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PSYCHOLOGICAL DISTRESS AND MORTALITY: EVIDENCE FROM THE ALAMEDA COUNTY STUDY

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Abstract—The relationship between psychological distress, in this case depression, and subsequent risk of mortality is examined using data from the Alameda County (California) Study, an 18-yr, three-wave prospective investigation of psychosocial risk factors and health. The results indicate no relationship between psychological morbidity and all-cause mortality or specific causes of death. While these results are discordant with those reported from a majority of studies of psychiatric patients, they are concordant with a majority of community-based studies of the general adult population. Possible methodologic explanations are discussed which might account for disparate results reported to date, in particular failure to control for the effects of co-morbidity of somatic disorders and socioeconomic status.

Key words—psychological disorder, depression, mortality, community survey

INTRODUCTION

One of the more consistent findings reported historically in the mental health literature is the elevated risk of mortality among persons with psychological disorders [1–3]. The bulk of the data on this issue emanates from studies of either patients in treatment or studies of highly selective nonpatient groups [4–9], usually males, selected initially as students or industrial workers. The consistency of the findings, both across diverse studies and across time, is remarkable. With few exceptions [10, 11], those persons classified as 'psychologically impaired' at baseline are subsequently at greater risk of premature death.

The association between psychological dysfunction and elevated risk of death has been found to be largely attributable to increased risk of death from 'unnatural' causes, particularly suicide [12, 13], although several recent studies in Israel [3] and in France [14] report increased risk of all-cause mortality as well as from 'natural' and 'unnatural' causes of death. The increased risk of unnatural deaths, in particular suicide, is in general greater for persons manifesting mood or affective disorders [15–17], although there are findings to the contrary [10, 11]. There also is an elevated risk of suicide for persons with eating disorders [18, 19]. There is some evidence that the trend over the past several decades has been toward a decrease in the risk of death, at least among psychiatric patients [1]. Even so, current evidence suggests that there remains an excess risk of death for persons with psychological disorders, based on the experience of persons diagnosed and treated for such disorders [3].

There is a third group of studies which are community-based epidemiologic investigations. In these studies, the subjects are unselected in terms of prior mental or physical morbidity or other characteristics (such as age, gender, occupation, or education) and therefore constitute a more accurate representation of the communities in which they live. Such studies probably reflect a more representative spectrum of illness as well. There are many fewer of these community studies of the relationship between psychological dysfunction and risk of all-cause mortality. Six of these studies have been conducted in the United States, one in Canada, and two in Europe. The results from these community-based research efforts do not present as coherent a picture as those from the other two groups of studies, nor are they consistent with them. Six of these community studies have failed to find any relationship between psychological dysfunction and all-cause mortality. These studies have been carried out at different times in diverse locations: Midtown Manhattan [20]; Missouri and Maryland [21]; New Haven, Connecticut [22]; Piedmont, North Carolina [23]; Denmark [24]; and Lundby, Sweden [25, 26]. Three studies have found such an association; these were carried out in Stirling County, Canada [27], New Haven [28], and Evans County, Georgia [29].

Interpretation of results from these community-based investigations is problematic from several perspectives. First, the findings are equivocal. Only three of the nine community surveys, Stirling County in Canada [27], the New Haven ECA [28] and Evans County, Georgia [29], found a risk of excess all-cause mortality. The Midtown Manhattan [20] and Lundby, Sweden [25, 26] studies found some elevated risk for causes of death such as suicides and accidents among those depressed at baseline, but not for overall mortality.

Second, there are design characteristics which *a priori* place limitations on the findings. One of these involves sample size. In all of the community studies the samples are rather small, ranging in size from 938 to 3007 at baseline. Normally these are not small samples, but in the case of mortality as an outcome, these samples yielded small numbers of deaths, particularly from unnatural causes. Another problem is

that only two studies [22, 27] assessed mental health status of subjects subsequent to baseline to predict future mortality. The fluctuations in mental health status reported in community surveys [30–33] suggest that lack of data on mental health status of subjects over a long period subsequent to baseline observation could attenuate the observed relationship with mortality. The studies also vary considerably in terms of case ascertainment procedures. In only five of the studies—the two ECA sites, Denmark, Lundby, and Sterling County—can it be said that more serious forms of mental illness were distinguished from milder forms.

Although most of the community-based studies attempted to assess the role of somatic illness in the relationship between psychological impairment and mortality, only two [23, 27] attempted to control for severity of illness and only one [23] of these incorporated a measure of functional impairment and disability.

