

Risk Factors for Depressive Symptomatology in a Drug Using Population

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Abstract: This study employs a prospective design to examine possible personality, drug use, stressful life event, and social support-related variables associated with the onset of a depressive episode in a cohort of psychoactive drug using young adults. Two waves of data, collected one year apart, were available on 942 individuals. Cases ($n = 62$) were free of depressive symptoms at time 1 but reported significant symptomatology at time 2 as measured by the depression subscale of the Brief Symptom Inventory. Controls (n

$= 490$) were those free of depressive symptoms at both time points. In multivariate analyses, users of the central nervous system depressant methaqualone had a nearly four-fold elevated risk for depressed mood as compared to nonusers. Additional risk factors significant after multivariate adjustment included lower self-esteem at time 1 and negative life events. These results highlight the multifactorial nature of depressive symptomatology. (*Am J Public Health* 1990; 80:580-585.)

Introduction

Using predominantly case-control comparisons on general population samples, the association between negative life events,¹⁻⁹ lack of social support,^{10,11,4,5,8,12} and self-esteem¹³⁻¹⁸ with depressive symptoms and disorder has been well documented. Analogous prospective studies are rare.¹⁷ While research has documented an association between alcohol use and depression,¹⁹⁻²¹ much less is known about the potential effects of other drugs or about the relative and possibly interactive effects of life events and drug use on depression symptomatology. One obstacle to such studies is the low base rate of negative life events and/or heavy substance use in most samples making it unfeasible to examine their relationships. By focusing on a population of young adults who frequently use a variety of psychoactive substances, we estimated the adjusted relative risk of various drug use, personality, psychosocial, and negative life event variables for developing an episode of depressed mood.

Methods

We utilized a case-control methodology nested within a prospective study^{22,23} to investigate risk factors for depressed mood using data from a study on the consequences of arrest for marijuana possession, a short-term longitudinal investigation of drug using young adults.*

Study Population

In 1981-82, 1,134 adults living in the State of Maryland were interviewed and 942 were relocated and agreed to a reinterview one year later. A primary aim was to evaluate the effects of having been arrested and tried for marijuana possession on subsequent drug use and mental health. An application for a certificate of confidentiality, which protects the identity of research study subjects, was approved by the US Secretary of Health and Human Services. Written consent was obtained of all research participants and each individual was informed as to the nature of the study and the

certificate which protects the information they provide from seizure by police or use in any civil or criminal proceeding.

The panel of 942 individuals which forms the basis of the present study was comprised, in part, of a random sample of 621 individuals, stratified by month of arrest, from a roster of all adults who had been tried in 1981 for marijuana possession within the district courts of Maryland. An additional 321 non-arrested individuals, nominated by their arrested counterparts as being friends or acquaintances, were selected as a neighborhood-based comparison group. This nomination procedure was done to ensure comparability between the two groups on demographic characteristics and drug use patterns.

Operationalization of Variables

The presence of depressive symptomatology in the week prior to interview was assessed using the depression subscale of the Brief Symptom Inventory (BSI)—the short form of the Symptom Checklist-90 (SCL-90).^{24,25} Internal consistency analysis of the depression sub-scale revealed coefficient alpha = .78 at the time of first interview (Time 1) and alpha = .80 at the second interview (Time 2). The depression subscale, consisting of six items, has been found to correlate (.95) with its corresponding longer version on the SCL-90.²⁵ There is evidence in support of the temporal stability and the convergent and discriminant validity of the BSI depression subscale.²⁶

Individuals were classified as a case if they did not report depressive symptoms at Time 1 but reported symptoms at Time 2 that placed them in the 90th centile or above on the non-patient norms for the BSI depression subscale.²⁶ This cut-point was selected to serve as a strong contrast between cases and controls while still retaining a sufficient sample size for purposes of statistical power. While this depression measure does not identify a case of dysthymia or major depressive disorder according to DSM-III-R²⁷ criteria, an individual defined as a case in this study would present clinically with at least moderate symptoms of depression. All individuals who reported no or minimal symptoms of depression at both time points were selected to form a control group. Using these criteria a total of 62 cases and 490 controls were selected. Individuals excluded from analyses were those who reported mild-to-severe depressive symptoms at Time 1. Inspection of the depression scores at both time points revealed that those selected as cases went, on average, from the 50th percentile at Time 1 to the 95th percentile at Time 2. In contrast, those selected as controls were at about the 45th percentile at Time 1 and remained there at Time 2.

