Chronically Depressed Mood and Cancer Risk in Older Persons

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Background: Depression has been proposed as a predisposing factor for cancer, but prospective studies have been inconclusive. We examined whether a high level of depressive symptoms, present for a long time, is associated with increased risk of cancer in the elderly.

Methods: Data were obtained and analyzed from persons who lived in three communities (Massachusetts, Iowa, and Connecticut) of the Established Populations for Epidemiologic Studies of the Elderly, a prospective cohort study with a mean follow-up of 3.8 years that included 4825 persons (1708 men and 3117 women) aged 71 years and older. Chronically depressed mood was defined as present when the number of depressive symptoms exceeded specific cut points on the Center for Epidemiologic Studies-Depression scale at baseline (1988) and 3 and 6 years before baseline. New cases of cancer were identified from Medicare hospitalization records and death certificates.

Results: Of the 4825 persons studied, 146 (3.0%) were chronically depressed. The incidence rate of cancer was 30.5 per 1000 person-years for the 146 persons with chronic depression and 21.9 per 1000 person-years for the 4679 nonchronically depressed persons. After adjustment for age, sex, race, disability, hospital admissions, alcohol intake, and smoking, the hazard ratio for cancer associated with chronically depressed mood was 1.88 (95% confidence interval = 1.13–3.14). The excess risk of cancer associated with chronic depression was consistent for most types of cancer and was not specific to cigarette smokers.

Conclusion: When present for at least 6 years, depression was associated with a generally increased risk of cancer. [J Natl Cancer Inst 1998;90:1888–93]

Depression, a mental state of depressed mood characterized by feelings of sadness, despair, and discouragement, has been demonstrated to cause immune suppression by increasing the synthesis and release of adrenal corticosteroids (1–4) and by decreasing lymphocyte proliferation and natural killer cell activity (5–8). Consequently, depression has been proposed as a predisposing factor for diseases that are more likely to develop with decreased surveillance activity of the immune system, such as cancer. However, the evidence of an association between depression and cancer occurrence from prospective cohort studies is inconclusive, with findings of both a lack of association (9–11) and a weak positive association (12–15). Furthermore, studies that did show an association did so mainly among smokers (13,14). This makes the overall interpretation of their findings complicated, since results could be attributed to the fact that, among smokers, depression is associated with specific patterns of smoking behavior that increase the risk of cancer. For example, it has been proposed that depressed smokers inhale more deeply and are less likely to quit than non-depressed smokers (16–18).

All previous prospective studies used a single measurement occasion to assess depressed mood. However, any hypothesis linking depression and later development of cancer presumably implies some element of chronic mental illness. Obtaining only a single measure of depression in the absence of assessment of duration may, therefore, provide a weakened test of the hypothesis. In addition, a single estimate of mental health will often classify persons as depressed as a result of temporary stressful life circumstances or health problems present at that moment. About 55% of the persons who fulfill screening criteria for depression appear to remit during the next 2 years (19). Therefore, using multiple measurement occasions enables better identification of persons with a serious, long-term depressed condition.

This study uses data collected at three different times (1982, 1985, and 1988) over a 6-year period to identify chronically depressed older persons and investigates whether these persons were subsequently at higher risk of developing cancer than nonchronically depressed persons. The study excludes persons with cancer at or before baseline and is population based, which may increase its generalizability.

METHODS

Study Population

This study uses data from three communities of the Established Populations for Epidemiologic Studies of the Elderly (EPESE), a prospective cohort study of persons aged 65 years and older that was supported by the National Institute on Aging, National Institutes of Health. The sampling procedures and data collection methods were described in depth previously (20). During the period from January 1982 through March 1983, a survey was conducted on the entire population aged 65 years and older living in East Boston, MA, and in two counties in Iowa and on a random sample stratified by housing type and sex of the New Haven, CT, population. More than 10,000 persons, 80%–85% of those eligible, were enrolled. At the initial EPESE interview and 3 and 6 years after the initial interview, trained
interviewers administered a 90-minute questionnaire that covered a wide range of psychosocial and health-related issues. For the present study, cancer events were assessed for the time between the sixth assessment in 1988 (which is considered the baseline for the analysis presented here) until December 31, 1992.

