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Substance use disorder (SUD) morbidity versus number of parents with SUD

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Abstract

Objectives: To assess the association between numbers of parents (i.e., 0, 1 or 2) with Substance Use Disorder (SUD) and proband's SUD severity and morbidity.
Design: Descriptive, cross-sectional.
Settings: Alcohol–drug treatment programs in two university medical centers.
Subjects: 597 voluntary patients aged 18 and older with SUD; adoptees excluded.
Results: On univariate analysis, parental SUD was associated with ten characteristics. On logistic regression analysis, having any parental SUD was associated with lower socioeconomic status, younger age at using tobacco, more severity on M-SAPS, and lower psychosocial function in the last year (Axis 5) as threshold effects. Logistic regression analysis comparing 1 versus 2 parents with SUD showed that those with 2 SUD parents began using alcohol at an earlier age as compared with having 1 SUD parent; this was an additive effect.
Conclusions: Parental SUD affects the proband's SUD severity in a threshold fashion.

Keywords: Family; Substance use disorder; Severity; Treatment history

1. Introduction

A positive family history is one of the most significant risk factors associated with Substance Use Disorders (SUD) (Bierut et al., 1998; Meller, Rinehart, Cadoret, & Troughton, 1988; Rounsaville et al., 1991). Merikangas et al. reported an 8-fold increased risk of SUD in relatives of probands with SUD (Merikangas et al., 1998), while the Collaborative Study on the Genetics of Alcoholism showed a 2-fold increased risk of alcohol dependence in relatives of probands with alcohol dependence (Nurnberger et al.,

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2004). Twin studies (Gynther, Carey, Gottesman, & Vogler, 1995; Kendler, Jacobson, Prescott, & Neale, 2003; Tsuang et al., 1996, 1998) and adoption studies (Cadoret, Troughton, O'Gorman, & Heywood, 1986; Cadoret, Yates, Troughton, Woodworth, & Stewart, 1996) have demonstrated that SUD is correlated with genetic and environmental factors. A large twin study demonstrated that only SUD had substantial disorder-specific genetic risk among seven psychiatric disorders (Kendler, Prescott, Myers, & Neale, 2003). Genetic factors have been seen as accounting for 31% to 34% of the variance in the clinical features of SUD (Tsuang et al., 1996, 1998).

Several studies have also indicated a relationship between family history and certain dimensions of addiction severity. For example, alcoholics with positive family history tend to have had earlier onset of alcoholism, more severe alcohol-related physical symptoms, more academic, social, and employment problems, more antisocial personality disorder, and more psychopathology (Conway, Swendsen, & Merikangas, 2003; Cook & Winokur, 1985; Frances, Timm, & Bucky, 1980; McKenna & Pickens, 1981; Schuckit, 1984; Templer, Ruff, & Ayers, 1974). In two studies, patients with two alcoholic parents have manifested more problems and more alcohol-related symptoms (Schuckit, 1984; Stabenau, 1984). Probands with two affected parents had higher alcohol abuse scores on the Michigan Alcohol Screening Test (MAST) and higher Addiction Severity Index (ASI) scores on drug, alcohol, family, and psychiatric scales than those with no parental history (Boyd, Plemons, Schwartz, Johnson, & Pickens, 1999), but no significant differences on the medical, employment, and legal scales of the ASI.

Studies of patients in methadone maintenance programs have provided similar evidence. Pickens et al. reported that opioid addicts with positive family histories for substance problems had earlier onset of heroin use and more opioid dependence symptoms, but no differences on duration of heroin use, onset of heroin dependence, and total number of dependence diagnoses (Pickens et al., 2001). Another study observed that more asocial behavior and medical problems accompanied familial SUD density (Coviello, Alterman, Cacciola, Rutherford, & Zanis, 2004).

Methods for ascertaining parental SUD have varied. In the study by Boyd and coworkers, parental substance use depended on the query, "State whether your mother/father had a drug or alcohol problem" (Boyd et al., 1999). In the Pickens and colleagues study, family history was defined as positive if either parent had "substance problems" (Pickens et al., 2001). Coviello et al. classified family history within three risk groups, i.e., high risk, medium risk, and low risk, based on subjects' self-report of their relatives' substance use (Coviello et al., 2004). In the Irish study of Alcohol Dependence on family history validity, probands provided family data that were highly consistent with multiple informants (Prescott et al., 2005).

