people of South Asian descent, including those from India, Pakistan, Bangladesh, Sri Lanka, and Nepal, make up a quarter of the world’s population. For over a century, the South Asian diaspora has also led to a substantial number of South Asians living in Western countries. South Asians represent 7% and 3% of the population in England and Canada, respectively.

In the US, South Asians have been one of the fastest growing population segments and now represent the second largest Asian American group. South Asians throughout the world have been shown to have a disproportionate risk of cardiovascular disease (CVD) and type 2 diabetes mellitus (DM). There are clearly lifestyle factors contributing to this increased cardiometabolic risk; however, surprisingly little is known about sleep in South Asians and whether it may also be contributing to the increased risk. This is of particular interest given that obstructive sleep apnea (OSA) and other causes of poor sleep are increasingly linked to CVD and DM.

Epidemiologic studies from India suggest the prevalence of OSA is similar to US whites, despite a lower level of obesity. However, few studies have directly compared OSA risk in South Asians to other groups. Questionnaire-based surveys suggest people of South Asian heritage are at higher risk than those of European or Chinese heritage, but the relative performance of these questionnaires across races has never been assessed.

In this issue of the Journal, Leong et al. report on differences in the prevalence of OSA in severely obese South Asians and whites in a UK bariatric clinic where all patients underwent limited-channel home sleep testing. South Asians had a higher prevalence of OSA (43%) than whites (22%) and a substantially greater apnea hypopnea index. Differences remained significant even after adjustment for potential confounders such as body mass index (BMI). These data confirm that severely obese South Asians are at elevated risk for OSA compared to whites. The authors are to be commended for performing the first cross-ethnic assessment including South Asians utilizing an objective assessment of OSA severity. While the possibility of differential biases for referral to a bariatric program needs to be considered, these findings support the need for additional studies in more representative South Asian populations in order to understand the reasons for the difference in OSA risk and to examine OSA as a potential driver of CVD and DM in South Asians.

It is well known that South Asians manifest greater visceral adiposity, insulin resistance, and DM at a lower BMI compared to whites. Because South Asians commonly develop adverse metabolic profiles at a lower BMI, the International Diabetes Federation has recommended race-specific thresholds to define central obesity. A similar approach might be needed to define the thresholds for OSA risk in South Asians and other high-risk racial/ethnic groups. In the meantime, clinicians need to recognize that the use of “one size fits all” BMI thresholds in OSA screening instruments may not accurately identify South Asians who are at increased risk for OSA.

In terms of obesity independent risk factors, a small cross-ethnic study identified bony craniofacial predictors of OSA in some ethnic groups but not Asian Indians. There is a notable paucity of work evaluating ethnic differences in physiological risk factors for OSA such as arousal threshold or loop gain. Further work is needed to identify both anatomic and non-anatomic risk factors for OSA in South Asians and other non-white groups.

An even more interesting question is whether the consequences of OSA may differ by race. The Sleep Heart Health Study suggested common OSA symptoms such as snoring, witnessed apnea, and sleepiness differ substantially by race/ethnicity. However, the difference in sleepiness between African Americans and whites was largely explained by differing responses to two questions in the Epworth Sleepiness Scale, suggesting that differences in the performance characteristics of this instrument may underlie racial/ethnic differences. No studies have used objective assessments to assess sleepiness across diverse OSA populations. Nevertheless, reports of genetic polymorphisms that modify the impact of OSA provide a mechanism for racial heterogeneity in OSA consequences. A prior study found South Asians with OSA had higher hemoglobin A1c levels and lower high density lipoprotein concentrations than whites with OSA. Interestingly, Leong et al found the prevalence of diabetes was twice as high in South Asians compared to whites in their cohort. These cross-sectional data provide important observations that need further investigation. Specifically, longitudinal multiethnic cohorts that include South Asians are needed to understand the causal pathways and strength of the association between OSA and cardiometabolic outcomes, and to answer questions about whether these pathways and consequences differ by race/ethnicity.
A final issue that should be considered is to what extent does race/ethnicity influence treatment options, adherence, and outcomes? There is increasing evidence that African Americans have disproportionate failure rates following adenotonsillectomy for OSA in children and reduced rates of CPAP adherence for OSA in adults.\(^{22,23}\) Other vulnerable groups have also been found to have lower CPAP adherence.\(^ {24}\) Little information is available regarding the success of specific OSA treatment options in South Asians. While the heterogeneous results of CPAP studies on metabolic syndrome may be due to differences in study design,\(^ {25-27}\) another possible explanation is that the metabolic impact of OSA therapy in South Asians may differ from whites.

Reducing the cardiometabolic risk of South Asians is a vital public health priority. The data from Leong et al. support the notion that sleep disorders may be contributing to this increased risk. Further well-designed studies are needed to determine if and how sleep disorders such as OSA are contributing to the elevated CVD and DM risk observed in South Asian populations and the potential of sleep interventions to minimize this disparity.

**REFERENCES**


**SUBMISSION & CORRESPONDENCE INFORMATION**

Submitted for publication July, 2013
Accepted for publication July, 2013
Address correspondence to: Sanjay R. Patel, M.D., Brigham and Women’s Hospital, Division of Sleep Medicine, 221 Longwood Avenue, Room 225C, Boston, MA 02115; Tel: (857) 307-0347; Fax: (617) 278-6946; E-mail: spatel@partners.org

**DISCLOSURE STATEMENT**

The authors have indicated no financial conflicts of interest.