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Disparities and Genetic Risk Factors in Obstructive Sleep Apnea

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Abstract

Obstructive sleep apnea (OSA) is an increasingly prevalent condition. A growing body of literature supports substantial racial disparities in the prevalence, risk factors, presentation, diagnosis and treatment of this disease. Craniofacial structure among Asians appears to confer an elevated risk of OSA despite lower rates of obesity. Among African Americans, Native Americans, and Hispanics, OSA prevalence is increased, likely due in part to obesity. Burden of symptoms, particularly excessive daytime sleepiness, is higher among African Americans, though Hispanics more often report snoring. Limited data suggest African Americans may be more susceptible to hypertension in the setting of OSA. While differences in genetic risk factors may explain disparities in OSA burden, no definitive genetic differences have yet been identified. In addition to disparities in OSA development, disparities in OSA diagnosis and treatment have also been identified. Increased severity of disease at diagnosis among African Americans suggests a delay in diagnosis. Treatment outcomes are also suboptimal among African Americans. In children, tonsillectomy is less likely to cure OSA and more commonly associated with complications in this group. Among adults, adherence to continuous positive airway pressure (CPAP) is substantially lower in African Americans. The reasons for these disparities, particularly in outcomes, are not well understood and should be a research priority.

Keywords

Obstructive sleep apnea; race; ethnicity; disparities; continuous positive airway pressure (CPAP)

Background

Obstructive sleep apnea (OSA) is one of the most prevalent sleep disorders with moderate to severe disease affecting up to 17% of middle-aged men and 9% of middle-aged women.[1] OSA is associated with numerous adverse consequences including excessive daytime sleepiness, motor vehicle accidents, hypertension and cardiovascular disease.[2] A large body of literature has identified risk factors for OSA, consequences of the disease and treatment

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options. However, studies evaluating the extent to which the development, presentation, consequences and management of OSA vary by race have not been as extensively considered. This article will review known differences in OSA by racial background as well as point out areas where further research is needed.

Disparities in OSA Prevalence

Few studies have directly compared the prevalence of OSA across racial groups. In addition, the lack of consistent criteria to define OSA limits comparisons of OSA prevalence across studies. Nevertheless, available data indicate an elevated prevalence of OSA among African Americans, Hispanics, and Native Americans as compared to US whites while the prevalence of OSA in Asians appears comparable to whites.

The strongest evidence for a racial disparity in OSA exists with regards to African Americans. Several studies have found a higher rate of OSA in African Americans particularly African American children.[3] Among pediatric patients evaluated in sleep clinic, African American race is associated with a 20% increase in OSA severity[4] and greater oxygen desaturation.[5] African American children are 4-6 times more likely to have OSA compared to white children.[6,7] Even among young adults less than 26 years of age, African Americans are 88% more likely to have OSA as compared to whites.[8] Among middle-aged populations, the evidence for a disparity in OSA prevalence is weaker as differences in OSA prevalence from community based studies are evident in some but not all studies.[9-12] In contrast, data from older populations suggests a disparity may re-emerge in this age group. While African Americans had similar prevalence of OSA as whites (32% and 30% respectively) in a community-based survey of individuals 65 years of age and older, this group was 2.1 times more likely to have severe OSA.[13]

Data are somewhat more limited regarding OSA prevalence in US Hispanics. The Hispanic Community Health Study (HCHS) used portable sleep monitoring to evaluate the prevalence of OSA in a diverse US Hispanic cohort of over 14,000 adults. The prevalence of mild, moderate and severe OSA in this cohort was 25.8%, 9.8%, and 3.9% but OSA risk was found to vary substantially by Hispanic background being greatest among Cuban men. Consistent with other racial groups, older age, male gender, and obesity were independent risk factors for OSA in this cohort.[14] Although the prevalence is somewhat greater than estimates of community-based white populations, the monitoring system used is very different making direct comparisons difficult.

