

NIH Public Access

Author Manuscript

Acta Neurol Scand. Author manuscript; available in PMC 2012 December 1.

Published in final edited form as:

Acta Neurol Scand. 2011 December ; 124(6): 396-402. doi:10.1111/j.1600-0404.2011.01491.x.

HYPERTENSION AND INCIDENT DEMENTIA IN COMMUNITY-DWELLING ELDERLY YORUBA NIGERIANS

A. Ogunniyi, MBChB¹, K.A. Lane, MS³, O. Baiyewu, MBBS², S. Gao, PhD³, O. Gureje, DSc², F.W. Unverzagt, PhD⁴, J.R. Murrell, PhD⁵, V. Smith-Gamble, MD⁸, K.S. Hall, PhD⁴, and H.C. Hendrie, DSc^{4,6,7}

¹Department of Medicine, University College Hospital, Ibadan, Nigeria

²Department of Psychiatry, University College Hospital, Ibadan, Nigeria

³Department of Medicine, Indiana University School of Medicine, Indianapolis, USA

⁴Department of Psychiatry, Indiana University School of Medicine, Indianapolis, USA

⁵Department of Pathology and Laboratory Medicine, Indiana University School of Medicine, Indianapolis, USA

⁶The Regenstrief Institute Inc., Indiana University School of Medicine, Indianapolis, USA

⁷Center for Aging Research, Indiana University School of Medicine, Indianapolis, USA

⁸Department of Psychiatry, Richard L. Roudebush Veterans Administration Medical Center, Indianapolis, IN, USA

Abstract

Objectives—To investigate the relationship between hypertension and dementia incidence in community-dwelling elderly Yoruba (age 70 years and above) because of sparse information on dementia and its risk factors in developing countries.

Materials & Methods—Community-based, prospective study of consenting elderly Yoruba using 2-stage design. Blood pressure was measured during the baseline evaluation at 2001 and hypertension was defined as BP \geq 140/90 mmHg. Diagnosis of dementia and normal cognition was by consensus using standard criteria. Non-demented subjects from the 2001 evaluation wave were re-evaluated during the 2004 and 2007 waves for dementia. Logistic regression was used to examine the association of baseline hypertension and incident dementia, after adjusting for age, gender, education, and histories of stroke and smoking. P-values <0.05 were considered significant.

Results—During the six year follow-up, 120 individuals developed dementia while 1633 remained non-demented. The frequency of hypertension in the demented group was significantly higher than in the non-demented (70.0% vs. 60.2%, p = 0.034). Baseline hypertension was a significant risk factor for dementia (OR = 1.52; 95% CI 1.01 – 2.30). Higher systolic, diastolic or pulse pressure was associated with increased risk (p<0.05). Participants with diastolic BP \geq 90 mm Hg were at a significantly greater risk than those with readings below 70 mm Hg (OR = 1.65; 95% CI 1.01–2.69).

Corresponding author: Dr. Adesola Ogunniyi, Department of Medicine, University College Hospital, Ibadan, Nigeria; aogunniyi892@gmail.com; Tel: +2348038094173. Description of authors' roles:

All the authors contributed equally to study design, data collection, data analysis, data interpretation, manuscript writing and editing. **Conflict of interest declaration:**

Conclusions—Hypertension was associated with increased risk of dementia in elderly Yoruba and its appropriate treatment may lower the risk.

Keywords

Dementia; Developing country; Higher cortical functions; Hypertension; Neurodegenerative diseases

INTRODUCTION

Dementia poses considerable public health challenge in developing countries because of the projected increase in the number of affected individuals that would occur in tandem with rapid ageing of the population [1]. The general fiscal allocation to the health sector in most of these countries is usually miniscule and would be overwhelmed by the attendant needs for optimal health care provision for the cases [2]. The right approach at curtailing the imminent epidemic is to place emphasis on preventive strategies against the modifiable risk factors. Hypertension and other cardiovascular risk factors like obesity, diabetes mellitus, and coronary artery disease have been reported to increase the risk of dementia and cognitive decline [3].