**Data Collection**

Symptoms of depression were assessed in the interviews at baseline (1988) and 3 years (1985) and 6 years (1982) before baseline with the use of the Center for Epidemiologic Studies-Depression (CES-D) scale (21). This scale measures depressive feelings and behaviors experienced during the past week (e.g., feelings of sadness or feelings that life had been a failure, lack of appetite, having a restless sleep, or having crying spells). New Haven used the standard 20-item version with a score range of 0 to 60. East Boston and Iowa used shorter versions (10 items and 11 items, respectively) in order to minimize the burden on the participants. The scores of these shorter versions were transformed by use of the procedure recommended by Kohout et al. (22). Briefly, for men and women separately, the scores from East Boston and Iowa were standardized against the 1974–1975 National Health and Nutrition Examination Survey (NHANES I), by means of the equation $x = (x - \bar{x})/s$ where $\bar{x}$ is the participant’s raw score; $\bar{x}$ and $s$ are the sex-specific mean and standard deviation of the raw scores, respectively; and $m$ and $sd$ are the sex-specific criterion mean (mean of the standard 20-item version) and standard deviation from the NHANES I, respectively. The sex-specific mean ($m$) and standard deviation ($sd$) were generated by use of the raw scores from the full sample from each of the two sites at the first interview. The criterion mean ($\bar{x}$) and standard deviation ($sd$) were used for the calculation of depression scores from NHANES I for men and 3117 women). Of the 4825 persons who were free of incident cancer was 4825 (1672 from East Boston, 3193 from Iowa) and was also confirmed in our study for individuals from the Iowa site. Their cancer outcomes were validated by matching with the State Health Registry of Iowa’s cancer registry, which is part of the National Cancer Institute’s Surveillance, Epidemiology, and End Results (SEER) Program (31). The kappa statistic for the agreement of the MEDPAR files and death certificates with the SEER registry in ascertaining prevalent, incident, and any prevalent or incident cancer cases was 0.77, 0.83, and 0.86, respectively.

Several potentially confounding factors were assessed at the baseline interview: age; sex; ethnic origin; cigarette smoking (nonsmoker, ex-smoker, or current smoker of 1–19 or ≥20 cigarettes per day); alcohol intake (≥28.35 g [one drink] per day, assessed by self-reported frequency and number of consumed drinks of beer, wine, and spirits); number of hospital admissions during follow-up until the qualifying event or before censoring; physical disability (no disability, mobility disability according to Rosow-Breslau scale (32), and mobility disability plus disability in activities in daily life (ADL) according to the scale of Katz et al. (33)); and body mass index. Information about use of antidepressants was obtained by interviewers’ observation of the containers for all prescription and nonprescription drugs taken during the past 2 weeks. Of the original population of more than 10 000 interviewed in 1982, 6566 participants were still alive and were interviewed in 1988. We excluded 298 participants who could not be matched with MEDPAR files; 1216 participants who reported a cancer at any of the interviews, who had a hospital discharge diagnosis of cancer in the 3 years before study baseline, or who were using antiancancer drugs such as tamoxifen; and participants with two ($n = 179$) or three ($n = 48$) missing depression scores. The remaining number of participants at risk for incident cancer was 4825 (1672 from East Boston, 1918 from Iowa, and 1235 from New Haven; 1708 men and 3117 women).

**Statistical Analyses**

Associations between population characteristics and depressed mood status were studied by use of the $\chi^2$ test for categorical variables and Student’s $t$ test or the Mann–Whitney test for comparisons of continuous variables. The first cancer event that occurred during follow-up was used as a primary end point. Subjects with no cancer events were censored at December 31, 1992, or at the time of death from other causes. Cancer incidence rates were calculated according to chronically depressed mood status. Hazard ratios (HRs) and 95% confidence intervals (CIs), adjusted for potential confounders, were used as the measure of association and were computed from proportional hazards regression models stratified by community (with the use of the STRATA option of SPSS COXREG procedure (34)) in order to obtain summary estimates across the three communities. For exposure (chronically depressed mood) and for age and sex, the assumption of proportionality of hazard was checked with log minus log plots and by tests of the interaction of the variable with time. In addition, Kaplan–Meier analyses were used to check the association between chronically depressed mood and cancer incidence without the assumption of proportionality of hazard. Additional indicator variables were used for missing data for cigarette smoking, alcohol intake, and physical disability (0.9%, 0.6%, and 6.3% missing data, respectively). All statistical tests were two-sided.