A cross-racial survey utilizing overnight oximetry, however, did find a higher rate of OSA in Hispanics compared to whites.[11] In contrast, an analysis of data from one site of the Multi-Ethnic Study of Atherosclerosis (MESA) found that the rate of OSA in Hispanics was similar to whites.[15] However, a more recent analysis evaluating subjects at all MESA sites has reported a higher prevalence in Hispanics.[16]

Information about OSA in Native Americans is sparse. The best evidence comes from SHHS where the odds of moderate to severe OSA was 1.7 times greater than that found in whites[10]

Unlike African Americans, Hispanics and Native Americans, the prevalence of OSA in Asians and Asian Americans appears similar or lower than that of whites. In a cross-study analysis comparing Japanese participants in the Circulatory Risk in Communities Study (CRICS) to whites in MESA, the prevalence of OSA among Japanese was roughly half that of whites (18.4% vs. 36.5%).[15] However, in other studies, Asians have been found to have similar OSA severity to whites.[17] In the Male Study of Osteoporosis (MrOS) cohort of older men, Asian American background was an independent risk factor for OSA.[18] This is consistent with population-based studies from Asia where high rates of OSA have been found in China, Japan, Korea and India, despite low rates of obesity.[19-22]

In summary, current data from population-based studies suggest the prevalence of OSA is greater among African Americans, Hispanics, and Native Americans although direct comparisons particularly for Hispanics and Native Americans compared to other groups are limited. The greater prevalence in African Americans is particularly notable in younger and older age groups. Asians and Asian Americans appear to have comparable rates of OSA to whites despite markedly lower levels of obesity.

OSA Risk Factors

Understanding the basis of disparities in OSA prevalence requires an evaluation of disparities in the risk factors for OSA as well as an assessment of racial heterogeneity in how risk factors contribute to OSA pathogenesis. Among the most studied OSA risk factors are craniofacial shape and obesity.

Craniofacial shape—Craniofacial shape has been recognized as an important contributor to OSA risk. Both skeletal features such as maxillary-mandibular shape, inferior hyoid position, and small cranial base, as well as soft tissue features such as size of the tongue, soft palate, tonsils, pharyngeal walls, and parapharyngeal fat pads have been identified as OSA risk factors. In general, studies suggest soft tissue factors may be more relevant to predicting risk in African Americans while skeletal features are more predictive in Asians.[23-25]

Studies comparing African Americans to whites have found tongue area is significantly larger in African Americans with OSA. In contrast, skeletal features such as brachycephaly (a skull shape with a greater lateral compared to antero-posterior dimension) were predictors of OSA severity in whites but not African Americans.[26] In contrast, Asians with OSA have more skeletal restriction than their white counterparts as measured by a shorter cranial base as well as difference in length and positioning of the maxilla and mandible.[27-31] In addition, both an inferiorly positioned hyoid and an extended craniocervical angle have been demonstrated to predict OSA risk in Asians.[28,29] However, it is important to note that heterogeneity does exist across Asian backgrounds in the relationship between craniofacial risk factors and OSA.[32]

As compared to African Americans and Asians, there is much sparser data on the relationship between craniofacial shape and OSA risk in Hispanics and Native American groups. Only a few studies have evaluated differences in craniofacial shape between Hispanics and whites that could contribute to differences in OSA risk and these have been inconclusive. One study found bi- maxillary retroposition to be more common among

Hispanics with OSA than apneics of other races,[33] however another study did not find any differences between Hispanics and whites.[34] Table 1 summarizes the contributing soft tissue and skeletal contributors to OSA, with racial differences noted where literature is available.

Obesity—Obesity is one of the strongest risk factors for OSA, with over 50% of OSA diagnoses attributable to overweight.[35] Obesity likely contributes to OSA through increased fat deposition in neck subcutaneous fat as well as other soft tissue structures as well as a reduction in lung volumes.

