

Depressive Disorder, Dysthymia, and Risk of Stroke

Thirteen-Year Follow-Up From the Baltimore Epidemiologic Catchment Area Study

Sharon L. Larson, PhD; Pamela L. Owens, PhD; Daniel Ford, MD, MPH; William Eaton, PhD

Background and Purpose—This study examined depressive disorder as a risk factor for incident stroke in a prospective, population-based design.

Methods—The Baltimore Epidemiologic Catchment Area Study is a prospective 13-year follow-up of a probability sample of household residents from Baltimore, Md. Depressive disorder was measured with the diagnostic interview schedule, and stroke was assessed by questions from the health interview survey or by documentation on a death certificate.

Results—During the 13-year follow-up of 1703 individuals, 66 strokes were reported and 29 strokes were identified by death certificate search. Individuals with a history of depressive disorder were 2.6 times more likely to report stroke than those without this disorder after controlling for heart disease, hypertension, diabetes, and current and previous use of tobacco. Medications used in the treatment of depressive disorder at baseline did not alter this finding. A history of dysthymia demonstrated a similar relationship to stroke, although the estimate was not statistically significant.

Conclusions—Depressive disorder is a risk factor for stroke that appears to be independent of traditional cardiovascular risk factors. Further research on mechanisms for the association and the impact of treatment for depressive disorder on subsequent stroke is needed. (*Stroke*. 2001;32:1979-1983.)

Key Words: affective disorders ■ cerebrovascular disorders ■ depression ■ depressive disorder ■ dysthymic disorder ■ stroke

Increased interest in the relationship between affective disorder and long-term health consequences has generated recent examinations of depressive symptoms and stroke. Recently, Ohira et al¹ reported that depressive symptoms were associated with an increased incidence of ischemic, but not hemorrhagic, strokes in an older Japanese sample. Similar results have been found in an Australian sample.^{2,3} Likewise, Jonas and Mussolino⁴ found that depressive symptoms were associated with incident stroke in a longitudinal study spanning 22 years. In an earlier study, researchers found that individuals reporting 5 or more symptoms of depression at baseline were 50% more likely to die of a stroke-related cause during a 29-year follow-up.⁵

There are plausible explanations for a prospective relationship between depressive disorder (or depression symptoms) and stroke. A growing body of research is targeting the effects of depression on platelet function. Platelet activation, measured by plasma levels of platelet factor 4 and β -thromboglobulin, are higher in individuals with major depression.⁶ Musselman et al⁷ found that depressed individuals experience a heightened susceptibility to platelet activation that is associated with cardiovascular disease. Interestingly, the results of a small study indicate that platelet

function normalized after 6 weeks of treatment with paroxetine.⁸ Similarly, cardiovascular disease and depression have been linked. In one study, respondents with a history of major depression were 4 times more likely to report a myocardial infarction than were individuals without a history of depression.⁹ Ford et al¹⁰ also found that men with clinical depression were at an increased risk for coronary artery disease. Because individuals who have had a myocardial infarction are at an increased risk for stroke, these findings suggest yet another pathway by which depression may be linked to stroke.¹¹ Further evidence that there may be a relationship between depression and vascular disease is found in studies reporting that individuals with major depression appear to have higher adrenocortical activity, a risk factor for atherosclerosis and in turn a risk factor for stroke.¹²⁻¹⁴

Although researchers have examined the association between depressive symptoms and stroke by using measures of current symptoms, no research was identified that examined the relationship between lifetime reports of depressive disorder assessed by rigorous diagnostic standards. Thus, the objective of our prospective study was to examine the relationship between the lifetime occurrence of depressive disorder measured by an instrument consistent with the

Received January 17, 2001; final revision received May 25, 2001; accepted June 1, 2001.

From Johns Hopkins University Bloomberg School of Public Health (S.L.L., P.L.O., W.E.) and School of Medicine (D.F.), Baltimore, Md.

This study was funded in part by National Institute of Mental Health grant MH 47447 from the National Institutes of Health, Bethesda, Md.

