AJH 2001; 14:660–664

Blood Pressure and Symptoms of Depression and Anxiety: A Prospective Study

Eileen Huh Shinn, Walker S. Carlos Poston, Kay T. Kimball, Sachiko T. St. Jeor, and John P. Foreyt

This study investigated whether symptoms of depression and anxiety were related to the development of elevated blood presure in initially normotensive adults. The study's hypothesis was addressed with an existing set of prospective data gathered from an age-, sex-, and weight-stratified sample of 508 adults. Four years of follow-up data were analyzed both with logistic analysis, which used hypertension (blood presssure ≥140 mm Hg systolic or 90 mm Hg diastolic) as the dependent variable, and with multiple regression analysis, which used change in blood pressure as the dependent variable. Five physical risk factors for hypertension (age, sex, baseline body mass index, family history of hypertension, and baseline blood pressure levels) were controlled for in the regression analyses. Use of antidepressant/antianxiety and antihypertensive medications were controlled for in the study.

Of the 433 normotensive participants who were eligible for our study, 15% had missing data in the logistic regression analysis focusing on depression ($n = 371$); similarly, 15% of the eligible sample had missing data in the logistic regression using anxiety as the psychological variable of interest ($n = 370$). Both logistic regression analyses showed no significant relationship for either depression or anxiety in the development of hypertension. The multiple regression analyses ($n = 369$ for the depression analysis; $n = 361$ for the anxiety analysis) similarly showed no relationship between either depression or anxiety in changes in blood pressure during the 4-year follow-up. Thus, our results do not support the role of depressive or anxiety symptoms in the development of hypertension in our sample of initially normotensive adults. Am J Hypertens 2001;14:660–664 © 2001 American Journal of Hypertension, Ltd.

Key Words: Depression, anxiety, psychosocial risk factors for hypertension.

Prospective studies that have examined the relationship of depressive or anxiety symptoms with blood pressure have been characterized by mixed results.[1–11] Of seven prospective studies that examined the risk of anxiety in normotensive and relatively healthy participants,[1–7] five found that anxious normotensives were significantly more likely to develop subsequent hypertension or clinically meaningful increases in blood pressure compared to their nonanxious counterparts.[1,3–6] These studies used well-validated measures of anxiety as well as multivariable techniques to independently assess anxiety's influence on subsequent hypertension.

The existing body of prospective studies for symptoms of depression is less consistent. Of six prospective studies that examined the relationship between depression and future elevations in blood pressure,[1,2,8–11] three found a significant effect.[1,2,8] The remaining three studies did not find any association between depression and hypertension. One of these studies even found that scores indicating nondepression at baseline (scores in the lowest tertile) were associated with hypertension.[10]

According to Jonas et al,[1] the lack of consensus among the prospective studies is due to three factors: insufficient follow-up time, inadequate sample size, and lack of well-validated standardized measures. Other methodologic issues to be considered are the use of multivariable analysis to control for biologic and genetic risk factors, and the characterization of the baseline sample in terms of hyper-

Received April 18, 2000. Accepted December 4, 2000.

From the Department of Behavioral Science (EHS), University of Texas—M.D. Anderson Cancer Center, Houston, Texas; Mid America Heart Institute (WSCP), St. Luke's Hospital, University of Missouri-Kansas City, Kansas City, Missouri; Department of Medicine (KTK), Baylor College of Medicine, Houston, Texas; Nutrition Education and Research Program (STSJ), University of Nevada School of Medicine at Reno, Reno, Nevada; and Behavioral Medicine Research Center (JPF), Baylor College of Medicine, Houston, Texas.

This work was supported by grants from the National Heart, Lung, and Blood Institute (HL34589), National Cancer Institute (2-R25-CA57712; R-25-CA57730), and a Minority Scientist Development Award from the American Heart Association and with funds contributed by the AHA, Puerto Rico Affiliate.

Address correspondence and reprint request to Dr. Eileen Huh Shinn, Box 243 1515 Holcombe Blvd, Houston, TX 77030; e-mail: eshinn@mdanderson.org

0895-7061/01/$20.00
PII S0895-7061(01)01304-8

© 2001 by the American Journal of Hypertension, Ltd.
Published by Elsevier Science Inc.