Obesity risk varies widely by race. The prevalence of obesity was 37.1% in African American men and 56.6% in African American women in the US in 2012.[36] As compared to other groups, African Americans have a 51% increased likelihood of obesity, even after age, sex, comorbidities, and socioeconomic factors are considered.[37] Reasons behind the higher obesity rates among African Americans are not fully understood. One potential explanation is decreased physical activity—particularly among African American women.[38-40] This may reflect competing time interests that leave no time for exercise or other physical activities. Indeed, data from the National Health and Nutrition Survey (NHANES) suggests that African Americans are more likely to have no leisure time activity as compared to whites; nearly three-quarters of middle-aged African Americans reported no physical activity during leisure time in 2010, as opposed to 43% of whites.[39]

Another potential explanation is a diminished basal metabolic rate and energy expenditure among African Americans as compared to whites.[41-43] In contrast, no consistent differences in caloric intake by race have been identified.[39] Socioeconomic factors may also contribute through effects on access (in terms of proximity, cost) to fresh fruits and vegetables versus processed foods, dietary choices, and walkability of neighborhoods. Finally, the impact of childhood obesity should not be overlooked, as one-third of obesity at age 20 was attributed to rapid weight gain during infancy among African Americans in one cohort.[44]

The impact of obesity among African Americans explains at least some degree of the increased prevalence of OSA. For example, in community-based surveys and cohorts, such as SHHS, once differences in body mass index (BMI) are accounted for, African American background is no longer an independent risk factor for OSA.[9,10]

US Hispanics are also disproportionately impacted by obesity – in 2012, the prevalence of obesity among Hispanics in the US was 42.5% (40.1% among men and 44.4% among women).[36] Similar to African Americans, rates of leisure time physical activity are low.[39] Other studies have demonstrated that although the aggregate rate of Hispanic obesity is greater than that of whites, there is marked heterogeneity within Hispanic subgroups.[45] Mexican and Puerto Ricans, for example, have higher rates of obesity as compared to other Hispanic groups, with widening disparities as individuals transition from children to adults.[45]

Native Americans, as a group, also have higher obesity rates than whites. In the Behavioral Risk Factor Surveillance System (BRFSS), 33.9% of Native American men and 35.5% of Native American women reported obesity as compared to 23.3% and 21.0% in white man

and women respectively.[46] Similar to African Americans and Hispanics in SHHS, after accounting for differences in adiposity, Native American background was not an independent risk factor for OSA, suggesting again that the increased OSA prevalence is a direct consequence of disparities in obesity.[10]

In contrast to these other groups, obesity rates in Asian Americans based on a BMI threshold of 30 kg/m^2 are markedly lower than whites. Data from NHANES demonstrates a prevalence of only 10.8% in Asian Americans.[36] Even after adjustment for socioeconomic factors and other potential confounders, Asian Americans are roughly one third as likely to be obese compared to whites.[37] However, because Asians have greater amounts of body fat for the same BMI,[47] the World Health Organization and other organizations have advocated a lower BMI threshold (e.g., 25 kg/m^2) for defining obesity in Asian populations.[48] The shape of the BMI – diabetes relationship in Asians supports use of a lower threshold for defining obesity in this group.[49] The BMI – OSA relationship in Asians similarly supports the use of a lower threshold to define elevated risk.

When matched to whites with similar OSA severity, Asians tend to have more bony restriction while whites have a greater BMI.[27] Though some studies have suggested a similar effect of BMI on AHI across race,[15] recent data from MESA suggest that each unit BMI increase may have a greater effect on AHI in Asian Americans than other US racial groups.[16] This is supported by data suggesting the prevalence and severity of OSA is much greater among Asians than whites when matched for BMI.[50]

Pulmonary function and ventilatory control—In children, other respiratory problems such as sinus disease and asthma may be risk factors for OSA. [7] In addition, secondhand tobacco exposure has been associated with elevated OSA risk in this age group.[51-53] Both asthma and secondhand tobacco exposure are more common among African American children and may contribute to the OSA disparity observed in this group.[54] Additional data suggests a possible role of lung function contributing to OSA. Among African Americans age 25 years of age and younger, lower vital capacities are observed after corrected for age, sex, and height.[8]