Correspondence to Sharon L. Larson, PhD, Agency for Healthcare Research and Quality (AHRQ), 2101 E Jefferson #500, Rockville, MD 20852. E-mail: slarson@jhsph.edu or slarson@ahrq.gov

© 2001 American Heart Association, Inc.

Stroke is available at <http://www.strokeaha.org>

TABLE 1. Sample Characteristics for Baltimore ECA Follow-Up, 1981–1993, 1996

	Dysthymia Only (N=27) n (%)	Depressive Disorder (N=101) n (%)	No Depressive Disorder (N=1575) n (%)
Self-reported stroke	1 (3.7)	7 (6.9)	87 (5.5)
Age, y			
18–29	7 (25.9)	35 (34.6)	535 (34.0)
30–44	13 (48.2)	44 (43.6)	461 (29.3)
45–54	3 (11.1)	10 (9.9)	172 (10.9)
55–64	3 (11.1)	9 (8.9)	219 (13.9)
≥65	1 (3.7)	3 (2.9)	188 (11.9)
Female	20 (74.1)	76 (75.2)	975 (61.9)
Race/ethnicity			
White	18 (66.7)	63 (62.3)	978 (62.1)
Black	9 (33.3)	35 (34.7)	544 (34.5)
Other	0 (0)	3 (3.0)	53 (3.4)
Low socioeconomic status (Nam-Powers index ≤35)	11 (39.4)	33 (32.7)	630 (40.0)
No high school diploma	13 (48.2)	36 (34.6)	675 (42.9)
Ever diabetes	2 (7.4)	3 (2.9)	77 (4.9)
Ever heart problem	2 (7.4)	10 (9.9)	143 (9.1)
Ever high blood pressure	6 (22.2)	28 (27.7)	420 (26.7)
Currently smoke tobacco	22 (71.0)	59 (58.4)	926 (58.8)
Ever smoke tobacco	21 (77.8)	76 (73.8)	1124 (71.4)
2 Or more medical risk factors*	7 (25.9)	26 (25.2)	405 (25.7)
Tricyclic use	9 (33.3)	40 (38.3)	150 (9.5)
MAOI use	1 (3.7)	0 (0)	2 (0.1)
Lithium use	1 (3.7)	4 (3.8)	4 (0.9)

*Hypertension, heart disease, diabetes, and tobacco use.

standards presented in the Diagnostic and Statistical Manual, 3rd edition (DSM-III) and stroke.¹⁵

Methods

Research Design

The Epidemiologic Catchment Area (ECA) project is a national study that was initially funded by the National Institute of Mental Health (NIMH) between 1980 and 1983 (wave 1). The purpose of this study was to measure the prevalence and incidence of psychiatric disorders in the general population. More than 20 000 adults >18 years old were interviewed in 5 US metropolitan areas. In the Baltimore site, 3481 persons were interviewed. These respondents were selected through probability sampling within the east Baltimore area, with oversampling of those >65 years of age. This site reported an interview completion rate of 82%. Participants gave informed consent before their interview, per the study protocol approved by the Committee on Human Research of the Johns Hopkins University School of Hygiene and Public Health. Further details of the ECA study and methods are described elsewhere.¹⁶

Study Sample

Between 1993 and 1996, a follow-up study of the original Baltimore ECA cohort was conducted.¹⁷ Of the 3481 participants interviewed at baseline, 1920 were located and reinterviewed. Eight hundred forty-eight of the original respondents died during follow-up, with death certificates including cause of death obtained for 663 participants. Of the remaining 713 individuals, 298 refused to participate and 415 were lost to follow-up.¹⁸ Our sample was restricted to those individuals who indicated that they had not had a stroke at baseline

and either responded to the question “Have you ever had a stroke?” at follow-up or had a death certificate on record.

In the follow-up study, 1731 of those surveyed in 1981 responded to the question, “Have you ever had a stroke?” Twenty-six of these individuals indicated either that they had had a stroke before wave 1 or that they did not know whether they had had a stroke before wave 1; these subjects were excluded from this analysis. There were 1705 cases at risk for incident stroke during the follow-up period. The final sample for analysis excluded mania (n=2) to examine only those mood disorders related to depression. The sample for analysis had 1703 cases.

