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Uncontrolled blood pressure and risks of sleep apnea among Blacks: Findings from the Metabolic Syndrome Outcome (MetSO) study

Azizi A. Seixas, **Joseph Ravenell**, **Natasha J. Williams**, **Stephen K. Williams**, **Ferdinand Zizi**, **Gbenga Ogedegbe**, and **Girardin Jean-Louis**

Center for Healthful Behavior Change, Department of Population Health, New York University School of Medicine

Abstract

Uncontrolled blood pressure (BP) is linked to increased risk of obstructive sleep apnea (OSA). However, few studies have assessed the impact of this relationship among blacks with metabolic syndrome. Data for this study were collected from 1,035 blacks (mean age = 62±13 years) enrolled in the Metabolic Syndrome Outcome (MetSO) study. Patients with a score ≥6 on the Apnea Risk Evaluation System (ARES™) were considered at risk for OSA. Of the sample, 77.1% were low-to-high OSA risk and 92.3% were hypertensive, of which 16.8% had uncontrolled BP levels. Analysis also showed 60.4% were diabetic, 8.9% had a stroke history, 74.3% had dyslipidemia, 69.8% were obese and 30.9% had a history of heart disease. Logistic regression analyses were employed to investigate associations between uncontrolled BP and OSA risk, while adjusting for known covariates. Findings showed that uncontrolled BP independently increased the odds of OSA risk twofold (OR = 2.02, 95% CI = 1.18–3.48, $p < 0.05$). Our findings show that uncontrolled BP was associated with a twofold greater risk of OSA among blacks, suggesting that those with metabolic syndrome and who have uncontrolled BP should be screened for the presence of OSA.

Keywords

uncontrolled blood pressure; hypertension; obstructive sleep apnea; metabolic syndrome; blacks

INTRODUCTION

According to the American Heart Association, 34% of US adults meet criteria for metabolic syndrome (MetS), an insulin-resistant condition that increases the risk of cardiovascular

Address correspondence and reprint requests to: Azizi Seixas, Ph.D., Center for Healthful Behavior Change, Department of Population Health, New York University School of Medicine, 550 First Avenue, VZ30 6th Floor, 629D, New York, New York 10016 p: 646-501-2672 f: 212.263.4201, ; Email: Azizi.Seixas@nyumc.org

CONFLICT OF INTEREST

All coauthors meet the criteria for authorship, including acceptance of responsibility for the scientific content of the paper. They have seen and agreed on the contents of the paper and there is no financial conflict or conflicts of interests to report. They certify that the submission is the original work and is not under review at any other publication.

disease (CVD), stroke and diabetes (T2DM).[1] Findings from the National Health and Nutrition Examination Survey indicate that blacks have a lower prevalence of MetS compared to whites.[2, 3] These findings are inconsistent with evidence that blacks are at increased risk for three of the five MetS components including, waist circumference >102 cm in men and >88 cm in women; blood pressure (BP) level ≥130/≥85 mmHg; and high fasting plasma glucose ≥100 mg/dL.[4,5] Risk for dyslipidemia among blacks tend to be lower relative to whites. Moreover, these findings are inconsistent with overwhelming evidence that indicates that blacks are at increased risk for obstructive sleep apnea (OSA) and cardiovascular disease, [6] commonly observed among individuals with metabolic syndrome.[7] Such findings should be interpreted cautiously, as they may lead to the misconception that blacks are at reduced risk for MetS and therefore less at risk for health correlates of MetS, like OSA. While there may be a need to redefine the diagnostic criteria of MetS to reflect a more accurate risk profile,[1] there appears to be a compelling rationale to assess the relative influence of each MetS component in predicting OSA risk.

