# **Depression and Unemployment: Panel Findings** from the Epidemiologic Catchment Area Study<sup>1</sup>

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Studies that have found an association between unemployment and psychological depression often fail to establish the direction of causal influence. Analyses of Epidemiologic Catchment Area panel data revealed that of employed respondents not diagnosed with major depression at first interview, those who became unemployed had over twice the risk of increased depressive symptoms and of becoming clinically depressed as those who continued employed. Although the increase in symptoms was statistically significant, the effect on clinical depression was not, possibly because of the low power of the test. The reverse causal path from clinical depression at Time 1 to becoming unemployed by Time 2 was not supported. The unemployment rate in the respondent's community at time of interview was not related directly to psychological depression but appeared associated indirectly with depression via its impact on the risk of becoming unemployed. Implications for policy and further research were discussed.

KEY WORDS: unemployment; depression; panel; stress; anxiety.

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# Unemployment as a Major Loss Event

A defining characteristic of community psychology is its attention to social stressors and their impact on individual psychological well-being (Dohrenwend, 1978). This perspective suggests different intervention points in the stress process. For example, evidence showing the adverse effects of a controllable social stressor, such as unemployment, could help persuade the government to engage in primary prevention by reducing the unemployment rate. Although past research has found a correlation between unemployment and depressive symptoms, the causal nature of this linkage is difficult to establish, and this uncertainty may aid the resistance of those who have successfully opposed full-employment policies. Moreover, little research has focused on diagnosed depression, a clinical category with severity and treatment cost implications that might arouse more political concern than small mean changes in depressive symptoms. At a more individual level, the stress model suggests interventions soon after the stressor has occurred for the secondary prevention of psychological symptoms. For example, recently unemployed participants in a series of training programs were found to suffer lower levels of depression at follow-up than nonparticipants (Vinokur, Price, & Caplan, 1991). However, not all job losers have access to such programs, and further documentation of the psychological effects of unemployment may be necessary to encourage the spread of such interventions.

Certain kinds of events have the potential to produce depression those involving loss and disappointment and threatening long-term consequences (G. W. Brown & Harris, 1978). Stressful events do not account entirely for depression as evidenced by those hardy individuals who withstand such events without becoming depressed. However, the diathesis for depression is sufficiently widespread that such events must be regarded as a nontrivial cause of this disorder (Monroe & Simons, 1991). Reviews of the literature consistently find that depressed patients experience more stressful events in the months prior to onset than do normal controls and that the risk of depression rises as much as fivefold in the 6 months after an event (Lloyd, 1980). Unfortunately, much of this research relies on retrospective methods and tends to mix stressors rather than to focus on a particular type of event such as job loss.

Losing a job seems a likely trigger of mental disorder generally and depression in particular. Scholars have observed the adverse mental health effects of unemployment throughout this century (Jahoda, Lazarsfeld, & Zeisel, 1971/1933; Rowntree & Lasker, 1911; Warr, 1987). Reviews have found that job losers exhibit more psychiatric symptoms than their employed counterparts and that communities experiencing high unemployment rates also have higher mental hospitalization and suicide rates (Dooley & Catalano, 1986).

To provide more diagnostic focus, the present study concentrates on depression as one of the most likely affective responses to a major loss such as unemployment. Although research has pointed to the comorbidity of depression and anxiety (Brady & Kendall, 1992; Clark & Watson, 1991), suggesting that they may be difficult to separate as outcomes of job loss, the literature on the psychological effects of unemployment has focused more on depression than on anxiety. Perhaps losing a job induces a greater sense of depression-triggering loss than of anxiety-provoking fear. Prior analyses of the Epidemiologic Catchment Area (ECA) data, which are used in this study, failed to mention either a cross-sectional or a longitudinal linkage between unemployment and anxiety disorder (Robbins & Regier, 1991). As noted later, our tests also found no relationship of unemployment with anxiety. As a result, the present study focuses on the symptoms and diagnosis of depression.

A major problem in establishing the causal connection between any life event and depression is ruling out reverse causation. Even after controlling for possible confounding variables, cross-sectional studies leave the possibility that preexisting depression causes the life events with which it correlates. To establish causal sequence, various researchers have conducted panel studies of life events and depression. Although not focusing on unemployment, these studies have established that simultaneous or prior life events correlate with depressive symptoms controlling for prior symptoms. These results have appeared consistently in studies of two interviews (Billings & Moos, 1982; Solomon, Smith, Robins, & Fischbach, 1987; Turner & Noh, 1988), three interviews (Ensel & Lin, 1991), and four interviews (Aneshensel & Frerichs, 1982) with between-interview lag periods ranging from 4 months to 4 years.