Both hypertension and dementia are age-associated conditions and are responsible for considerable disability [1,4]. The lifetime risk of developing hypertension for people who are not hypertensive at age 55 years is 90% [5]. The relationship between hypertension and dementia as well as cognitive decline appears inconsistent from results of cross-sectional and longitudinal studies where both positive and negative associations have been reported [3,6–8]. However, the results of these observational studies might have been biased because of the confounding effects of treatment. Adding to the confusion is the observation that a decrease in blood pressure occurred in the late interval preceding the onset of dementia and afterwards [9].

Dementia research is still in its infancy in developing countries and no study, to our knowledge, has addressed the relationship between hypertension and risk of dementia in people in the African continent. In a recent study in rural China, greater decline in cognitive function was found in subjects who were hypertensive, especially those not taking medication [10]. In a systematic review of hypertension in sub-Saharan Africa, the prevalence ranged between 7.5 and 29.4% using JNC 7 criteria, and was reported to be higher in urban areas with progressive increase as the population aged. There was considerable evidence of under-diagnosis, treatment and control with less than 20% of hypertensives having blood pressure within the normal range [4]. Awareness and treatment of hypertension are particularly poor in Nigeria where only about a third of the hypertensive individuals were reported to be aware of the disease and only two-thirds of those aware were on treatment according to a national survey on communicable diseases [11]. In this communication, we report the relationship between hypertension and dementia in a largely untreated community-dwelling elderly Yoruba from the Indianapolis-Ibadan dementia study.

METHODS

The Indianapolis-Ibadan Dementia Research Project is a comparative epidemiological study of prevalence, incidence and risk factors for dementia that commenced in 1992. The study participants are elderly Yoruba residents in Idikan Ward of Ibadan City, Nigeria. Recruitment into the study was conducted at two time points: 1992 and 2001. The cohort enrolled in 1992 included subjects 65 years or older and those in the 2001 cohort included subjects that were 70 years and older. Details of the study methodology have been described

elsewhere [12]. The study was approved by the Ethics Committee of the University of Ibadan/University College Hospital, Ibadan, Nigeria as well as the Indiana University-Purdue University of Indianapolis Institutional Review Board. All enrolled participants provided informed consent for each phase of the study.

Evaluation of participants

Participants enrolled in 1992 had additional cognitive assessment and clinical evaluations done in 1995, 1997, 2001, 2004 and 2007 while those recruited in 2001 were further evaluated in 2004 and 2007. All the subjects were screened in their homes. At each wave of the study, a two-stage assessment was utilized. The first stage consisted of cognitive assessment using the Community Screening Interview for Dementia (CSI-D) that included items which tested memory, orientation, language, attention, calculation, and reasoning. The results were combined to produce a Cognitive Score. A concurrent structured interview with an informant (usually a close relative) provided information on the onset and progression of any cognitive symptoms and the adequacy of the subject's daily functioning which are combined to produce an Informant Score. The informant also provided information on medical and social habits. Other information collected during the baseline evaluation in 2001 included age, gender, whether the participant attended school. Information on alcohol consumption and smoking history, history of stroke and heart attack or angina were collected by self and informant report.

Based on the cognitive and Informants' scores, the subjects were classified into three performance groups of good, intermediate and poor. They were then randomly sampled for the clinical assessment stage from prior analysis for high likelihood of dementia as follows: all subjects in the poor performance group, 75% of those in the intermediate performance category, and 5% of those identified as good performers.