Patients with metabolic syndrome are particularly at high risk for OSA. Of the five MetS components, body mass index (BMI)/large waist circumference (central fat) and impaired glucose tolerance[8] historically have been chiefly implicated in the MetS-OSA relationship, and therefore have been the two most studied drivers of the MetS-OSA association.[9] However, recent evidence indicates that obesity and diabetes may not be the strongest predictors responsible for the association between MetS and OSA. Despite the strong evidence for obesity and large waist circumference in OSA, some studies have shown that surgical[10] and dietary[11] weight loss did not reduce the amount of apnea/hypopnea index AHI, a marker of OSA severity. Additionally, Ronksley et al. found that the diabetes-OSA relationship primarily exists with patients who report excessive daytime sleepiness [12] and Barcelo et al.[13] observed a decrease in insulin resistance after three months of continuous positive airway pressure only in patients who reported excessive daytime sleepiness but not for patients who reported no daytime sleepiness. Consequently, these limitations point to other possible causes of the MetS-OSA association. Of the two remaining MetS components (dyslipidemia and hypertension), hypertension appears to offer a higher possibility of explaining the MetS-OSA relationship. Recent evidence on the negative effects of resistant hypertension on OSA provide a new framework for understanding the role hypertension plays in the development and maintenance of OSA.[7]

However, little is known about: a) how abnormal BP levels impacts OSA risk; and b) how this association manifests in diverse populations such as blacks who are at greater risk for uncontrolled BP and hypertension.[14] This study ascertained the independent associations of uncontrolled BP with the risk of OSA. It also assessed whether dyslipidemia, diabetes, and obesity have significant covarying effects on hypothesized associations between uncontrolled BP and risk of OSA.

MATERIALS AND METHODS

Study Population

Data for the present study were collected from 1,035 blacks (mean age = 62±13 years) enrolled in the Metabolic Syndrome Outcome (MetSO) study, a cohort study of patients with

metabolic syndrome. All participants were recruited from four primary-care clinics in Brooklyn, NY. During initial interviews, patients provided sociodemographic variables, health risk factors, and history of comorbid diseases, which were verified using an electronic medical record system (Allscripts). The study was approved by the SUNY Downstate Medical Center research ethics board and informed consents were obtained from all participants.

Measures and Procedures

Patients who fit the study's inclusion criteria were recruited by study staff from participating primary-care settings. Participants who identified as Black, African-American or of African ancestry, 18 years and older, who fit metabolic syndrome diagnosis were targeted. Also, patients who are pregnant or breastfeeding, involved in another study, unable to provide consent, and those who recently had a heart attack within the past 122 weeks were all excluded. Those who agreed to participate provided an informed consent before enrollment. This was followed by administration of a brief questionnaire on sleep problems, medical history, and use of medications. They also provided responses to the Apnea Risk Evaluation System (ARES™) Questionnaire, which has superior reliability in diverse groups, as compared to other apnea risk instruments.[15] Patients with a score ≥ 6 on the Apnea Risk Evaluation System (ARES™) were considered at risk for OSA.[16] The ARES questionnaire gathers information on: a) demographic and anthropometric information; b) diseases associated with risk for sleep apnea (hypertension, diabetes, heart disease, or stroke), and c) prior diagnosis of sleep apnea, the Epworth Sleepiness Scale, and frequency rating for snoring, waking up choking, and having been told that patients stopped breathing during sleep. The psychometric properties of ARES are robust with sensitivity of 0.94, moderate specificity of 0.79, positive predictive value of 0.91, and negative predictive value of 0.86.[16]

Information on body mass index (BMI), BP, high-density lipoprotein cholesterol (HDL-), low-density lipoprotein cholesterol (LDL), and fasting plasma glucose (FPG) or hemoglobin (HbA1c) for diabetic patients were obtained from electronic medical records. BMI was used instead of waist circumference because preliminary analyses indicate similar findings for BMI and waist circumference. Additionally ROC (area under the curve) analysis confirmed that both BMI and waist circumference had similar sensitivity and specificity in predicting OSA risk. Following the guidelines set by the Seventh Joint National Committee on High Blood Pressure (JNC 7), uncontrolled BP was defined as average systolic and diastolic BP ≥140/90 mmHg (for those without comorbidity), or average clinic SBP ≥130 mmHg or DBP ≥ 80 mmHg (for those with diabetes or kidney disease).[14, 17–19] BP was measured three times on two different visits at an office setting and averaged for a single BP, which was used to derive whether the participant had uncontrolled BP or not.

Statistical Analysis

To test the hypothesis that patients with uncontrolled BP were more likely to be at high OSA risk, compared with those with whose BP is we utilized multivariate-adjusted logistic regression modeling. Covariates entered in the model were age, sex, obese (defined as BMI ≥ 30 Kg/m^2), a history of diabetes, dyslipidemia, heart disease, and depression. Before constructing the model, correlational analyses were performed to assess associations

between hypothesized predictors and the dependent variable—obstructive sleep apnea; only factors showing significant associations at $p<0.05$ were entered in the final model. All analyses were performed using SPSS (version 19.0; SPSS Inc. Chicago).