The wording of the BSI allowed for a determination of the current level of depressed mood experienced by a study participant, although, it could not help determine depressed

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mood in the months prior to each administration. While an imperfect and somewhat less specific measure for determining case status than use of an instrument such as the Diagnostic Interview Schedule,²⁸ it is unlikely that a bias was produced by this manner of case-control selection. Because the BSI was administered at Time 1, some control for prior depressed mood was achieved; more than is the case in many other case-control comparisons.

Participants were asked about frequency of use of a wide variety of licit and illicit drugs in the two months prior to each interview. Alcohol and marijuana were used with enough regularity to justify coding on an interval level (3 = daily; 2 = two–three times a week; 1 = one time a week or less; 0 = no use). Tobacco use was measured in packs of cigarettes smoked per day. The vast majority of use of such substances as amphetamines, barbiturates, methaqualone (Quaalude, Sopor), cocaine, heroin, hallucinogens, and anxiolytics (e.g., Valium, Librium) occurred on a weekly or less frequent basis. Therefore, these drug variables were dichotomously coded with 1 representing any reported use of the drug during the two months prior to interview and 0 indicating no use.

The occurrence of any of five negative stressful life events (arrest, conviction, hospitalization, traffic accident, fired from job) in the 12 months between interviews was assessed and a summative count score was created. Prior research has shown that weighting of events before creation of an overall score does not increase prediction of the dependent variable.^{29–32} These negative life events are typical of those found on life event inventories³³ and represent life stressors that occur with relative frequency in this population.

Separate questions tapping perceived social support, or social embeddedness,³⁴ with family members and with peer group were asked of all respondents at both interviews: “Do you feel a sense of solidarity, closeness, belonging to your family?” Four dichotomous variables were created from the Time 1 and Time 2 questions representing perceived social

embeddedness with family or friends or lack of support/embeddedness. The presence of a spouse or living together with a partner was also determined and dichotomously coded as another important indicant of social support.^{32,35}

Finally, the Rosenberg Self-Esteem Scale,³⁶ a widely used 10-item measure of self-esteem, was administered at both interviews. Extensive research has demonstrated the predictive and construct validity of this instrument.³⁷ With the present sample, the instrument displayed good internal consistency (Coefficient alpha = .75) and the one year test-retest stability was $r(938) = .49$.

Univariate logistic regression models that estimate the crude relative risk ratio for the development of an episode of depression were first calculated for variables from each of the demographic, drug use, life events, social support, and personality domains. Using multivariate logistic regression techniques, variables predictive of case status in univariate analyses were simultaneously evaluated to estimate adjusted relative risks. The presence of multiplicative interaction was examined for pairwise combinations of variables that remained significant in the multivariate analysis.

Results

Descriptive statistics for the 62 depressed mood cases and 490 controls and crude relative risk estimates based on univariable logistic regression analyses are presented in Table 1 for categorical variables and Table 2 for variables measured on a continuous scale. Cases were fairly similar to controls with respect to age, race, sex, marital status, being employed or in school, recent history of marijuana arrest, and socioeconomic status (not shown in tables). Univariate relative risk (RR) estimates for the development of depressive symptomatology were found for any use during the two months prior to the second interview for methaqualone, anxiolytics, and less strongly for heroin, tobacco, and hallucinogens.

TABLE 1—Estimated Relative Risk for an Episode of Depressed Mood Based on Univariate Logistic Regression Analysis with Categorical Variables