The second stage included an in-home interview using the Clinician Home-based Interview to assess Function [13]; a neuropsychological battery adapted from the Consortium to Establish a Registry of Alzheimer's Disease [14]; a standardized neurologic and physical examination and a structured interview with a close relative adapted from the Cambridge Examination for Mental Disorders of the Elderly informant interview [15]. Following the second stage evaluation, participants were diagnosed as either having normal cognitive function or dementia. The diagnoses were made during consensus diagnostic conference of clinicians who reviewed the CERAD neuropsychological test battery, the physician's assessment and the informant interview. Study participants diagnosed as having dementia satisfied the International Classification of Diseases, 10th Revision [16] and the Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition criteria [17].

Study Outcome

This analysis reports on the association between incident dementia observed between 2001, when blood pressure measures were systematically obtained from the entire cohort, to 2007. Participants diagnosed with dementia in 2001 were excluded from this analysis. The study outcome was incident dementia determined by the comprehensive clinical assessment at either follow-up waves in 2004 and 2007. Participants who had a clinical assessment and diagnosed as non-demented and those who were not clinically assessed post-baseline but were in the good performance group on screening in 2007 were in the non-demented comparison group.

Blood Pressure Measurements

During the administration of the CSI'D at the 2001 evaluation wave, blood pressure was measured by trained interviewers with Omron digital units (Omron Health-care Inc.,

Bannockburn, Ill). The study participants were seated with the right arm resting at heart level and three consecutive readings were obtained. The first record was obtained twenty minutes after the commencement of the screening interview, while the second and the third readings were obtained fifteen minutes apart. The average of the three blood pressure readings was used in this analysis.

Apolipoprotein E analysis

This was conducted on a subsample of the participants who consented to this blood-drawing procedure. Blood samples were drawn in 10-ml EDTA vacutainer tubes and the specimens were transferred to the laboratory at the University College hospital, Ibadan where the buffy coat, plasma and erythrocytes were separated and then stored in -70 degree Celsius freezer. DNA was extracted using standard protocols and APOE genotyping was determined by *Hhal* digestion.

Statistical Methods

Hypertension was defined as the mean of three readings of systolic $BP \ge 140$ mmHg or diastolic $BP \ge 90$ mm Hg. Student's t-tests and chi-squared tests were used to compare the baseline demographic characteristics and hypertension status of those included in our analysis with non-demented subjects who were excluded for various reasons. We compared the baseline demographic characteristics and hypertension status of the groups with and without incident dementia using Student's t-tests for continuous variables and chi-squared tests for categorical variables. The associations between incident dementia and hypertension were analyzed using logistic regression models that were adjusted for gender, education, age at diagnosis and co-morbidities. Logistic regression models were also used to analyze for association of incident dementia with continuous measurement of systolic and diastolic blood pressure values as well as pulse pressure, defined as the difference between systolic and diastolic blood pressures. Covariates were included in the final model if their p-values for incident dementia were less than 0.10 after adjusting for gender, education and age at diagnosis. Odds ratios (OR), 95% confidence intervals (CI) and p-values are reported from the final models. The statistical software SAS version 9.2 was used for the analysis.

RESULTS

There were 2718 non-demented participants at 2001, however, 965 of them were excluded from the analysis for various reasons: 13 had no blood pressure measurements, 634 died, 95 moved, 88 were lost, 8 refused, 14 were too sick, 29 were not in the good performance group in 2007 and 84 for other reasons. Table 1 shows a comparison of the baseline characteristics between the 1753 subjects included in this analysis and the 965 non-demented subjects excluded. Those included were younger, more likely to be females, less likely to have attended school, or used alcohol than those not included in the analysis. They also had higher cognitive scores and lower systolic as well as diastolic blood pressure measurements at baseline. There were no significant differences between the two groups in history of heart attack, stroke or APOE genotypes.