The purpose of this study is to extend our knowledge regarding the relationship between addiction severity and the number of biological parents with SUD. In particular, areas not addressed in earlier studies have been emphasized (i.e., periods of abstinence, treatment history, psychosocial function). In order to relate our findings to earlier studies, data on course and symptom severity were undertaken. The subjects were classified into three groups according to the number of parents with SUD (0, 1, or 2), using a structured interview. Based on published data, our hypotheses were as follows:

No parental effect: Number of parents with SUD is not associated with proband's demographic characteristics (i.e., age, gender, education, marital and employment status, socioeconomic status).

Threshold parental effect: Having any parent with SUD is associated with course, severity, and treatment of SUD, but number of parents with SUD does not affect course or treatment.

Additive parental effect: The number of parents with SUD (0, 1, 2) is associated with some types of SUD severity on symptom severity measures of SUD.

2. Method

2.1. Procedure for determining parental SUD

Patients who had been adopted or were under age 18 were not included in this analysis. Trained research associates (RAs) at the master's level (MSW, MNS, MS) obtained data regarding the parents' substance use history. Using a decision tree, the RAs tabulated a parent as having an SUD (using DSM-III-R criteria (American Psychiatric Association, 1987)) if any one of the following characteristics obtained: (1) the parent received treatment for SUD; (2) the parent died of an SUD-related disease (e.g., alcoholic cirrhosis, drug overdose); and/or (3) the parent met DSM criteria for substance use disorder.

In cases of uncertain classification, the research associate consulted with an addiction psychiatrist. If they could not come to a consensus decision, the entire team discussed the matter, which was then decided by consensus.

We have used the Boyd convention as follows: P-0 = no parents with SUD, P-1 = one parent with SUD, and P-2 = two parents with SUD (Boyd et al., 1999).

2.2. Subjects

This clinical sample of 642 consecutive, voluntary patients had been referred with an alcohol and/or drug related problem. Adopted patients (n = 45) were excluded, leaving 597 study patients. Settings included two similar programs located within Departments of Psychiatry at two university medical centers. Referrals came from primary care clinicians, mental health clinicians, and substance abuse clinicians. About 90% were initially assessed as outpatients. Patients were sober and not manifesting withdrawal signs or symptoms at the time of evaluation. They represented a broad socioeconomic group, with recompense including private fee-for-service, referrals from Health Maintenance Organizations, Medicare-Medicaid, and Medical Assistance; each source of finance represented about 20% to 30% of the sample.

Substance use disorders (SUD) included substance abuse and substance dependence, with the exception of nicotine and caffeine abuse/dependence. Types of current 1-year SUD in this sample of 597 patients were as follows:

- any dependence diagnosis = 391 (65.5%); abuse diagnosis only = 206 (34.5%);
- alcohol abuse or dependence diagnoses = 451 (75.5%);
- abuse or dependence of other drugs: cannabis = 189 (31.7%), opiates = 76 (12.7%), cocaine = 66 (11.1%), amphetamine = 56 (9.4%), sedatives = 36 (6.0%), hallucinogens = 15 (2.5%).

2.3. Data collection

2.3.1. Patient-rated scales

Patients completed the self-rated Modified Michigan Alcohol–Drug Screening Test (MAST/AD) (Westermeyer, Yargic, & Thuras, 2004), based on Selzer's MAST (Selzer, 1971).

2.3.2. Research associate scales

The RAs routinely obtained the following information:

- *Demographic data*: Gender, current age, current employment, current marital status, years of education, and current socioeconomic status (using the Hollingshead and Redlich classification, with 1 = upper class to 5 = lower class (Redlich & Hollingshead, 1953)).
- *Parental loss during childhood*: The RA inquired about parental loss of mother and/or father during childhood, whether due to death, divorce, or separation, since parental substance abuse could be associated with greater likelihood of subsequent morbidity during adulthood.
- *Severity of substance disorder*: Measured by interview-based, clinician-rated data from the Minnesota Substance Abuse Problems Scale (M-SAPS) (Westermeyer, Nugent, & Crosby, 1998).
- *Substance related treatment*: Treatment facility type, number of admission, number of lifetime days in treatment, and attributed cost of treatment.