Variables

The incident stroke measure represents those who reported a first stroke during the 13-year follow-up period or for whom stroke was a cause of death reported on the death certificate. Within this sample of those at risk for a first-ever stroke, there were 66 self-reports of stroke during the follow-up period and 29 stroke-related deaths reported on death certificates. Forty-six (70%) respondents who self-reported stroke also reported an inability to perform at least 1 activity of daily living.

The diagnostic interview schedule (DIS) used in the ECA study is a structured survey instrument developed by the NIMH for this study. The interview is intended to produce diagnoses of specific mental disorders according to standards established by the American Psychiatric Association and presented in the DSM-III.¹⁵ The DIS consists of dichotomously coded questions about the presence of signs and symptoms of psychiatric illness. The psychiatric diagnoses of interest to this study were depressive disorders. Based on responses to the DIS, individuals were categorized into 3 groups. The

first group included those with dysthymia only ($n=27$). The second group consisted of those with major depression ($n=61$), as well as those reporting dysphoria and meeting the criteria in 2 or more other depression symptom groups but without reporting distress and/or impairment in important areas of functioning ($n=40$). Elsewhere this latter subgroup has been labeled "depression syndrome,"¹⁹ and we refer to the combined group (of 101 cases) as "depressive disorder." Respondents with dysthymia were included in this group if they also met the criteria for depression syndrome ($n=6$). The third group included all individuals who did not meet criteria for either depressive disorder or dysthymia ($n=1575$).

Demographic characteristics from wave 1 were controlled for in this analysis, including age, race/ethnicity, education, and sex. Age was treated as a categorical variable (18 to 29, 30 to 44, 45 to 54, 55 to 64, and 65 years or older). The youngest group, 18 to 29 years old, served as the reference group. Indicator variables were created to control for differential effects of race/ethnicity on stroke. Blacks and other minorities were compared with whites. Educational attainment was dichotomously coded, and those with no high school diploma served as the reference group. Female sex is the indicator variable for gender. The Nam-Powers socioeconomic index was calculated by using income, occupation, and prestige ratings, and this index ranges between 0 and 100.²⁰ This variable was coded as an indicator, and those with a score >35 (middle and upper socioeconomic status) served as the reference group. At baseline, respondents were also asked to indicate whether they had ever had diabetes, heart problems, high blood pressure, or currently or ever used tobacco products including cigarettes, cigars, or pipes. Current smoking indicated smoking any time during the last 7 days.

In addition, respondents were queried regarding their use of medications prescribed for the treatment of depression and depressive disorder at baseline. These included tricyclic/heterocyclic drugs, mood stabilizers such as lithium, and monoamine oxidase inhibitors (MAOIs). Analysis of the use of these medications provides information about the effect of pharmacological treatment for depression disorder at baseline on the risk for stroke.

Analytic Strategy

The 3 exposure groups, those with dysthymia only, those with depressive disorder, and those with no depressive disorder or dysthymia, were compared with respect to baseline characteristics (Table 1). Stroke incidence and relative risk (RR) were calculated according to risk factors assessed at baseline (Table 2). RR for stroke was calculated for dysthymia only and depressive disorder (Table 3, models 1 and 2) by using logistic regression and adjusting for age, sex, race, and educational attainment. Model 3 (Table 3) controlled for the presence of medical risk factors, including a history of diabetes, heart problems, high blood pressure, and current use of tobacco. We also estimated the population-attributable risk to generate some notion of how important depression might be as a risk factor for incident stroke.

Results

Over the 13-year follow-up, few individuals with dysthymia alone or depressive disorder reported a first stroke during this period (3.7% and 6.9%, respectively; Table 1). In the group of respondents with neither dysthymia nor depressive disorder, there were 87 cases of incident stroke (5.5%). Those with dysthymia only and those with depressive disorder were younger and more likely to be female, consistent with research on the epidemiology of depressive disorder.¹⁶ They were also more likely to have used psychotropic medications.