RESULTS

Sixty-nine percent (69%) were female, 42.9% had an annual income lower than $10K (significantly lower than the median family income in the United States).[20] A significant number of the study sample had hypertension (92.3%) of which 16.8% had uncontrolled BP. Of the sample, the average age was 62.26 years ± 13.53, average weight of 197.78 lbs ± 48.8, average BMI of 33.8 ± 8.56, average waist circumference (central obesity) of 43.47 inches ± 12.96. 77.1% had moderate to high OSA risk, 60.4% were diabetic, 8.9% had history of stroke, 74.3% had dyslipidemia, 69.8% were obese (BMI ≥ 30 Kg/m^2) and 30.9% had a history of heart disease. Mean systolic BP was 134.98±16.39 mm Hg; diastolic BP was 75.77±10.55 mm Hg; LDL cholesterol was 105.60±36.88 mg/dL; HDL cholesterol was 48.03±16.49 mg/dL; triglyceride was 134.98±73.24 mg/dL; fasting glucose was 138.38±68.27 mg/dL; HbA1c was 7.93±1.63 % mmol/L; and BMI was 197.78±48.98 lbs.

Table 2 shows differences in the clinical characteristics of the participants with and without uncontrolled BP. Rates of OSA risk and Type 2 diabetes were significantly higher among individuals with uncontrolled BP. Table 3 shows differences in the clinical characteristics of participants with high OSA risk versus those without. Fifty-four percent of patients with uncontrolled BP were at risk for OSA. Multivariate-adjusted logistic regression analysis showed that uncontrolled BP independently increased the odds of OSA risk twofold (OR = 2.02, 95% CI = 1.18–3.48, $p < 0.05$) which was greater than an overweight and obese BMIs (OR = 1.63, 95% CI= 1.05–2.52, $p<0.05$) and triglycerides (OR = 1.003, 95% CI = 1.000–1.050, $p<0.05$) (see Table 4).

DISCUSSION

Our findings indicate that patients with uncontrolled BP are at greater OSA risk, independent of other well-established MetS risk factors like obesity (elevated BMI), Type II diabetes and triglyceride levels. Our investigation of the potential impact uncontrolled BP has on OSA among blacks is novel because previous studies primarily focused on how OSA causes hypertension. And studies that investigated the influence BP has on OSA primarily focus on resistant hypertension, a less prevalent hypertension phenotype and a condition largely defined by patients' level of responsiveness to antihypertensive medications. Identifying uncontrolled BP as a predictor of OSA, which is the main purpose of our paper, provides a unique and more economical opportunity to treat OSA, in a metabolic syndrome (MetS) population, through proper and more effective BP management.

Though our study is not the first to investigate the prevalence of MetS among blacks and the shared risk between OSA and MetS through a hypertension lens;[21] to our knowledge, it is the first to investigate the association between uncontrolled BP and OSA, using a black sample. Our approach is informed by compelling evidence that OSA is one of the strongest predictors of cardiometabolic risk and plays a central role in the pathophysiological

relationship between metabolic syndrome and cardiovascular disease and diabetes.[9] These studies have shown that approximately 60% of individuals with metabolic syndrome have moderate to severe OSA.[22] There is even stronger evidence that OSA impacts some of the five metabolic syndrome components.[2,13] Hypertension, specifically uncontrolled blood pressure (SBP≥140 mm Hg and DBP ≥90 mmHg), is strongly associated with OSA despite aggressive antihypertensive medication regimen.[14,23] OSA is highly prevalent among patients with uncontrolled blood pressure levels[25,26] and treatment of OSA through continuous positive airway pressure reduces daytime and nocturnal blood pressure.[26,27]