2.3.3. Psychiatrist-rated scales

Addiction psychiatrists made current psychiatric diagnoses using the Diagnostic and Statistical Manual of Mental Disorders, 3rd edition, revised or DSM-III-R (American Psychiatric Association, 1987). The psychiatrist then tabulated whether any of the SUD diagnoses involved substance abuse only versus any substance dependence. They also completed the Axis 5 Psychosocial Function scale for the last year from DSM-III-R.

2.3.4. Consent

Patients provided consent that this data be collected confidentially for research. Copies of the consent were provided to the patient, installed in the chart, and kept with the research file. The IRB at the respective study sites approved the study.

2.4. Statistical analyses

The Statistical Package for the Social Sciences (SPSS) was used for the analysis, using the current updated version. The first analysis consisted of all demographic and clinical characteristics, comparing patients with 0, 1, and 2 SUD parents. For univariate analysis, Chi Square tests were used for categorical data; Kruskal–Wallis for ordinal or skewed continuous data; and ANOVA for normally distributed continuous data. Level of statistical significance for these univariate comparisons was set at .01 due to the large number of subjects, so as to avoid the Meehl effect (Meehl, 1978) observed in large samples (i.e., small differences of little practical significance being statistically significant).

Two logistic regression analyses were performed to further assess threshold and additive effects. The first involved a comparison of no SUD parent versus any SUD parent; and the second involved 1 SUD parent vs. 2 SUD parents. The ten variables found to be statistically significant on the univariate analysis were entered into the regression analysis. A backwards stepwise logistic regression was used: all variables were entered in the initial step. At the subsequent step, the least statistically significant variable was removed until the removal criterion ($p > .1$) was no longer met. Level of statistical significance for these regression comparisons was set at .05. Although logistic stepwise procedures have been criticized for inflating Type I error (Hosmer & Lemshow, 2000), we feel that they are justified by the exploratory nature

of this analysis and counterbalanced to some degree by the more stringent criterion used in the univariate tests.

3. Findings

3.1. Univariate analysis 1: P-0 vs. P-1 vs. P-2 (see Table 1)

3.1.1. Numbers of patients by category

P-0 comprised the largest group, with 269 patients (45.1% of 597). P-1 occurred in 234 cases (39.2% of 597). P-2 occurred in 94 cases (15.7% of 597).

Among those in the P-1 group, the number of those with father-SUD was three times higher than the number of those with mother-SUD (175 w/father-SUD and 59 w/mother-SUD).

Among all of those who had a mother with SUD ($n=153$), 61.4% ($n=94$) also had a father with SUD. Among all of those who had a father with SUD ($n=269$), 34.9% ($n=94$) also had a mother with SUD. Compared to having a father with SUD, having a mother with SUD was more strongly associated with having two SUD parents. This difference was highly statistically significant at $p<.001$ ($\chi^2=70.292$, $df=1$).

Loss of a parent before age 18 (by divorce, separation, death, imprisonment, etc.) was frequent in all three groups, as follows:

- P-0 ($n=269$): 40 (14.9%) lost mother, 67 (24.9%) lost father;
- P-1 ($n=234$): 56 (23.9%) lost mother, 113 (48.3%) lost father;
- P-2 ($n=94$): 34 (36.2%) lost mother, 51 (54.3%) lost father.

Note the increased rate of parental loss with the progression from P-0 to P-1. Mother-SUD was highly associated with mother loss during childhood ($\chi^2=19.604$, $df=2$, $p<.001$). Likewise, father-SUD was highly associated with father loss during childhood ($\chi^2=40.235$, $df=2$, $p<.001$).