Downloaded from https://academic.oup.com/ajh/article/14/7/660/258839 by guest on 23 April 2024

tensive and general health status. In response to these methodologic criticisms, the present study used well-validated psychological measures[12–14] in conjunction with biological measures in a multivariable analysis. The present study followed normotensive participants drawn from a study sample previously screened for past medical and psychiatric illness. The length of follow-up, 4 years, is longer than the minimum 3 years recommended by Jonas et al.[1] It was hypothesized that symptoms of depression and anxiety are independent risk factors for developing hypertension or elevated blood pressure in initially normotensive individuals.

Methods

Participants

The first 4 years of prospective data from the Reno Diet Heart Study were analyzed on subjects who were normotensive at baseline. The original data set included measures of diet, weight, blood pressure, medical, and psychological data from a sample of 508 participants, aged 20 to 75 years.[15] The original sample was stratified for age group (20s, 30s, etc.), sex, and obesity classification (defined as 20% above the ideal weight as listed in the Metropolitan Insurance tables).[16] The participants' eligibility in the Reno Diet Heart Study was determined by the following criteria: 1) self-assessed to be in good physical health, 2) 12 or fewer sick days in the previous year, 3) employed at least half time, 4) no major psychological problems, and 5) no major current illnesses. Baseline demographic data revealed low prevalence of serious illnesses among the original sample: less than 5% for kidney disease, diabetes, history of cancer, and less than 1% for history of severe mental illness. Participants for the present study met additional criteria: 1) systolic blood pressure levels $<$140 mm Hg and diastolic blood pressure levels $<$90 mm Hg at baseline, and not on antihypertensive medication before or during baseline, and 2) complete data on the psychological measures at baseline. Because the number of missing cases increased dramatically in our study by the fifth year of the Reno Diet Heart Study, it was decided to limit the length of follow-up to the first 4 years.

The sample in the present study was very well educated, with more than half of the participants being college graduates. The sample was also primarily white, comprising 95% of the sample. Three percent of the sample was Hispanic and less than 1% of the sample was Asian American. A large majority was married (75%) and earned over $24,000 a year after taxes.

Baseline Measures

The Center for Epidemiologic Studies-Depression Scale (CES-D) was designed to measure current levels of depressive symptomatology, and should be used as a screening instrument rather than a diagnostic tool for detecting cases of major depression.[17] In community and patient samples, the CES-D had much higher sensitivity than specificity for detecting depression (64% sensitivity *v* 6% specificity,[18] 59% sensitivity *v* 14% specificity,[19] 90% sensitivity *v* 55% specificity[20]).

Table 1. Participants who were coded as depressed or anxious at baseline (n = 370)

	No. of Participants	%
Depressed (CES-D $>$15 or GWB-D $<$13)	43	11.7
Anxious (GWB-A $<$13)	57	15.4

CES-D = Center for Epidemiologic Studies-Depression scale; GWB-D = general well-being depression subscale; GWB-A = general well-being anxiety subscale.

Studies comparing the performance of the General Well-Being Subscales against standardized clinical interviews are not available. However, convergent validity studies for the General Well-Being Anxiety (GWB-A) and General Well-Being Depression (GWB-D) Subscales obtained high correlations with various standardized instruments, such as the Zung Self-Rating Depression Scale (r = 0.62), and with the Psychiatric Symptoms Anxiety Subscale (r = 0.76).[21] Reliabilities for the two subscales have been high in both community and patient samples, with r ranging from 0.77 to 0.78 for the GWB-D, and 0.71 to 0.78 for the GWB-A.[1,22]

Scores on the CES-D, GWB-D, and GWB-A were used to measure symptoms of depression and anxiety for each participant at time zero. Questionnaires were administered by the staff and checked for completeness to minimize missing data. To measure depression, scores on the CES-D and the GWB-D were recoded as a dichotomous variable. Using the recommended cutoff scores, a subject was coded as depressed if he or she scored either 1) 16 or higher on the CES-D scale or 2) 12 or lower on the GWB-D. To measure anxiety, scores on the GWB-A were recoded as a dichotomous variable. Using the recommended cutoff score, subjects who scored 12 or lower on GWB-A were coded as anxious. Table 1 presents the number of participants who were coded as depressed or anxious during baseline year (time zero).