# Recent Research on Job Loss and Depression

Of special interest here is evidence on the link between depression and job loss and the causal direction of any observed association. Unfortunately, most of the research on this question uses cross-sectional designs that cannot establish causal sequence, for example, by comparing unemployed people with matched controls who have jobs. A related problem often appearing in this cross-sectional literature is the failure to distinguish two types of unemployment: job losers versus people seeking work after being out of the labor force, such as students. Such studies typically find elevated symptom counts in the unemployed whether men (Melville, Hope, Bennison, & Barraclough, 1985) or women (Hall & Johnson, 1988). Another variant on the cross-sectional design relies on a random community sample and typically finds that depressive symptoms correlate with unemployment controlling for other variables (Dressler, 1986). The common use of symptom count measures leaves in doubt whether unemployment causes clinical depression or only elevates depressed mood in the normal range. One exception to this reliance on symptom count measures comes from the ECA study that found higher levels of diagnosed clinical depression in those not employed (including both unemployed and those not seeking work) than those who were employed in a cross-sectional study based on five sites (Weissman, Bruce, Leaf, Florio, & Holzer, 1991). Another approach to the question of unemployment and case-level depression compares diagnosed patients with matched controls. In one such study using 300 nonendogenous depressed patients and 300 controls, the former were more likely to be unemployed before onset (Roy, 1987). In the same study, unemployment did not distinguish endogenous depressed patients from their controls.

Retrospective reports of the timing and nature of job loss both clarify the nature of the unemployment (job loser vs. new job seeker entering the labor force) and partially address the reverse causation problem in cross-sectional research. For example, one survey of stably employed, unemployed, and previously unemployed people included items to distinguish those who might have caused their unemployment from those who had not contributed to their job loss (Kessler, Turner, & House, 1988). The results indicated that even controlling for voluntariness, job loss was associated with more symptoms of depression. Another cross-sectional study relying on retrospection focused on 80 unemployed men (Eales, 1988). Using the Present State Examination, that study diagnosed 22 (28%) of the men as having affective disorder at the case level and a further 16 (20%) as borderline for caseness. Pointing to the risk of reverse causation, 18 of these 38 cases or near cases reported that their affective disorders arose before job loss. However, of the remaining 20 who reported themselves well before unemployment, 14 (70%) became depressed within 6 months of losing their jobs, and in most of these cases (10), job loss was the only severe event preceding onset.

Prospective designs would provide better evidence on this question. Unfortunately, the few recent panel studies in this area typically begin with people who have lost a job and follow them to reemployment. Such studies find that the continuing unemployed have more symptoms of depression than those who find work (Bolton & Oatley, 1987; Kessler, Turner, & House, 1989; Shamir, 1986). This longitudinal association could arise if the most depressed job losers fail to seek reemployment. However, one such study found that the more depressed unemployed were more likely to find reemployment than the less depressed (Kessler et al., 1989). One prospective panel study started with employed persons and followed them for 2 years (Hamilton, Hoffman, Broman, & Rauma, 1993). That study found that losing a job was related to prior frequency of symptoms of depression and that depressive symptoms decreased when employment outcomes (either obtained a wanted job, lost a

disliked job, or remained willfully unemployed) matched desires. That study focused on frequency of depressive symptoms rather than major depressive disorder, and it defined unemployment as not working for whatever reason rather than the narrower category of collecting unemployment, both differences from the present study.

Aside from the Hamilton et al. study, the literature usually reports that job loss causes depression. Unfortunately, few unemployment studies have used longitudinal methods and even fewer (Eales, 1988; Roy, 1987) have operationalized case-level depression rather than symptom counts. While symptom count studies imply that job loss can cause one to feel blue, they may not be able to demonstrate connection to clinical levels of depression. From an epidemiologic perspective, it would help to know the impact of job loss on case production as well as on distress and demoralization. Finally, this literature entirely omits one potentially important variable, that of the surrounding economic climate. Some research has suggested a direct impact of the prevailing unemployment rate, controlling for personal unemployment, on psychological distress (Dooley, Catalano, & Rook, 1988). Other studies have pointed to a moderating effect of unemployment rates, either buffering the adverse effect of job loss in the unemployed (R. M. Cohn, 1978) or increasing the symptoms of the employed (R. L. Brown, 1982). Seldom replicated, these effects have not been tested for cases of depression.