Having a parent with SUD was associated with parental loss to a statistically significant extent. For example, 35.3% (54/153) of the patients with mother-SUD experienced loss of mother, whereas only 17.1% (77/444) of the patients whose mother did not have SUD experienced loss of mother ($\chi^2=21.017$, $p<.001$). Similarly, 58.9% (136/269) of the patients with father-SUD experienced loss of father, whereas only 29.0% (95/328) whose fathers did not have SUD experienced loss of father ($\chi^2=28.149$, $p<.001$).

SUD in mothers also accompanied father loss (51.6% vs. 34.2%. $\chi^2=13.798$, $p<.001$). However, SUD in fathers did not attend mother loss (26.0% vs. 18.3%, $\chi^2=4.740$, $p=.03$).

3.1.2. Demography

Only the Hollingshead–Redlich socioeconomic status showed a statistically significant difference (see Table 1). The P-0 group had the highest current SES of 3.58, which fell between white collar/college educated and blue collar/skilled-artisan. The P-2 group had the lowest SES of 4.12 (between blue collar and unemployed-institutionalized). Sex, age, years of education, marital status, employment status, and residence were not statistically significant between the three groups.

Table 1
Number of SUD parents vs. demographic and clinical characteristics

Characteristics	Number of parents with SUD			Statistics
	None (P-0)	One (P-1)	Two (P-2)	
Number of patients	269	234	94	
Parent with SUD				
SUD Mother, $n=153$	0 (0%)	59 (25.2%)	94 (100%)	
SUD Father, $n=269$	0 (0%)	175 (74.8%)	94 (100%)	
Demography				
Sex				
Male	161 (59.9%)	135 (57.7%)	45 (47.9%)	$\chi^2=4.13$, $df=2$, $p=.13$
Female	108 (40.1%)	99 (42.3%)	49 (52.1%)	
Age	31.4 (12.4)	30.2 (9.5)	28.5 (8.1)	KW=1.46, $p=.48$
Education (in years)	12.7 (2.8)	12.6 (2.5)	12.2 (2.9)	$F=1.05$, $p=.35$
Marital status				$\chi^2=7.75$, $df=6$, $p=.26$
Single	152 (57.1%)	128 (55.4%)	50 (53.2%)	
Married	47 (17.7%)	31 (13.4%)	9 (9.6%)	
Divorced, separated	52 (19.5%)	55 (23.8%)	28 (29.8%)	
Widowed, other	15 (5.6%)	17 (7.4%)	7 (7.4%)	
Unknown	3	3	0	
Employment				$\chi^2=13.95$,
Full-time	54 (21.3%)	50 (21.8%)	13 (14.4%)	$df=8$, $p=.08$
Part-time, episodic	34 (13.4%)	31 (13.5%)	8 (8.9%)	
Homemaker, student	42 (16.6%)	19 (8.3%)	12 (13.3%)	
Retired, disabled	22 (8.7%)	25 (10.9%)	14 (15.6%)	
Unemployed	101 (39.9%)	104 (45.4%)	43 (47.8%)	
Unknown	16	5	4	
Residence				$\chi^2=8.74$,
Alone	59 (22.4%)	55 (24.1%)	24 (26.1%)	$df=10$, $p=.56$
With parents	63 (24.0%)	39 (17.1%)	17 (18.5%)	
With spouse	40 (15.2%)	28 (12.3%)	9 (9.8%)	
With friends	40 (15.2%)	35 (15.4%)	16 (17.4%)	
Institutional	19 (7.2%)	24 (10.5%)	10 (10.9%)	
None, other	42 (16.0%)	47 (20.6%)	16 (17.4%)	
Unknown	6	6	2	
Socioeconomic status	3.6 (1.0)	3.9 (0.9)	4.1 (0.8)	$F=12.44$, $p<.001$
Course of substance use				
Age onset alcohol use, $n=544$	15.6 (4.7)	14.8 (4.1)	13.6 (4.3)	KW=16.57, $p<.001$
Age onset tobacco use, $n=493$	16.3 (6.2)	15.3 (5.1)	13.5 (4.9)	KW=18.80, $p<.001$
Lifetime, alcohol, years $n=544$	13.3 (10.6)	13.6 (8.5)	12.7 (7.6)	KW=2.58, $p=.28$
Last year, alcohol, days $n=555$	131.4 (120.6)	132.7 (118.3)	122.3 (117.7)	$F=0.26$, $p=.78$
Number of drugs used, lifetime	6.3 (2.5)	7.0 (2.5)	7.4 (2.3)	$F=9.55$, $p<.001$
Longest abstinence in last years	53.2 days (78.7)	57.5 days (85.1)	58.7 days (81.0)	KW=0.41, $p=.82$
Longest abstinence, 5 years	8.5 months (11.8)	9.4 months (12.6)	7.6 months (11.8)	KW=1.99, $p=.37$
Longest abstinence, 10 years	18.8 months (27.7)	18.5 months (25.5)	15.3 months (24.3)	KW=1.27, $p=.53$
Use of self help methods	2.5 (2.1)	2.9 (2.3)	3.1 (2.3)	KW=5.65, $p=.06$