The group of respondents with depressive disorder was slightly more likely than those with no disorder to report an incident stroke (RR=1.2) in unadjusted analyses (Table 2). Age is a significant risk factor for stroke. After age 44, there is a steady increase in the incidence of stroke. About one

TABLE 2. Stroke Incidence by Risk Factor, Baltimore ECA Follow-Up 1981–1993, 1996

Risk Factors in 1981	Frequency of Stroke	Incidence of Stroke per 100	RR
History of depressive disorder			
No history	87	5.5	1.0
History of dysthymia	1	3.7	0.7
History of other depressive disorder	7	6.8	1.2
Age, y			
18–29	7	1.2	1.0
30–44	8	1.5	1.3
45–54	12	6.5	5.4
55–64	23	10.0	8.3
≥ 65	45	23.4	19.5
Female sex	68	6.3	1.5
Race/ethnicity			
White	66	6.2	1.0
Black	26	4.4	0.7
Other	3	5.4	0.9
Low socioeconomic status (Nam-Powers index ≤ 35)	46	6.9	1.5
No high school diploma	72	9.9	4.3
Ever diabetes	11	13.4	2.6
Ever heart problem	25	16.1	3.6
Ever high blood pressure	55	12.1	3.8
Currently smoke tobacco	57	5.8	1.1
Ever smoked tobacco	69	5.1	1.1
History of 2 or more medical risk factors	50	11.4	3.2

fourth (23.4 per 100) of respondents aged 65 years and older had a first stroke during the follow-up period. Educational attainment is also related to stroke: those who did not complete high school had a cumulative incidence of 9.9 strokes per 100 while those with a high school education had a cumulative incidence of only 2.3 per 100. Lower socioeconomic status was associated with a stroke incidence of 6.9 per 100 while those with a higher socioeconomic status reported 4.8 strokes per 100. A history of heart problems, hypertension, and diabetes appeared to be associated with an increased incidence of stroke. Medications used as treatment for depressive disorder were not associated with a change in the incidence of stroke.

We created a multivariate logistic model with incident stroke as the dependent variable (Table 3). After controlling for baseline characteristics of age, sex, and educational attainment, dysthymia demonstrated a tendency to be a risk factor for stroke, but confidence intervals were wide. (RR 2.65; 95% CI, 0.69 to 10.11). However, depressive disorder was significantly related to stroke after adjusting for these baseline characteristics. Those with depressive disorder were 3.08 (95% CI, 1.26 to 7.52) times more likely to report a first stroke during the 13-year follow-up than were those who did not have a depressive disorder.

TABLE 3. Affective Disorder and Incidence of Stroke, Baltimore ECA Follow-Up, 1981–1993, 1996

Model	RR (95% CI)		
	Dysthymia Only	Depressive Disorder	Depression and Medical Risk Factors
Dysthymia only	2.65 (0.69, 10.11)		
Depressive disorder		3.08 (1.26, 7.52)	2.67 (1.08, 6.63)
Age, y*			
30–44	0.62 (0.18, 2.11)	0.59 (0.17, 2.02)	0.54 (0.15, 1.86)
45–54	4.52 (1.70, 12.06)	4.48 (1.68, 11.97)	3.39 (1.25, 9.24)
55–64	7.54 (3.08, 18.45)	7.48 (3.04, 18.37)	5.77 (2.30, 14.51)
≥65	29.95 (12.71, 70.62)	25.43 (10.62, 60.88)	19.62 (7.95, 48.43)
Female sex	1.22 (0.75, 1.99)	1.38 (0.82, 2.31)	1.46 (0.85, 2.52)
Race/ethnicity (black)†	1.42 (0.84, 2.40)	1.22 (0.71, 2.11)	1.01 (0.58, 1.77)
Other ethnic/racial groups‡	1.07 (0.34, 3.33)	0.74 (0.21, 2.68)	0.64 (0.17, 2.35)
Socioeconomic status (Nam-Powers index ≤35)	0.97 (0.60, 1.59)	0.85 (0.51, 1.41)	0.90 (0.53, 1.50)
No high school diploma	1.57 (0.92, 2.65)	1.77 (1.03, 3.06)	1.55 (0.89, 2.71)
Ever diabetes			1.35 (0.62, 2.97)
Ever heart problem			2.07 (1.19, 3.60)
Ever high blood pressure			2.14 (1.32, 3.48)
Currently smoke tobacco			2.03 (1.23, 3.33)
Model χ^2	190.10	165.9	191.15
df	10	10	14

*Reference age is 18–29 years.