Every year, blood pressure levels were measured by trained and certified technicians according to the standardized method using the random zero sphygmomanometer and hypertension detection and follow-up procedure.[23] Before the measurement session began, participants fasted for 12 h and had not smoked for at least 1 h. The participant was seated quietly for 5 min with both feet resting on the floor and the right arm resting at heart level on a table. Blood pressure measurements were taken three times, with the last two measurements separated by at least 1 full min. The average of the last two measurements was recorded. Proper cuff size was ensured by having a variety of cuff sizes available. Data on the other physical variables were

Downloaded from https://academic.oup.com/ajh/article/14/7/660/258839 by guest on 23 April 2024

collected by health questionnaires and by height and weight measurements taken at the University of Nevada School of Medicine.

Follow-Up Variables

Two types of outcome variables were used. First, dichotomous variables of hypertensive status after 4 years of follow-up were defined by using the cutoff of 140 mm Hg systolic blood pressure or 90 mm Hg diastolic blood pressure. Because clinically significant increases that fell short of the cutoff criteria would not be counted as hypertension, we also calculated changes in blood pressure and used the change scores as a continuous outcome variable. Continuous measures of changes in systolic blood pressure and in diastolic blood pressure were calculated by subtracting the participant's blood pressure level at baseline from their final blood pressure level.

The following procedures were used in those cases where participants were placed on antihypertensive medication. If participants were taking hypertensive medication at baseline (time zero), they were dropped from the analyses. If persons were put on hypertensive medication during their follow-up period, then the highest blood pressure level reached before they were placed on medication was used as their final blood pressure level. Therefore, for participants who started antihypertensive medications after time zero, follow-up periods were less than 4 years. However, $<5\%$ of the participants were affected by this rule and the average length of follow-up was 3.94 years. Regarding the use of antidepressants or anxiolytics, surprisingly few participants ($n = 12$) admitted to taking these types of medications at follow-up. Of these, four participants were coded as depressed at baseline. The low number of participants on psychoactive medication may have been in conjunction with the prescreening that excluded people who self-reported being depressed or reported having been hospitalized for psychiatric disorders.

Statistical Analyses

Separate analyses were conducted to evaluate the effects of depressive and anxiety symptoms on hypertension and on changes in systolic and diastolic blood pressure. First, two logistic regression analyses were conducted to determine whether symptoms of depression or anxiety were significant risk factors for the development of hypertension. Next, four multiple regression analyses were conducted to determine whether 1) depression was associated with changes in systolic blood pressure after follow-up, 2) anxiety was associated with changes in systolic blood pressure after follow-up, 3) depression was associated with changes in diastolic blood pressure after follow-up, and 4) anxiety was associated with changes in diastolic blood pressure after follow-up.

The set of multiple regression analyses were performed to avoid losing information regarding the development of hypertension. For example, a participant who entered the study with a systolic blood pressure level of 120 mm Hg may have experienced an increase of 19 mm Hg systolic blood pressure by the end of follow-up. But because the participant's final systolic blood pressure level of 139 mm Hg does not meet the cutoff criterion of 140 mm Hg systolic blood pressure, this participant would not have been coded as hypertensive, although the participant had a clinically meaningful increase of 19 mm Hg systolic blood pressure.[3] In contrast, when blood pressure is analyzed as a continuous variable, clinically significant increases that fall short of the cutoff criteria for hypertension are tracked.

For both the logistic and multiple regression equation models, age, sex, family history, baseline obesity, baseline blood pressure level, and use of either antidepressant or anxiolytic medication at follow-up were entered first as a block of variables. Sex, family history of hypertension, and use of psychoactive medications were scored as dichotomous variables. Family history for hypertension was defined as one or both of the participant's parents having a diagnosis of hypertension. Baseline systolic and diastolic blood pressure levels, age, and baseline body mass index (BMI) were continuous variables. BMI was defined as weight/(height)^2, where weight was measured in kilograms and height in meters. Finally, depression or anxiety was entered into the regression equation to determine whether symptoms of depression or anxiety predicted any change in blood pressure level independent of the physical risk factors.