Table 1 (*continued*)

Characteristics	Number of parents with SUD			Statistics
	None (P-0)	One (P-1)	Two (P-2)	
Severity of SUD, rating scales and psychiatrists' assessments				
MAST/AD	25.6 (13.3)	29.1 (12.2.)	28.3 (12.6)	$F=4.31$, $p=.014$
M-SAPS – total score	25.9 (11.5)	30.9 (11.2)	31.8 (12.0)	$F=14.76$, $p<.001$
Axis 5 Psychosocial Function, last year	4.3 (1.1)	4.6 (1.1)	4.8 (1.0)	$F=7.32$, $p=.001$
Abuse vs. Dependence				
Abuse	109 (40.5%)	66 (28.2%)	31 (33.0%)	$\chi^2=8.51$,
Dependence	160 (59.5%)	168 (71.8%)	63 (67.0%)	$df=2$, $p=.014$
Lifetime treatment history				
Number of treatment, SRD facility types	1.6 (1.6)	2.1 (2.0)	2.1 (1.8)	KW=11.95, $p=.003$
Number of admissions, all SUD facilities	3.6 (7.3)	6.6 (14.5)	4.1 (6.4)	KW=12.62, $p=.002$
Number of days in SRD treatment	88.0 (180.9)	134.9 (226.8)	114.4 (196.3)	KW=11.81, $p=.003$
Cost of SRD treatment (attributed)	$21,576 (43,932)	$28,954 (46,141)	$24,015 (38,512)	KW=9.87, $p=.007$

3.1.3. SUD course: Age at onset of use, years of use, days of use in last year, longest abstinence, and attempts at self help

3.1.3.1. Age at first use. Number of parents with SUD was strongly and inversely associated with age at first beginning to use alcohol, the one substance used by virtually all patients (see Table 1, $p<.001$). Earlier age at tobacco use was also associated with parental SUD ($p<.001$). Among substances reported by only some of the patients (e.g., opioids, cocaine, amphetamine, cannabis), age at beginning use was not associated with parental SUD.

3.1.3.2. Lifetime number of substances used. A greater number of psychoactive substances were associated with parental SUD (see Table 1; $p<.001$).

3.1.3.3. Duration of use. Duration of lifetime use and number of days of use during the last year for alcohol did not show any difference in relation to parental SUD. We also analyzed other substances, which also did not show a difference.

3.1.3.4. Abstinence. Longest periods of abstinence in the last year, 5 years, and decade bore no relationship to parental SUD.

3.1.3.5. Self-help. The number of self-help methods used, out of a possible seven methods (i.e., change dose, change frequency, change substance, changes residence, change friends, change work/school, join self-help group) was also not associated with parental SUD.

3.1.4. SUD severity

These measures consisted of lifetime prevalence of substance-related phenomena, experiences, events, and symptoms.

3.1.4.1. Michigan Assessment/Screening Test – Alcohol/Drug (MAST/AD). This patient-rated scale showed the lowest severity among those who had no parents with SUD. Those with one SUD parent and two SUD parents scored very close to one another. The difference fell short of being statistically significant, but was close at $p=.014$ (see Table 1).