†Reference group is white.

‡Reference group is white.

A history of heart problems and hypertension was also significantly related to stroke in the adjusted analyses. Those with a history of heart problems were 2.07 (95% CI, 1.19 to 3.60) times more likely to report stroke, and those with a history of hypertension were 2.14 (95% CI, 1.32 to 3.48) times more likely to report a first stroke during the follow-up period. A history of diabetes was associated with an increased risk for stroke (RR 1.35; 95% CI, 0.62 to 2) but was not statistically significant. Current tobacco use was associated with an increased risk for stroke (RR 2.03; 95% CI, 1.23 to 3.33).

The final logistic regression model included history of diabetes, heart problems, high blood pressure, and current tobacco use. Depressive disorder persisted as a significant predictor of incident stroke during the 13-year follow-up (RR 2.67; 95% CI, 1.08 to 6.63). In an additional logistic regression analysis (not shown) controlling for pharmacological depression treatment at baseline, including tricyclics/heterocyclics, MAOIs, and lithium, depressive disorder continued to significantly predict stroke, and the use of these medications was not associated with a change in the risk for stroke.

The prevalence of these 2 forms of depression was 0.0752 (128 cases of depressive disorder and dysthymia in this sample of 1703; 7.52 per 100). By using the adjusted RR of 2.67, the attributable risk for depression was found to be 0.108, or 10.8%. Therefore, if these 2 forms of depression were causing the stroke, then the maximum impact of removing depression exposure is a decrease in stroke of ≈11%.

Discussion

A lifetime history of depressive disorder is associated with a 2- to 3-times greater risk for incident stroke. This increased risk

was independent of a history of diabetes, hypertension, heart problems, or current tobacco use; the use of psychotropic drugs did not alter the association of depressive disorder and stroke. The relationship became stronger after controlling for the effect of age on stroke incidence. Dysthymia was similar to depressive disorder in its relationship to stroke. Our findings, based on an instrument designed to generate information of the type obtained in psychiatric interviews, add support to those reported by Jonas and Mussolino⁴ and Ohira et al.,¹ as well as others who have reported a positive relationship between stroke and recent symptoms of psychological distress.^{2,3,5}

A significant body of literature has found linkages that may help to explain the relationship between stroke and depression. Symptom scales of depression such as the Centers for Epidemiologic Studies-Depression instrument are associated with brain changes similar to those experienced by stroke patients.^{21,22} Cardiovascular disease is concomitantly associated with depression, providing another vehicle for a relationship between stroke and depression.^{9,10,23}

Multiple studies are documenting that depression acts as a risk factor for many physical diseases. The strongest evidence is for an association between depression and cardiovascular disease.^{7,9,10,23} This observational study, though based only on self-reported data, provides evidence that stroke should be included as a potential consequence of depressive disorder. Other research will be necessary to determine whether depression causes, results from, or is concurrent with vascular changes that lead to stroke. Additional studies that identify the mechanisms underlying the association are needed to guide possible prevention efforts.

The predictive strength of depressive disorder is not trivial. The estimated population-attributable risk, which is the maximum proportion of the disease that would be eliminated in the population if the risk factor were a necessary cause, is $\approx 11\%$. In a similar analysis, Klungel et al²⁴ estimated that 22.8% to 25.4% of strokes in the Netherlands were attributable to untreated hypertension.