During the 6-year study interval, we diagnosed 120 subjects with incident dementia which included 99 cases of Alzheimer's Disease (AD), 11 with Vascular Dementia, 5 cases of dementia with depression, 2 Parkinson's dementia, and 3 with other dementia. These were compared with 1633 non-demented subjects who comprised 625 subjects diagnosed as non-demented post-baseline and an additional 1008 that were not sampled for clinical assessment but were in the good performance group at screening in 2007. The baseline characteristics between those with and without incident dementia are shown in Table 2. There was no gender difference between the two groups. However, the demented subjects were

significantly older at diagnosis and had more self reported histories of stroke and smoking than the non-demented subjects (p<0.05). There was no difference in the frequencies of APOE e4 allele between the two groups.

Eighty four of the demented subjects (70.0%) had hypertension and this was significantly higher than the proportion of 60.2% encountered in those who were not demented (p = 0.034). On average, the systolic BP readings were 10 mmHg higher, the diastolic readings were 4 mmHg higher, and the pulse pressure was 6 mmHg higher for demented subjects (p<0.01) as shown in Table 3. Significant differences were observed between the demented and the non-demented groups for both categorical systolic and diastolic blood pressure readings (p < 0.05).

We evaluated a list of co-morbidities as potential confounders and found that only histories of smoking and stroke were significant risks for incident dementia after adjusting for age at diagnosis, gender and education (p < 0.05). Table 4 shows the results from a series of logistic regression models examining the association between various baseline blood pressure measures and incident dementia each adjusting for age at diagnosis, gender, education, and baseline histories of smoking and stroke. Hypertension was significantly associated with incident dementia after adjusting for the baseline histories of smoking and stroke, age at diagnosis, gender, and education(p=0.0450). In separate models using continuous blood pressure measures, higher systolic blood pressure, diastolic blood pressure and pulse pressure were also associated with significantly higher rate of incident dementia (p<0.05 for all). A categorical breakdown of systolic blood pressure just missed the significance level (p=0.0502), but showed a trend of subjects with systolic BP 160 mmHg or higher with increased risk for incident dementia compared to those with systolic BP of less than 120 mmHg. Breaking diastolic blood pressure into categories showed that subjects with diastolic pressure ≥90 mmHg had a significantly increased risk for incident dementia compared to those with blood pressure of 70–79 mmHg (p=0.0469).

When we re-analyzed the data using AD as the outcome, the results were similar to those for dementia, but with weaker significance levels. Higher systolic, diastolic and pulse pressures were all significantly associated with higher risk of incident AD (p=0.0176, p=0.0376 and p=0.0377, respectively). But all the categorical variables for hypertension were no longer significant for AD.

DISCUSSION

In this community-based study, we found a significant association between incident dementia and hypertension. A significantly higher proportion of the demented individuals were hypertensive and both the systolic and diastolic blood pressure readings were significantly higher in the incident dementia cases when compared with the non-demented individuals. Study participants whose blood pressure readings were above 140/90 mm Hg at baseline had 52% increased risk of developing dementia during the six-year follow-up. The association of hypertension and incident dementia in this cohort agreed with the findings in 3 longitudinal studies that reported positive correlation between the two conditions [9,18,19]. Our finding, however, differed from the report of a prospective study in a biracial population in Chicago, USA, that reported no association between both systolic or high diastolic blood pressure and cognitive decline over a 6-year period [20]. In the Chicago study, 50% of the participants took some type of anti-hypertensive medication at baseline.

The increased risk of dementia associated with hypertension thus means that more individuals are at risk of developing dementia as they get older since blood pressure increases with age. This portends a gloomy outlook since a large proportion of the affected

individuals may be unaware of the diagnosis and fewer still may be receiving optimal treatment.

The frequency of hypertension in the non-demented cohort (60.2%) is much higher than the reported crude prevalence rates ranging between 25% and 35% for individuals aged 65 years or over in the non-communicable disease survey in Nigeria [11]. However, in that study, the cut-off value used for the diagnosis of hypertension was >160 mm Hg (systolic) and >95 mm Hg. (diastolic). Our study definition conformed to the JNC 7 Report [5] for the diagnosis of hypertension as most recent studies have done. The frequency of hypertension in our study of 60% is similar to the proportion of 63.2% obtained in rural China with a median age of 70 years [10]. However, higher frequencies were obtained in European populations and in urban population in Beijing, China [21,22]. For the population of 70 year old and over in the United States, gender and ethnic variations are evident with the prevalence rate above 80% in non-Hispanic Black men and women [23].