Finally, several of the studies experienced substantial attrition. For example, only 68% of the Midtown sample were located at follow-up, while in the first New Haven study, data were available at all three time periods for only 55% of the original sample. Attrition reduces effective sample size, thereby reducing the number of deaths available for study. This is a serious problem in all but the Lundby and ECA studies. Attrition also is a problem because of the bias that may be introduced, although in several of the studies the effect of attrition was assessed and found to be negligible [20, 22, 28].

The purpose here is to use data from the Alameda County longitudinal health study to reexamine the question of whether impaired psychological functioning is associated with an increased risk of death. The Alameda County Study provides a good opportunity for such an assessment. The study represents the largest sample at baseline of all the community-based surveys yet reported. In addition, there were assessments of mental health status subsequent to the baseline survey which can be used to predict mortality over a long follow-up period. Rigorous tracing procedures yielded a much lower rate of subjects lost to follow-up than in previous studies [20, 21, 22, 27]. Finally, our measures of physical health status provide data on both milder and more severe impairment, including disability. Taken together, these design attributes should permit a rigorous reexamination of this research question.

METHODS

Study population

The data in this report come from ongoing studies conducted by the Human Population Laboratory in Alameda County, California. In 1965, 8023 noninstitutionalized adults (aged 20 yr and over, or 16 yr and over if ever married) in 4452 housing units were selected on the basis of a stratified systematic sample of Alameda County housing units. The sampling procedures are discussed in greater detail in Hochstim [34] and Berkman and Breslow [35]. The sample subjects were asked to complete an extensive questionnaire about behavioral, social, and psychologic aspects of their lives. The goal of the Alameda County Study has been to examine the contribution of these factors to subsequent morbidity and mortality. Completed questionnaires were received from 6928 (86%) of the respondents. When compared with respondents, the nonrespondent group contained more older people, males, whites, and single or widowed persons. This original group of 6928 persons included 3158 men and 3770 women, aged 16–94 yr.

In 1974, an attempt was made to recontact those who responded in 1965. Vital status of the 1965 respondents was ascertained by means of a computerized record linkage to the state of California's registry of deaths and through attempted contacts with the respondents or references previously supplied by them. Only 302 (4.4%) of the original panel members were lost to follow-up over this 9-yr period. After these previous respondents were located, questionnaires were placed with them, personally or by mail, depending on location, and vigorous efforts to maximize response were followed [35]. Completed questionnaires were received from 4864 (85.1%) of the 5714 surviving respondents who were located.

Further information on the tracing, mortality ascertainment, and differences between panel members who participated in 1965–1974 and those who participated only in 1965 is presented in Belloc and Arellano [36], Wiley and Camacho [37], and Berkman and Breslow [35].

The same procedures used to ascertain mortality in 1974 were employed in a 1983 follow-up. Thus, outcome data on all-cause mortality were available for 1965–1982. Of the 6928 subjects who completed questionnaires in 1965, 1174 were known to have died through 1983; respondents not known to have died are assumed to be alive. Underascertainment of deaths appears to be slight; during the first 9 yr of follow-up it was 4.4% and during the next 8 yr it was 5.7% [38].

Measures

The measure of psychological distress used here has been utilized in several other studies [39–41]. A score is generated by assigning one point for each true or false answer which is indicative of a 'distressed' response and for each 'often' or 'never' response (whichever is appropriate). The 18 items primarily relate to depressive symptomatology (see Table 1)

Table 1. Items in Human Population Laboratory Index of psychological dysfunction

Felt depressed or very unhappy
Appetite poor
Trouble getting to sleep or staying asleep
Felt lonely or remote from other people
Felt on top of the world
Felt too tired even to do things I enjoy
Little enjoyment from leisure time
Less energy than other people
Felt pleased about accomplishing something
Felt bored
Felt so restless, could not sit still long
Felt left out, even in a group
Felt excited or interested in something
Hard to feel close to others
Never satisfied with performance
Cannot relax easily
Bothered by getting tired in a short time
Felt vaguely uneasy without knowing why

and have item-total correlations ranging from 0.18 to 0.45 as well as acceptable internal consistency reliability. Coefficient alpha was 0.77 for the total sample, 0.75 for males, and 0.77 for females. Reliabilities using the Spearman–Brown split-half procedure were 0.77 for the total sample, 0.74 for males, and 0.79 for females [38].

Comparison of these 18 items with those contained in other brief symptom checklists such as the Langner 22-item index [42], the Center for Epidemiologic Studies Depression Scale [43], the Health Opinion Survey [44], and the Symptom Check List 90 [45] suggest that these various measures can be considered conceptually equivalent. Indeed, most such symptom checklists probably tap a common underlying dimension (demoralization or psychological distress) and, as a result, can be expected to correlate about as highly as their reliabilities permit. In fact, our own research indicates that the Human Population Laboratory 18-item index is correlated 0.66 with the Beck Depression Inventory [46] in an outpatient clinical population. This correlation is about as high as can be expected based on the reliability of these two instruments.