Variables	Cases No. (%) (N = 62)	Controls No. (%) (N = 490)	Referent Category	Relative Risk Estimate (unadjusted)	(95%CI)	p value
Sociodemographic						
Male	52 (84)	382 (78)	female	1.47	(.72, 2.99)	.29
White	24 (39)	166 (34)	black	1.23	(.72, 2.12)	.45
Employed or in school	40 (65)	322 (66)	no	.94	(.54, 1.64)	.84
Recent marijuana arrest	45 (73)	321 (66)	no	1.39	(.77, 2.51)	.27
Drug Use						
Amphetamines (Time 1)	7 (11)	49 (10)	no use	1.15	(.49, 2.65)	.75
Amphetamines (Time 2)	9 (15)	52 (11)	no use	1.43	(.67, 3.06)	.36
Barbiturates (Time 1)	5 (8)	32 (7)	no use	1.26	(.47, 3.35)	.65
Barbiturates (Time 2)	6 (10)	28 (6)	no use	1.77	(.70, 4.45)	.3
Methaqualone (Time 1)	4 (6)	31 (6)	no use	1.02	(.35, 2.99)	.97
Methaqualone (Time 2)	10 (16)	23 (5)	no use	3.90	(1.76, 8.65)	.0008
Anxiolytics (Time 1)	3 (5)	29 (6)	no use	.81	(.24, 2.74)	.73
Anxiolytics (Time 2)	8 (14)	26 (5)	no use	2.64	(1.14, 6.12)	.02
Cocaine (Time 1)	16 (26)	96 (20)	no use	1.43	(.77, 2.63)	.25
Cocaine (Time 2)	14 (23)	99 (20)	no use	1.15	(.61, 2.17)	.66
Hallucinogens (Time 1)	6 (10)	26 (5)	no use	1.91	(.75, 4.84)	.17
Hallucinogens (Time 2)	7 (11)	25 (5)	no use	2.37	(.98, 5.73)	.06
Heroin (Time 1)	4 (6)	16 (3)	no use	2.04	(.66, 6.32)	.21
Heroin (Time 2)	7 (11)	24 (5)	no use	2.47	(1.02, 6.00)	.046
Social Support (Time 2)						
Family Embeddedness (yes)	53 (85)	453 (93)	no	.41	(.19, .91)	.03
Peer Group Embeddedness (yes)	42 (68)	400 (82)	no	.47	(.27, .85)	.01
Married or Living Together	10 (16)	120 (24)	no	.59	(.29, 1.20)	.15

TABLE 2—Estimated Relative Risk for an Episode of Depressed Mood Based on Univariate Logistic Regression Analyses with Continuous-scale Variables

Variables	Cases (N = 62) Mean (S.D.)	Controls (N = 490) Mean (S.D.)	Relative Risk Estimate (unadjusted)	(95%CI)	p value
Sociodemographic					
Age	24.50 (6.21)	24.92 (6.88)	.99	(.96, 1.04)	.92
Drug Use					
Alcohol (Time 1)*	1.51 (1.01)	1.37 (.96)	1.16	(.88, 1.53)	.28
Alcohol (Time 2)*	1.62 (1.02)	1.43 (.97)	1.22	(.93, 1.61)	.15
Marijuana (Time 1)*	1.85 (1.10)	1.77 (1.12)	1.04	(.84, 1.30)	.72
Marijuana (Time 2)*	1.81 (1.25)	1.56 (1.15)	1.20	(.95, 1.51)	.12
Tobacco (Time 2)**	3.89 (2.07)	3.35 (1.86)	1.17	(1.01, 1.35)	.04
Personality (coded 1-4)					
Self-esteem (Time 1)	3.24 (.49)	3.45 (.40)	.32	(.17, .59)	.0003
Self-esteem (Time 2)	3.08 (.45)	3.46 (.42)	.14	(.07, .26)	<.0001
Life Events: (count 0-4)					
Negative Events	.79 (.91)	.44 (.72)	1.67	(1.24, 2.26)	.0009

*These frequency codes range from 0-3: (0 = no use; 1 = 1 time a week or less; 2 = 2-3 times a week; 3 = daily use).

**These frequency codes range from 1-7: (1 = no use; 2 = >1 cigarette a day; 3 = 1-5 cigarettes a day; 4 = 10 cigarettes a day; 5 = 20 cigarettes a day; 6 = 30 cigarettes a day; 7 = 40 cigarettes a day).