Of the five MetS components, obesity and diabetes have been considered the most likely causes for the MetS-OSA association.[28] Obesity is considered the most established risk factor, as several studies have found that body mass index (BMI), neck circumference[28] and visceral fat[29] increase an individual's risk of OSA. Most of these studies indicate physiological (mechanical), inflammatory and hormonal explanations for the effect obesity have on OSA. Genomic research shows that shared and non-shared biological factors, such as percentage of body fat, serum/leptin levels, waist: hip ratio and lipid metabolism, are linked to apnea/hypopnea index --a marker of OSA risk-- and BMI among whites[11] but not among blacks because they have lower rates of abdominal fat than their white counterparts. Inflammatory mechanisms are suggested by a strong association between OSA, obesity and inflammatory biomarkers. Specifically, OSA is associated with aberrant lipid and glycemic homeostasis and systemic inflammation in both non-obese and obese individuals. Physiological research suggests that obesity increases hypoventilation,[30] which can cause a cascade of events such as pharyngeal collapse, decreased lung volume and the production of adipokines (signaling proteins in the central nervous system), which restricts normal breathing pattern while sleeping[31] and eventually leads to apneic events.

Despite convincing evidence that obesity is the primary predictor of OSA risk,[21] recent studies show that surgical[32] and dietary[33] weight loss did not reduce the amount of AHI, which opens the possibility of a more robust OSA predictor. The current findings make a compelling case that uncontrolled blood pressure is a robust OSA risk factor and clinical correlate. Uncontrolled BP was the strongest predictor of OSA compared with other MetS indicators and the use of antihypertensive medication. Therefore, it could be inferred that individuals who engage in blood-pressure-lowering activities reduce their risk for OSA; as well as treating OSA could reduce BP to normotensive ranges.[34] Perhaps secondary level treatment of OSA through blood pressure education, monitoring, and management might yield greater clinical outcomes as compared to primary OSA treatments, such as surgery and continuous positive airway pressure treatment.[35–39] Blood pressure treatment has a higher adherence rate and lower non-adherence rate (BP: 28% vs CPAP: 28–83%) [40–42] and is often considered more economical than treatment with continuous positive airway pressure.[41,42]

There are several limitations that should be considered in interpreting our results. First, since our sample only included blacks, our findings cannot be generalized to other racial and ethnic groups. Second, the study does not offer a causal mechanism for the relationship between uncontrolled BP and OSA. Despite these limitations, the current findings provide

initial evidence needed to establish how uncontrolled BP might contribute to increased OSA risk.

CONCLUSION

In conclusion, our study shows that uncontrolled BP is independently associated with OSA in a sample of black patients with MetS. Specifically, these data demonstrate that blacks with uncontrolled BP are two times more likely to be at risk for OSA, as compared to those with controlled BP. Additionally, it appears that blacks with uncontrolled BP appear to be more at risk for OSA than those who are obese, have diabetes or glucose resistance problems, or dyslipidemia. Our findings provide initial evidence for addition of uncontrolled BP as a risk factor for screening of OSA risk. Such addition should improve diagnostic screening for OSA and may have potential significance for making treatment decision, such that individuals with metabolic syndrome and uncontrolled BP will be screened for OSA.

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REFERENCES

1. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C. Definition of Metabolic Syndrome: Report of the National Heart, Lung and Blood Institute/American Heart Association Conference on Scientific Issues Related to Definition. Circulation. 2004; 109:433–438. [PubMed: 14744958]
2. Reaven GM. Pathophysiology of insulin resistance in human disease. Physiol Rev. 1995; 75:473–486. [PubMed: 7624391]
3. Meis SB, Schuster D, Gaillard T, Osei K. Metabolic Syndrome in nondiabetic, obese, first-degree relatives of African American patients with type 2 diabetes: African American triglycerides-HDL-C and insulin resistance paradox. Ethnic Disparities. 2006; 16:830–836.
4. DeFronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia and atherosclerotic cardiovascular disease. Diabetes Care. 1991; 14:173–194. [PubMed: 2044434]
5. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001; 285:2486–2497. [PubMed: 11368702]
6. Olafiranye O, Akinboboye O, Mitchell JE, Ogedegbe G, Jean-Louis G. Obstructive sleep apnea and cardiovascular disease in blacks: A call to action from the Association of Black Cardiologists. American Heart Journal. 2013; 165:469–476.
7. Jean-Louis G, Zizi F, Brown DB, Ogedegbe G, Borer JS, McFarlane SI. Obstructive sleep apnea and cardiovascular disease: evidence and underlying mechanisms. Minerva Pneumol. 2009; 48:277–293. [PubMed: 21643544]
8. Relchmuth KJ, Austin D, Skatrud JB, Young T. Association of sleep apnea and Type II Diabetes: A population-based study. Am J Respir Crit Care Med. 2005; 172:1590–1595. [PubMed: 16192452]
9. Calvin AD, Albuquerque FN, Lopez-Jimenez F, Somer VK. Obstructive Sleep Apnea, Inflammation and Metabolic Syndrome. Metabolic Syndrome and Related Disorders. 2009; 7:271–277. [PubMed: 19344228]
10. Lettieri CJ, Eliasson AH, Greenburg DL. Persistence of Obstructive Sleep Apnea After Surgical Weight Loss. Journal of Clinical Sleep Medicine. 2008; 4:333–338. [PubMed: 18763424]