Depressive status in 1965 and 1974 is scored as a categorical variable for purposes of analysis: low (0–1 symptoms), moderate (2–4 symptoms), and marked (5 or more symptoms). Using this scoring procedure, 14.8% of the 1965 sample were classified as having experienced marked distress and 36.2% moderate distress; in 1974 15.6% reported marked distress and 33.4% reported moderate distress.

To examine change in distress we use the nine-fold classification that results from all possible combinations of the 1965 and 1974 scores (e.g. low/low, low/marked, moderate/marked, etc.); this approach allows us to examine magnitude and direction of change while controlling for absolute levels of distress at both times.

Other measures used as covariates in the analysis of the association between depressive symptoms and mortality include age in years in 1965; sex; years of education (0–8, 9–11, 12, 13+ yr); self-reports of heart disease, high blood pressure, diabetes, cancer, chest pains, or shortness of breath within the past year; functional disability in the form of difficulty climbing stairs, getting outdoors, or being unable to work for the preceding 6 months or longer; perceived overall health rated as excellent, good, fair, or poor. All of these have been shown to be associated with both mortality and depression in this sample [38, 41, 47, 48]. In analyses of baseline depression and 1965–1974 mortality all covariate measures are from the baseline data collection; in the analyses of 1965–1974 change in depression and 1974–1983 mortality we use measures of education and of physical health and disability taken at the 1974 follow-up.

Analyses

This analysis addresses the following three questions:

(1) Is there an association between level of depressive symptoms measured at baseline (1965) and all-cause mortality in the subsequent 9 yr (1965–1974)?
(2) Is there an association between *changes* in level of depressive symptoms between 1965 and 1974 and all-cause mortality in the next 9 yr (1974–1983)?
(3) Does adjustment for demographic, socioeconomic, and physical health covariates of depression alter the association(s) found in (1) or (2) above?

In addressing questions (1) and (2) we begin by examining sex-specific age-adjusted mortality rates for the appropriate time periods first by 1965 depression and then by change in depression between 1965 and 1974. In each case we then proceed with a series of logistic regression analyses in which the relationship of depression or depression change to subsequent mortality is modeled with adjustments for socioeconomic and physical health measures in successive steps.

In logistic analyses of baseline depressive symptoms and 1965–1974 mortality the trichotomous depression variable is represented by two dummy variables, one each for moderate and marked depression, referenced to low depression. Similarly, the nine-category depression change variable is represented by eight dummy variables referenced to the category representing low depression in both 1965 and 1974. This use of dummy variables allows us to look for any nonlinear associations that may be present.

RESULTS

In Table 2 we present the crude and age-adjusted 9-yr all-cause mortality rates for men and women at each of the three levels of the 1965 depression score. The very high crude mortality rate observed among markedly depressed men and women is reduced with age-adjustment, but the age-adjusted rates also show a clear positive association with depression for both genders.

The same pattern is observed in the logistic regression analysis shown in Table 3 (Model No. 1), where depression (now represented by two dummy variables), age and gender are entered simultaneously into the model; the odds ratios for moderate and marked depression, compared to low depression, are 1.4 and 2.1 respectively, and both are significant ($P < 0.05$).

Table 2. 1965 Depression and 9 yr mortality experience by gender, crude, and age-adjusted rates

	All			Men			Women		
	N	%	Age-adjusted rate	*N*	%	Age-adjusted rate	*N*	%	Age-adjusted rate
Low (0–1)	3375	7.4	8.6	1641	8.3	9.7	1734	6.5	7.6
Moderate (2–4)	2488	11.3	10.6	1114	13.6	12.8	1374	9.3	9.0
Marked (5+)	1015	17.0	13.8	375	20.5	16.2	640	15.0	12.5

