Published in final edited form as:
Hypertension. 2010 February ; 55(2): 408. doi:10.1161/HYPERTENSIONAHA.109.139824.

Baseline depressive symptoms are not associated with clinically important levels of incident hypertension during two years of follow-up: the Multi-Ethnic Study of Atherosclerosis

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Abstract

Previous longitudinal cohort studies have suggested an association between baseline depressive symptoms and incident hypertension. We assessed this possible association using data from the Multi-ethnic Study of Atherosclerosis, a population-based prospective cohort study of 6,814 US adults from 4 different racial/ethnic groups. Baseline users of antihypertensive medications and participants lost to follow-up were excluded leaving 3914 participants. Patients with baseline depressive symptoms (n=622) were defined using a high score on the Center for Epidemiologic Studies Depression Scale (≥16) or the use of an antidepressant medication. Hypertension was defined as systolic blood pressure ≥ 140, diastolic blood pressure ≥90 or new use of antihypertensive medications plus physician diagnosis. Estimates were adjusted for known risk factors including: age, sex, baseline blood pressure, diabetes, and body mass index. Untreated blood pressure was estimated using an imputation approach. A total of 477 participants developed hypertension. Using relative risk regression, patients with baseline depressive symptoms did not have an increased risk of incident hypertension (Relative Risk = 1.02; 95% Confidence Interval (CI):0.99 to 1.05) although an association between tricyclic antidepressants and hypertension (Relative Risk 1.20; 95% CI:1.05 to 1.37) was observed in sub-group analysis. Depression, even after adjustment for covariates, was associated with small changes in systolic (+2.4 mmHG; 95% CI: 0.2 to 4.7) and diastolic (+0.8 mmHG; 95% CI: −0.6 to 2.3) blood pressure. Depressive symptoms may be associated with slight increases in blood pressure in this multi-ethnic cohort but it is premature to conclude much without longer studies in other populations.

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Disclosures:
None. No authors have any relevant financial, personal or professional relationships.

Keywords

Multi-Ethnic Study of Atherosclerosis; depression; hypertension; blood pressure; imputation; censored normal regression

BACKGROUND

Depressive symptoms have been associated with incident hypertension in multiple epidemiological studies [1]. Although this association is not consistently documented in cross-sectional studies [2], several longitudinal studies have supported an association between depressive symptoms and hypertension. These studies have been in multiple populations and all show an approximately two-fold increase in the odds of hypertension among adults with baseline depressive symptoms [3–6].

However, all of these previous studies have limitations. These include: being among young healthy patients, a lack of information on confounders and being set in an earlier time period where clinical care was quite different [3–6]. These issues of study design make it unclear how relevant previous estimates of this association are to current populations.

In order to assess this association in diverse populations, we examined the association between baseline depressive symptoms and increases in participant blood pressure after an average of 1.60 years in a multi-ethnic, population-based study. We collected information on depressive symptoms [7], as reported by Center for Epidemiologic Studies Depression Scale (CES-D) score. There was also information on other psychological variables such as anger [8] and anxiety [9] that might be potential confounders as these measures are available in the MESA cohort [10].

METHODS

The Multi-Ethnic Study of Atherosclerosis (MESA) is a population-based study. The study was initiated in order to determine the risk factors for the development and progression of subclinical and clinical cardiovascular disease [11]. This prospective cohort study consisted of 6,814 participants (between the ages of 45 and 84 years of age at baseline) who were recruited from six field centers across the United States: Baltimore, MD; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; New York, NY and St. Paul, MN. The MESA study categorized all participants into four ethnic groups: African-American, Caucasian, Chinese and Hispanic based on the categories from the 2000 census questionnaire [11].

The baseline exam for the MESA participants occurred between July 2000 and April 2002 and included medical screening tests for sub-clinical atherosclerosis. The follow up exam occurred between October 2002 and October 2003 with an average of 595 days of follow-up. Medication use was determined at both baseline and follow-up using a previously validated medication inventory approach [12]. Blood pressure was measured on all participants in a standardized fashion. After a five-minute rest, blood pressure was taken three times in the seated position using a Dinamap model Pro 100 automated oscillometric sphygmomanometer (Critikon, Tampa, Florida) with the average of the last two measurements used as our measure of blood pressure.