### Hypotheses

The present study measures the relation of job loss and depression in a panel design that begins with a large sample of nonclinically depressed, employed people. Based on previous studies linking stressful events and depressive symptoms, Hypothesis 1 predicts that job loss by the time of reinterview will be associated with an increased risk of being diagnosed with major depression, controlling for the prevailing unemployment rate and the interaction of unemployment rate and personal job loss and for potential confounding variables such as gender and age. On the same basis, even if unemployment does not result in increased risk of clinical depression, Hypothesis 2 predicts that job loss by reinterview will be associated with an increased risk of having depressive symptoms adjusting for the number of depressive symptoms at first interview, for the main and interaction effects of unemployment rate, and for potential confounding variables. Finally, reverse causation is explored using a slightly larger sample that includes all employed people at first interview including those diagnosed as clinically depressed. Hypothesis 3 predicts that compared to nonclinically depressed people, the depressed cases will be at increased risk of job loss within a year controlling for other disorders and for unemployment rate.

### **METHODS**

# Epidemiological Catchment Area Survey

The ECA research program measured the prevalence of behavioral disorders in five sites in the United States in the 1980s. Survey data were collected in Baltimore, Los Angeles, New Haven, Raleigh-Durham, and St. Louis. Researchers at each site used similar survey designs and identical core questions to assess the prevalence and incidence of specific mental disorders, use of health services, and socioeconomic and demographic factors. A minimum of 3,000 adult community residents at each site were given two face-to-face interviews at a 1-year interval. More detailed information describing the survey methods and research design of the project is available in several publications (Eaton & Kessler, 1985; Robins & Regier, 1991). The present hypotheses were tested on panel data from three of these sites, each measured during different but overlapping time periods. These were Johns Hopkins (the Eastern Health District of Baltimore, 1981–1984), Duke (five primarily rural counties in North Carolina, 1982-1985), and UCLA (two noncontiguous mental health center catchment areas in Los Angeles, 1983-1986). First interview data from the other two sites, New Haven and St. Louis, could be used in cross-sectional analyses, but some key variables were not available at reinterview in these sites making them unusable for the longitudinal tests.

Multistage probability sampling procedures were used at all sites differing only in the intentional oversampling of some subgroups in certain locations. The Johns Hopkins and Duke surveys oversampled the elderly, and one of the UCLA catchment areas provided an oversample of Hispanic respondents. The first interviews in the three sites used in the present panel analyses yielded 10,534 noninstitutionalized respondents for completion rates ranging from 68 to 79%. Of these, 8,278 (79%) were reinterviewed approximately 1 year later. The data from these three sites were pooled to provide a sufficiently large sample to study two relatively infrequent occurrences: incidence of job loss and major depression. Even though each site sample appears large, the convergence of these two events is sufficiently rare to threaten the power to detect effects. In addition, pooling the three sites and their respective time periods offered the widest possible range of unemployment rates.

# Diagnostic Interview Schedule Measures of Depression

The ECA instruments provided an array of standard demographic and other information including age, gender, socioeconomic status (SES, based on household income, education, and occupation), and ethnicity. However, the core instrument was the Diagnostic Interview Schedule (DIS) administered at both interviews and intended to assign respondents to specific categories of the DSM-III, the official diagnostic system of the American Psychiatric Association during the field phase of the ECA (Eaton & Kessler, 1985). The DIS is a structured symptom checklist designed for use by lay interviewers to yield lifetime (ever met the criteria) and more recent diagnoses based on most recent symptom including current (past 2 weeks), last month, last 6 months, and last year.

The DSM-III criteria for major depression require an episode of dysphoric mood persisting at least 2 weeks and at least four of eight other symptoms (e.g., sleep disturbance, feelings of worthlessness or guilt, suicidal ideation; see Weissman et al., 1991). Beyond these symptoms, the diagnosis required that the respondent did not meet the criteria indicative of another disorder (e.g., manic episode pointing to bipolar disorder) and that the symptoms were sufficiently severe to impair functioning. Subsequent revisions of the DSM have retained the same basic criteria for a major depressive episode (5 of 9 symptoms in a 2-week period, not attributable to another disorder). The DSM-III-R added a coding of degree of impairment in the fifth digit of the diagnosis code (American Psychiatric Association, 1987), and the DSM-IV added a criterion to ensure the clinical significance of the depressive episode (American Psychiatric Association, 1994). In sum, the criteria for the major depressive diagnosis have remained relatively stable since the DIS was employed to operationalize the DSM-III in the ECA study, and there is no evidence that these definitional variations would have yielded substantially different estimates of incidence of depression.