TABLE 3—Estimated Relative Risk for an Episode of Depressed Mood Based on Multivariate Logistic Regression Model with Drug Variables Only

Variable	Referent Category	Estimated Relative Risk	(95%CI)	p value
Drug Use (Time 2)				
Amphetamines	no use	.81	(.29, 2.30)	.69
Barbiturates	no use	.54	(.14, 7.08)	.37
Methaqualone	no use	5.27	(1.86, 14.96)	.002
Anxiolytics	no use	2.07	(.75, 5.71)	.16
Cocaine	no use	.53	(.24, 1.19)	.13
Hallucinogens	no use	2.14	(.77, 6.00)	.15
Heroin	no use	3.32	(1.18, 9.30)	.02
Alcohol frequency	NA*	1.06	(.79, 1.43)	.67
Marijuana frequency	NA*	1.07	(.82, 1.40)	.59
Tobacco frequency	NA**	1.08	(.93, 1.25)	.34

*Not applicable. These frequency codes range from 0-3: (0 = no use; 1 = 1 time a week or less; 2 = 2-3 times a week; 3 = daily use).

**Not applicable. These frequency codes range from 1-7: (1 = no use; 2 = >1 cigarette a day; 3 = 1-5 cigarettes a day; 4 = 10 cigarettes a day; 5 = 20 cigarettes a day; 6 = 30 cigarettes a day; 7 = 40 cigarettes a day).

To statistically control for multiple drug use, a multivariate logistic regression model containing main effect terms for all Time 2 drug variables (see Table 3) was created. The relative risk estimates show a clear excess risk for those reporting any use of methaqualone in the prior two months (RR = 5.27; 95% CI = 1.86, 14.96) as well as those reporting any use of heroin (RR = 3.32; 95% CI = 1.18, 9.30). Two-way interaction terms were then evaluated for methaqualone and heroin with combinations of the other drug variables. None were found to be statistically significant at ($p < .05$) although the interaction term for methaqualone and barbiturate use was significant at ($p < .10$) and was further evaluated in subsequent analyses.

Crude relative risk estimates for the life event, social support, and self-esteem measures are apparent in Tables 1 and 2. Higher levels of self-esteem at Time 1 appear to act as a protective factor with the risk of depressed mood dropping by an estimated .32 (95% CI = .17, .59) with each unit increase in self-esteem. Conversely, individuals with lower levels of self-esteem appear more vulnerable to the development of an episode of depressed mood. A strong cross-

sectional relationship between Time 2 self-esteem and depression was also present. Negative life events happening in the year before follow-up served to elevate the risk for depression with each occurrence of such an event (arrest, conviction, fired from job, or hospitalization) increasing the risk for developing depressed mood 1.67-fold (95% CI = 1.24, 2.26). Risk for depressed mood was less if the subjects reported that they felt embedded within their family (RR = .41; 95% CI = .19, .91) or within a peer group (RR = .47; 95% CI = .27, .85). Being married or living with someone was not associated with case-status.

In a multivariate logistic regression model (see Table 4) containing main effect terms for life events, social support and self-esteem, the relative risk estimate for family as well as for peer group embeddedness were sharply reduced suggesting that the univariate relationships were due to associations with other variables. Self-esteem at the earlier period and the occurrence of negative events during the year remained strong predictors of case status. All possible two-way interactions among statistically significant variables in this model were tested but no interaction was detected.

We then tested a multivariable function (table not shown) that combined the drug use and psychosocial varia-

TABLE 4—Estimated Relative Risk for an Episode of Depressed Mood based on Multivariate Logistic Regression Model with Social Support, Self-esteem, and Negative Event Variables Only

Variable	Referent Category	Estimated Relative Risk	(95%CI)	p value
Family embeddedness (yes)	no	.50	(.21, 1.16)	.11
Peer group embeddedness (yes)	no	.58	(.35, 1.22)	.18
Self-esteem (Time 1)	NA*	.39	(.21, .75)	.005
Negative events	NA**	1.60	(1.15, 2.15)	.005

*Not applicable. This variable is measured on a continuous scale.

**Not applicable. This variable ranges from 0–4 and was not categorized.

bles that had retained p values of .05 or less in the prior multivariate models as well as the interaction term for methaqualone and barbiturate use. The prior association between heroin use and depressed mood no longer held in this model nor did the methaqualone-barbiturate interaction term. Negative events, self-esteem, and methaqualone use maintained their independent effects. A final model was tested that retained these three main effect terms and produced the following estimates: negative events (RR = 1.45; 95% CI = 1.06, 2.00); self-esteem (RR = .32; 95% CI = .17, .59); and methaqualone use (RR = 3.70; 95% CI = 1.59, 8.63). Interaction was not detected among pairwise combination of these three variables.