Several limitations of the study should be considered when drawing conclusions. First, these data are self-reported. Individuals were asked whether they had ever had a stroke in each of the survey periods. The individual reporting the stroke may have been unclear about whether a stroke or another medical phenomenon had occurred. However, the general population has been found to be reasonably accurate in identifying strokes.²⁵ A recent study found that individuals with depression self-report stroke as accurately as individuals without a history of depression, with a positive predictive value equal to 0.79.²⁶ Misclassification concerning transient ischemic attacks is possible. No information was available to assess the type of stroke that may have occurred, nor were confirming brain imaging studies available. However, a high percentage (70%) of those individuals reporting a stroke did report at least 1 limitation of activities of daily living in the follow-up study.

Another possible limitation may be found in the potential misclassification of the exposure variables, depressive disorder and dysthymia. The DIS has been shown to have good reliability and is generally valid. One study that examined the concordance between clinical diagnoses and the DIS found that the DIS errs in the direction of underidentification of those with depression.²⁷

Of all established cardiovascular risk factors, depression is most highly associated with tobacco smoking. We considered both current and past tobacco smoking in this analysis, and found current tobacco use to be associated with future stroke. It is possible that depressive disorder could have led to the development of hypertension between baseline and stroke occurrence. Similar to most of the investigators in this field, we also could not have accounted for this possibility. Future studies should carefully measure both blood pressure and tobacco smoking over time to refine our understanding of the relationship between depressive disorder and stroke.

Finally, although the sample is relatively large, the number of individuals with depressive disorder at baseline was more limited, and among these, only a handful had new strokes. The problem here is not one of sample size, strictly speaking, because the findings are statistically significant. The potential problem is that errors of measurement in only a few cases could markedly change the results. The potential importance of these results, even though they are consistent with prior research, albeit with weaker measures,^{1–5} argues for the need to replicate them further in other samples. With these caveats, this prospective, population-based study has found that depressive disorder is a risk factor for stroke.