For the first hypothesis, respondents were diagnosed as meeting the maior depression criteria using three different dichotomous variables: lifetime depression based on first interview, current depression at first interview, and current depression at reinterview. To control for comorbidity, another dichotomous variable was used: any other current diagnosis at first interview. For the second hypothesis, a current symptom variable was based on the presence in the past 2 weeks of the eight depressive symptoms noted above. The count of these symptoms at first interview served as a control variable. Depressive symptom count had a modal value of zero and was very badly skewed, and a dichotomous version (0 = no symptoms, 1 = one or more) was used when depressive symptom count at reinterview was used as the dependent variable in logistic regression analyses. Because skewness in an independent variable is less problematic than skewness in the dependent variable (Hanushek & Jackson, 1977), and in order not to lose information through unnecessary categorization, the undichotomized symptom count measure is used as a control although analyses using the dichotomous version as a control gave similar results. To check the possibility of comorbidity between anxiety and depression, parallel measures of clinical anxiety disorder and anxiety symptom counts were created for analyses parallel to the first two hypotheses for depression. For the third hypothesis, current major depression and lifetime major depression at first interview were tested as the predictors (0 = absent, 1 = present) of becoming unemployed by second interview.

# Measures of Aggregate Unemployment and Job Loss

The ECA study has the advantage of having contacted respondents not only at different sites but also over a range of months within each site. Thus, the survey sampled people experiencing a variety of different economic climates. The most common way of representing the aggregate economic experience of a community is the unemployment rate, defined as the proportion of the work force (those working plus those out of work but seeking a job) who are not currently employed. Unemployment rates were obtained from archival sources for each month of interviewing at each site. A respondent's unemployment rate variable consisted of the average of the monthly unemployment rate for the month of the respondent's interview and the 2 earlier months for his or her site. The first interviews for the three sites used in this panel analysis began near the end of the 1982 recession with the result that first interview unemployment rates were fairly high (M = 8%, range = 6.4 to 11%). As the nation recovered economically, the second interview unemployment rates fell on average 0.5% with between-interview change ranging from a decrease of 3.9% to an increase of 2.3%.

An earlier test of the relationship of employment and major depression in the ECA data contrasts those currently working with those not currently working (Weissman et al., 1991). That study found that those not working had a 48% greater risk of having major depression adjusting for sex, age, and ethnicity (p. 74, Table 4-17) but noted that the cross-sectional design could not determine causal direction. However, people who are not employed may be neither unemployed (i.e., not working but looking for a job) nor job losers. It is possible that many depressed people who are not working are not even in the work force. The stressor we are concerned with is losing a job for economic reasons rather than for personal behavior. For Hypotheses 1 and 2, the crucial independent variable is job loss due to slack demand for labor operationalized as moving from being employed at first interview to receiving unemployment compensation at reinterview. To receive unemployment compensation, a worker is supposed to have lost a job due to slack demand for labor. This job loss variable also serves as the dependent variable for Hypothesis 3.

This measure of job loss is not intended to capture all people who are unemployed at reinterview. The proportion of people receiving unemployment insurance in the reinterview sample was, therefore, lower (4.1%) than the official unemployment rate in the surveyed communities at Time 2 (7.5%). This arises for different reasons. First, the discrepancy between archival unemployment rates and observed rates of receiving unemployment compensation derives in part from the definitional differences in these variables. To be counted as unemployed in the official unemployment rate, one must be unemployed but actively seeking work, regardless of whether one has worked in the past or is currently receiving unemployment compensation. Thus people who had left jobs to become full-time students or homemakers after Time 1 but reentered the work force by Time 2 to seek work would not be immediately eligible for unemployment insurance although they would be counted in the official unemployment rate. Since the focus of this study is on the effects of job loss, this subgroup of new job seekers is not crucial. Second, although the reinterview completion rate

was reasonably good for such surveys (79%), there may have been some bias in which more of the unemployed than the continuing employed were unreachable or uncooperative at Time 2. If the uncontacted job losers tended to have more distress, the analyses would underestimate the adverse effects of job loss.

However, there are two types of unemployed job losers who are missed by the unemployment insurance measure. One subgroup includes individuals who have unstable jobs in which they do not work long enough to qualify for compensation or who work in fields not covered by unemployment insurance. The other subgroup consists of insured job losers who reach the time limit for coverage. The duration of coverage varies by state from 14 to 26 weeks, although these benefits can be extended when the insured unemployment rate in a state reaches a critically high level (Hansen, 1988). In the three studied sites, the standard coverage extended for 26 weeks (6 months). In one site (Los Angeles, there was one extension for 12 weeks during part of the second interview phase, and in another site (Baltimore) there were two extensions during the survey period for an additional 21 weeks. Thus, respondents who lost jobs shortly after the first interview would no longer be able to receive unemployment insurance by the second interview even if still unemployed. Such long-term unemployed could be expected to feel more emotional distress from their financial condition, and their absence would tend to underestimate the adverse effects in studied job losers. As a result of these limitations, the job loss effect measured by the unemployment insurance variable is a very conservative estimate of the true impact of job loss in the population.