Participants with a self reported history of either prevalent cardiovascular disease (including stroke) or previous surgery for cardiovascular disease were excluded from participation in the MESA study. There was no information available on the dementia status of the MESA participants at baseline. From the 5970 participants with complete data and who participated

in both exams, an additional 2056 participants were excluded for baseline use of anti-hypertensive drugs.

The baseline MESA data included scores from the CES-D instrument [7,13] which can be used as marker of depressive symptoms. This instrument is a 20-item self-report questionnaire covering self report of depressed mood, feelings of worthlessness, feelings of hopelessness, poor concentration, loss of appetite, and sleep disturbance. In the MESA study population, the CES-D was administered in English, Spanish, Cantonese, and Mandarin. Higher scores for the CES-D suggest more evidence of depressive symptoms; in previous work, a CES-D score of ≥16 was proposed as an indicator of propensity to depressive symptoms [7]. In addition to self-reported depressive symptoms scales, MESA data also included self-reported measures of anger and anxiety using the Spielberger anger and anxiety scores [8,9].

We used an operational definition of depressive symptoms (n=622) which we defined as a score on the Center for Epidemiologic Studies Depression Scale ≥ 16 [14] or baseline use of any antidepressant medications. This operational definition refers to participants reporting depressive symptoms and is not equivalent to a clinical diagnosis of depressive symptoms. The primary study outcome was defined as change in SBP or DBP (defined as a continuous variable). Participants returned for a follow-up exam after an average of 595 days (range 385 to 1224 days) and underwent similar blood pressure measures and medication inventories as in the baseline exam. New physician diagnosis of hypertension was reported by the participant at telephone contacts, the most recent of which averaged 35 days before the follow-up exam. Based on the exam 2 medication inventory, 442 participants began new anti-hypertensive drug use. Hypertension was defined according to the seventh Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7) which recommends SBP ≤140 and DBP ≤90 for low risk patients over age 50 [15].

Statistical Analysis

Descriptive statistics were given as mean [SD] for continuous variables or frequencies for dichotomous variables. We used multivariable linear regression to model systolic and diastolic blood pressure at the first follow-up exam. In these models, we controlled for age, sex, ethnicity, baseline blood pressure, body mass index, diabetes, smoking, physical activity, alcohol use, anger, anxiety, income and health insurance.

Primary Analysis: Change in Blood Pressure—For the primary analysis, all participants were included regardless of baseline hypertension status (although we excluded baseline medication users). This required us to consider the influence of incident medication use on blood pressure. The primary strategy to handle the 442 participants who started an antihypertensive medication at the second visit was multiple imputation. Here we treat the blood pressure values of new users as missing data and use standard imputation techniques to estimate their untreated blood pressure. We used a chained imputation approach where treated blood pressure at exam 2 was considered to be missing and imputed using the following covariates: age (linear and non-linear forms), sex, diabetes, impaired fasting glucose, body mass index, ethnicity, study site, smoking status, alcohol use, intentional exercise, sedentary activities, anger or anxiety scores, depressive symptoms score, health insurance status, household income and baseline blood pressure. We used 5 imputed datasets and derived regression results using the SAS procedure MIANALYZE. Imputation has been previously used to estimate missing blood pressure information [16–17] as well as missing biomarker data [18].

As sensitivity analysis, we also considered two other approaches to accounting for the presence of treatment with anti-hypertensive drugs. One, naïve restriction, was to exclude all new users

from the analysis. The second was to use a censored normal regression model with treated blood pressures considered as being censored [19].

Secondary Analysis: Incident Hypertension—The primary definition of incident hypertension was SBP ≥ 140, DBP ≥90 (the criteria from JNC7) or new use of anti-hypertensive medications plus physician diagnosis of high blood pressure. We also considered the criterion of Davidson *et al.* [4] who defined hypertension as SBP ≥ 160, DBP ≥ 95 or use of an anti-hypertensive medication (the old world health organization definition) and a very liberal definition of hypertension (SBP ≥ 130, DBP ≥80 or new use of anti-hypertensive medications plus physician diagnosis of high blood pressure) as sensitivity analyses. When considering incident hypertension, we excluded participants with prevalent hypertension (by the corresponding definition) at baseline.