11. Palmer LJ, Buxbaum SG, Larkin E, Patel SR, Elston C, Tishler PV, et al. A whole-genome scan for obstructive sleep apnea and obesity. American Journal of Human Genetics. 2003; 72:340–350. [PubMed: 12506338]
12. Ronksley PE, Hemmelgarn BR, Heitman SJ, Hanly PJ, Faris PD, Quan H, et al. Obstructive sleep apnea is associated with diabetes in sleepy subjects. Thorax. 2009; 64:834–839. [PubMed: 19679579]
13. Barcelo A, Barbe F, De la Pena M, Martinez P, Soriano JB, Pierola J, Agusti AG. Insulin resistance and daytime sleepiness in patients with sleep apnea. Thorax. 2008; 63:946–995. [PubMed: 18535117]
14. Demede M, Pandey A, Zizi F, Bachmann R, Donat M, McFarlane SI, Jean-Louis G, Ogedegbe G. Resistant Hypertension and Obstructive Sleep Apnea in the Primary-Care Setting. International Journal of Hypertension. 2011; 20:1–5. (2011).
15. Levendowski DJ, Morgan T, Montague J, Melzer V, Berka C. Westbrook, PR Prevalence of probable obstructive sleep apnea risk and severity in a population of dental patients. Sleep and Breathing. 2008; 12:303–309. [PubMed: 18398630]
16. Levendowski DJ, Olmstead EM, Popovich D, Carper D, Berka C, Westbrook PR. Assessment of obstructive sleep apnea risk and severity in truck drivers: validation of a screening questionnaire. Sleep Diagnosis and Therapy. 2007; 2:20–26.
17. Calhoun DA, Jones D, Textor S, Goff DC, Murphy TP, Toto RD, et al. Resistant hypertension: diagnosis, evaluation, and treatment a scientific statement from the American Heart Association Professional Education Committee of the Council for High Blood Pressure Research. Circulation. 2008; 51:1403–1419.
18. Persell SD. Prevalence of resistant hypertension in the United States, 2003–2008. Hypertension. 2011; 57:1076–1080. [PubMed: 21502568]
19. de la Sierra A, Segura J, Banegas JR, Gorostidi M, de la Cruz JJ, Armario P, et al. Clinical features of 8295 patients with resistant hypertension classified on the basis of ambulatory blood pressure monitoring. Hypertension. 2011; 57:898–902. [PubMed: 21444835]
20. Noss, A. American Community Survey Briefs. Table H-9 Race of Head of Household by Median and Mean Income, US Census Bureau. U.S. Department of Commerce: Economics and Statistics Administration; 2013. Household Income: 2012; p. 1-2.
21. Parish JM, Adam T, Facchiano L. Relationship of metabolic syndrome and obstructive sleep apnea. Journal of Clinical Sleep Medicine. 2007; 3:467–472. [PubMed: 17803009]
22. Drager LF, Togerio SM, Polotsky VY, Lorenzi-Filho G. Obstructive sleep apnea: A cardiometabolic risk in obesity and the metabolic syndrome. Journal of the American College of Cardiology. 2013; 62:569–576. http://dx.doi.org/10.1016/j.jacc.2013.05.045. [PubMed: 23770180]
23. Walia HK, Li H, Rueschman M, Bhatt DL, Patel SR, Quan SF, et al. Association of severe obstructive sleep apnea and elevated blood pressure despite antihypertensive medication use. J Clin Sleep Med. 2014; 10:835–843. [PubMed: 25126027]
24. Logan AG, Perlikowski SM, Mente A. High prevalence of obstructive sleep apnea in drug resistant hypertension. Journal of Hypertension. 2001; 19:2271–2277. [PubMed: 11725173]
25. Sumner AE. Ethnic differences in triglyceride levels and high-density lipoprotein lead to underdiagnosis of the metabolic syndrome in black children and adults. Journal of Pediatrics. 2009; 155:S7–e7. [PubMed: 19732569]
26. Pedrosa RP, Drager LF, Gonzaga CC, Sousa MG, de Paula LK, Amaro AC, et al. Obstructive sleep apnea: the most common secondary cause of hypertension associated with resistant hypertension. Hypertension. 2011; 58:811–817. [PubMed: 21968750]
27. Logan AG, Tkacova R, Pelikowski SM, Tisle A, Floras JS, Bradley TD. Refractory hypertension and sleep apnea: effects of CPAP on blood pressure and baroreflex. European Respiratory Journal. 2003; 21:241–247. [PubMed: 12608436]
28. Davies RJO, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnea syndrome. European Respiratory Journal. 1990; 3:509–514. [PubMed: 2376247]
29. Pillar G, Shehadeh N. Abdominal fat and sleep apnea: The chicken or the egg? Diabetes Care. 2008; 31:303–309.