Recent studies have demonstrated novel phenotypes such as upper airway muscle responsiveness, loop gain, and arousal threshold may contribute to OSA risk.[55] Whether there are differences in these phenotypes by race has not yet been studied. However, at least one small study suggests differences in chemoreflex responses during sleep may exist between African Americans and whites.[56]

Genetics—Family history has long been identified as a strong risk factor for OSA. In both whites and African Americans participating in the Cleveland Family Study (CFS), roughly one third of the total variability in apnea hypopnea index (AHI) was explained by shared genetic factors.[57,58] The corresponding estimates for BMI in this cohort are 0.53 for whites and 0.54 for African Americans, while for minimum cross-sectional area of the upper airway as assessed by acoustic pharyngometry is 0.34 and 0.39 respectively.[57-59]

In contrast to these very similar global findings of the importance of genetics across races, identification of candidate loci for OSA has differed by race. Linkage analyses from the CFS identified regions on chromosomes 2 and 19 as the best candidates for an OSA locus in whites but a region on chromosome 8 was the best candidate in African Americans.[57,58] Of note, however, none of these regions met genome wide significance levels so the meaning of this heterogeneity is unclear. With regards to specific loci, many loci have been reported as OSA risk variants in the literature with little replication within or across races. To date, no variant has been identified as meeting a genome-wide level of statistical significance ($p < 5\times10^{-8}$). However, several analyses have directly compared results in whites and African Americans within the same study. A candidate gene analysis found variants in C-reactive protein (CRP) and glial cell line-derived neurotrophic factor (GDNF) were associated with OSA phenotypes in whites while a SNP in serotonin receptor 2a (HTR2A) was associated with OSA in African Americans.[60] In a broader assessment across multiple cohorts, a SNP in prostaglandin E2 receptor (PTGER3) was associated with OSA phenotypes in whites while a SNP in lysophosphatidic acid receptor 1 (LPAR1) was associated with OSA phenotypes in African Americans.[61] These differences in association may reflect true differences in the underlying genetic basis for OSA across races. However, there are other potential reasons for these heterogeneous findings. It is important to remember that the index SNP identified at each locus is most likely not the causal SNP but simply a surrogate for the causal SNP. As such, differences in the prevalence of the index SNP across races may result in differences in power to detect an association. Furthermore, differences in the underlying genetic architecture between races means that the strength of association between the index SNP and true causal SNP will vary. Studies assessing the transferability of genetic findings for BMI in whites to other races are informative in this regards. When studied on a SNP by SNP comparison, roughly half the SNPs identified as influencing BMI in whites were found to also be relevant in an Asian population.[62] However, when analyses were conducted at a locus level to account for differing genetic architecture, evidence was found for an association in Asians for most of the loci identified in whites. Similar results have been reported comparing whites and African Americans as well.[63] Of note, the two strongest BMI loci, in the fat mass and obesity-associated protein (FTO) and melanocortin 4 receptor (MC4R) gene regions, have been replicated across multiple races including whites, African Americans, Asians, and Hispanics, suggesting that for obesity, at least, the underlying genetic basis is similar across human populations.[62]

OSA Symptoms and Consequences

While a number of studies have established the disparities that exist in OSA prevalence across racial groups, fewer studies have examined differences in the consequences of OSA including how presentation of the disease may vary by race. However, given the cultural differences that exist regarding sleep, it is no surprise that reports of common OSA-related symptoms might vary. With regards to cardiovascular disease (CVD), the substantial disparity in CVD borne by certain racial groups, particularly African Americans, has made it a research priority to understand the extent to which differences in OSA prevalence or susceptibility to the effects of OSA might explain disparities in CVD.