Discussion

Prior research evidence has suggested associations between self-esteem, the occurrence of negative life events, and indices of social support with depressed symptomatology. With the exception of social support, which only evidenced a trend in the predicted direction in multivariate analysis, this was corroborated in our findings; and, importantly, our results indicate that, when controlling for each other, these variables remain independent predictors for who will develop a new episode of depression. It is conceivable that a more refined measure of social support than the ones used in this study might have detected a support-depression association in multivariate, not just univariate, analyses. The measure of life events was limited by the available data to a count of five negative occurrences (arrest, conviction, hospitalization, traffic accident, fired from job). A more exhaustive index of life events would likely have produced an even stronger association.

This study has documented that a similar pattern of risk factor relationships for depressed affect exists among a sample of psychoactive drug users as has typically been found in general population samples.^{1–9,15,17} The final model suggests that individuals who have low self-esteem (measured a year prior), or who experience negative life events, or who use methaqualone, are at elevated risk for developing depressed mood. An issue to consider was the effectiveness of the nested case-control design in controlling for Time 1 depressed mood. We examined this by re-analyzing all models reported in Tables 1–4 and the final model. While the Time 1 measure of depressed mood was a strong predictor of the same variable at Time 2, the same findings emerged from the reanalysis with only slight and trivial changes in the relative risk estimates. Thus, sufficient control for prior depressed mood appears to have been achieved in the design alone.

In this sample, methaqualone use appears to substantially increase the risk for depressed symptoms independently of self-esteem or negative life events. The majority of methaqualone users who became depressed at Time 2 may have initiated their use of this substance during the study period: Of the 10 individuals with depressed symptoms who reported use of methaqualone at Time 2, seven (70 percent) had not reported use at Time 1 while three (30 percent) reported use at both time points. This methaqualone-depression association cannot be explained by the potential confounding effects of other drug use assessed in this study or other predictive variables. In fact, the adjusted relative risk estimates for methaqualone use were higher than the crude estimate. Nevertheless, the existence of a third variable which causes both a propensity to use methaqualone and to become depressed cannot be ruled out. When examined with other drug variables, heroin use was also related to depression. However, when considered together with psychosocial variables, heroin use failed to retain its importance in predicting an episode of depression.

Our review of the literature indicates that the association found between methaqualone use and depressed mood in this drug using sample is a new finding and, therefore, deserves further comment. Methaqualone is a synthetic central nervous system depressant compound that is similar to barbiturates in its sedative and hypnotic action.^{38,39} Until January 1984, when it was made a controlled substance,⁴⁰ the drug was marketed in the United States under the trade names Quaalude, Sopor, Parest, Optimal, and Somnafac.⁴¹ Its use reached epidemic proportions in the 1970s and early 1980s^{42–46}; perhaps, due in part to its purported aphrodisiac qualities.⁴⁷ Although its use today has diminished as a result of discontinuation of legal production in the United States,⁴⁸ quantities produced by clandestine domestic or small foreign laboratories are still available.^{47,48}

Methaqualone users are reported to be more likely to also use other illicit and licit psychoactive substances than are non-users of the drug.^{44,49} This was confirmed in the present sample. The multi-drug involvement of methaqualone users could account for this study's finding of univariable associations between depressed mood and various other substances (marijuana, tobacco, hallucinogens) but lack of association when controlling for methaqualone use. Our results suggest the possible prudence of adjusting for the potential influence of methaqualone use when examining the relation between other drugs and depression.