**RESULTS**

The mean age of the participants at baseline was 79.0 years (range, 71–96 years); 64.6% were women and 5.2% were blacks. Of the 4825 participants, 146 (3.0%) were classified as having a chronically depressed mood, scoring above the depression cutoff of 20 at baseline and 3 and 6 years before baseline. Compared with the nonchronically depressed subjects, chronically depressed persons were older, more often female, and less often smokers or occasional drinkers (Table 1). They were more likely to be admitted to a hospital during follow-up, to use antidepressants (primarily amitriptyline), and to be physically disabled.

Of the 4825 persons who were free of cancer at baseline, 402 developed cancer during follow-up (mean follow-up time, 3.8 years). The crude incidence rate of cancer for the total population was 22.1 per 1000 person-years. Persons with a chronically depressed mood had a significantly higher crude rate of cancer than nonchronically depressed persons ($HR = 1.68; 95% CI = 1.01–2.79$) (Table 2). The interaction term between age group and sex in the proportional hazards model was nonsignificant ($P = .74$), which supports the assumption of proportionality. Also, Kaplan–Meier analyses, pooled over age and sex categories, confirmed the increased crude cancer risk associated with chronic depression ($P$ value of log-rank test = .028). Since ethnic origin, physical disability, number of hospital admissions during follow-up, smoking, and alcohol intake were associated with
Table 1. Population characteristics according to chronically depressed mood status

| Characteristic                              | No chronically depressed mood (n = 4679) | Chronically depressed mood (n = 146) | P*  
|--------------------------------------------|----------------------------------------|-------------------------------------|------
| Mean (standard deviation [SD]) age, y       | 78.9 (5.8)                             | 80.7 (5.9)                          | <.001
| Women                                      | 63.9%                                  | 85.6%                               | <.001
| Black                                      | 5.3%                                   | 0.9%                                | .014
| Cigarette smoking                          |                                        |                                     |      
| Non-smoker                                 | 61.7%                                  | 73.6%                               | .018
| Ex-smoker                                  | 29.2%                                  | 17.4%                               |      
| Current smoker                             | 5.2%                                   | 5.6%                                |      
| ≥ 20 cigarettes/day                        | 3.9%                                   | 3.5%                                |      
| Alcohol intake, ≥ 28.35 g/day              | 5.4%                                   | 0.0%                                | .004
| Physical disability                        |                                        |                                     |      
| No disability                              | 52.3%                                  | 13.0%                               | <.001
| Mobility disability                        | 33.5%                                  | 55.7%                               |      
| Mobility and disability in activities daily| 14.2%                                  | 31.3%                               |      
| Mean (SD) No. of hospital admissions during follow-up| 1.25 (2.0) | 1.96 (2.7) | <.001
| Mean (SD) body mass index in kg/m²         | 24.6 (6.3)                             | 24.4 (7.0)                          | .71
| Use of antidepressants                     | 4.3%                                   | 15.1%                               | <.001

*x² test for categorical variables; Student’s t test or Mann-Whitney test for comparison of means. All statistical tests were two-sided.

chronically depressed mood and with the incidence of cancer (P<.1), analyses were adjusted for these variables. The fully adjusted HR for chronically depressed mood in the multivariate community-stratified analyses was 1.88 (95% CI = 1.13–3.14). Use of antidepressants and body mass index were not associated with cancer incidence, and additional adjustment for these variables did not change the HR for chronically depressed mood. In a further subanalysis, it was found that the risk for the chronically depressed person was even higher when this group was compared with the 186 persons who scored zero on the CES-D scale at all three measurement occasions (fully adjusted HR = 2.43; 95% CI = 1.17–5.07).

To compare our results with results of earlier prospective studies, we examined HRs for cancer incidence in persons who were depressed at baseline (n = 575) compared with those who were not depressed at baseline (n = 3837). These analyses do not consider the depression scores at 3 and 6 years before baseline. In univariate and multivariate analyses, baseline depressed mood was not associated with an increased risk of developing cancer (age- and sex-adjusted and fully adjusted HRs were 0.95 and 1.02, respectively) (Table 2).

The most frequent cancers were those of the colon (n = 60), lung (n = 56), prostate (n = 53), lymphatic and hematopoietic organs (n = 42), urinary tract (n = 37), and breast (n = 31) (Table 3). Although the small numbers did not permit meaningful site-specific analyses, these analyses nevertheless seem to indicate that the association with chronically depressed mood was not specific to any particular site or type of cancer. In women, a significantly increased HR of 4.80 was found for cancers of the uterus and adnexa uteri. For all other cancer sites, except for breast cancer, HRs were increased but did not reach statistical significance.