# Analytic Approach

The dependent variables for all three hypotheses are dichotomous: (a) current major depression at reinterview or not. (b) one or more versus no current symptoms of depression at reinterview (controlling for first interview continuous count of symptoms of depression), (c) losing a job or still employed by reinterview. Logistic regression was used to test these hypotheses instead of ordinary least squares or structural equation procedures because these dependent variables were badly skewed (Hosmer & Lemeshow, 1989). Logistic regression provides coefficients that are easily interpretable as odds ratios, or the effect of each predictor variable on the risk of occurrence of the target level of the dependent variable. The standard control variables of gender, age, ethnicity, and SES were included in each analysis along with their interactions and appropriate controls for lifetime major depression and other current diagnosis at first interview. Change in unemployment rate was measured for each respondent as his or her reinterview unemployment rate minus his or her first interview unemployment rate. To separate the effects of change from initial position and to help control for possible regression to the mean in the unemployment rate measure, unemployment rate at first interview was always included when testing the effect of unemployment rate change (Kessler & Greenberg, 1981; Lin & Ensel, 1984).

The ECA project intentionally oversampled subgroups at each site, and consequently, the data are weighted to yield samples representative of the nation as a whole. In addition, because the samples were drawn in several stages, the estimated variance from this weighted data is somewhat larger than would be expected from simple random sampling (Kessler et al., 1985). To ensure proper variance estimation in the logistic regression models, the SUDAAN statistical package for variance estimation in complex survey data was used to compute the regression coefficients and pooled variance estimates (Korn & Graubard, 1991; Shah, 1982).

#### RESULTS

### Cross-Sectional Findings

Earlier analyses of the ECA data failed to note a cross-sectional association between employment status and anxiety disorder (Robins & Regier, 1991) but did report such an association for depression (Weissman et al., 1991). Before testing the three hypotheses with the panel data, we confirmed the cross-sectional association between employment status and major depression. The first interview data from all five sites, cross-tabulated in Table I,

Depression diagnosis	Working	Collecting unemployment	Out of the work force
Not currently depressed	7,953	330	9,360
Currently depressed	145	12	248
	$\chi^2(2) = 15.06, p$	< .001	

Table I. Major Depression by Employment Status: First Interviews Only from Five Sites

reveal a positive association between job loss (i.e., receiving unemployment compensation) and current major depression consistent with the earlier report. For example, the odds of being depressed if one is unemployed (12/330 = .036) are twice the odds of being depressed if one is working (145/7953 = .018) or an odds ratio of 2. In contrast, in the same data, the odds of being diagnosed with an anxiety disorder for the unemployed (23/316 = .073) only slightly exceed the odds of being so diagnosed for those working (504/7555 = .067, odds ratio = 1.09).

The data in Table I underscore three important aspects of these data. First the point prevalence of depression in this study is low, just 2.2% (405 of 18,048 respondents). Second, more people are out of the work force (53.2%, 9,608) than in it (employed 44.9%, 8,098 plus collecting unemployment compensation 1.9%, 342). Third, because job loss is low in this sample (4.1% of those in the work force, 342 of 8,440), very few people experience both major depression and unemployment (12).

The cross-sectional association between employment status and clinical depression remained even after controlling by logistic regression for the significant effects of potential confounders such as gender, age, and site. Those receiving unemployment compensation remained about twice as likely as all other respondents to suffer current major depression (odds ratio = 1.99). Some out-of-work-force categories were also significantly associated with depression including the disabled (odds ratio = 4.89), housewives (odds ratio = 1.41), welfare recipients (odds ratio = 2.93), and others not working without specific reason given (odds ratio = 1.76). Because these cross-sectional analyses cannot rule out reverse causation, the following three hypothesis tests address the temporal sequence between job loss and depression in the three sites for which panel data are available.