30. Kostolglou-Athanassiou I, Athanassiou P. Metabolic syndrome and sleep apnea. Hippokrata. 2008; 12:81–86.
31. Schwartz AR, Patil SP, Laffan AM, Polotsky V, Smith PL. Obesity and obstructive sleep apnea: Pathogenic mechanisms and therapeutic approaches. Proceedings of the American Thoracic Society. 2008; 5:185–192. [PubMed: 18250211]
32. Lettieri CJ, Eliasson AH, Greenburgh DL. Persistence of obstructive sleep apnea after surgical weight loss. Journal of Clinical Sleep Medicine. 2008; 4:333–338. [PubMed: 18763424]
33. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea. American Journal of Respiratory Critical Care Medicine. 2002; 165:1217–1239. [PubMed: 11991871]
34. Schein ASO, Kerkhoff AC, Coronel CC, Plentz RDM, Sbruzzi G. Continuous positive airway pressure reduces blood pressure in patients with obstructive sleep apnea; a systematic review and meta-analysis with 1000 patients. J Hypertens. 2014; 32:1762–1773. [PubMed: 24979300]
35. Levine DM, Green LW, Deeds SG, Chwalow J, Russell RP, Finlay J. Health education for hypertensive patients. JAMA. 1979; 241:1700–1703. [PubMed: 430732]
36. Gonzalez-Fernandez RA, Rivera M, Torres D, Quiles J, Jackson A. Usefulness of a systemic hypertension in-hospital educational program. Am J Cardiol. 1990; 65:1384–1386. [PubMed: 2188495]
37. Farquhar JW, Fortmann SP, Flora JA, Taylor CB, Haskell WL, Williams PT, Maccoby N, Wood PD. Effects of communitywide education on cardiovascular disease risk factors: the Stanford Five-City Project. JAMA. 1990; 264:359–365. [PubMed: 2362332]
38. Hill MN, Bone LR, Hilton SC, Roary MC, Kelen GD, Levine DM. A clinical trial to improve high blood pressure care in young urban black men: recruitment, follow-up, and outcomes. Am J Hypertens. 1999; 12:548–554. [PubMed: 10371363]
39. Sclar DA, Chin A, Skaer TL, Okamoto MP, Nakahiro RK, Gill MA. Effect of health education in promoting prescription refill compliance among patients with hypertension. Clin Ther. 1991; 13:489–495. [PubMed: 1934001]
40. Tomaszewski M, White C, Patel P, Masca N, Damani R, Hepworth J, et al. High rates of non-adherence to antihypertensive treatment revealed by high-performance liquid chromatography-tandem mass spectrometry (HP LC-MS/MS) urine analysis. Heart. 2014; 100:855–861. [PubMed: 24694797]
41. Weaver TE, Kribbs NB, Pack AI Kline LR, Chugh DH, Maislin G, et al. Night-to-night variability in CPAP use over the first three months of treatment. Sleep. 1997; 20:278–283. [PubMed: 9231953]
42. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. Proc AM Thorac Soc. 2008; 5:173–178. [PubMed: 18250209]