A relation between methaqualone use and depressed symptomatology has been hinted at in two prior studies. Schwirian and colleagues,⁴⁹ in a study of personality characteristics of methaqualone users, noted a cross-sectional association between duration of methaqualone use and mea-

tures of "lethargy and introspectiveness." Craig and Van Natta⁵⁰ looked at cross-sectional, unadjusted associations between recent use of a variety of prescription medicines and the Center for Epidemiologic Studies' depression (CES-D) scale. Users of anxiolytics and sedatives (which included but were not limited to methaqualone) had significantly greater proportions of high CES-D scores than did non-users of either class of drugs with stronger associations for females than for males. Barbiturates, a class of drugs with a similar sedative-hypnotic action to methaqualone, have also been linked to depression. Recent case reports indicate that phenobarbital may cause severe depression in patients taking the drug to control seizure disorders.^{51,52} At a more general level, Judd and Grant⁵³ found that central nervous system depressant users, especially sedative users, displayed pronounced neuropsychological deficits on cognitive tasks which assessed abstract thinking, nonverbal learning, eye-hand coordination, and perceptual accuracy.

The temporal ordering of methaqualone use and depressed mood is important in terms of etiology. In a survey of college student methaqualone users that is relevant to this issue, Kochansky and colleagues⁴⁴ report that while no particular emotion encouraged the use of methaqualone, "moods named as clearly aversive to use were depression and anger." This study suggests that use of methaqualone is more likely to be an antecedent of depressed mood than a consequence. Until more conclusive research is conducted, it would be premature to infer that methaqualone use causally contributes to the development of depressed mood. Also, it should be noted that methaqualone is an infrequently used drug. Therefore, despite the almost four-fold relative risk for depressed mood, its attributable risk both in the general population and among drug user samples would be less than that of other explanatory variables including other potential etiologic factors examined in this study.

In this sample, lower self-esteem was associated with the development of a depressive episode one year later regardless of drug use behavior or the occurrence of negative life events. Likewise, the occurrence of negative life events also independently predicted depressed mood. Such findings may suggest alternative pathways to depressed mood in drug users, with those individuals low in self-esteem, or who use methaqualone, or who experience one or more stressful life events at higher risk. A combination of these risk factors would appear to increase risk in a multiplicative manner. Each one of these three variable's ability to predict subsequent depressed affect does not depend on a particular level of the other two; that is, effect modification was not detected.

A study limitation was reliance on a measure of depressed symptomatology as opposed to a diagnostic classification for depressive disorder. Nevertheless, a strong consistency exists between psychosocial risk factors for depressed symptoms and unipolar depressive disorder indicating a possible continuity between these two conditions.⁵⁴