We found that systolic blood pressure of 160 mm Hg or higher relative to 120 mm Hg or lower. increased the risk of incident dementia almost two and half times; whilst a diastolic value of 90 mm Hg or higher was associated with 65% increased risk relative to diastolic blood pressure of 70–79 mmHg. The effect of both systolic and high diastolic pressure readings persisted after controlling for stroke and history of smoking. Hypertension results in dementia and cognitive decline through promotion of arteriosclerosis and lipohyalinosis of small cerebral vessels resulting in ischemic lesions and increased volume of white matter hyperintensities in later life [3,24], Hypertension also predisposes to strokes and is also responsible for endothelial dysfunctions. Ischemic lesions tend to worsen cognitive performance as reported in the Nun study and in another study in Oxford by Esiri and coworkers [25,26].

We found greater pulse pressure as a predictor of dementia and we ascribe this to atherosclerosis. In our data, pulse pressure was highly correlated with systolic BP (correlation coefficient r=0.91) and moderately correlated with diastolic BP (correlation coefficient r=0.49). Hence subjects with greater pulse pressure also tended to have higher systolic BP. It was however impossible to assess the independent contribution of pulse pressure in addition to systolic BP in this data set due to the colinearity problem. Our study showed no association between APOE ε 4 allele and incident dementia which agreed with our previous report of lack of association between APOE e4 allele and dementia in elderly Yoruba contrary to the findings in African Americans [27].

To our knowledge, ours is the first study to examine the relationship between hypertension and dementia in a population of community-dwelling older persons in Africa. The data were based on actual blood pressure measurements rather than self reports or antihypertensive medication use. Lack of awareness and possible recall bias would have affected the quality of data if self-report had been used; while relying on those on treatment would have been unrepresentative with the attendant problem of small number reducing statistical power. Our follow-up period of 6 years falls within the range of 3 and 30 years for most longitudinal studies on this area of research [6]. We took an average of three blood pressure readings obtained from digital instruments by specially trained field workers. However, as was noted in the JNC 7 report, ambulatory blood pressure monitoring could have provided a better profile in instances of episodic hypertension and autonomic dysfunction among others [5]. The logistics of doing this in a community where compliance may be low made this impossible.

Our analyses were based on subjects with follow-up data and we did not adjust for missing data. As we have shown in the results, the 965 subjects not included in the analysis (66%

lost to follow-up due to death) had lower cognitive scores and higher BP at baseline. Therefore, our current results provide conservative estimates of the association between BP and incident dementia.

In conclusion, this study showed significant association between hypertension and incident dementia. In particular, subjects with systolic $BP \ge 160$ or diastolic $BP \ge 90$ were shown to have significant increase in risk for dementia. It may thus be worthwhile to treat individuals with hypertension to reduce the risk of dementia in elderly Yoruba. In support of this suggestion is the report that antihypertensive drug use was associated with 8% risk reduction of dementia per year in persons less than 75 years of age [28]. Antihypertensive medication use has also been reported to result in less Alzheimer disease neuropathology [29] Embarking on such treatment in developing economies is an onerous task and not likely to be sustained quite apart from the issue of compliance. Another approach is reduction of salt in the diet as primary prevention; the feasibility of which was shown in a study in Nigeria by Adeyemo and others [30]. Advocacy campaigns on community-based programs for hypertension control appear necessary and supported by our data for reducing the burden of dementia in elderly Yoruba.

Acknowledgments

Funding: The research was supported by NIH grant R01 AG09956.

We thank our field interviewers and supervisors of the Ibadan Dementia Research Project for data collection.