Snoring/Apnea—Analyses of SHHS data indicate that self-report of snoring varies substantially by race with important modification by gender. Among women, African Americans are most likely to report frequent snoring, while among men, Hispanics are most likely.[64,65] Data from NHANES also suggests non-Mexican Hispanics are the most likely to report both snoring as well as snorting/gasping symptoms.[66] Interestingly, snoring in SHHS was associated with an increased risk of OSA in all racial groups except Hispanics. Similarly, in pediatric cohorts, Hispanic children are more likely to be reported to snore but snoring in this group is not associated with OSA risk. [67,68] In contrast, among African-American children, snoring is increased in prevalence and predictive of OSA.[69] It is important to note heterogeneity within racial groups. For example, Caribbean-born blacks appear to have higher rates of snoring than African Americans.[70]

In contrast to snoring, breathing pauses in SHHS were most frequently reported by Native Americans, both among women and men. However, unlike in all other races, breathing pauses during sleep were not associated with elevated OSA risk in American Indians.[64]

Studies comparing OSA symptoms in Asians to other groups are sparse. The Honolulu-Asia Aging Study of older Japanese-American men found 12% reported snoring loudly on a regular basis. Interestingly, prevalence of snoring decreased with age in this cohort.[71] Among Asian populations outside of the United States, reported rates of snoring and witnessed apneas are quite variable. Snoring prevalence varies from 4.6% in Thailand to as high as 59.1% in Taiwan[72] while prevalence of witnessed apneas ranges from 2.6% in Taiwan to 15.2% in Malaysia.

Sleepiness—Excessive daytime sleepiness is the most common daytime symptom of OSA.[73] Several studies have demonstrated that there are racial differences in sleepiness as assessed by the Epworth Sleepiness Scale (ESS). In the SHHS, ESS scores were nearly one point higher among African Americans compared to whites. Among OSA populations, this finding has been confirmed with an ESS score 1.5-2.3 points higher in African Americans than whites.[74-77]

On closer examination of ESS responses, the difference in total scores was found to be due primarily to differing responses to two of the eight questions suggesting there may be variation in the performance characteristics of the ESS between African Americans and whites.[78] Another study found US-born African Americans are 68% more likely to report excessive daytime sleepiness compared to foreign-born blacks in the US suggesting cultural effects on report of sleepiness.[79] Whether objective differences in sleepiness exists between African Americans and whites is not known and if differences are found, it is unclear the extent to which such differences are due to differing susceptibility to OSA or differing prevalences of competing causes of daytime sleepiness such as chronic sleep restriction and shiftwork. [79]

In contrast to African Americans, there is not strong evidence that Hispanics suffer from more daytime sleepiness. In adjusted analyses, Hispanics in MESA were less likely to report frequent excessive daytime sleepiness than whites.[76] Asian-American women in MESA were also not sleepier than white women. Among older Japanese-American men, the

Honolulu-Asian Aging Study found that 8% reported excessive daytime sleepiness; interestingly, this was associated with other chronic diseases and not self-report of snoring.[71] However, studies from Asia suggest heterogeneity across regions in Asia in excessive daytime sleepiness with prevalences ranging from 3.9% to 24%.[72] A cross-ethnic study from Singapore found higher rates of daytime sleepiness among those of Malay and Chinese background compared to those of Indian heritage.[80]

Cardiovascular Consequences of OSA—OSA is a strong risk factor for hypertension and has also been linked to other cardiovascular disease including stroke, coronary artery disease and heart failure. There is a striking difference in the prevalence of these cardiovascular outcomes when examined by race. For example, among African Americans, severe hypertension is five to seven times more prevalent as compared to whites.[81] The extent to which OSA may play explain some of this disparity is unclear. However, the fact that OSA is typically associated with a 'non-dipping' blood pressure pattern where blood pressure doesn't fall during sleep and African Americans with OSA are more likely to have a 'non-dipping' blood pressure profile than whites. [82] Furthermore, the relationship between 'non-dipping' and OSA severity appears to vary by race and use of antihypertensive medications.[82]