Predictor	β	Error	Odds ratio		
Gender	-1.40 <sup>a</sup>	0.50	0.25		
Ever clinically depressed at Time 1	2.09 <sup>b</sup>	0.59	8.12		
Became unemployed	0.80	0.78	2.23		
Intercept	-4.75 <sup>b</sup>	0.28			

 Table II. Clinical Depression at Reinterview: Logistic Regression Panel Analysis for Three Sites

Overall model: Satterthwaite-adjusted F(3.30) = 85.49, p < .001

 $\overline{a \atop p} < .01.$  $b \atop p < .001.$ 

# Hypothesis 1

This hypothesis predicted that people who lost their jobs between interviews would have a higher risk of major depression by reinterview than those who did not lose jobs. For this test, the sample consisted of those who were working and not currently diagnosed as major depressives at first interview and still in the work force (i.e., working or receiving unemployment compensation) at reinterview (n = 3,574 with nonmissing data on all variables). The results are shown in Table II. Although people losing a job appeared to have more than twice the risk of becoming clinically depressed, this effect was not significant, perhaps because of the low frequencies and resulting lowered power. Becoming clinically depressed at reinterview was significantly associated with just two variables: gender and lifetime depression. Women were much more likely to become depressed than men, and people who had major depression before the first interview were much more likely to become clinically depressed at second interview. The sizes of these effects were virtually the same regardless of whether the nonsignificant effect of becoming unemployed was included in the model. When both Time 1 unemployment rate and change in unemployment rate were entered simultaneously, neither approached significance. Nor were there any significant interactions. For example, history of depression did not interact with job loss in affecting Time 2 clinical depression.

Predictor	β	Error	Odds ratio			
Gender	-0.47 <sup>b</sup>	0.14	0.63			
Depression symptom count at Time 1	0.78 <sup>b</sup>	0.08	2.16			
Became unemployed	0.73 <sup>a</sup>	0.33	2.08			
Intercept	-2.30 <sup>b</sup>	0.10				

 
 Table III. Symptoms of Depression at Reinterview: Logistic Regression Panel Analysis for Three Sites

Overall model: Satterthwaite-adjusted F(3.99) = 290.06, p < .001

 ${}^{a}_{b}p < .05.$  ${}^{b}_{b}p < .001.$ 

Because of the possibility that job loss might affect anxiety either alone or in conjunction with depression, the above analysis was applied twice more. Becoming unemployed was significantly related neither to clinical anxiety alone nor to the combination of anxiety or depression. Moreover, the odds ratio of the job loss effect was smaller (<1.7) for anxiety or anxiety plus depression than it was for depression alone (>2.2).

### Hypothesis 2

This hypothesis predicted that job loss would raise the risk of having one or more symptoms of depression on reinterview. This hypothesis received support as shown in Table III. Those respondents who were working at Time 1 but who were receiving unemployment compensation by Time 2 were more than twice as likely as those still employed to have one or more current symptoms of depression by Time 2. This association persisted despite controls for gender and Time 1 symptom count. Another control variable approached significance, Other Diagnoses at first interview (if entered next,  $\beta = .37$ , p < .06), but its inclusion had little effect on the coefficients in Table III. As with the test for the first hypothesis, aggregate unemployment did not relate significantly to depressive symptoms nor were there any significant interactions.

Predictor	β	Error	Odds ratio
Diagnosis other than depression	0.72 <sup>b</sup>	0.31	2.05
Unemployment rate at first interview	0.45 <sup>c</sup>	0.16	1.57
Change in unemployment rate Time 2-Time 1	0.86 <sup>d</sup>	0.14	2.36
Intercept	-7.35 <sup>d</sup>	1.31	

 
 Table IV. Becoming Unemployed by Reinterview: Logistic Regression Panel Analysis for Three Sites<sup>a</sup>

Overall model: Satterthwaite-adjusted F(3.93) = 223.44, p < .001

<sup>a</sup> If Major Depression at Time 1 is entered next,  $\beta = -0.23$ , error = 0.98 and odds ratio = 0.79. Power of this test = .24. <sup>b</sup> p < .05.

 $p^{c} p < .01.$  $p^{d} p < .001.$ 

When the same type of analysis was repeated for symptoms of anxiety, there was no significant effect of job loss. This negative finding held true both in logistic regression using a dichotomous dependent variable and in the more powerful least squares regression using a continuous measure of anxiety symptoms (made possible because anxiety symptoms were more normally distributed than were depressive symptoms).

## Hypothesis 3

The third hypothesis predicted that those who were clinically depressed at first interview would be more likely to lose a job than their nondepressed counterparts. The sample of this analysis included all those working at Time 1 both with and without major depression diagnoses (n = 4,075 with complete data for all variables). Table IV shows the best fit model for this hypothesis, in which major depression at Time 1 made no significant contribution. In separate and joint analyses, neither lifetime major depression nor current major depression at Time 1 predicted becoming unemployed. In contrast, diagnoses other than depression and both measures of aggregate unemployment did predict becoming unemployed.