REFERENCES

- 1. Ferri CP, Prince M, Brayne C, et al. Global prevalence of dementia: a Delphi consensus study. Lancet. 2005; 366:2112–2117. [PubMed: 16360788]
- 2. Wimo A, Winblad B, Jonsson L. An estimate of the total worldwide societal costs of dementia in 2005. Alzheimer's & Dementia. 2007; 3
- Duron E, Hanon O. Vascular risk factors, cognitive decline, and dementia. Vasc. Health and Risk Management. 2008; 4:363–381.
- 4. Addo J, Smeeth L, Leon DA. Hypertension in Sub-Saharan Africa: a systemic review. Hypertension. 2007; 50:1012–1018. [PubMed: 17954720]
- Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. The JNC 7 Report. JAMA. 2003; 289:2560–2572. [PubMed: 12748199]
- Hanon O, Forette F. Treatment of hypertension and prevention of dementia. Alzheimer's & Dementia. 2005; 1:30–37.
- 7. Seux ML, Thijs L, Forette F, et al. Correlates of cognitive status of old patients with isolated hypertension: the Sys-Eur Vascular Dementia Project. J Hypertension. 1998; 16:963–969.
- Scherr PA, Hebert LE, Smith LA, Evans DA. Relation of blood pressure to cognitive function in the elderly. Am J Epidemiol. 1991; 134:1305–1315.
- Skoog I, Lernfelt B, Landahl S, et al. 15-year longitudinal study of blood pressure and dementia. Lancet. 1996; 347:1141–1145. [PubMed: 8609748]
- Gao S, Jin Y, Unverzagt FW, et al. Hypertension and cognitive decline in rural elderly Chinese. J Am Geriatr Soc. 2009; 57:1051–1057. [PubMed: 19507297]
- National Expert Committee on NCD. Non-communicable diseases in Nigeria: Final report of a national survey. Chapter 5. In: Akinkugbe, OO., editor. Federal Ministry of Health and Social Services. Lagos: 1997.
- Hendrie HC, Ogunniyi A, Hall KS, et al. Incidence of dementia and Alzheimer disease in 2 communities: Yoruba residing in Ibadan, Nigeria, and African Americans residing in Indianapolis, Indiana. JAMA. 2001; 285:739–747. [PubMed: 11176911]