Table 3. 1965–74 Mortality by 1965 depression score adjusted for age, gender, education, and various physical health measures ($N = 6755$, deaths = 673)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.
Age	1.10	1.09–1.11	1.10	1.09–1.11	1.10	1.09–1.10	1.09	1.08–1.10	1.09	1.08–1.10
Male	1.80	1.49–2.17	1.79	1.49–2.17	1.84	1.52–2.23	1.89	1.56–2.30	1.88	1.55–2.28
Moderate depression	1.42	1.16–1.75	1.42	1.15–1.74	1.29	1.04–1.59	1.18	1.95–1.46	1.11	0.89–1.38
Marked depression	2.12	1.65–2.72	2.10	1.63–2.71	1.56	1.18–2.05	1.28	0.96–1.71	1.07	0.79–1.45
0–8 yr school			1.07	0.83–1.39	1.01	1.78–1.31	1.05	1.80–1.37	0.96	0.74–1.26
9–11 yr school			1.12	1.84–1.50	1.08	0.80–1.44	1.10	1.82–1.48	1.03	0.77–1.39
12 yr school			0.96	1.73–1.26	0.96	0.73–1.26	0.99	0.75–1.30	0.97	0.73–1.28
Conditions										
High blood pressure					1.27	1.00–1.62	1.23	0.97–1.57	1.19	0.93–1.51
Heart disease					1.81	1.26–2.59	1.51	1.04–2.18	1.43	0.99–2.07
Diabetes					2.00	1.31–3.04	1.94	1.28–2.96	1.77	1.16–2.70
Cancer					1.97	1.15–3.36	1.83	1.07–3.14	1.69	0.98–2.90
Chest pains					0.85	0.60–1.18	0.78	1.56–1.10	0.74	0.53–1.05
Shortness of breath					1.79	1.37–2.34	1.65	1.25–2.15	1.53	1.16–2.01
Disabled							1.88	1.49–2.38	1.64	1.29–2.10
Perceived health									1.40	1.19–1.63

Beginning with Model No. 2 in Table 3 we introduce other covariates of depression into the logistic model to see whether the mutual associations of depression and mortality with these covariates can account for the relationships we observed in earlier models. The addition of education level to the model does not change the previously observed relationships appreciably (Model No. 2). When adjustment is made for chronic physical conditions and symptoms (Model No. 3) the odds ratio (OR) associated with marked depression decreases to 1.6, although the OR for moderate depression remains very nearly the same as before, and both variables are still significant predictors. However, when the functional disability measure is added to this model (Model No. 4), the ORs associated with the two depression variables decline even further, and become nonsignificant. Finally, the addition of perceived health, another strong predictor of mortality in this sample, to the logistic analysis results in even smaller ORs for moderate and, especially, marked depression, both now approx. 1.1 and nonsignificant (Model No. 5). In sum, it appears from this series of models that the observed relationship between baseline depression and all-cause mortality over the subsequent 9 yr can be explained by the covariance of depression and a set of parameters of physical health, most notably functional disability and perceived health.

Depression change

Table 4 shows age-adjusted mortality rates for the nine possible combinations of 1965 and 1974 depression scores. Overall, the association between 1974 depression and 1974–1983 mortality is the same as that between 1965 depression and 1965–1974 mortality, i.e. there is a positive association between 1974 depression and 1974–1983 mortality; in general, this is true regardless of the level of depression in 1965 (although for men with low or moderate levels of depression in 1965 this relationship is not completely linear).

For both genders the very lowest mortality rates are found in the group of respondents who went from marked depression in 1965 to low depression in 1974, suggesting that a lessening of depressive symptoms, rather than an absence of a history of depression has the greater impact on subsequent mortality. However, further examination of this table reveals that, for both genders, all three of the groups with low depression in 1974 are included in the set of four groups with the lowest mortality rates—marked/low, moderate/low, low/low, and moderate/moderate—suggesting that mortality is predicted by recent depression levels irrespective of prior states. At the other end of the spectrum, the very highest mortality rates are found among those who go from moderate depression in 1965 to marked in 1974; and, for both genders the two categories with the next highest mortality rates are marked/marked and marked/moderate; these figures would support a view that chronicity of depression, as well as recent high levels, takes its toll in terms of subsequent mortality.

In Table 5 we examine these same relationships in a logistic model, adding various potential confounding variables in successive steps. Model No. 1 reveals the same relationships as seen in the age-adjusted rates, but allows us to see that the only depression change measures which are significant predictors of mortality are those that represent continuous high levels of depression—moderate/marked and marked/marked. Respondents who move to low levels of depression, from either moderate or marked levels in 1965 have odds ratios less than 1, but they are not significant when compared to the reference category of those who had low levels of depression at both

Table 4. Age-adjusted mortality rates, 1974–82 by 1965 and 1974 depression scores for men and women

	All ($N = 4767$)			Men ($N = 2109$)			Women ($N = 2658$)		
1965 Depression score	1974 Depression scale			1974 Depression scale			1974 Depression scale		
	Low	Moderate	Marked	Low	Moderate	Marked	Low	Moderate	Marked
Low	0.121	0.133	0.139	0.129	0.168	0.155	0.111	0.113	0.125
Moderate	0.113	0.121	0.168	0.143	0.134	0.213	0.094	0.110	0.143
Marked	0.073	0.143	0.145	0.106	0.170	0.198	0.032	0.133	0.131