### DISCUSSION

#### Summary of Findings

Tests of the first two hypotheses give mixed results. Respondents who were not diagnosed as having major depression at Time 1, were not significantly more likely to be so diagnosed after losing a job. In contrast, becoming unemployed did raise significantly the risk of depressive symptoms. Although these tests give different answers in terms of inferential statistics, they yield similar findings in descriptive terms. In both cases (Tables II and III), becoming unemployed appears to double the risk of depression, whether measured as clinical disorder or in terms of symptoms.

The test for Hypothesis 3 reveals no support for the reverse causal direction from clinical depression to job loss. The present findings suggest two kinds of explanations. The first, or full compliance argument, is that the intent of labor law is being met in that persons who collect unemployment compensation are supposed to have lost jobs due to slack demand for labor rather than personal characteristics. If the law were being applied correctly, persons with psychological problems such as major depression would, as found here, be no more likely to collect unemployment compensation than other workers. At odds with this view is the finding (Table IV) that diagnosis other than depression predicts becoming unemployed. The second, or selective layoff argument, suspects that employers do not comply with labor law and in fact lay off persons with behavioral problems while attributing the action to slack demand for labor. Our findings could be consistent with this possibility if major depression does not lead to behaviors that make workers so undesirable that an employer would use the claim of slack work to lay them off but that other kinds of disorders do entail such symptoms. Other research based on the ECA data has reported that clinically significant alcohol abuse puts workers at elevated risk of moving from employment to receiving unemployment compensation (Dooley, Catalano, & Hough, 1992). The combined findings therefore support the selective lavoff argument.

Tests for both Hypotheses 1 and 2 find no evidence for a direct or interactive effect of aggregate unemployment rate on depression controlling for job loss. However, as shown in the test of Hypothesis 3, workers living in communities with higher unemployment rates at Time 1 or experiencing greater increases in local unemployment rates from Time 1 to Time 2 are at greater risk of job loss by Time 2. Thus, aggregate unemployment rates appear to have an indirect effect, via the intervening variable of job loss, on depressive symptoms.

### Interpretive Problems

The first interpretive difficulty, apparent in the differences in findings for the first two hypotheses, involves the risk of Type II error (J. Cohen, 1977). Although the odds ratios for the effect of job loss were similar in these two tests (both >2), the first failed to reach significance while the second did. The power to detect a significant effect in these analyses depends crucially on the frequencies of rarely occurring events: job loss and diagnosed depression (see Table I). Neither test had good power (usually set at .80), but the first test was much lower (.15) than the second (.53). Given the effect size observed in the first hypothesis test, a sample about eight times as large would have been required to raise the power to .80. Given the present sample of 3,574, over 28,000 respondents would have been needed, each meeting the requirement of being employed at Time 1, not clinically depressed at Time 1, and still in the work force at Time 2. We know of no panel study that meets these requirements. This power analysis suggests that the negative findings for Hypothesis 1 may well be a case of Type II error and that future analyses of clinical depression and unemployment must await a much larger scale survey.

The second interpretive problem involves the conservative nature of the job loss variable operationalized in this study as receiving unemployment compensation at reinterview. As indicated in the Methods section, this measure omits an unknown number of job losers who either ran out of unemployment insurance or who were not qualified to receive it. Thus the relatively strong effect observed for this measure on clinical depression (odds ratio of 2.23) probably underestimates the true adverse impact of job loss in the population. Efforts to estimate empirically the extent of this underestimation were unsuccessful. Although the ECA data do not identify nonworking people who are looking for work, these data can be used to assign nonworking people to categories such as full-time student, retired, or disabled. By elimination of people in these categories, it is possible to identify individuals who were employed at Time 1, not working or receiving unemployment compensation at Time 2, and not falling in any of the known out of the labor force categories. These "other" nonworking respondents should include those individuals who ran out of unemployment insurance or never qualified for it. Unfortunately, this category would also include other persons such as those taking voluntary leave from their jobs to travel or work on personal projects and those fired for cause, two groups which we would not regard as victims of economic recession. Nevertheless, we tested the effect of membership in this other nonworking group on depressive symptoms (the higher power test) and found it both lower in magnitude than the effect of being in the unemployment compensation group and also not statistically significant. In sum, there is good reason to suppose the depressive effect of job loss to be greater than the estimate provided here, but the available data do not allow us to specify it.