- Hendrie HC, Lane KA, Ogunniyi A, et al. The development of a semi-structured home interview (CHIF) to directly assess function in cognitively impaired elderly people in two cultures. Int. Psychogeriatr. 2006; 18(4):653–666. [PubMed: 16640794]
- Morris JC, Mohs RC, Rogers H, Fillenbaum G, Heyman A. Consortium to establish a registry for Alzheimer's disease (CERAD) clinical and neuropsychological assessment of Alzheimer's disease. Psychopharmacol Bull. 1988; 24(4):641–652. [PubMed: 3249766]
- Hendrie HC, Hall KS, Brittain HM, et al. The CAMDEX: a standardized instrument for the diagnosis of mental disorder in the elderly: a replication with a US sample. J. Am. Geriatr Soc. 1988; 36(5):402–408. [PubMed: 3361042]
- American Psychiatric Association Press. ICD-10. The International Statistical Classification of Diseases and Related Health Problems: 1 and 2. Report No.: V.3. 1992.
- 17. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 3rd Edition. Washington, DC: American Psychiatric Association; 1987. Rev.
- Launer LJ, Ross GW, Petrovitch H, et al. Midlife blood pressure and dementia: the Honolulu-Asia aging study. Neurobiol Aging. 2000; 21:49–55. [PubMed: 10794848]
- 19. Whitmer RA, Sidney S, Selby J, Johnston SC, Yaffe K. Midlife cardiovascular risk factors and risk of dementia in later life. Neurology. 2005; 64:277–281. [PubMed: 15668425]
- 20. Hebert LE, Scherr PA, Bennett DA, et al. Blood pressure and lat-life cognitive function change: a biracial longitudinal population study. Neurology. 2004; 62:2021–2024. [PubMed: 15184608]
- Wolf-Maier K, Cooper RS, Banegas JR, et al. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. JAMA. 2003; 289:2363–2365. [PubMed: 12746359]
- He Y, Jiang B, Wang J, et al. Prevalence of metabolic syndrome and its relation to cardiovascular disease in an elderly Chinese population. J Am Coll Cardiol. 2006; 47:1588–1594. [PubMed: 16630995]
- Cutler JA, Sorlie PD, Wolz M, Thom T, Fields LE, Rocella EJ. Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988–1994 and 1999– 2004. Hypertension. 2008; 52:818–827. [PubMed: 18852389]
- 24. Swan GE, DeCarli C, Miller BL, et al. Association of midlife blood pressure in later-life cognitive decline and brain morphology. Neurology. 1998; 51:986–993. [PubMed: 9781518]
- Snowdon DA, Greiner LH, Mortimer JA, Riley KP, Greiner PA, Marksberry WR. Brain infarction and clinical expression of Alzheimer's disease: The Nun study. JAMA. 1997; 12:813–817. [PubMed: 9052711]
- Esiri MM, Nagy Z, Smith MZ, Barnetson L, Smith AD. Cerebrovascular disease and threshold for dementia in the early stages of Alzheimer's disease. Lancet. 1999; 354:919–920. [PubMed: 10489957]
- 27. Gureje O, Ogunniyi A, Baiyewu O, et al. APOE e4 is not associated with Alzheimer's disease in elderly Nigerians. Ann Neurol. 2006; 59:182–185. [PubMed: 16278853]
- Haag MDM, Hofman A, Koudstaal PI, Breteler MMB, Stricker BHC. Duration of antihypertensive drug use and risk of dementia: a prospective cohort study. Neurology. 2009; 72:1727–1734. [PubMed: 19228584]
- Hoffman LB, Scheidler J, Lesser GT, et al. Less Alzheimer disease neuropathology in medicated hypertensive than non-hypertensive persons. Neurology. 2009; 72:1720–1726. [PubMed: 19228583]
- Adeyemo AA, Prewitt TE, Luke A, et al. The feasibility of implementing a dietary sodium reduction intervention among free-living normotensive individuals in south west Nigeria. Ethn Dis. 2002; 12:207–212. [PubMed: 12019929]

Comparisons of baseline characteristics between subjects included in the analysis and those excluded from the analysis.

	Included in Analysis	Not Included	
Baseline Characteristics	(N=1753)	(N=965)	P-value
Female, n (%)	1210 (69.02%)	616 (63.83%)	0.0058
Education (any school), n (%)	224 (12.78%)	150 (15.56%)	0.0440
Age, mean \pm sd	76.18 ± 5.35	77.51 ± 6.03	<.0001
History of Heart Attack/Angina, n (%)	143 (8.16%)	93 (9.64%)	0.1898
History of Stroke, n (%)	35 (2.00%)	27 (2.80%)	0.1813
History of Smoking, n (%)	684 (39.02%)	410 (42.49%)	0.0777
History of Alcohol Use, n (%)	664 (37.88%)	415 (43.01%)	0.0089
Cognitive Score, mean \pm sd	25.81 ± 3.12	25.29 ± 3.66	0.0001
Hypertension (BP≥140/90), n (%)	1067 (60.87%)	614 (64.50%)	0.0631
Systolic BP, mmHg, mean \pm sd	150.09 ± 30.28	154.50 ± 33.18	0.0005
Diastolic BP, mmHg, mean \pm sd	83.62 ± 14.72	85.94 ± 16.10	0.0002
Systolic BP, mmHg, n (%)			0.0006
≥160	593 (33.83%)	391 (41.07%)	
140–159	463 (26.41%)	201 (21.11%)	
120–139	414 (23.62%)	224 (23.53%)	
<120	283 (16.14%)	136 (14.29%)	
Diastolic BP, mmHg, n (%)			0.0288
≥90	572 (32.63%)	365 (38.34%)	
80–89	434 (24.76%)	219 (23.00%)	
70–79	437 (24.93%)	219 (23.00%)	
<70	310 (17.68%)	149 (15.65%)	
	(N=1136)	(N=566)	
ApoE E4 allele present, n (%)	453 (39.88%)	209 (36.93%)	0.2394