Table 5. 1974–83 Mortality by combinations of 1965 and 1974 (trichotomous) depression scores adjusted for age, gender, and education ($N = 4692$, deaths = 466)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.
Age (in yr)	1.11	1.10–1.11	1.10	1.09–1.11	1.10	1.09–1.11	1.10	1.09–1.11	1.10	1.09–1.11
Gender = male	1.75	1.40–2.18	1.76	1.41–2.20	1.86	1.48–2.33	1.88	1.50–2.36	1.87	1.49–2.35
1965/74 Depression scores										
(Low/low)	1.00		1.00		1.00		1.00		1.00	
Low/moderate	1.20	0.86–1.69	1.19	0.85–1.66	1.07	0.76–1.50	1.00	0.71–1.42	0.92	0.65–1.30
Low/marked	1.56	0.83–2.93	1.50	0.80–2.82	1.13	0.60–2.16	1.00	0.52–1.92	0.81	0.42–1.57
Moderate/low	0.92	0.63–1.34	0.91	0.63–1.33	0.87	0.59–1.27	0.85	0.58–1.25	0.80	0.55–1.18
Moderate/moderate	0.99	0.71–1.38	0.98	0.70–1.36	0.83	0.59–1.17	0.79	0.56–1.11	0.70	0.49–0.99
Moderate/marked	1.85	1.22–2.80	1.79	1.18–2.72	1.24	0.80–1.94	1.09	0.70–1.72	0.87	0.55–1.39
Marked/low	0.71	0.26–1.93	0.71	0.26–1.92	0.69	0.25–1.88	0.66	0.24–1.81	0.61	0.22–1.68
Marked/moderate	1.41	0.82–2.41	1.39	0.81–2.38	1.12	0.65–1.94	1.03	0.59–1.80	0.92	0.53–1.62
Marked/marked	1.70	1.11–2.59	1.66	1.08–2.53	1.10	0.70–1.73	0.97	0.61–1.55	0.71	0.44–1.16
Education										
0–8 yr school			1.11	0.82–1.51	1.09	0.80–1.48	1.10	0.81–1.51	0.99	0.72–1.36
9–11 yr school			1.33	0.96–1.83	1.24	0.90–1.72	1.25	0.90–1.73	1.16	0.84–1.62
12 yr school			1.01	0.75–1.35	0.99	0.74–1.34	1.01	0.75–1.36	0.97	0.71–1.30
(13+ yr school)			1.00		1.00		1.00		1.00	
Conditions										
High blood pressure					1.26	0.98–1.62	1.22	0.95–1.57	1.16	0.90–1.49
Heart disease					1.62	1.16–2.28	1.55	1.10–2.18	1.43	1.02–2.01
Diabetes					1.63	1.11–2.40	1.54	1.04–2.28	1.44	0.97–2.12
Cancer					2.21	1.28–3.82	2.18	1.26–3.76	2.03	1.17–3.52
Chest pains					0.93	0.65–1.33	0.89	0.62–1.28	0.84	0.59–1.21
Shortness of breath					1.88	1.39–2.54	1.72	1.26–2.35	1.55	1.13–2.11
Disabled							1.45	1.10–1.89	1.17	0.88–1.55
Perceived health									1.57	1.31–1.87

times. When education is added to the model (Model No. 2) the effects associated with depression change variables are virtually the same as in the first model. However, when 1974 physical conditions are added (Model No. 3), the association between marked depression in 1974 and 1974–1983 mortality is weakened considerably, and none of the depression change dummies is significant. Adding disability to the model (Model No. 4) has very little effect; however, in the full model (No. 5) all odds ratios associated with depression change variables are less than 1.0 and, surprisingly, the odds ratio for the group with moderate levels of depression in both waves (0.70) is significant at $P < 0.05$.

We also analyzed our data using 1965 baseline data to predict mortality over the full 18-yr follow-up period. Doing so permitted a more direct comparison with the Midtown Manhattan Study, which had a 20-yr follow-up. We obtained basically the same results as for 1965–1974, although the association was even weaker for the 1965–1983 interval (data not shown).

As a further check on the robustness of our findings using logistic regression analysis, we also analyzed the data using the depression scale as a continuous measure. Again, the results were essentially the same using the two different analytic procedures (data not shown).

The role of physical health

The data presented here suggest that the apparent association between depression and subsequent mortality which has been observed in various studies may well be spurious, and that it may be accounted for by a possibly causal relationship between physical illness and depression. We know from our previous work that global measures of physical well-being do predict subsequent depression [40]. Based on the results reported above, we have examined this relationship in more detail, with special attention to the measures of chronic disease and of functional status used in the present analysis and focusing on changes in physical status that are associated with changes in depression level (see Table 6).

