems are similar and where they are unique, as discussed by others (e.g., Heath et al., 1994; Sher et al., 1995; Zuckerman & Cloninger, 1996). Heath et al. argued that the two systems are not merely alternative descriptions of the same dimensions of personality. It is not yet obvious whether differences across the EPQ and TPQ represent gaps in the constructs used within each system or psychometric limitations of the individual scales.

Our hypothesis that traits related to behavioral undercontrol are most relevant for predicting addictive phenomena was supported by the data linking NS and P to later SUDs. That these results were still obtained after controlling for baseline diagnoses provides strong evidence against the hypothesis that these personality–SUD relations are spurious. However, the exact meaning of these findings requires further clarification.

First, there appears to be noticeable inconsistency across forms of SUD, which may be attributable to important differences in the constructs assessed by NS and P. Some recent evidence bears on these potential differences. For example, although in our previous conjoint factor analysis (Sher et al., 1995) we found that P loaded strongly on an NS factor, substantial cross-loadings also were evident with TPQ-SS. This finding is consistent with the Big Five factor interpretation of P (e.g., Costa & McCrae, 1995), which suggests both an agreeableness and a conscientiousness component. In contrast, NS appears to be more reflective of impulsivity and sensation seeking (Zuckerman & Cloninger, 1996). Furthermore, behavioral genetic research indicates that NS may be a more heritable dimension than P (Heath et al., 1994; Zuckerman, 1994) and that the underlying coherence of P is not genetically based

(Heath & Martin, 1990). Thus, it is not surprising that although NS and P were moderately correlated in the present study, the correlation is far from unity, and the patterns of correlations with external criteria differ.

However, even if seeming differences in the SUD correlates of NS and P could be detailed, the etiological significance of obtained personality correlates must be examined in the context of specific motivational processes. As we have discussed elsewhere (Sher et al., 1999; Sher & Trull, 1994), personality constructs are probably best viewed as quite distal to drug use and abuse, and several alternative models relating behavioral undercontrol can be considered. For example, the psychobiological underpinnings of behavioral control could conceivably represent a vulnerability to the disinhibiting (McDougall, 1929), hypnotic (H. J. Eysenck, 1957), or stress-reducing (Sher, 1987) effects of sedative drugs. Alternatively, facets of behavioral undercontrol such as sensation seeking could relate to reward seeking (Cooper, Frone, Russell, & Mudar, 1995) and consequent use of drugs to enhance experience. Evaluation of specific mechanisms mediating personality effects is beyond the scope of the current article. Nevertheless, it is important to emphasize that correlations between traits and behaviors represent mere associations and do not provide an accounting of etiological process. It may be best to direct research efforts toward placing aspects of temperament within the context of larger, more comprehensive psychosocial models (see Cooper et al., 1995; Sher, 1991; Sher et al., 1999).

The use of a large, mixed-gender sample, multidimensional personality systems, structured diagnostic interviews, and lengthy follow-up intervals are important strengths of this research. Moreover, by controlling for baseline associations, other concurrent diagnoses, and sex differences, we were able to provide a more stringent test of the etiologic relevance of temperament than has typically been examined. Nonetheless, some limitations of the present study should be noted.

First, although the sample was systematically ascertained using a known sampling frame (all first-time freshmen at a large university), college enrollment requires a degree of academic success in secondary school and, consequently, certain aspects of educational achievement and its correlates (e.g., conduct problems, lower intelligence) may be underrepresented in this sample. Also, all forms of SUD are relatively prevalent in young adulthood, suggesting that perhaps substance involvement is more related to developmental and social factors associated with this stage of life as opposed to stable individual differences. Thus it is possible that stronger or even different personality correlates would be evident in older samples. Further follow-up of the sample will provide additional opportunities to observe possible changes in SUD–personality relations. It also should be noted that because the age of onset of many substance-related problems may predate college enrollment (Warner et al., 1995), and given the high prevalence of SUD diagnoses in our baseline data, the prospective personality–SUD relations we report may indicate both persistence of diagnoses and onset of new diagnoses, rather than purely onset. Finally, all of the data reported in this article are based on self-reports and the diagnostic data derived from structured interviews. Consequently, various self-report biases could influence the levels of both predictor and criterion variables and represent a “third variable” confound affecting the magnitude of personality–SUD correlations. However, any such effects should be minimized in all

prospective analyses, because the bias would need to be maintained over 6 years. When baseline diagnoses are statistically controlled, effects of such a confound should be effectively eliminated. It is for these reasons that we have greatest confidence in our prospective analyses that control for baseline diagnosis. However, such highly conservative prospective analyses can fail to detect genuine causal effects, and care must be taken not to equate negative findings in these analyses with the lack of an effect, especially because less conservative cross-sectional and prospective (excluding baseline control) models do show patterns of hypothesized relations.

Despite potential limitations, the current study represents an important advance in the search for personality correlates and predictors of SUDs. Several weaknesses in the extant literature have been addressed in this study, and our findings suggest that the systems of Cloninger (e.g., 1987a, 1987b) and Eysenck (e.g., H. J. Eysenck & Eysenck, 1975; S. B. G. Eysenck et al., 1985) provide unique prediction of problems with alcohol and other drugs. These results provide further evidence of the etiologic relevance of traits related to behavioral undercontrol for SUDs. Future research could profitably extend these findings to other developmental periods and further explicate the potential mediational processes by which personality dimensions influence SUDs.

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