Most of the other studies of hypertension and depressive symptoms had much longer follow-up periods than the time between two MESA exams. In order to look at the influence of distance from the initial measurement to the diagnosis of hypertension, we also considered the 5421 participants who returned for the third MESA follow-up exam. This exam was similar to the first follow-up exam and the mean follow-up time by this exam was 1763 days (range 1218 to 2430 days). For this analysis we used the JNC7 definition of hypertension as described above.

We compared the incidence of hypertension between those participants with and without baseline depressive symptoms using relative risk regression [20]. We tested variables for their relationship with the outcome and included those that were associated with the outcome as candidate confounders: namely age, sex, ethnicity, diabetes, body mass index and baseline blood pressure. The inclusion of baseline blood pressure was required in order to avoid possible bias in the estimation of the association between hypertension and depressive symptoms due to regression to the mean [21] as measurement error in participants with borderline blood pressure might generate some outcomes and adjusting for baseline blood pressure is a technique for reducing this error [21]. As starting an anti-hypertensive medication was part of the outcome definition for these models, we only excluded baseline anti-hypertensive medication users. We tested for interactions between depressive symptoms and both ethnicity and sex. As previous research suggests that hypertension is not a predictor of loss to follow-up in the MESA cohort [22], we did not apply any corrections for loss to follow-up.

All analyses were done in either SAS version 9.1.3 or STATA 10 and all tests are two sided at the 5% level of significance.

RESULTS

The baseline characteristics of the study population are presented in Table 1. The participants who were depressed at baseline were younger, more likely to be Caucasian, exercise less and have slightly lower blood pressure readings. We also show the characteristics of the participants who met our entry criterion but did not return for the second examination. These lost to follow-up participants did not have important differences in blood pressure levels but were notably poorer, less well insured and less likely to be of European descent. Baseline depressive symptoms were more common among those participants lost to follow-up (19%) as compared to participants who remained in the study (16%).

The difference in SBP blood pressure attributable to depressive symptoms was small regardless of the approach used to handle untreated blood pressure, as seen in Table 2. The estimate of the association of depressive symptoms with SBP at exam 2, unadjusted for covariates other than baseline SBP, was 2.16 mmHG (95% Confidence Interval (CI): 0.04 to 4.29) and, adjusted for candidate confounders, was 2.45 mmHG (95% CI: 0.15 to 4.67). Both naïve restriction

(1.91 mmHG; 95% CI: 0.49 to 3.13) and censored normal regression (1.92 mmHG; 95% CI: 0.63 to 3.21) approaches gave similar adjusted results.

The estimates of the association of depressive symptoms with DBP blood pressure were smaller and less uniformly significant than those observed for SBP, as seen in Table 3. The estimate of the association of depressive symptoms with DBP at exam 2, unadjusted for covariates other than baseline DBP, was 0.93 mmHG (95% CI—0.30 to 2.17) and, adjusted for candidate confounders, was 0.82 mmHG (95% CI: −0.62 to 2.27). Both naïve restriction (0.71 mmHG; 95% CI: 0.04 to 1.39) and censored normal regression (0.91 mmHG; 95% CI: 0.27 to 1.55) approaches gave similar adjusted results suggesting that all approaches gave reasonably consistent estimates of blood pressure associations in the presence of treatment.

Based on the JNC7 definition of hypertension, there were 3130 participants who met the entry criteria for the incident hypertension analysis of whom 409 (12%) developed incident hypertension between the first and second exam. The main definition of hypertension (JNC7) showed no statistically significant association between depressive symptoms and incident hypertension [Relative Risk (RR): 1.02; 95% CI: 0.99 to 1.05] when adjusted for variable thought to be the most critical confounders. This estimate was unchanged (RR: 1.02; 95% CI: 0.98 to 1.06) when we expanded the pool of covariates to include: age, sex, ethnicity, smoking alcohol use, diabetes, body mass index, exercise (intentional and sedentary), Spielberger anxiety and anger scores, health insurance, income and baseline systolic and diastolic blood pressure

Use of the Davidson *et al.* [4] definition of incident hypertension resulted in no association between baseline depressive symptoms and incident hypertension (RR: 1.01; 95% CI: 0.98 to 1.04). After adjustment for potential risk factors, there is a small association between baseline depressive symptoms and incident hypertension using the very liberal definition of either SBP 130, DBP 80 or anti-hypertensive medication (RR: 1.05; 95% CI: 1.01 to 1.08) but the importance of this association is unclear. Considering the main definition of hypertension over a different time period (between the baseline exam and the third follow-up exam, five years later) also did not yield a statistically significant association despite longer follow-up (RR 1.03; 95% CI: 0.99 to 1.07).