3.1.4.2. Minnesota Substance Abuse Problem Scale (M-SAPS) total score. This scale, based on a structured interview with a trained master-level clinician, showed a statistically significant difference ($p<.001$). Again, the greatest difference occurred between 0 and 1 parents (mean difference of 5.0), with a small mean difference between 1 and 2 parents (0.9) (see Table 1).

The following SUD-related problem subscales revealed the same pattern of findings, statistically significant at $p<.001$: Family ($F=16{,}869$); Interpersonal ($F=12.059$); Financial ($F=9.312$); and Occupational–Academic ($F=7.390$). A similar pattern occurred for the following SUD-related subscales at a lower probability level: Psychological Problems ($F=6.312$, $p=.002$) and Pharmacological Problems ($F=5.372$, $p=.005$). Only one scale failed to reach significance, i.e., SUD-related Legal Problems (KW: $\chi^2=6.490$, $p=.04$). (Note that the Kruskal–Wallis was used for Legal Problems due to skewed data, with most patients having few or no SUD-related legal problems.)

3.1.4.3. Psychosocial function. This variable was assessed using the DSM-III-R Axis 5 measure of psychosocial function, rated for the last year. The Axis 5 measure was significantly poorer in association with more parental SUD ($p=.001$).

3.1.4.4. Substance abuse vs. substance dependence. The P-1 and P-2 groups had a higher rates of substance dependence diagnoses as compared to the P-0 group. This classification, made by the addiction psychiatrist, was close to being statistically significant ($p=.014$). Again, the mean difference was greatest between P-0 and P-1 groups (12.3% higher rate of dependence). Between the P-1 and P-2 parent groups, the percentage of patients manifesting substance dependence actually declined slightly (by 4.8%).

3.1.5. SUD treatment history

Parental SUD was associated with care in more types of facilities ($p=.003$; see Table 1). Parental SUD was also associated with a greater number of separate admissions to all forms of SUD treatment ($p=.002$). Total lifetime days in SUD treatment was related to parental SUD ($p=.003$). Finally, total

Table 2
Logistic regression analysis comparison of P-0 vs. (P-1+P-2)

Characteristic	(*B*)	S.E.	Wald's	Odds ratio	95% CI	Significance
Socioeconomic status	.249	.129	3.732	1.283	.996–1.652	.053
Age at onset of tobacco use	−.048	.024	3.986	.953	.909–.999	.046
M-SAPS total score	.024	.01	6.189	1.025	1.005–1.044	.013
Axis 5 psychosocial function, last year	.268	.109	6.062	1.307	1.056–1.619	.014

Table 3
Logistic regression analysis comparison of P-1 vs. P-2

Characteristic	(*B*)	S.E.	Wald's	Odds ratio	95% CI	Significance
Age at onset of alcohol use	−.082	.039	4.533	.921	.854–.993	.033

lifetime cost of treatment, used attributed cost data, was associated with parental SUD ($p = .007$). Each of these treatment variables showed the greatest difference between P-0 and P-1, with somewhat less use of treatment among P-2 patients when compared to P-1 patients.

3.2. Logistic regression analyses

3.2.1. P-0 vs. (P-1 + P-2) (see Table 2)

The ten characteristics that were statistically significant from the original analysis above were analyzed in order to assess their relative contribution to differences between those with no parental SUD versus those with any parental SUD. This would enable us to identify variables that were threshold in nature. The ten characteristics included the following: socioeconomic status (SES), age at first alcohol use, age at first tobacco use, lifetime number of drugs used, Minnesota Substance Abuse Problems Scale, Axis 5 Psychosocial Function (previous year), and four lifetime SUD treatment variables (number of types, admissions, days, and cost). As shown in Table 2, this analysis identified four characteristics associated with any parental SUD: namely, lower current socioeconomic status, earlier age at first use of tobacco, higher score on the Minnesota-SAPS, and lower psychosocial coping over the last year (Axis 5).

3.2.2. P-1 vs. P-2 (see Table 3)

The ten characteristics that were statistically significant from the original analysis above were again analyzed in order to assess their relative contribution to differences between those with 1 SUD parent versus those with 2 SUD parents. This would enable us to identify variables that were additive in nature. As shown in Table 3, this analysis identified one characteristic associated with having an increasing number of parents with SUD, i.e., earlier age at first use of alcohol.