Original Article Table Fact Sheet

What is known on this topic
• Individuals with metabolic syndrome (MetS) are at increased risk for obstructive sleep apnea (OSA)
• Of the five MetS components, obesity/abdominal fat (central fat)/waist circumference is considered the most established risk factor of OSA. However, recent evidence suggests that obesity reduction did not reduce OSA risk opening the case for another risk factor such as hypertension.
What this study adds
• Uncontrolled hypertension among Blacks with metabolic syndrome increases their risk for OSA
• Uncontrolled hypertension is the strongest predictor of OSA, compared to obese BMI, antihypertensive medication, cholesterol, triglycerides, blood glucose and HbA1c.

Table 1

Metabolic characteristics of the study participants

Variable	Mean	SD
Systolic BP (mmHg)	134.98	16.39
Diastolic BP (mmHg)	75.77	10.55
LDL Cholesterol (mg/dL)	105.6	36.88
HDL Cholesterol (mg/dL)	48.03	16.49
Triglycerides (mg/dL)	134.98	73.24
Glucose (mg/dL)	138.38	68.27
HbA1c (mmol/L)	7.93	1.63
BMI	197.78	48.98

Note: BP= Blood Pressure; LDL= Low-density lipoprotein, HDL = High-density lipoprotein; HbA1c= glycated hemoglobin; BMI= Body Mass Index in pounds.

Table 2

Differences between Health Risks and Medical Characteristics of Participants with Uncontrolled BP and Controlled BP

Variable	Uncontrolled BP (%)	
	YES	NO
OSA risk *	25.9	16.0
BMI ≥30kg/m^2	18.6	21.7
T2DM **	23.7	7.20
Stroke	17.8	17.1
DLA	17.6	16.5
CHD	15.0	18.1

Note. Uncontrolled BP= average systolic and diastolic BP ≥140/90 mmHg (for those without comorbidity), or average clinic SBP ≥130 mmHg or DBP ≥ 80 mmHg; OSA risk=obstructive sleep apnea risk ARES ≥ 6; BMI = body mass index; T2DM = Type 2 Diabetes; CHD = Coronary Heart Disease; DLA= Dyslipidemia; Significance was determined by Fisher's Exact test

* $p \leq .05$;

** $p \leq .001$

Table 3

MetS indicators and OSA risk

Variables	OSA risk	No OSA risk	Fisher Exact Significance (*p* value)
Insulin/Glucose	46.7%	53.3%	N.S.
Dyslipidemia	48.6%	51.4%	N.S.
Uncontrolled BP/Hypertension	54.0%	46.0%	.068†
BMI (Overweight-obese)	49.0%	51.0%	.009

Insulin/Glucose=Fasting plasma glucose > 100 mg/dL; Dyslipidemia=Plasma triglycerides > 150 mg/dL, HDL cholesterol < 40 mg/dL in men and < 50 mg/dL in women; Elevated BP/Hypertension=BP medication or BP > 130/85 mm/Hg; Waist Obesity=Waist circumference > 40 inches in men and > 35 inches in women.

† trending to significance.

Table 4

Multivariate logistic regression analysis indicating odds ratios (ORs) for Uncontrolled BP associated with OSA risk in the MetS; N= 1,035.

Variables	OR (Odds Ratio)	95% CI		*p*
Uncontrolled BP	2.020	1.177	3.480	0.011
Anti-Hypertensive	1.021	0.541	1.920	0.950
LDL Cholesterol	0.996	0.989	1.000	0.290
HDL Cholesterol	0.998	0.984	1.010	0.832
Triglycerides	1.003	1.000	1.005	0.028
Glucose	0.998	0.994	1.000	0.310
HbA1c	0.890	0.756	1.050	0.157
BMI	1.630	1.050	2.520	0.029

Note: Uncontrolled BP= average systolic and diastolic BP ≥140/90 mmHg (for those without comorbidity), or average clinic SBP ≥130 mmHg or DBP ≥ 80 mmHg; LDL= Low-density lipoprotein; HDL = High-density lipoprotein; HbA1c= glycated hemoglobin; BMI (Obese) = BMI ≥30kg/m^2