As a post-hoc analysis, we considered the individual components of our operational definition of depressive symptoms. Table 4 shows the association of the individual components of our composite depressive symptoms endpoint with changes in blood pressure. Here CES-D is used as a continuous covariate but the inference would be the same with a dichotomous cut-point of 16 for depressive symptoms. Table 5 shows the association of the individual components of our composite depressive symptoms endpoint with incident hypertension (JNC7 defintion). These components were separated into: tricyclic antidepressant use, non-tricyclic antidepressant use and CES-D score. Neither non-tricyclic antidepressant use nor CES-D score was significantly associated with incident hypertension. However, tricyclic antidepressant use was associated with incident hypertension, even after the application of a full Bonferroni adjustment for multiple comparisons (p=0.03). Restriction of the sample to the 220 antidepressant users (182 non-tricyclic antidepressant users, 27 tricyclic antidepressant users, 11 users of both agents) is an approach that may partially control for antidepressant indication (and thus reduce confounding by indication) as all participants would have had an indication for medication use. In this restricted sample of 220 participants we observed an association between tricyclic antidepressant use (RR: 1.21; 95% CI: 1.04 to 1.42) and incident hypertension using non-tricyclic antidepressants as reference (adjusting for age, sex, ethnicity and CES-D score).

We tested for interactions between both ethnicity and sex with baseline depressive symptoms when estimating the relative risk of incident hypertension. There was no observed interaction between depressive symptoms and Asian (p=0.43), African-American (p=0.59) or Hispanic (p=0.78) descent. Nor was there an interaction between male sex and depressive symptoms (p=0.15). We show the estimates of this association when stratified by ethnicity in Table 6. We could not test for tricyclic effects by ethnicity as most tricyclic users were of European descent (there were 4 users of African descent, 2 users of Asian descent, 24 users of European descent and 8 users of Hispanic descent). We also noted that a 10 point change in either the Spielberger anger (RR: 0.99; 95% CI:0.95 to 1.03) or anxiety (RR: 1.01; 95% CI: 0.98 to 1.05) scores was not associated with incident hypertension (using the JNC7 definition) in this cohort.

DISCUSSION

We found a small association between baseline depressive symptoms and increases in blood pressure in the MESA population and no statistically significant association with incident hypertension. We did not find any evidence that this association between baseline depressive symptoms and increased systolic blood pressure differed by sex or ethnicity. However, it is important to note that the actual magnitude of changes in blood pressure associated with baseline depressive symptoms do not meet previous definitions of clinically significant blood pressure changes [17].

Several previous longitudinal studies have reported much stronger associations than found in the MESA population (although typically over a longer period of follow-up; ranging from 5 years to 16 years). These estimates ranged from a hazard ratio of 1.7 to an odds ratio of 2.1. These reports include a study in CARDIA by Davidson *et al.* which defined depressive symptoms and hypertension in ways similar to ours [4] although the participants were much younger. They found an association of odds ratio 2.10 between baseline depressive symptoms (as measured by CES-D score of >16) and incident hypertension among both Caucasian and African-American participants [4]. This study, in contrast to the current report, also offered some evidence that the effect of depressive symptoms might be higher in African-American participants [4].

It is also possible that some of the previous studies of depressive symptoms and hypertension may have overstated the size of the association as these papers state that they used an odds ratio to estimate this association [3,5]. Odds ratios only approximate the relative risk when the outcome is rare [23]; in the MESA example, 440 of 3194 participants (14%) develop incident hypertension based on the JNC7 criterion over the course of one follow-up visit. If we had used an odds ratio to estimate the association using the JNC7 criterion then we would have reported an odds ratio of 1.32 (95% CI: 0.98 to 1.76) which would have overstated the magnitude of the association considerably were this estimate to be interpreted as a relative risk. Some of the difference between MESA and other studies could be explained by this difference in analytical approach.