4. Discussion

4.1. No parental effects

Parental SUD showed "no effect" on 14 out of the following 24 variables studied. These "no SUD parental effect" variables showing no effect on univariate comparisons included the following:

- 6 out of 7 demographic variables: i.e., gender, age, education, marital status, employment, residence;
- 6 out of 9 variables related to course of SUD: i.e., years of alcohol use, days of alcohol use in the last year, 3 periods of abstinence, and use of self-help methods;
- 2 out of 4 variables related to SUD severity, i.e., MAST/A-D score and abuse vs. dependence (although the statistical analysis for both was close to meeting significance at $p = .014$).

Coviello et al. likewise found no difference in low vs. high familial "density" of substance abuse as related to gender, education, marital status, days worked in the last month, years of substance use, and days of substance use in the last month (Coviello et al., 2004). Coviello did find a small age difference, with "high familial" patients aged 39.2 on average vs. "low familial" mean age of 41.4 years ($p = .011$). However, the mean age difference observed by Coviello et al. was small, only 1.2 years.

Both Coviello and Boyd, and their coworkers, found no association between parental SUD and the ASI-Legal scale (Boyd et al., 1999). We also confirmed Boyd et al.'s observation regarding absence of legal problems in association with parental SUD. Our findings and those of the Coviello team agree in terms of three demographic characteristics (gender, education, marital status) and two aspects of clinical course (lifelong and recent alcohol use). Our data supplement the published data by showing that several additional variables manifested no association with parental SUD, i.e., residence, periods of abstinence, and use of self-help methods.

Explanations for absence of parental SUD effects on these demographic and course variables are not obvious from the data. It is likely that more than one factor accounts for the findings. On one hand, certain demographic risk factors (such as male gender, unmarried marital status) may be associated with SUD regardless of parental SUD. On the other hand, certain elements of course may ensue from SUD itself, again with no or minimal influence from parental SUD.

4.2. Threshold parental effects

We defined "threshold effect" as indicating that any parental SUD produces greater morbidity as compared to no parental SUD. Based on the logistic regression analysis, threshold effects associated with any parental SUD in our study included the following:

- demography: lower socioeconomic status;
- course: earlier age at first using tobacco;
- symptom severity: higher Minnesota Substance Abuse Problems Scale (M-SAPS) total score, lower DSM-III-R Axis 5 psychosocial coping over the previous year.

Boyd and coworkers found an association with parental SUD and the Michigan Alcoholism Screening Test (MAST) score ($p < .001$) (Boyd et al., 1999). Coviello also observed that parental SUD was associated with higher scores on the ASI Medical subscale ($p = .052$), and higher rates of alcohol dependence as determined by the SCID ($p = .001$) (Coviello et al., 2004). Our findings thus agree with the study of Boyd et al. in regard to rating scale severity.

Findings from these studies do support the notion that parental SUD affects the proband's SUD severity, in a "threshold" fashion. However, our findings were considerably stronger and more extensive than the findings of the other two teams. Potential reasons for these differences include the following:

- Differences could be due to sampling differences, as our sample included younger, more educated, and relatively more female patients, and a minority of opiate dependent patients. Coviello's sample consisted entirely of methadone patients.
- Our M-SAPS grounds all of its ratings in substance use only (Westermeyer et al., 1998), whereas the ASI does not require that substance use be related to some variables, such as psychiatric symptoms (McLellan et al., 1985). In addition, the M-SAPS is weighted more heavily towards

psychosocial function and relationships and less toward medical complications, as compared to the ASI.

4.3. Additive parental effects

"Additive" parental effect occurs when each addition of an SUD parent, from P-1 to P-2, produces progressively more morbid findings. On the logistic regression analysis, each additional parent had a statistically significant "additive effect" for only one variable related to substance use history:

– Younger age at beginning alcohol use.