Results

Logistic Regression

Neither depressive nor anxiety symptoms were significantly related to the development of hypertension in the logistic regression analyses. In the logistic regression analysis that focused on depression (Table 1), 62 participants of 433 eligible participants were dropped from the analysis because of missing data, resulting in 371 participants. Men ($P < .01$) and those with higher BMI ($P < .05$) were more likely to have hypertension at the end of follow-up, compared to women and those with lower BMI. During follow-up, 55 normotensive participants became hypertensive.

In the logistic regression analysis that focused on anxiety (Table 2), 63 participants of the 433 eligible participants were dropped because of missing data, resulting in 370 participants. Men ($P = .01$), and those participants with higher BMI ($P = .01$) were more likely to have hypertension at the end of follow-up, compared to women, and those participants with lower BMI. During follow-up, 54 normotensive participants became hypertensive. Tables 2 and 3 present the results of logistic regression models.

Multiple Regression

For the multiple regression analyses, 72 participants were dropped from the analyses that focused on anxiety and 64

Downloaded from https://academic.oup.com/ajh/article/14/7/660/258839 by guest on 23 April 2024

Table 2. Logistic regression predicting hypertension with depression

Factor	Odds Ratio	CI	*P*
Baseline systolic blood pressure	1.04	(1.01, 1.07)	.01
Baseline BMI	1.09	(1.02, 1.16)	.02
Age at entry	1.02	(1.0, 1.05)	.07
Sex	.41	(.22, .79)	.01
Parental history of hypertension	.62	(.33, 1.2)	.14
Antidepressant/ Anxiolytic use	.60	(.14, 2.52)	.48
Depression	.64	(.14, 1.45)	.29
Constant			.00

CI = confidence interval; BMI = body mass index.

Note: Dependent variable coded hypertensive = 1, normotensive = 0; $n = 371$.

participants were dropped from the analyses that focused on depression due to their hypertensive status at baseline. Both depression and anxiety were unrelated to changes in both systolic and diastolic blood pressure in the multiple regression analyses. All four of the multiple regression equations were significant ($P < .001$) and predicted between 13 and 15% of the total variance (adjusted $R^2 = 0.13$ to 0.15). The combination of significant physical predictor variables varied for each of the four multiple regression analyses, and were in the expected direction. The range of change in systolic and diastolic blood pressure was broad and normally distributed. Age ($P < .001$) and higher baseline BMI ($P < .001$) were associated with increases in systolic blood pressure, whereas higher baseline BMI ($P < .005$) and being male ($P < .05$) were associated with increases in diastolic blood pressure.

Table 3. Logistic regression predicting hypertension with anxiety

Factor	Odds Ratio	CI	*P*
Baseline systolic blood pressure	1.04	(1.01, 1.07)	.02
Baseline BMI	1.09	(1.03, 1.17)	.01
Age at entry	1.03	(1.02, 1.17)	.06
Sex	.39	(.20, .75)	.01
Parental history of hypertension	.65	(.34, 1.24)	.19
Antidepressant/ Anxiolytic use	.57	(.14, 2.38)	.44
Anxiety	.70	(.30, 1.64)	.45
Constant			.00

Abbreviations as in Table 2.

Note: Dependent variable coded hypertensive = 1, normotensive = 0; $n = 370$.

Discussion

The logistic analyses in our study do not support the hypothesis that symptoms of depression or anxiety are risk factors for the future development of hypertension. Thus, our results are consistent with three of the six prospective studies that examined the risk associated with depression,[9–11] and with three of the nine studies that examined the risk associated with anxiety.[2,7,10] Our study also calculated changes in blood pressure and used the change scores as a continuous outcome variable in multiple regression analysis. Three other prospective studies similarly used change scores as a continuous outcome variable.[3,5,7] Of the three studies, two found that depression or anxiety symptoms at baseline significantly predicted increases in blood pressure at follow-up.[3,5] The third study found no relationship between psychological factors and future blood pressure change.[7] Our study was similar to the three studies in that a wide range of changes from sizeable decreases to sizeable increases in blood pressure occurred over time. The range of changes in systolic and diastolic blood pressure reported by Markovitz et al[3] was similar to the range of changes in our study.