In general, previous longitudinal studies reported stronger associations than we observed and were over longer time periods [3–6]. It is not known if these differences could be due to age differences in the populations, sampling variation, differences in follow-up time or some unknown factor. In the Meyer *et al.* and the Patton *et al.* studies, hypertension was purely self report and it is unclear exactly how comparable this definition of hypertension may be compared with systematically measured blood pressure and inventoried medications [5,6]. In addition, Patton *et al.* had limited ability to adjust for covariates due to the nature of the data, with not reporting any information on confounding factors such as body mass index and physical activity [6].

It is also possible that different measures of depressive symptoms played a role; only the Davidson *et al.* study used CES-D to define depressive symptoms; the others used the General Well-being Schedule (GWB-D) [3], the Diagnostic Interview Schedule (DIS) [5] or the Composite International Diagnostic Interview Short Form (CIDI-SF) [6]. These measures may define depressive symptoms slightly differently than the CES-D and be a further source of study heterogeneity. Finally, the different composition of the MESA study might also have contributed to study differences despite the lack of any measurable effect modification by ethnicity.

In the post-hoc analysis of the individual components of our operational definition of depressive symptoms we saw a strong association with tricyclic antidepressants. This replicates the report of Licht and colleagues that tricyclic antidepressants are associated with hypertension [24]. While the number of exposed participants in this report is small and the analysis is post-hoc, it is suggestive of an adverse drug effect for tricyclic antidepressant users. Tricylic antidepressants can also cause orthostatic hypotension [25] through alpha 1 blockage [26]; these results reinforce previous findings that increased hypertension may be the more prevalent side effect in general population cohorts [27] but it is unclear if these results would generalize to older cohorts or among populations with prevalent cardiovascular disease.

There are limitations to this study. The definition used for depressive symptoms [7], while widely used in epidemiological research, is not equivalent to a full clinical diagnosis of depressive symptoms and some misclassification of depressive symptoms is possible. The reporting gap between self report of new hypertension and the second clinic exam could lead to some misclassification of exposure status and attenuate the results for incident hypertension. MESA is a multi-ethnic study and it may also be the case that different ethnicities initiate drug therapy for depressive symptoms [22] or hypertension [28] at different levels of disease severity. These differences in drug utilization could be a source of mild misclassification as medication use was part of our definitions for both depressive symptoms and hypertension. While this study does show some small associations, residual confounding [29] or an adverse drug effect [25] may explain most of the association between baseline depressive symptoms and increased systolic blood pressure. As the definition of prevalent cardiovascular disease was determined by participant self-report and there could have been some under-ascertainment of these conditions. We looked only at incident hypertension as cardiovascular conditions have been shown to predict incident depressive symptoms [30] and we wanted to avoid any concerns of reverse causality.

Perspectives

We found a small relation between baseline depressive symptoms and increases in participant blood pressure during follow-up. The magnitude of this association was smaller than that observed in previous longitudinal cohort studies in different (often younger) populations and at different time periods. There was no statistically significant association between baseline depressive symptoms and incident hypertension for the widely accepted JNC7 definition of hypertension. Increasing the follow-up time to 5 years, as a sensitivity analysis, did not change this lack of a significant association. However, the evidence that this association might be driven by tricyclic antidepressants gives reason for caution at considering treating depressive symptoms as a prevention of hypertension (as the opposite result may be achieved).

Previous longitudinal research has found much larger effect sizes than those reported in this study [3–6] and cross-sectional reports cannot distinguish whether depressive symptoms predated the diagnosis of hypertensions. The small magnitude of the associations seen in MESA would not warrant clinical concern about increased hypertension risk among patients presenting with depressive symptoms, especially in the absence of other risk factors. Additional study of these associations with high quality studies with large sample sizes, and relatively

long longitudinal surveillance periods is essential We also recommend further exploration of the possible role of tricyclic antidepressants in any such association. This additional research, especially in other populations, could contribute to further understanding any possible links between depressive symptoms and the development of hypertension.

Acknowledgments

Funding Support:

This research was supported by contracts N01-HC-95159 through N01-HC-95165 and N01-HC-95169 from the National Heart, Lung, and Blood Institute. The authors thank the other investigators, the staff, and the participants of the MESA study for their valuable contributions. A full list of participating MESA investigators and institutions can be found at http://www.mesa-nhlbi.org.