No study has yet assessed the relationship between polysomnographically established OSA and hypertension risk across races. However, an analysis of NHANES data using a definition of OSA based on self-reported diagnosis or symptoms found OSA was more strongly associated with hypertension in African Americans (OR = 4.7) than in Hispanics (OR = 2.0) or whites (OR = 1.7). [83] Furthermore, observational data suggests treatment of OSA with CPAP may result in greater reduction of blood pressure in African Americans compared to whites.[84]

Despite substantial evidence that African Americans are at much higher risk for other adverse cardiovascular outcomes, in particular stroke and heart failure, there has been virtually no research on racial heterogeneity on the impact of OSA on these outcomes. The SHHS has reported on the impact of OSA on incident cardiovascular outcomes in a racially diverse population,[85,86] but no stratification by race has yet been conducted. A small study did report that among patients presenting with stroke, Hispanics were more likely to be at risk for OSA based on the Berlin questionnaire as compared to whites and African Americans.[87] However, differences in stroke risk among OSA patients by race have not been assessed prospectively.

OSA Evaluation and Management

Because many of the tools used to screen for OSA and associated symptoms were developed in primarily white populations, the validity of these instruments in other races is unclear. For example, both the Berlin and STOP-BANG instruments use a BMI threshold derived for non-Asian populations. Cultural differences in the understanding of normal sleep and breathing likely impact the presentation of OSA as well. Reduced access to healthcare in general among certain populations likely impacts access to OSA evaluation and care as well, though little data exist in this regard. With regards to treatment, emerging evidence suggests

African Americans in particular may have poorer outcomes from standard OSA therapies. The reasons for this are not as yet clear.

Diagnosis and Treatment—Data suggests that racial and other minority groups may have reduced access to health care.[88] These disparities in access to care and outcomes are best described among African Americans. However they have been recognized among all minority groups. Therefore, assessment and recognition of obstructive sleep apnea likely varies by race.

One small study examined source of referral within an urban academic medical center and found that African Americans are less likely to self-refer to sleep compared to whites, though this difference only approached significance.[74] This could be due to misconceptions about OSA. Focus groups among African Americans have shown a perception of OSA as a type of insomnia or related to aging.[89] Perhaps due in part to this, African Americans are less likely to follow through on a recommendation to have a sleep study, with only 38% presenting for a sleep study appointment.[90] This may reflect perceived unfamiliarity and discomfort with the sleep laboratory environment.[89] However, once CPAP is recommended, some studies suggest CPAP acceptance is equal to that of whites.[74,89]

There may also be social and cultural differences that contribute to differences in diagnosis and treatment. Fewer African Americans are married at the time of diagnosis as compared to whites,[74] which could account for some of the differences in initiation of referral (e.g. by spouse). Additionally, some research suggests that African American bed partners are more accepting of snoring as compared to whites, and more often felt that snoring was normal.[91] Delay in diagnosis for all of these reasons may explain the increased severity of OSA found among both African American adults and children upon presentation to the sleep clinic.[90,92,93]

Adenotonsillectomy—Adenotonsillectomy is first line treatment for pediatric OSA. However, African American children with OSA are less likely to receive adenotonsillectomy than white children.[94] This may be due to barriers to accessing health care, insurance type, and other socioeconomic factors.[95,96]

Among those who do receive adenotonsillectomy, African American children are more likely to have residual disease after surgery than white children and more likely to have recurrent disease 1 year after surgery.[97,98] In addition, African American children are more likely to have higher pain medication requirements after surgery for OSA[99] and have higher rates of respiratory complications.[100]

Continuous Positive Airway Pressure (CPAP)—CPAP is first line therapy for OSA in adults. Research suggests African Americans with OSA are equally accepting of a CPAP titration study and home CPAP set-up as whites.[74] Despite this, numerous studies have demonstrated decreased CPAP adherence among African American populations.[101-106] On a nightly basis, African Americans use CPAP an average of 60-90 minutes less than whites.[101,103,105-108] One study found that African Americans were approximately five times more likely to be non-adherent to CPAP than whites.[106] The reasons behind this

disparity in CPAP adherence are not completely understood, however shorter nightly sleep duration among African Americans may be a contributor.[109] Long sleep onset latency and sleep onset insomnia symptoms also appear to be more prevalent among African American with OSA, and may contribute to poorer adherence.[103,109] Low socioeconomic status (SES) has explained racial differences in some but not all studies.[110] Low SES may explain poorer adherence through effects on sleep patterns or other pathways such as reduced knowledge about OSA. Although self-efficacy is an important predictor of adherence among whites, no studies have evaluated whether differences in self-efficacy explain racial disparities in CPAP usage.