Age- and sex-adjusted analyses were calculated for separate demographic strata. HRs were 2.51 (95% CI = 1.36–4.62) in persons aged 71–79 years and 1.01 (95% CI = 0.41–2.46) in persons aged 80 years and over; 1.95 (95% CI = 1.00–1.05) in men and 1.61 (95% CI = 0.87–2.97) in women; 0.86 (95% CI = 0.32–2.36) in East Boston, 2.94 (95% CI = 1.47–5.88) in Iowa, and 3.68 (95% CI = 1.12–12.10) in New Haven; and 1.60 (95% CI = 1.00–2.69) in whites and 5.54 (95% CI = 0.72–42.56) in blacks. Chronically depressed mood was associated with an increased HR for cancer in all smoking status strata. Fully adjusted HRs for chronically depressed mood were 2.13 (95% CI = 1.15–3.96) in nonsmokers, 1.19 (95% CI = 0.38–3.74) in ex-smokers, and 1.40 (95% CI = 0.32–6.10) in current smokers. Analyses stratified by cigarette smoking and chronically depressed mood status (Table 4) showed that the HR for chronically depressed nonsmokers (2.34) exceeded the HR for smokers without chronic depression (1.96). No statistically significant interaction between cigarette smoking and depressed mood status was observed.

Separate analyses used the Iowa SEER cancer registry data rather than the MEDPAR files or death certificates to define incident cases of cancer. In these analyses, the incidence rate of cancer was 12.9 per 1000 person-years among non-chronically depressed persons (95 events, 1743 participants) and 26.7 per 1000 person-years among chronically depressed persons (95 events, 146 participants) and 26.7 per 1000 person-years among chronically depressed persons (95 events, 146 participants).

Table 2. Association between cancer incidence and depressed mood

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. of events</th>
<th>Person-years</th>
<th>Rate per 1000 person-years</th>
<th>Hazard ratio* (95% confidence interval)</th>
<th>Hazard ratio, adjusted† (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronically depressed mood</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (n = 4679)</td>
<td>386</td>
<td>17630</td>
<td>21.9</td>
<td>1.0 (referent)</td>
<td>1.0 (referent)</td>
</tr>
<tr>
<td>Yes (n = 146)</td>
<td>16</td>
<td>524</td>
<td>30.5</td>
<td>1.68 (1.01–2.79)</td>
<td>1.88 (1.13–3.14)</td>
</tr>
<tr>
<td>Depressed mood at baseline</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (n = 3837)</td>
<td>345</td>
<td>14882</td>
<td>23.2</td>
<td>1.0 (referent)</td>
<td>1.0 (referent)</td>
</tr>
<tr>
<td>Yes (n = 575)</td>
<td>41</td>
<td>2098</td>
<td>19.5</td>
<td>0.95 (0.68–1.31)</td>
<td>1.02 (0.73–1.42)</td>
</tr>
</tbody>
</table>

*Adjusted for age and sex: community-stratified summary.
†Adjusted for age, sex, ethnic origin, physical disability, number of hospital admissions during follow-up, cigarette smoking, and alcohol intake: community-stratified summary.
‡Chronically depressed are those who are depressed at baseline and 3 and 6 years before baseline.
son-years among chronically depressed persons (five events, 48 participants). After adjustment for confounding factors, the HR of cancer associated with chronically depressed mood was 3.23 (95% CI = 1.26–8.23).

Finally, we analyzed the cancer mortality data. Of the 146 chronically depressed persons, 7.5% (n = 11) died because of cancer, compared with 4.1% (n = 190) of the 4679 nonchronically depressed persons. The adjusted cancer mortality risk for chronically depressed mood was 2.22 (95% CI = 1.19–4.16).

**DISCUSSION**

In this prospective study of persons aged 71 years and older, chronically depressed persons had a statistically significant 88% excess risk of developing cancer during the follow-up of, on average, 3.8 years compared with nonchronically depressed older persons. The excess risk of cancer associated with chronically depressed mood was consistent for most types of cancer and was not limited to cigarette smokers. This is the first time that the association with cancer incidence has been analyzed for *chronically depressed mood*.