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REFERENCES

- Dohrenwend BS, Dohrenwend BP (eds): *Stressful Life Events: Their Nature and Effects*. New York: John Wiley, 1974.
- Lloyd C: Life events and depressive disorder reviewed: II. Events as precipitating factors. *Arch Gen Psychiatry* 1980; 37:541-548.
- Finlay-Jones R: Showing that life events are a cause of depression—A review. *Aust NZ J Psychiatry* 1981; 15:229-238.
- Billings AG, Cronkite RC, Moos RH: Social-environmental factors in unipolar depression: Comparisons of depressed patients and nondepressed controls. *J Abnorm Psychol* 1983; 92:119-133.
- Mitchell RE, Cronkite RC, Moos RH: Stress, coping, and depression among married couples. *J Abnorm Psychol* 1983; 92:433-448.
- Murrell SA, Norris FH: Resources, life events, and changes in positive affect and depression in older adults. *Am J Community Psychol* 1984; 12:445-464.
- Monroe SM, Bromet EJ, Connell MM, Steiner SC: Social support, life events, and depressive symptoms: A 1-year prospective study. *J Consult Clin Psychol* 1986; 54:424-431.
- Phifer JF, Murrell SA: Etiologic factors in the onset of depressive symptoms in older adults. *J Abnorm Psychol* 1986; 95:282-291.
- Dohrenwend BP, Shrout PE, Link BG, Martin JL, Skodol AE: Overview of initial results from a risk-factor study of depression and schizophrenia. In: Barrett JE, Rose RM (eds): *Mental Disorders in the Community: Progress and Challenge*. New York: Guilford Press, 1986.
- Brown GW, Harris T: *Social Origins of Depression: A Study of Psychiatric Disorder in Women*. New York: Free Press, 1978.
- Henderson SP: The social network, social support, and neurosis: The function of attachment in adult life. *Br J Psychiatry* 1977; 131:185-191.
- House JS, Landis KR, Umberson D: Social relationships and health. *Science* 1988; 241:540-545.
- Bibring E: The mechanism of depression. In: Greenacre P (ed): *Affective Disorders*. New York: International Universities Press, 1953.
- Jacobson E: *Depression*. New York: International Universities Press, 1971.
- Blatt S, Quinlan D, Chevron E, McDondald C, Zuroff D: Dependency and self-criticism: Psychological dimensions of depression. *J Consult Clin Psychol* 1982; 50:113-124.
- Dobson KS, Shaw BF: Specificity and stability of self-referent encoding in clinical depression. *J Abnorm Psychol* 1987; 96:34-40.
- Barnett PA, Gotlib IH: Psychosocial functioning and depression: Distinguishing among antecedents, concomitants, and consequences. *Psychol Bull* 1988; 104:97-126.
- Segal ZV: Appraisal of the self-schema construct in cognitive models of depression. *Psychol Bull* 1988; 104:147-162.
- Aneshensel CS, Huba GJ: Depression, alcohol use, and smoking over one year: A four wave longitudinal causal model. *J Abnorm Psychol* 1983; 92:134-150.
- Deykin EY, Levy JC, Wells V: Additional depression, alcohol and drug abuse. *Am J Public Health* 1987; 77:178-182.
- Parker DA, Parker ES, Harford TC, Farmer GC: Alcohol use and depression symptoms among employed men and women. *Am J Public Health* 1987; 77:704-707.
- Rothman KJ: *Modern Epidemiology*. Boston: Little, Brown, 1986.
- Kelsey JL, Thompson WD, Evans AS: *Methods in Observational Epidemiology*. New York: Oxford, 1986.
- Derogatis LR, Cleary PA: Confirmation of the dimensional structure of the SCL-90: A study in construct validation. *J Clin Psychol* 1977; 33:981-989.
- Derogatis LR: *The SCL-90 Manual I: Scoring Administration and Procedures for the SCL-90*. Baltimore, MD: Clinical Psychometrics Unit, Johns Hopkins University School of Medicine, 1977.
- Derogatis L, Spencer P: *The Brief Symptom Inventory (BSI): Administration, Scoring, & Procedures Manual-I*. Baltimore, MD: Division of Medical Psychology, Johns Hopkins University School of Medicine, 1982.
- American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*. 3rd Ed. Washington, DC: American Psychiatric Association, May 1987.
- Robins LN, Helzer JE, Orvaschel H, Anthony JC, Blazer DG, Burnam A, Burke JD Jr: The Diagnostic Interview Schedule. In: Eaton WW, Kessler LG (eds): *Epidemiologic Field Methods in Psychiatry: The NIMH Epidemiologic Catchment Area Program*. New York: Academic Press, 1985.
- Ross CF, Mirowsky J: II. A comparison of life-event weighting schemes: Change, undesirability, and effect-proportional indices. *J Health Soc Behav* 1979; 21:166-177.
- Kessler RL: A comment on 'A comparison of life-event-weighting schemes.' *J Health Soc Behav* 1980; 20:293-296.
- Lei H, Skinner HA: A psychometric study of life events and social readjustment. *J Psychosom Res* 1980; 24:57-65.
- Thoits PA: Conceptual, methodological, and theoretical problems in studying social support as a buffer against life stress. *J Health Soc Behav* 1982; 23:145-159.
- Holmes TH, Rahe RH: The social readjustment rating scale. *J Psychosom*