Boyd et al. (1999), using parental data similar to our own, reported two Addiction Severity Index (ASI) subscales that would fit into an "additive" parental SUD effect. Coviello and colleagues (Coviello et al., 2004), using a more complex familial substance abuse scale, also reported data that would fit an "additive" model for the ASI Alcohol Subscale ($p=.04$) and days of use to intoxication within the last month ($p=.05$). These three studies concur that the "additive model" of parental SUD accounts for relatively little clinical manifestations. Perhaps clinical variables related to alcohol use are more apt to be "additive" in nature, whereas most characteristics related to parental SUD are threshold in nature.

4.4. Geometric parental effects

A few reports in the literature have indicated small severity increases in going from P-0 to P-1, with much greater severity increases going from P-1 to P-2, following a geometric rather than an additive model (i.e., P-2 ≫ P-1 > P-0). Boyd and coworkers observed this pattern for the Drug subscale of the Addiction Severity Index at $p=.005$ (Boyd et al., 1999). Boyd et al.'s sample, like ours, was obtained at a university-based clinical facility. However, their subjects were older, included fewer women and fewer Caucasians, with MAST scores notably lower than our MAST/AD scores (i.e., less than 50% as severe). Coviello and colleagues found a similar pattern in the Family-Social subscale of the Addiction Severity Index at $p=.053$ (Coviello et al., 2004). Their sample involved only opioid-dependent patients in methadone treatment, predominantly male, almost a decade older, and fewer Caucasians. A rationale for the "geometric" model might consist of greater effects from a combination of both "nature" liabilities (with genetic contributions from two parents with SUD) plus "nurture" disadvantages associated with having two SUD parents (e.g., greater risk to parental loss, fetal damage, neglect, and abuse). None of our variables manifested this phenomenon.

4.5. Comment

We have suggested four models from examining the association of parental SUD to proband SUD morbidity, i.e., no parental effect, threshold effect, additive effect, and geometric effect. Our findings plus those of others suggest that parental SUD effect does not affect, or minimally affects demographic characteristics. Threshold parental SUD effects occurred for one demographic characteristic (socio-economic status), one substance use characteristic (age at first use of tobacco), SUD symptom severity (as measured by M-SAPS), and coping in the last year (as measured by Axis 5). The additive model was infrequent in our sample, occurring with only one variable (age at onset of alcohol use).

4.6. Caveats

One limitation was the nature of the sample. A clinical sample such as this one may not represent all patients with SUD. Or it may represent those with more severe or treatment refractory SUD. A community-based sample of those with SUD would address this limitation.

A second limitation was our inability to distinguish between genetic, intrauterine, and environmental factors. To make this distinction, one would need to use other methods, such as monozygotic vs. dizygotic twins, or adopted vs. non-adopted children from the same sibship.

A third limitation was our not distinguishing between father-SUD and mother-SUD in the one-parent cases. Father-SUD can affect offspring through the effects of alcohol and other drugs on spermatogenesis (Cicero, 1994; Goodwin, 1985). Mother-SUD can affect offspring through intra-uterine exposure to psychoactive substances during pregnancy, as well as associated nutritional, infectious, or traumatic events that may be associated with SUD (Little & Wendt, 1991; Mills, Granbard, Harley, Rhoads, & Berendes, 1984; Streissguth, Barr, & Sampson, 1990). However, it is not likely that these substance-related contributions to progeny are necessarily equivalent (Kendler, Prescott et al., 2003). We plan to subsequently compare father-SUD and mother-SUD patients, but that comparison went beyond the bounds of the current study.

A fourth limitation was the use of parental SUD history alone, rather than other relatives. Use of siblings creates an analytic problem, since the number of siblings may vary from none to many. In addition, many siblings would not yet have lived through the years when a SUD is apt to have begun. Using modern methods of genetic data analysis, these confounds can be addressed. Use of grandparents introduces validity issues, since patients often know little about grandparents who may have died or left the family even before they were born.

A fifth limitation was the absence of nicotine diagnoses in the data set, although we did present data on age of first tobacco use (which was related to parental category). In addition, we have no data on parental use of nicotine use (a substance that can affect offspring when used by parents).

Unlike some familial studies, we relied primarily on the patients themselves for this information. Although family history data from patients is specific, it is not particularly sensitive (especially regarding mothers). Thus, these results are apt to be conservative, with an undercount of parental SUD.

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