Previous prospective studies measured depressed mood at one point in time only. In these measures, information about the chronicity of the condition is lacking. In addition, a single estimate of depressed mood is not particularly stable and is more likely to misclassify persons as being depressed as a result of temporary stressful life circumstances or health problems present at that moment. In our study, only 25% of the 575 persons depressed at baseline were also depressed 3 and 6 years before baseline. When using the single measure of baseline depressed mood, we found no increased HR of developing cancer, which conforms to results of previous prospective studies (9–11). However, when three different measurement occasions were used over a 6-year period of follow-up, chronically depressed mood was associated with the development of cancer. Thus, depressed mood persisting over a long period seems to be an essential element in linking depression and later development of cancer.

How can chronically depressed mood increase the risk of cancer? The consistent associations of chronically depressed mood with increase in most types of cancer found in this study suggest that the risk of cancer is increased through a common underlying biologic process. Some current evidence suggests that the immune system may play an important role in the link between depression and cancer. Several, but not all, studies on humans found decreases in mitogen-induced lymphocyte proliferation and peripheral blood natural killer cell activity among depressed versus nondepressed subjects (4,7). The underlying biologic mechanisms may be related to alterations in central nervous system activity, especially of the hypothalamic pituitary–adrenal (HPA) axis. Depressed patients have been shown to exhibit hypersecretion of the adrenal steroid cortisol, adrenal hypertrophy, and an increased cortisol response to adrenocorticotropic hormone (35). Since adrenal corticosteroids have potent immunoregulatory effects that influence leukocyte traffic and function and the immune response, increases in adrenal corticoesteroid excretion can cause immunosuppression (4,7). In addition to the role of the HPA axis, other neuroendocrine systems could play a role. Although the mechanisms are not understood in detail, depression is associated with diminished secretion of the immunostimulants prolactin and growth hormone (36).

Epidemiologic evidence suggests that immunosuppression plays a role in certain cancers. Immune-suppressed patients (patients with congenital, acquired, or iatrogenic immunosuppression) show a consistent, increased risk for a subset of cancers that have a likely viral etiology, including non-Hodgkin’s lymphoma, skin cancer, Kaposi’s sarcoma, and vulvar, anal, and cervical cancers (37). However, immune-suppressed patients have so far not shown increases in the most common types of cancer (adenocarcinomas of the lung, breast, prostate, and colon), which places limitations on the immunosuppression hypothesis.

Therefore, other alternative explanations linking depression and cancer should be considered. First, the depression and cancer link may be due to a third factor that is related to both conditions. A shared genetic predisposition can be such a factor, although genetic forces are generally more limited in an older population.

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**Table 3.** Association between chronically depressed mood and specific types of cancer

<table>
<thead>
<tr>
<th>Type of cancer (ICD-9* code)</th>
<th>No. of events</th>
<th>Hazard ratio (95% confidence interval)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stomach (151)</td>
<td>13</td>
<td>2.38 (0.29–19.65)</td>
</tr>
<tr>
<td>Colon (153)</td>
<td>60</td>
<td>1.37 (0.33–5.74)</td>
</tr>
<tr>
<td>Rectum (154)</td>
<td>22</td>
<td>2.82 (0.35–22.83)</td>
</tr>
<tr>
<td>Liver, gallbladder, pancreas (155–157)</td>
<td>24</td>
<td>1.65 (0.21–12.69)</td>
</tr>
<tr>
<td>Lung (162)</td>
<td>56</td>
<td>2.10 (0.49–8.92)</td>
</tr>
<tr>
<td>Skin (172–173)‡</td>
<td>12</td>
<td>3.35 (0.39–28.52)</td>
</tr>
<tr>
<td>Breast (174‡)</td>
<td>31</td>
<td>No depressed cases</td>
</tr>
<tr>
<td>Uterus, adnexa (182–183)‡</td>
<td>21</td>
<td>4.80 (1.01–22.79)</td>
</tr>
<tr>
<td>Prostate (185)§</td>
<td>53</td>
<td>1.47 (0.20–10.83)</td>
</tr>
<tr>
<td>Bladder, ureter, kidney (188–189)</td>
<td>37</td>
<td>2.89 (0.66–12.59)</td>
</tr>
<tr>
<td>Lymphatic, hematopoietic (200–208)</td>
<td>42</td>
<td>2.85 (0.66–12.30)</td>
</tr>
</tbody>
</table>

*International Classification of Diseases, 9th revision.
†Adjusted for age, sex, ethnic origin, physical disability, number of hospital admissions during follow-up, cigarette smoking, and alcohol intake: community-stratified summary.
‡Analyses for women only.
§Analyses for men only.