Baseline Comparisons of Subjects by Incident Dementia Status

Baseline Characteristics	Non-Demented (N=1633)	Incident Dementia (N=120)	P-value
Female, n (%)	1120 (68.59%)	90 (75.00%)	0.1424
Education (any school), n (%)	214 (13.10%)	10 (8.33%)	0.1308
Age at diagnosis, mean \pm sd	80.82 ± 5.14	83.81 ± 7.33	<.0001
History of Heart Attack/Angina, n (%)	136 (8.33%)	7 (5.83%)	0.3352
History of Stroke, n (%)	28 (1.72%)	7 (5.83%)	0.0019
History of Smoking, n (%)	624 (38.21%)	60 (50.00%)	0.0106
History of Alcohol Use, n (%)	627 (38.40%)	37 (30.83%)	0.0993
	(N=1058)	(N=78)	
ApoE E4 allele present, n (%)	422 (39.89%)	31 (39.74%)	0.9801

Comparison of Hypertension and BP Information by Incident Dementia Status

Blood Pressure	Non-Demented (N=1633)	Incident Dementia (N=120)	P-value
Hypertension (BP≥140/90), n (%)	983 (60.20%)	84 (70.00%)	0.0337*
Systolic BP, mmHg, mean \pm sd.	149.41 ± 30.07	159.38 ± 31.69	0.0005^{*}
Diastolic BP, mmHg, mean \pm sd.	83.34 ± 14.60	87.43 ± 15.81	0.0033*
Pulse Pressure, mmHg, mean \pm sd.	66.06 ± 20.08	71.94 ± 21.18	0.0021*
Systolic BP, mmHg, n (%)			0.0111*
≥160	537 (32.88%)	56 (46.67%)	
140–159	436 (26.70%)	27 (22.50%)	
120–139	388 (23.76%)	26 (21.67%)	
<120	272 (16.66%)	11 (9.17%)	
Diastolic BP, mmHg, n (%)			0.0130*
≥90	517 (31.66%)	55 (45.83%)	
80-89	411 (25.17%)	23 (19.17%)	
70–79	410 (25.11%)	27 (22.50%)	
<70	295 (18.06%)	15 (12.50%)	

* Statistically significant

Results from Six Separate Logistic Regression Models on Incident Dementia by Various Blood Pressure Measures Adjusted for Age at Diagnosis, Gender, Education, and History of Smoking and Stroke Collected at Baseline.

Model Number	Effect	Odds Ratio	95% CI	P-value
1	Hypertension	1.52	1.01-2.30	0.0450
2	Systolic BP, x 10 mmHg	1.09	1.03-1.16	0.0033*
3	Diastolic BP, x 10 mmHg	1.22	1.07-1.38	0.0021*
4	Pulse Pressure, x 10 mmHg	1.10	1.01-1.21	0.0278^{*}
5	Systolic BP, mmHg			0.0502
	120–139 vs. <120	1.59	0.77-4.62	0.2119
	140–159 vs. <120	1.57	0.76-3.25	0.2219
	≥160 vs. <120	2.36	1.20-4.62	0.0125*
6	Diastolic BP, mmHg			0.0109
	<70 vs. 70–79	0.73	0.38-1.42	0.3570
	80–89 vs. 70–79	0.84	0.47-1.51	0.5618
	≥90 vs. 70–79	1.65	1.01-2.69	0.0469*

* Statistically significant