References

- Ohira T, Iso H, Satoh S, Sankai T, Tanigawa R, Ogawa Y, Imano H, Sato S, Kitamura A, Shimamoto T. Prospective study of depressive symptoms and risk of stroke among Japanese. *Stroke*. 2001;32:903–908.
- Simons LA, McCallum J, Friendlander Y, Simons J. Risk factors for ischemic stroke: Dubbo Study of the Elderly. *Stroke*. 1998;29:1341–1346.
- Simons LA. Is depression a risk factor for ischemic stroke? *Stroke*. 2001;32:907–908.
- Jonas BS, Mussolino ME. Symptoms of depression as a prospective risk factor for stroke. *Psychosom Med*. 2000;62:463–471.
- Everson S, Robert R, Goldberg D, Kaplan G. Depressive symptoms and increased risk of stroke mortality over a 29-year period. *Arch Intern Med*. 1998;158:1133–1138.
- Laghrissi-Thode F, Wagner WR, Pollock BG, Johnson PC, Finkel MS. Elevated platelet factor-4 and β -thromboglobulin plasma levels in depressed patients with ischemic heart disease. *Biol Psychiatry*. 1997;42:290–295.
- Musselman D, Tomer A, Manatunga A, Knight B, Porter M, Kasey S, Marzec U, Harker L, Nemeroff C. Exaggerated platelet reactivity in major depression. *Am J Psychiatry*. 1996;153:1313–1317.
- Musselman DL, Marzec UM, Manatunga A, Penna S, Reemsnyder A, Knight BT, Baron A, Hanson SR, Nemeroff CB. Platelet reactivity in depressed patients treated with paroxetine: preliminary findings. *Arch Gen Psychiatry*. 2000;57:875–882.
- Pratt L, Ford D, Crum R, Amerenian H, Gallo J, Eaton W. Depression, psychotropic medication and risk of myocardial infarction: prospective data from the Baltimore ECA follow-up. *Circulation*. 1996;94:3123–3129.
- Ford DE, Mead LA, Chang PP, Cooper-Patrick L, Wang NY, Klag MJ. Depression is a risk factor for coronary artery disease in men: the precursors study. *Arch Intern Med*. 1998;158:1422–1426.
- American Heart Association. *Heart and Stroke Statistical Update*. Dallas, Tex: American Heart Association; 1999.
- Nemeroff C, Widerlov E, Bissette G, Wallwus H, Karlsson I, Eklund K, Kilts C, Loosen P, Vale W. Elevated concentrations of CSF corticotropin-releasing factor-like immunoreactivity in depressed patients. *Science*. 1984;226:1342–1344.
- Arato M, Banki C, Nemeroff C, Bissette G. Hypothalamic-pituitary-adrenal axis and suicide. *Ann NY Acad Sci*. 1986;487:263–270.
- Troxler R, Sprague E, Albanese R, Fuchs R, Thompson A. The association of elevated plasma cortisol and early atherosclerosis as demonstrated by coronary angiography. *Atherosclerosis*. 1977;26:151–162.
- American Psychiatric Association. *Diagnostic and Statistical Manual*. 3rd ed. Washington, DC: American Psychiatric Association; 1980.
- Eaton WW, Kessler L. *Epidemiologic Field Methods in Psychiatry: The NIMH Epidemiologic Catchment Area Program*. Orlando, Fla: Academic Press; 1985.
- Eaton WW, Anthony JC, Gallo J, Cai G, Tien A, Romanoski A, Lyketsos C, Chen L. Natural history of DIS/DSM major depression: the Baltimore ECA follow-up. *Arch Gen Psychiatry*. 1997;54:993–999.
- Badawi MA, Eaton WW, Myllylouoma J, Weimer L, Gallo J. Psychopathology and attrition in the Baltimore ECA follow-up 1981–1996. *Soc Psychiatry Psychiatr Epidemiol*. 1999;34:91–98.
- Chen LS, Eaton WW, Gallo JJ, Nestadt G, Crum RM. Empirical examination of current depression categories in a population based study: symptoms, course and risk factors. *Am J Psychiatry*. 2000;157:573–580.
- Terrie EW, Nam CB. 1990 and 1980 Nam-Power-Terrie Occupational Status Scores. Tallahassee, Fla: Center for Study of Population, Florida State University; 1994.
- Krishnan KR, Hays JC, Blazer DG. MRI-defined vascular depression. *Am J Psychiatry*. 1997;154:497–501.
- Steffens D, Helms M, Krishnan K, Burke G. Cerebrovascular disease and depression symptoms in the Cardiovascular Health Study. *Stroke*. 1999;30:2159–2166.
- Glassman AH, Shapiro PA. Depression and the course of coronary artery disease. *Am J Psychiatry*. 1998;155:4–11.
- Klungel OH, Stricker B, Paes A, Seidell JC, Bakker A, Voko Z, Breteler MM, deBoer A. Excess stroke among hypertensive men and women attributable to undertreatment of hypertension. *Stroke*. 1999;30:1312–1318.
- Bergmann MM, Byers T, Freedman D, Mokdad A. Validity of self-reported diagnoses leading to hospitalization: a comparison of self-reports with hospital records in a prospective study of American adults. *Am J Epidemiol*. 1998;147:969–977.
- Engstad T, Bonaa KH, Viitanen M. Validity of self-reported stroke: the Tromso study. *Stroke*. 2000;31:1602–1607.
- Eaton WW, Neufeld K, Chen L, Cai G. A comparison of self-report and clinical diagnostic interviews for depression: diagnostic interview schedule and schedules for clinical assessment in neuropsychiatry in the Baltimore epidemiologic catchment area follow-up. *Arch Gen Psychiatry*. 2000;57:217–222.

Depressive Disorder, Dysthymia, and Risk of Stroke: Thirteen-Year Follow-Up From the Baltimore Epidemiologic Catchment Area Study
Sharon L. Larson, Pamela L. Owens, Daniel Ford and William Eaton

Stroke. 2001;32:1979-1983

doi: 10.1161/hs0901.094623

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2001 American Heart Association, Inc. All rights reserved.

Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://stroke.ahajournals.org/content/32/9/1979>

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Stroke* is online at:
<http://stroke.ahajournals.org/subscriptions/>