**Table 4.** Adjusted hazard ratios* (95% confidence interval) of cancer incidence, by cigarette smoking and chronically depressed mood status

<table>
<thead>
<tr>
<th>Hazard ratio (95% confidence interval)</th>
<th>No chronically depressed mood</th>
<th>Chronically depressed mood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonsmoker</td>
<td>1.0 (referent)</td>
<td>2.34 (1.26–4.35)</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>1.65 (1.29–2.10)</td>
<td>2.17 (0.68–6.88)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.96 (1.16–3.34)‡</td>
<td>2.80 (0.65–11.94)†</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, ethnic origin, physical disability, number of hospital admissions during follow-up, and alcohol intake: community-stratified summary.
†Also adjusted for number of cigarettes smoked per day.
Also, common alterations in specific central nervous system pathways (stimulated production of corticotropin-releasing hormone and immune cytokines, such as interleukins) have been shown to cause emotional disturbances as well as a decreased function of the immune system. (36).

Second, since most previous prospective studies showing a statistically significant association between depression and cancer did so mainly among smokers, differences in smoking behavior between depressed and nondepressed persons have been considered as a main explanation for the increased cancer risk among depressed persons. Depressed smokers are found to be less likely to quit smoking (17) and might inhale more deeply and smoke more of the cigarette (16) than nondepressed smokers. In our study, however, there was no interaction between depression and smoking, and increased cancer risks were found for both depressed smokers and depressed nonsmokers. Also, the sites at which chronically depressed persons showed excess cancers were not predominantly tobacco related. Consequently, differences in smoking habits are not likely to explain the increased cancer risk among chronically depressed persons in our study.

Third, it has been suggested that use of antidepressants may cause an increase in cancer risk (38). However, as in other studies (39,40), our analyses did not find an association between use of antidepressants and cancer, and use of antidepressants did not change the association between chronically depressed mood and cancer. Finally, depression may increase the probability that the individual will engage in behaviors that indirectly increase the risk of developing cancer (e.g., lack of physical activity, excessive alcohol consumption, and over-eating disorders) (41,42). Additionally, body mass index, disability, and alcohol use did not change the HR found for chronically depressed mood. Nevertheless, the preference of specific diets by depressed persons, such as foods high in fat, might partly explain our findings, although associations between fat intake and cancer incidence are generally weak (43,44).

Our study is limited by the small number of chronically depressed persons, which severely limited our ability to perform cancer site-specific analyses. In addition, occult cancer may have caused depressive symptoms (10). However, this causal explanation is not very likely, since our depressed mood measure included psychological information collected at 6 and 3 years before baseline and our study population had no reported cancer for 6 years before baseline (and no hospital records of cancer for 3 years before baseline). Chronically depressed persons may overrepresent depressed persons who did not obtain effective therapy for their condition, rather than all persons with depression. Therefore, residual confounding with cancer risk factors associated with failure to obtain treatment cannot be completely excluded. Furthermore, chronically depressed persons had higher hospitalization rates than persons without a depressed mood. In the present study, cancers were identified mainly through cancer hospital discharge records. Thus, it is possible that chronically depressed persons had a greater chance of being diagnosed earlier with cancer and also of having their cancer brought to our attention as a result of hospitalizations for other conditions. However, adjusting models for the number of hospital admissions during follow-up is not related to cancer yielded unchanged results. Also, additional adjustment for prevalent chronic diseases (e.g., coronary heart disease, stroke, and diabetes) did not change the results. The consistent results of the models based on the SEER data for persons living in Iowa as well as the finding of increased cancer mortality further suggest that such a potential ascertainment bias was limited and did not confound the results.

As in all studies, little credence can be given to our findings until they have been replicated in different populations. Our findings should stimulate further experimental and epidemiologic examination of the association between psychological factors and cancer. These studies should focus on persons with long-term depressive symptoms, the group found to be at risk in this study.

REFERENCES


NOTES

1Editor’s note: SEER is a set of geographically defined, population-based central tumor registries in the United States, operated by local nonprofit organizations under contract to the National Cancer Institute (NCI). Each registry annually submits its cases to the NCI on a computer tape. These computer tapes are then edited by the NCI and made available for analysis.

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