The third interpretive problem pertains to the inference of causal direction. Taken together, the results for Hypotheses 2 and 3 seem to support the view that job loss can cause depressive symptoms by a stress process over the view that disorder causes job loss by a process of selection. In fact, although consistent with this interpretation, the present data cannot themselves decide this question. The findings for Hypothesis 2 show an association between job loss and experiencing increased symptoms of depression in the year since the first interview, but the exact sequence of these phenomena cannot be established from the interview items used in the ECA project. That is, one could entertain the rival view that respondents had an increase in depressive symptoms shortly after the first interview and that this change in their mental status led later to job loss. Nevertheless, there are several circumstantial reasons to favor the stress model interpretation of these data over the rival selection view.

First, the findings for Hypothesis 3 show no connection between major depression at Time 1 and job loss although other diagnostic categories did predict job loss. If major depression does not predict job loss, it seems implausible that the increase of a few depressive symptoms would lead to job loss.

Second, it is possible that those who were depressed at first interview did lose jobs due to their psychological status but that employers fired them for cause rather than laid them off due to slack demand. This should preclude the job loser from collecting unemployment compensation. We tested this possibility indirectly by retesting Hypothesis 3 with the dependent variable being change from being employed at Time 1 to not working but not collecting unemployment compensation at Time 2. For people entering welfare or unknown not-working (no other role specified such as student, homemaker, retired) categories, the results were the same as the original test in that depression did not increase the risk of job loss. Firstinterview depression predicted only one nonemployed outcome, that of going onto disability.

Finally, the best evidence of the sequence of depressive symptoms and job loss would consist of frequent, diary-like monitoring of feelings and events. Absent such an analysis, the best available data come from retrospective reports about the timing of depression onset relative to job loss. Such studies imply that, for some people, the unemployment experience precedes depression onset, typically with a lag of a few months (Eales, 1988).

### Implications

In sum, the results favor the social causation explanation that losing a job leads, in the short term of a year or less, to an increase in symptoms of depression. This study does not establish, perhaps for want of analytic power, that this depressive reaction to job loss would become so severe or pervasive as to warrant a diagnosis of major depression. It should be noted that this study only followed respondents for 1 year and, in many cases, the unemployment experience of the job losers had lasted for only a few months because of the 26-week limitation on unemployment insurance. This study cannot tell the long-term effects of extended unemployment or of skidding into a permanently lower income level upon reemployment in a lower status occupation. The moderate increase in depressive symptoms observed in short-term job losers in this study could, without satisfactory reemployment, well turn into more severe clinical depression over time. The present study should prompt further research on possible long-term effects of job loss on severe depression.

Pending such longer term follow-ups, the present findings may not heighten public policy concerns regarding the social costs of unemployment. No one will be surprised to find that job loss is followed by depressive symptoms. The free enterprise system presumes that workers can tolerate such subclinical levels of distress, but it promises that such workers will eventually be more than compensated for their discomfort by the production efficiencies that accompany economic restructuring. Depressive mood can, of course, be politically important insofar as it motivates its victims and those who care about them to demand economic change at the ballot box. However, political institutions react to pressure from assertive constituents, and increases in the incidence of clinical depression may have relatively little effect on policy to the extent that its victims are unable to act forcefully on their own behalf.

Without evidence that unemployment significantly raises the risk of clinical depression, the case for reducing the social costs of unemployment may depend on other disorders. Evidence exists that alcohol abuse is increased among job losers, although the findings suggest that the connection between the incidence of this disorder and the economy is a complicated one (Catalano, Dooley, Wilson, & Hough, 1993). The reasons for the apparent differences in the strength of effects of job loss on alcohol abuse and major depression are not clear. However, one potentially relevant difference between these disorders involves gender. Women are much more at risk for depression, men are more at risk for alcohol disorder, and stressful events appear to operate in outcome-specific ways that differ by gender (Aneshensel, Rutter, & Lachenbruch, 1991). Although no unemployment by gender interaction appeared in the present analysis, future research might consider how the particular stressor of job loss could produce different kinds of diagnostic outcomes by gender. Such further studies could also explore the intergender transmission of economic stress as from unemployed husband to spouse (Rook, Dooley, & Catalano, 1991).

Finally, even if the linkage of job loss and depressed mood does not guide economic policy for primary prevention, it can guide mental health practitioners. Therapists working in economically depressed communities can expect increased complaints of demoralization and might consider targeting interventions to families coping with a member's job loss. Such secondary prevention could try to reduce maladaptive responses to unemployment that might lead to more severe or long-lasting depression or other disorders (Vinokur et al., 1991).

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