Using a sample consisting only of those respondents who reported low depression levels and none of the six chronic conditions in 1965, and adjusting for age, gender and education, we found that persons who reported incident diabetes, chest pain, or shortness of breath between 1965 and 1974 were significantly more likely to also report moderate or marked depression in 1974. The odds ratios for these conditions ranged from 1.9 (chest pain) to 2.3 for both diabetes and shortness of breath. For men the association of depression with diabetes was even stronger than for the sample as a whole (OR = 3.0), while that association was nonsignificant for women. Similarly, the odds ratio for shortness of breath for men was 2.9, while for women it was 1.8. However, chest pain was a stronger predictor for women (OR = 2.4) than for men (OR = 1.7). Similar results (Table 6.II) were obtained using a sample which included respondents reporting one or more conditions in 1965, and including these conditions as adjustment variables in the model. Using this same sample, we also found that the number (0–6) of conditions reported in 1974, with adjustment for the number reported in 1965 was a highly significant predictor of incident depression in 1974; the odds ratio for a 1-unit increase in the number of conditions was 1.52 (Table 6.III). Thus an individual who developed two new conditions between 1965 and 1974 would be 2.3 times as likely to become depressed by 1974 as would the individual who developed no new conditions during that time; an increase of three conditions of the six included in this analysis would result in an odds ratio of 3.5 compared to the respondent with none of these incident conditions.

Table 6. Relationship of moderate or marked depression to 1965–1974 chronic conditions and disability 1965–1974, adjusted for age and education

	All*		Men		Women	
	OR	P	OR	P	OR	P
I. Excluding those with any conditions, 1965						
	(N = 2046)		(N = 1012)		(N = 1034)	
1974 Conditions						
Heart disease	0.7	0.23	0.8	0.65	0.6	0.34
High blood pressure	1.0	0.94	1.1	0.83	1.0	0.83
Diabetes	2.3	0.01	3.0	0.01	1.7	0.28
Cancer	1.8	0.18	1.2	0.78	2.4	0.14
Chest pain	1.9	0.01	1.7	0.11	2.4	0.01
Shortness of breath	2.3	0.001	2.9	0.001	1.8	0.05
II. Including those with conditions in 1965						
	(N = 2443)		(N = 1169)		(N = 1274)	
1965 Conditions						
Heart disease	1.1	0.76	2.0	0.30	0.8	0.71
High blood pressure	1.4	0.07	1.3	0.40	1.4	0.17
Diabetes	0.6	0.24	0.6	0.39	0.7	0.58
Cancer	1.3	0.57	1.0	0.98	1.5	0.53
Chest pain	0.9	0.77	0.7	0.27	1.2	0.57
Shortness of breath	1.2	0.40	1.6	0.16	1.0	0.91
New Conditions 1965–1974						
Heart disease	0.9	0.60	0.7	0.39	1.1	0.84
High blood pressure	1.1	0.59	1.1	0.52	1.0	0.88
Diabetes	2.1	0.01	2.8	0.01	1.4	0.44
Cancer	1.5	0.27	0.9	0.85	2.0	0.13
Chest pain	1.8	0.01	1.7	0.05	2.0	0.01
Shortness of breath	2.5	0.001	2.9	0.001	2.1	0.001
III. Including those with conditions in 1965						
No. of conditions, 1965	1.1	0.29	1.1	0.56	1.1	0.45
No. of conditions, 1974	1.5	0.001	1.5	0.001	1.5	0.001
IV. Excluding those with disability in 1965 and adjusting for 1965 conditions						
	(N = 2343)		(N = 1133)		(N = 1210)	
Disabled 1974	3.2	0.001	4.1	0.001	2.5	0.001
V. Including those with disability in 1965 and adjusting for 1965 conditions						
	(N = 2465)		(N = 1176)		(N = 1289)	
Disabled 1965	2.3	0.001	1.4	0.32	3.1	0.001
Disabled 1974	3.1	0.001	4.2	0.001	2.3	0.001

*Adjusted for gender also.

The relationship between disability and depression is even stronger (Table 6.IV). Among respondents reporting little or no depression and no recent disability in 1965, those who reported a disability in 1974 were more than 3 times as likely (4 times for men) to also report increased depression than those who had no such disability. This association persists in the presence of controls for chronic conditions in 1965 and 1974, and when baseline disability is used as an adjustment variable rather than a basis for exclusion from the sample.