- Res 1967; 11:213-218.
34. Barrera M: Distinctions between social support concepts, measures, and models. *Am J Community Psychol* 1986; 14:413-445.
 35. Eaton WW: Life events, social supports, and psychiatric symptoms: A re-analysis of the New Haven data. *J Health Soc Behav* 1978; 19:230-234.
 36. Rosenberg M: *Conceiving the Self*. New York: Basic Books, 1979.
 37. Corcoran K, Fischer J: *Measures for Clinical Practice: A Sourcebook*. New York: Fress Press, 1987.
 38. Lewis AJ (ed): *Modern Drug Encyclopedia*. New York: Yorke Medical Books, 1979.
 39. Goodman and Gilman: *The Pharmacological Basis of Therapeutics* (7th ed). New York: Macmillan, 1985.
 40. Slak S: A dozen and two years of methaqualone: A case report. *Psychol Reports* 1985; 57:1097-1098.
 41. Gerald MC, Schwirian PM: Nonmedical use of methaqualone. *Arch Gen Psychiatry* 1973; 28:627-631.
 42. Pascarelli EF: Methaqualone abuse, the quiet epidemic. *JAMA* 1973; 224:1512-1514.
 43. Bridge TP, Ellinwood EH Jr: Quaalude alley: A one-way street. *Am J Psychiatry* 1973; 130:217-219.
 44. Kochansky GE, Hemenway TS III, Salzman C, Shader RI: Methaqualone abusers: A preliminary survey of college students. *Dis Nerv System* 1975; 36:348-351.
 45. Ryser PE: Students and drug abuse, 1974 and 1980. *J Sch Health* 1983; 53:435-436.
 46. Johnston LD, O'Malley PM, Bachman JG: *National Trends in Drug Use and Related Factors among American High School Students and Young Adults, 1975-1986*. DHHS Pub No. (ADM) 87-1535. Rockville, MD: National Institute on Drug Abuse, 1987.
 47. Nicholi AM Jr: The nontherapeutic use of psychoactive drugs. *N Engl J Med* 1983; 308:925-933.
 48. O'Malley PM, Bachman JG, Johnston LD: Period, age, and cohort effects on substance use among young Americans: A decade of change, 1976-86. *Am J Public Health* 1988; 78:1315-1321.
 49. Schwirian PM, Gerland MC: Personalities and attitudes of nonmedical users of methaqualone. *Arch Gen Psychiatry* 1974; 30:525-530.
 50. Craig TJ, Van Natta PA: Medication use and depressive symptoms. *NY State J Med* 1982; 82:1439-1443.
 51. Brent DA, Crumrine PK, Varma RR: Phenobarbital treatment and major depressive disorder in children with epilepsy. *Pediatrics* 1987; 80:909-917.
 52. Barabas G: Barbiturate anticonvulsants as a cause of severe depression. *Pediatrics* 1988; 82:284.
 53. Judd LL, Grant I: Brain dysfunction in chronic sedative users. *J Psychedelic Drugs* 1975; 7:143-149.
 54. Hirschfeld RMA, Cross CK: Epidemiology of affective disorders. *Arch Gen Psychiatry* 1982; 39:35-46.

European Charter on Environment and Health

Following 12 months of intense diplomatic activity and detailed technical consultations among the 32 countries of the European Region of the World Health Organization, a ministerial conference was held in Frankfurt on December 7-8, 1989, which formally approved a Charter on Environment and Health. The European Region of WHO represents 825 million people and covers an area from Greenland in the west to the Pacific coast of the Soviet Union in the east. The conference was attended by Ministers of both Health and Environment; observers were present from Canada and the United States.

Efforts are underway to secure adoption of the Charter by all sections of society in the countries of the WHO European Region.

The decision to develop the European Charter was based on a growing perception that links between the environment and health were not clearly perceived at international, national, and local levels and that insufficient attention had been given to the integration of health dimensions into policy decisions in such fields as physical planning, energy, transport, and agriculture, nor were potential health consequences always adequately addressed at the planning stage of new developments.

The conference not only addressed specific biological, chemical and physical hazards but placed considerable emphasis on the importance of a salubrious overall environment in terms of housing, the community, and the workplace. In brief, the Charter:

- Acknowledges the dependence of human health on a wide range of environmental factors;
- Devotes a section to entitlements and responsibilities, i.e., of individuals as well as public and private bodies;
- Encompasses principles for public policy—including new policies, technologies and developments—that safeguard the environment and human health;
- Enumerates strategic elements essential to its objectives, i.e., control measures, standards, information systems, and the appropriate use of fiscal/administrative/economic instruments and land-use planning;
- Delineates priority areas to which governments, the European community, and intergovernmental organizations should pay particular attention;
- Lays down steps necessary to reverse negative trends and maintain and increase improvements already taking place, in a section titled "The Way Forward."

A copy of the Charter, as well as a detailed commentary and other relevant materials, may be obtained from the WHO Regional Office of Europe, 8 Sherfigsvej, 2100 Copenhagen, Denmark.