Our findings do not negate the possibility that symptoms of depression and anxiety are risk factors for hypertension. One possible reason for our results may have been that participants 60 years and older were not as well represented in our sample compared to participants who were in the four younger age groups (20s, 30s, 40s, and 50s). The lower participation rate among the 60-year olds (60% compared to 86% to 99% among the five younger age groups) was primarily due to many of the 60-year olds being hypertensive at baseline, making them ineligible for our study. Thus, the sample in our study tended to be younger in age and may not have had sufficient follow-up time to develop increases in blood pressure. It is also possible that our study failed to find a relationship due to the higher rates of dropouts among depressed participants compared to nondepressed participants ($t = 3.307$; $P < .005$).

Another consideration to make when evaluating our study is the intense scrutiny under which Reno Diet Heart Study participants were placed at baseline recruitment. Compared to the majority of prospective studies that measured psychological factors at baseline and measured blood pressure only at baseline and final year, the Reno Diet Heart Study schedule of measurements was much more intense. The annual measures included blood pressure readings, physical examinations that required fasting and blood drawing, skin caliper measurements, 24-h and 7-day recall nutritional diaries, as well as a number of health and psychological questionnaires. This intense scrutiny, which originally was seen as a strength, may in fact have affected the outcome of the study by continually keeping the participants vigilant of their health status and motivating them to continually work on improving their health habits. In addition, the study was biased toward the

Downloaded from https://academic.oup.com/ajh/article/14/7/660/258839 by guest on 23 April 2024

recruitment of "healthy" participants who were not depressed by self-report or hospitalized for a psychological disorder within the past 5 years.

In conclusion, in light of the several prospective studies that found a high risk associated with depressive symptoms for mortality in patients already diagnosed with coronary artery disease,[24–27] there has been increased interest in major depression and its effects on other chronic diseases, such as asthma, diabetes, and cancer. The body of research regarding depression as a possible risk factor for hypertension is still relatively small and should remain open to question. Our finding that symptoms of depression and anxiety were unrelated to changes in blood pressure was contrary to our hypothesis that they were risk factors for hypertension or elevated blood pressure. This finding may be spurious and the possibility remains that there is a relationship between depression and anxiety and the development of hypertension. Despite its limitations, our study addressed some of the prevailing critiques of the literature by following an age- and sex-stratified sample of adequate size (n ranged from 361 to 371), for 4 years, longer than the minimum 3 years advocated by Jonas et al.[1] Three instruments that have demonstrated good reliability and validity, that is, the Center for Epidemiologic Studies Depression scale, the General Well-Being Anxiety Subscale, and the General Well-Being Depression Subscale, were used to measure depression and anxiety. Our study also controlled for a number of physical risk factors such as age, sex, BMI, family history of hypertension, and baseline blood pressure.