Cause of death

We also examined the issue of whether depression was related to cause of death, specifically whether depression was related to increased risk of death ostensibly the consequence of psychopathology, such as senile dementia, suicide, accidents, or alcoholism. We followed the procedure of Singer *et al.* [20] in classifying deaths. For the most part, the results were negative (data not shown) largely reflecting our analyses of all-cause mortality. Part of the problem was the small number of 'unnatural' deaths. There were only 36 such deaths during the 1974–1983 interval. Analyses of the relationship between depression in 1965 (low, moderate, marked) and 'unnatural' deaths from 1965 to 1974 yielded no significant result, even before adjustment for values of variables at baseline. Analyses of depression change between 1965 and 1974 and subsequent mortality from 'unnatural' causes did identify a significant increase in risk of death for those subjects who changed from moderate to marked levels of depression during the 1965–1974 interval. Although this effect remained after adjustment for physical health status, the very small numbers of deaths involved in the 1974–1983 interval (there were 36) makes us cautious in our interpretation of these results.

Our results lend strong support to the hypothesis that depression is associated with mortality primarily because it is a product of chronic disease and/or disability. On a cautionary note, we should point out that these data do not include information on the exact point of onset, in the 1965–1974 follow-up period, of either the illness/disability events or the change in depression level and therefore we cannot say with certainty whether the illness/disability preceded the depression change or vice versa. However, given that the two events are strongly linked, as has been demonstrated here, the explanation that depression is primarily a product of the disease or disability seems plausible.

DISCUSSION

In contrast to most studies of patients [1–3], this general population investigation finds no evidence that psychological dysfunction, in this case, depressive symptomatology, is associated with increased

all-cause mortality. This finding, however, is consistent with a number of community-based surveys of nontreatment populations published to date. These studies, conducted in the U.S. [20–23], in Denmark [24], and in Sweden [25, 26] also reported no overall excess mortality among persons classified as psychologically impaired.

There also have been several studies of mortality and psychiatric disorder using data from population-based, psychiatric case registries. The results from these investigations are mixed as well. Babigian and Odoroff [49], using data from New York, found that the psychiatric population had an excess of mortality, with the risk of suicide particularly dramatic. In Scotland, Innes and Miller [50] also found excess mortality among psychiatric patients, largely attributable to suicide. In Iceland, Helgason [51] found a mortality excess among psychiatric patients, but only for severe, chronic mental disorders.

Results from Alameda County also provide little evidence of an elevated risk of death from unnatural causes from persons who are psychologically impaired. We found no effect at all in the 1965–1974 interval and an effect for only one category of depression change in the 1974–1983 interval. Only four of the previous community-based studies examined this question, with mixed results. Singer *et al.* [20] found a weak relationship, Rorsman *et al.* [26] found a relationship, and two found no relationship. On the subject of cause of death, there is additional negative evidence as well. In an earlier analysis of the Alameda County data, Kaplan and Reynolds [52] reported no association between depression at baseline and subsequent cancer morbidity or mortality. It is worth noting that the HPL results are consistent with three other studies which address this general association for men [53–55].

Only three of the longitudinal community studies reviewed have found elevated risk of all-cause mortality associated with psychiatric disorder [27–29]. What might account for this apparent lack of coherence?

These disparate findings might be attributable to differences in period, place, or procedure. It does not appear that temporal variation is a likely explanation, since three of the studies are contemporaneous: Lundby (1947–1972), Midtown (1954–1974), and Stirling County (1952–1968). The Alameda County Study, while covering a more recent time period (1965–1983), overlaps the other studies during the first follow-up interval (1965–1974), as does the Danish Study (1965–1975). The first New Haven Study (1967–1975) is congruent with the Alameda County Study. The two ECA studies are the most recent in origin (they began in 1980–1981). Geographic differences also appear to be an unlikely explanation. For such to be the case, an argument would have to be made that the mortality experience in Georgia, Stirling County, and in the New Haven ECA sample was significantly different from the other sites. Perusal of the available information suggests that this is not a plausible explanation. There is nothing to suggest that the mortality experience in Stirling County should be any more different from Midtown Manhattan, Lundby, Maryland/Missouri, the earlier New Haven sample or Alameda County, for example, than these sites are different from one another.

What about differences in procedure? There clearly was considerable variation in how psychiatric caseness was determined across the studies. Again, however, this does not seem to be a likely explanation. Six studies, all with diverse procedures for case identication (Lundby, Midtown, Alameda County, Maryland/Missouri, Denmark, New Haven, Piedmont ECA) report no overall increased risk of death for persons judged psychologically impaired. The Stirling County investigators, and those from Georgia, and the New Haven ECA used still other case-ascertainment procedures, and find such an effect.