There is little data regarding CPAP adherence in Hispanics. One study of a public healthcare system in southern California found that Hispanics may be less accepting of CPAP home set up compared to whites.[111] Once set up with CPAP, data are conflicting on whether adherence rates in Hispanics are similar or worse than whites[103,109,112] A possible explanation for poorer CPAP adherence in Hispanics may be poor communication with healthcare providers due to language barriers. In one study, only about half of Hispanics reported that their sleep study results had been explained to them in their language.[113]

There is a paucity of data available to characterize adherence with CPAP treatment among Asian American individuals. Data available from other countries such as China suggests that adherence rates may be around 50-75%.[114,115] Barriers to usage appear to be similar to those encountered by other racial groups such as difficulty acclimatizing to CPAP after titration, lack of perceived benefit, or need for treatment.[114] CPAP acceptance rate was low, with a large minority (one-third) declining to obtain CPAP set up; this may reflect lack of insurance coverage for equipment and high out of pocket expenses for the patient. Among Chinese and Taiwanese individuals, as in whites, OSA severity is a predictor of adherence.[115] In an analysis comparing participants in a multi-center clinical trial, individuals in China had comparable CPAP adherence rates to participants in Australia and New Zealand.[116] Within New Zealand, the indigenous Māori have lower CPAP adherence than whites and this disparity appeared to be driven particularly by differences in education and socioeconomic status. [117,118]

Conclusion

Obstructive sleep apnea is a common disease across races. However research has demonstrated racial differences in OSA prevalence. Risk factors, including craniofacial structure and obesity, may explain some of the differences in prevalence, particularly among Asians and African Americans. Report of symptoms, including sleepiness and snoring, also clearly varies by race. This may reflect cultural differences in tolerance and report of symptoms. Finally, diagnosis and treatment disparities are known to exist, although the reasons behind these are not well characterized. Areas in need of further research include a more thorough evaluation of the heterogeneity that exists within races, validation of screening instruments in non-white groups, a better understanding of potential differences in susceptibility to cardiovascular consequences of OSA, and the development of strategies to reduce the barriers to diagnosis and optimal treatment in minorities. Given the unequal burden of OSA and related co-morbidities among racial minorities, improvements in the

recognition and treatment of OSA would have far reaching benefits to population health and so should be a priority for the sleep medicine field.

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Highlights

- OSA prevalence is higher among African Americans, Native Americans, and Hispanics.
- Obesity, craniofacial structure, and genetics contribute to prevalence differences.
- Presenting symptoms and consequences of OSA vary by racial background.
- OSA treatment outcomes vary by race, being worst among African Americans.

Table 1

Soft tissue and skeletal risk features associated with OSA, with racial/ethnic differences noted.

Craniofacial Risk Factors for OSA
Soft Tissue
Tongue
• Enlarged in African Americans with OSA
Skeletal
Brachycephaly
• Predictor of OSA among Caucasians, not among African Americans
Midface length
• Shorter in Asians with OSA
Cranial base
• Shorter and extended angle in Asians with OSA
Maxilla
• Shorter length predicts OSA in Asians
• Retro-position may be associated with OSA in Hispanics and Asians
Mandible
• Length and position predict OSA in Asians
Hyoid
• Inferiorly positioned in Asians and Caucasians with OSA
Anatomical imbalance (tongue area relative to intermaxillary length)
• Large tongue area relative to intermaxillary length associated with OSA in Caucasians but not African Americans or Asians