http://dx.doi.org/10.5665/sleep.2518

# The Effects of Body Fat Distribution on Obstructive Sleep Apnea: Are Older and Younger Adults the Same?

Commentary on Degache et al. Relation of central fat mass to obstructive sleep apnea in the elderly. SLEEP 2013;36:501-507.

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There are known variations in the distribution of adipose tissue between individuals. Two main subtypes are peripheral obesity and central obesity, also colloquially known as pear- vs. apple-body types, respectively. Central and peripheral patterns are most commonly implicated as having strong associations with respect to health of the non-elderly, and representative metrics such as waist-hip ratio generally exhibit strong associations with all-cause mortality,[1-4] cardiovascular mortality,[5-7] mortality in type 2 diabetes,[8] incident cardiovascular disease (CVD),[3,4,9,10] incident diabetes,[10] and incident metabolic syndrome.[10] In addition, central obesity bears a clear relationship to the severity of OSA,[11,12] which may be particularly strong in men[13,14] and to incident OSA.[15] In some studies, central obesity demonstrates a greater association than the well-known correlation with neck circumference (NC).[16,17]

Although it is easy to intuit a pathogenetic linkage between NC and OSA (increased NC implies more adipose tissue adjacent to the upper airway, reducing upper airway caliber and predisposing to OSA), a mechanism by which central obesity could increase the incidence of OSA is less obvious. Postulated mechanisms have included the following: (1) Reduced lung volumes due to central obesity, leading to reduced "tracheal tug" and therefore diminished upper airway caliber.[18,19] (2) The involvement of humoral factors associated with central obesity. Particular interest has focused on leptin and the leptin-resistant state, since this molecule augments respiratory drive.[20] A direct effect of leptin deficiency on the passive mechanical characteristics and active neuromuscular function of the upper airway has recently been demonstrated in mice.[21] Moreover, there are a host of other cytokines and humoral agents whose levels are known to vary with the presence of central obesity, and could potentially affect control of breathing and upper airway function.[22-24] (3) Finally, an effect mediated by the increased risk for CVD associated with central obesity, which can lead to congestive heart failure (CHF), should not be ruled out. CHF could induce OSA by producing rostral fluid shifts during sleep that reduce upper airway caliber.[25,26]

In this issue of *SLEEP*, Degache and colleagues add to our knowledge of the association between central obesity and OSA with an investigation involving elderly subjects, a population little studied in this regard.[27] It has long been known that the elderly suffer from a significantly higher prevalence of OSA, which has been postulated to be the result of obesity and/or decreased muscle tone and/or impaired pharyngeal sensory input, all consequent to aging.[28] Central obesity specifically has not often been proposed as another important pathogenetic mechanism in this population, and clarifying this issue is in itself an important goal. A second goal stated by the authors was to demonstrate whether dual-energy X-ray absorptiometry (DEXA) assessment of central obesity might help diagnose OSA in elderly individuals. That, I believe, was a less achievable goal, and the data from this investigation seems to support that opinion.

Degache and colleagues[27] enrolled 758 subjects (450 women) randomly selected from the electoral list of a city in France. Subjects received a general clinical assessment and administration of the Epworth Sleepiness Scale (ESS), and were excluded if found to have a history of myocardial infarction, heart failure, cerebrovascular accident, or previous diagnosis or treatment for OSA. Anthropometric data collected included body mass index (BMI), NC, and waist circumference (WC), Evaluation for OSA consisted of unattended cardiopulmonary monitoring (type 3 methodology); and regional (arms, legs, trunk and head) body fat utilizing DEXA. Central fat mass (CFM) was defined as the sum of that measurement for the trunk and head. An apnea-hypopnea index (AHI) > 15/hour, with ≥ 85% of events being classified as obstructive, was used to define the presence of OSA. OSA severity was classified as mild for AHI between 15/hour and 30/hour and moderate to severe for AHI > 30/hour.

The authors report on a subject group of modestly older individuals averaging just over 65 years of age. Moderate to severe OSA was more prevalent in men; neither gender exhibited significant degrees of excessive daytime sleepiness by ESS. Modest, but significant increases in odds ratios were found after fully adjusted logistic regression modeling for predicting AHI > 15/hour from CFM and BMI. When mild vs. moderate to severe OSA were analyzed separately, only moderate to severe OSA maintained a modest relationship to CFM, while only mild OSA maintained a modest association with BMI. The authors concluded that CFM measured by DEXA predicted an "enhanced risk" of more severe OSA in their subjects that was superior to BMI.[27]

As Degache et al. correctly acknowledge, DEXA did not prove to be a particularly useful screening tool for identifying elderly individuals with OSA. However, their data do lead to some interesting conclusions with respect to the involvement of

**Submitted for publication February, 2013**
**Accepted for publication February, 2013**
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CFM in the pathogenesis of OSA in this population: namely, that given the modest degree of association, we should probably look elsewhere for factors to explain the high prevalence of OSA, such as those enumerated above. Indices of central obesity have proven to exhibit a more robust relationship to OSA in younger adult populations.[29-32] In those studies reporting fully adjusted odds ratios for measures of central obesity, values were found to be roughly twice as high as those reported by Degache et al.[29,30] Moreover, the findings of Degache et al.[27] tend to reinforce other data concerning the relative unimportance of CFM in the elderly. Multiple studies have appeared demonstrating that all-cause mortality[33-36] bears little relationship to central obesity in both genders, and (surprisingly) even tends to improve with increasing central obesity in men.[33-37] When examined specifically, cardiovascular mortality also does not seem to be associated with central adiposity in both genders.[35] Paradoxically, a recent systematic review of data from 25 morbidity studies found that risk for many conditions, including type 2 diabetes, hypertension, cardiovascular disease, and some cancers increases with central obesity, while analysis of 17 mortality studies failed to consistently demonstrate a relationship between central obesity and mortality.[38] Interestingly, the authors concluded that the lowest mortality in the elderly may be achieved when such individuals are overweight or mildly obese.[38]

In conclusion, it appears that the distribution of adipose tissue in older adults may not have the same implications, and be involved in all of the same pathogenetic mechanisms for OSA, as in a younger population. This dichotomy may well help to dissect the various etiologic mechanisms involved in OSA pathogenesis in the future.

## CITATION

Brown LK. The effects of body fat distribution on obstructive sleep apnea: are older and younger adults the same? *SLEEP* 2013;36(4):457-459.

## DISCLOSURE STATEMENT

The author has indicated no financial conflicts of interest.

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