The results from Alameda County, and the majority of the other community-based studies clearly do not corroborate the general results obtained from studies of psychiatric treatment populations. We think that there are two possible explanations for the lack of convergence between community and patient studies. One has to do with severity and chronicity of psychological dysfunction and the other with a failure to control for the potentially confounding effects of socioeconomic status and somatic illness.

The issue of severity and chronicity relates to the nature of the study populations. Community surveys generally exclude institutionalized individuals. In addition, surveys which rely on direct interviews with study subjects (as was the case with studies such as Stirling County, Midtown Manhattan, Maryland/Missouri, and both New Haven studies) typically exclude subjects who are too impaired physically or psychologically to participate. By contrast, the subjects in treatment-based studies, particularly inpatients, ostensibly represent those individuals disproportionately distributed toward the more chronically and severely impaired end of the illness continuum. Medically, and logically, one should expect the physical health and mortality experience of this latter group to be more unfavorable, particularly in view of the demonstrated association between psychiatric and somatic disorders [41, 56–60].

Tsuang and Simpson [1], in their commentary on mortality studies in psychiatry, stress the importance of including physical morbidity as a covariate in studies of the mortality risks of psychiatric populations. Goldberg *et al.* [21] also have noted the need for multivariate analyses focusing on psychiatric disorder and mortality, pointing out that the relationship is stronger in studies that do not control for, say, socioeconomic status and physical health.

As noted above, there is strong evidence for the association between somatic and psychological morbidity. Likewise, there is strong evidence for an association between socioeconomic status and health outcomes, both somatic and psychologic [57, 60, 61].

Failure to adjust for the effects of physical health status and socioeconomic status is one of the most likely explanations for the oft-reported association between psychiatric disorder and increased risk of mortality in patient-based studies. Typically such studies do not control for the effect of differential physical morbidity or social class in their samples. As a case in point, recent studies using national data from Israel [3] and from France [14] do not assess the

impact on their results of either of these variables. A reasonable hypothesis is that the observed relationship between psychiatric morbidity and mortality is at least partially a function of associated comorbid somatic health problems. Furthermore, to the extent patients differ from the general population in terms of social class, the results are likely to be a function of this differential as well.

Most of the community-based studies, on the other hand, have attempted to control for either socioeconomic status or physical health status, or both. In the case of the Alameda County data, the findings are clear. We find little evidence for a relationship between mental health status and risk for death, net of these covariates. Similar results have been reported by Singer *et al.* [20] and by Goldberg *et al.* [21], and by Rorsman *et al.* [26] and Fredman *et al.* [23]. Murphy *et al.* [27] found that the relationship between depression and mortality persisted even after controlling for age, gender, and physical health. They did not control for the effects of socioeconomic factors. Bruce and Leaf [28] also report similar findings for depression (but not other disorders). Like the Stirling County investigators, Bruce and Leaf controlled for age, gender, and physical health, but not socioeconomic status. Somervell *et al.* [29] controlled for a large number of potentially confounding factors, including indicators of physical health and socioeconomic status and still observed a relationship (albeit very weak).

Overall, three studies have found no zero-order effect of mental health on mortality risk, four have found a zero-order effect that was explained by the covariation between mental health measures and other factors, primarily somatic health problems, and three studies have found that introducing controls for covariates did not eliminate the observed zero-order relationship.

CONCLUSION

At this point, there appears to be minimal support from community-based studies concerning the deleterious health effects of psychological distress. Only three of ten studies thus far find an elevated risk of dying among persons determined to be psychologically impaired at baseline. Furthermore, the results from the community studies are discordant with the general findings from studies of treatment populations. We believe the observed discrepancies are fundamentally a function of methodological inadequacies in most studies of this topic. These inadequacies involve representativeness of the samples studied, insufficient sample sizes, lack of data on duration and severity of psychiatric disorders, lack of data on treatment received (presence, quality), and failure to examine the interaction of socioeconomic status, somatic illness, and psychological dysfunction. These design deficiencies describe in particular patient-based studies but apply to some community-based studies as well. We suspect that if studies were carried out which overcame these problems there would be no elevated risk of death associated with psychiatric disorder *per se*, with the possible exception of suicide. There is a relationship between somatic and psychic well-being, and the relationship is reciprocal and asymmetric [41, 58–60, 63, 64], but not in the direction implied by studies of psychiatric disorder and mortality. We suggest that, based on available evidence, the more tenable hypothesis is that physical illness is a major risk factor for psychological disorder, but that psychological disorder is relatively unimportant as a risk factor for somatic illness and particularly for its extreme manifestation—mortality.

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