References

1. Jonas BS, Franks P, Ingram DD: Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. Arch Family Med 1997;6:43–49.
2. Kahn HA, Medalie JH, Neufeld HN, Riss E, Goldbourt U: The incidence of hypertension and associated factors: The Israel ischemic heart disease study. Am Heart J 1972;84:171–182.
3. Markovitz JH, Matthews KA, Wing RR, Kuller LH, Meilahn EN: Psychological, biological, and health behavior predictors of blood pressure changes in middle-aged women. J Hypertens 1991;9:399–406.
4. Markovitz JH, Matthews KA, Kannel WB, Cobb JL, D'Agostino RB: Psychological predictors of hypertension in the Framingham Study. JAMA 1993;270:2439–2443.
5. Perini C, Muller FB, Buhler FR: Suppressed aggression accelerates early development of essential hypertension. J Hypertens 1991;9:499–503.
6. Russek LG, King SH, Russek SJ, Russek HJ: The Harvard mastery of stress study 35-year follow up: Prognostic significance of patterns of psychophysiological arousal and adaptation. Psychosom Med 1990;52:271–285.
7. Sparrow D, Garvey AJ, Rosner B, Thomas HE: Factors in predicting blood pressure change. Circulation 1982;65:789–794.
8. Heine B: Approaches to the etiology and treatment of psychosomatic disorder. Proc Royal Soc Med 1970;63:1267–1270.
9. Paffenbarger RS, Thorne MC, Wing AL: Chronic disease in former college students: VIII. Characteristics in youth predisposing to hypertension in later years. Am J Epidemiol 1968;88:25–32.
10. Vogt T, Pope C, Mullooly J, Hollis J: Mental health status as a predictor of morbidity and mortality: A 15-year follow-up of members of a health maintenance organization. Am J Public Health 1994;84:227–231.
11. Goldberg EL, Comstock GW, Graves CG: Psychosocial factors and blood pressure. Psych Med 1980;10:243–255.
12. McDowell I, Newell C: The general well-being schedule, *in* McDowell I, Newell C (eds): Measuring Health: A Guide to Rating Scales and Questionnaires. New York, Oxford University Press, 1987, pp 125–133.
13. Ware JE, Johnston SA, Davies-Avery A, Brook RH: Conceptualization and Measurement of Health for Adults in the Health Insurance Study: Vol. III, Mental Health (Publication No. R-1987/3-HEW). Santa Monica, Rand Corporation, 1979.
14. Devins G, Orme C: Center for epidemiologic studies depression scale, in Kaiser D, Sweetland R (eds): Test Critiques, Vol II. 1985, pp 144–160.
15. St. Jeor ST, Simpson MT, Daugherty SA: Subject recruitment and retention, *in* St. Jeor (ed.): Obesity Assessment: Tools, Methods, Interpretations. New York, Chapman and Hall, 1997, pp 19–29.
16. Metropolitan Insurance Company: Metropolitan Height and Weight Tables. New York, Metropolitan Insurance Company, 1983.
17. Radloff LS: The center for epidemiologic studies—depression scale: A self-report depression scale for research in the general population. App Psych Measure 1977;1:385–401.
18. Myers JK, Weissman MM: Use of a self-report symptom scale to detect depression in a community sample. Am J Psych 1980;137:1081–1084.
19. Roberts RR, Vernon SW: The Center for Epidemiologic Studies Depression Scale: Its use in a community sample. Am J Psych 1983;140:41–46.
20. Weissman MM, Sholomskas D, Pottenger M, Prusoff BA, Locke BZ: Assessing depressive symptoms in five psychiatric populations: A validation study. Am J Epidemiol 1977;106:203–214.
21. Fazio AF: A concurrent validational study of the NCHS General Well-Being Schedule (Vital and Health Statistics Series 2, No.73. DHEW Publication No(HRA) 78-1347). Hyattsville, MD, U.S. Department of Health, Education, and Welfare, National Center for Health Statistics, 1977.
22. Zonderman AB, Costa PT, McCrae RR: Depression as a risk factor for cancer morbidity and mortality in a nationally representative sample. JAMA 1989;262:1191–1195.
23. Daugherty SA, Zahrt H: Blood pressure and pulse measurement, *in* St. Jeor ST (ed): Obesity Assessment: Tools, Methods, Interpretations. New York, Chapman and Hall, 1997, pp 114–121.
24. Frasure-Smith N, Lesperance F, Talajic J: Depression following myocardial infarction: Impact on 6 month survival. JAMA 1993;270:1819–1825.
25. Frasure-Smith N, Lesperance F, Talajic J: Depression and 18-month prognosis after myocardial infarction. Circulation 1995; 91:99–105.
26. Carney RM, Rich MW, Freedland KW, Saini J, TeVelde A, Simeone C, Clark K: Major depressive disorder predicts cardiac events in patients with coronary artery disease. Psychosom Med 1988;50:627–633.
27. Barefoot JC, Helms MJ, Mark DB, Blumenthal JA, Califf RM, Haney TL, O'Connor CM, Siegler IC, Williams RB: Depression and long-term mortality risk in patients with coronary artery disease. Am J Cardiol 1996;78:613–617.

Downloaded from https://academic.oup.com/ajh/article/14/7/660/258839 by guest on 23 April 2024