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Table 1

Descriptive statistics for the baseline characteristics of the 3185 participants from the Multi-Ethnic Study of Atherosclerosis with no anti-hypertensive medication use at either exam 1 or exam 2 based on depressive symptoms status; depressive symptoms are defined as a Center for Epidemiological Studies Depression score ≥ 16 or self-report of antidepressant medication use

Participant Characteristic [Standard Deviation]	Baseline: Depression (n=557)	Baseline: No Depression (n=3020)	Lost to Follow-up (n=346)
Age (years)	57.9 [9.8]	60.3 [9.9]	61.5 [10.7]
Male	32.1%	50.8%	47.7%
Caucasian	46.4%	41.1%	29.3%
Chinese	6.8%	15.5%	14.4%
African American	15.6%	22.6%	25.4%
Hispanic	31.2%	20.8%	30.9%
Current Smoker	17.4%	12.9%	19.1%
Ex-Smoker	36.1%	36.0%	33.8%
Alcohol (drinks/week)	4.3 [9.4]	3.9 [8.0]	3.5 [7.5]
Diabetes	7.9%	8.3%	13.0%
Impaired Glucose	22.6%	25.6%	29.2%
Body Mass Index (kg/m^2)	28.1 [5.6]	27.5 [5.1]	27.9 [5.7]
Sedentary Activities (MET-min/wk)	1720 [1163]	1612 [1115]	1671 [1191]
Exercise (MET-min/wk)	2272 [2716]	2515 [3043]	2398 [3464]
Anger Score	16.6 [4.5]	14.6 [3.4]	14.6 [4.1]
Anxiety Score	20.4 [5.2]	15.0 [3.8]	15.8 [4.7]
No Health Insurance	10.6%	9.2%	21.2%
Median Household Income, in thousands of $ [Inter-quartile range]	37.5 [18.0 to 62.5]	45 [22.5 to 87.5]	27.5 [14.0 to 62.5]
SBP, exam 1 (mmHG)	119.0 [21.1]	122.0 [19.8]	125.6 [22.7]
DBP, exam 1 (mmHG)	69.8 [9.8]	71.3 [9.9]	71.6 [10.9]
Depressive Symptoms	100%	0%	18.8%

Table 2

Association of Depression (anti-depressant medication use or Center for Epidemiological Studies Depression score ≥ 16) with Exam 2 Systolic Blood Pressure (adjusted linear regression model) in the Multi-Ethnic Study of Atherosclerosis using three different strategies for handling new users of anti-hypertensive medications at Exam 2

Variable	ΔSystolic Blood Pressure (mmHG)*	95% Confidence Limits		p-value
Restricted Analysis				
Not Depressed (CESD < 16 and no baseline anti-depressant use)	+0	Reference		n/a
Baseline anti-depressant use or CESD ≥ 16	+1.81	+0.49	+3.13	0.007
Imputation for Untreated Blood Pressure				
Not Depressed (CESD < 16 and no baseline anti-depressant use)	+0	Reference		n/a
Baseline anti-depressant use or CESD ≥ 16	+2.41	+0.15	+4.67	0.036
Censored Normal Regression				
Not Depressed (CESD < 16 and no baseline anti-depressant use)	+0	Reference		n/a
Baseline anti-depressant use or CESD ≥ 16	+1.92	+0.63	+ 3.58	0.003

* adjusted for age, sex, ethnicity, smoking, alcohol use, diabetes, body mass index, intentional exercise, sedentary activities (e.g. TV watching), anxiety and anger scores, health insurance, income and baseline systolic and diastolic blood pressure

Table 3

Association of Depression (anti-depressant medication use or Center for Epidemiological Studies Depression score ≥ 16) with Exam 2 Diastolic Blood Pressure (adjusted linear regression model) in the Multi-Ethnic Study of Atherosclerosis using three different strategies for handling new users of anti-hypertensive medications at Exam 2

Variable	ΔDiastolic Blood Pressure (mmHG)*	95% Confidence Limits		p-value
Restricted Analysis				
Not Depressed (CESD < 16 and no baseline anti-depressant use)	+0	Reference		n/a
Baseline anti-depressant use or CESD ≥ 16	+0.71	+0.04	+1.39	0.038
Imputation for Untreated Blood Pressure				
Not Depressed (CESD < 16 and no baseline anti-depressant use)	+0	Reference		n/a
Baseline anti-depressant use or CESD ≥ 16	+0.82	−0.62	+2.27	0.281
Censored Normal Regression				
Not Depressed (CESD < 16 and no baseline anti-depressant use)	+0	Reference		n/a
Baseline anti-depressant use or CESD ≥ 16	+0.91	+0.27	+1.55	0.006

* adjusted for age, sex, ethnicity, smoking, alcohol use, diabetes, body mass index, exercise (intentional and sedentary), Spielberger anxiety and anger scores, health insurance, income and baseline systolic and diastolic blood pressure

Table 4

Association of individual markers of depression with blood pressure among 3911 participants in the Multi-Ethnic Study of Atherosclerosis with no baseline anti-hypertensive medication use; change is between exam 1 and exam 2 and all estimates are done using the multiple imputation approach

Predictor	Δ Blood Pressure (mmHG)*	p-value	95% Confidence Limits	
	Systolic Blood Pressure			
Tricyclic Antidepressant Use	0.78	0.8407	−7.22	+8.79
Non-Tricyclic Antidepressant Use	0.53	0.7513	−2.83	+3.89
CESD Score (per unit) †	0.096	0.0508	−0.0003	+0.192
	Diastolic Blood Pressure			
Tricyclic Antidepressant Use	−1.36	0.6258	−7.18	+4.46
Non-Tricyclic Antidepressant Use	0.27	0.7929	−1.77	+2.31
CESD Score (per unit) †	0.071	0.1287	−0.022	+0.163

* adjusted for age, sex, ethnicity, smoking, alcohol use, diabetes, body mass index, exercise (intentional and sedentary), Spielberger anxiety and anger scores, health insurance, income and baseline systolic and diastolic blood pressure

† Note that CESD is a continuous variable in this analysis and not an indicator of CES-D ≥ 16; the binary variable would also have tested as non-significant.

Table 5

Association of individual markers of depression with Incident Hypertension (JNC7 definition; n=409) among 3130 participants in the Multi-Ethnic Study of Atherosclerosis between exam 1 and exam 2

Parameter	Relative Risk (RR)	Confidence Limits	P-value
Lightly adjusted model*			
Baseline Tricyclic Antidepressant Use (n=38)	1.24	1.06 to 1.44	0.0065
Baseline non-Tricyclic Antidepressant Use (n=193)	1.02	0.96 to 1.08	0.5976
CESD Score (per unit) ‡	1.000	0.998 to 1.002	0.8840
Fully Adjusted Model†			
Baseline Tricyclic Antidepressant Use (n=38)	1.20	1.05 to 1.37	0.0090
Baseline non-Tricyclic Antidepressant Use (n=193)	0.99	0.93 to 1.04	0.6282
CESD Score (per unit)‡	1.000	0.997 to 1.002	0.8071

*adjusted for age, sex and ethnicity.

†adjusted for age, sex, ethnicity, smoking, alcohol use, diabetes, body mass index, exercise (intentional and sedentary), Spielberger anxiety and anger scores, health insurance, income and baseline systolic and diastolic blood pressure

‡Note that CESD is a continuous variable in this analysis and not a indicator of CES-D ≥ 16; the binary variable would also have tested as non-significant.

Table 6

Association of Depression (anti-depressant medication use or Center for Epidemiological Studies Depression score ≥ 16) with incident Hypertension by ethnicity; participants with baseline hypertension are excluded

Ethnic Origin	Relative Risk (RR)*	Confidence Limits	P-value
African Descent (n=636)			
Depression	1.01	0.90 to 1.12	0.8814
Asian Descent (n=423)			
Depression	0.94	0.83 to 1.06	0.3243
European Descent (n=1390)			
Depression	1.02	0.97 to 1.08	0.4461
Hispanic Descent (n=681)			
Depression	1.06	0.98 to 1.16	0.1484
All Participants (n=3130)			
Depression	1.02	0.99 to 1.05	0.2985

* adjusted for age, sex, ethnicity, smoking, alcohol use, diabetes, body mass index, exercise (intentional and sedentary), Spielberger anxiety and anger scores, health insurance, income and baseline systolic and diastolic blood pressure