Methods: The Men Androgen Inflammation Lifestyle Environment (MAILES) Study is a population-based biomedical cohort of men aged ≥35y in Adelaide, South Australia. In 2008–10, men underwent biomedical assessment (including blood pressure, anthropometry, fasting glucose) and in 2010–12, 837 men underwent home-based full polysomnography (Emblett X100). ArTH was estimated using a 2014 published equation based on clinical and PSG data as follows: -65.391 + 0.6366*age + 3.692*1 (male sex) - 0.0314*BMI - 0.108 *apnea hypopnea index + 0.533 *oxygen saturation nadir + 0.0906 * hypopnea fraction. Pulse pressure was defined as systolic blood pressure (SBP, mmHg) - diastolic blood pressure (DBP, mmHg).

Results: OSA (AHI≥10/hr) was present in 52.9% (n=443). The mean (SD) ArTH was -17.8 cmH2O (6.5), median (IQR) was -16.2 (-19.1, -14.3) cmH2O, n=423. ArTH was significantly correlated with DBP (r = -0.143, p<0.05) and pulse pressure (r = -0.126, p<0.05). No significant correlations were evident with SBP (r = 0.024, p=0.63) or fasting glucose (r = 0.025, p=0.60). In multiple regression models adjusted for age, BMI, smoking and alcohol use, the significant associations with DBP [unstandardized B (SE): -0.15 (0.07), p=0.037] and pulse pressure persisted [0.24 (0.09), p=0.008].

Conclusion: The ArTH phenotype may contribute to cardiovascular risk in OSA via different mechanisms. Large negative intrathoracic pressure swings may increase blood pressure in patients with a high ArTH whereas repetitive surges in sympathetic activity may contribute to increased pulse pressure in those with a low ArTH.

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0446 INCREASED SYMPATHETIC ACTIVITY IS ASSOCIATED WITH HYPERTENSION IN SLEEP APNEA: BMI EFFECT

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Introduction: Hypertension is highly prevalent in obstructive sleep apnea (OSA). OSA patients are known to have increased sympathetic activity that induced by intermittent hypoxia and sleep fragmentation during sleep. It has been suggested that excessive sympathetic activity is one of the underlying mechanisms that associated with hypertension in OSA. However, previous findings of the association between sympathetic activity and hypertension in OSA were inconsistent. The aim of this study was to examine the association between sympathetic activity and hypertension in OSA.

Methods: We studied 114 OSA male patients (43.81 ± 9.46y) who underwent one night polysomnography. Twenty-four-hour urinary catecholamines, including dopamine, epinephrine and noradrenaline levels, were used to measure sympathetic activity. Hypertension was defined based either on blood pressure measures or on diagnosis treatment.

Results: Pearson’s correlation analyses showed that 24-hour noradrenaline levels were significantly correlated with systolic and diastolic blood pressure (SBP, r=0.18, p=0.05; DBP, r=0.24, p=0.001) and mean arterial pressure (MAP, r=0.22, p=0.02). Interestingly, this association was modified by overweight (BMI ≥ 25 kg/m2, all p-interaction=0.05). After adjusting for multiple potential confounders, increased noradrenaline levels were significantly associated with increased odds of hypertension in lean OSA patients (OR=1.08, 95%CI=1.01–1.16, P=0.03), whereas this association was lost in overweight or obese patients (OR=0.99, 95%CI=0.98–1.01, P=0.57). Similarly, increased noradrenaline levels were significantly associated with increased SBP (β=0.48, p=0.01), DBP (β=0.39, p=0.03) and MAP (β=0.44, p=0.01) in lean patients but not in overweight or obese patients (all p-values>0.1) while adjusting for multiple potential confounders. No association has been observed between dopamine, epinephrine levels and hypertension.

Conclusion: Increased sympathetic activity is associated with increased odds of hypertension in lean but not in overweight or obese OSA male patients. This finding suggests that underlying mechanisms of hypertension may different between lean and overweight patients: lean patients may more relate to excessive sympathetic activity whereas overweight and obese patients may more relate to inflammation.

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0447 UPPER AIRWAY AND SURROUNDING SOFT-TISSUE CHANGES DURING SLEEP IN APNEICS AND CONTROLS

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Introduction: Previous studies indicated the upper airway narrows in the retropalatal (RP) region during sleep in normal subjects due to thickening of the lateral pharyngeal walls. We hypothesized that apneics would exhibit state-dependent upper airway narrowing in the RP and retroglossal (RG) regions and narrowing in apneics would be greater than controls.

Methods: Upper airway MRIs were obtained in 15 sleep-deprived OSA (AHI≥36.2 ± 19.9) during wake and sleep and analyzed for airway measures and soft-tissue movement. We evaluated whether there were significant changes during sleep and if changes differed between groups.

Results: Apneics were older (39.2 ± 12.2 vs. 26.4 ± 6.9 years, p=0.005) and more obese (33.0 ± 6.2 vs. 24.2 ± 3.5 kg/m², p=0.0003) than controls; 96.3% of subjects were male. In the RP region, controls showed reduced average cross-sectional airway area (CSA; -18.7%, p=0.003) and minimum CSA (-40.5%, p=0.0001), anteroposterior (AP; -25.0%, p=0.004), and lateral (-17.7%, p=0.037) dimensions. Apneics showed reduced average CSA (-31.3%, p<0.0001) and minimum CSA (-65.4%, p<0.0001), AP (-29.2%, p=0.0016), and lateral (-44.2%, p=0.0004) measures. Apneics had greater reductions in RP minimal CSA (p=0.0204) and lateral dimensions (p=0.0326) than controls. In the RG region, controls showed no significant changes with sleep. Apneics had reductions in minimal CSA (-45.2%, p=0.0011), lateral dimensions (-26.3%, p=0.0137), and trending changes in AP dimension (-18.9%, p=0.070). Reductions in RG minimal CSA (p=0.0096) and lateral dimension (p=0.0291) were greater in apneics than controls. The soft palate (p=0.0451), anterior-superior (p=0.0001), and posterior-inferior (p=0.0113) tongue quadrants exhibited greater posterior movement in apneics. Similar increases in RP lateral wall thickness (p=0.3332) were seen in controls (10.8%, p=0.0214) and apneics (21.8%, p=0.0549).

Conclusion: Controls and apneics showed significant reductions in RP airway caliber during sleep, while only apneics showed RG airway changes. Apneics had greater narrowing in both regions compared to controls. State-dependent reductions in AP and lateral airway dimensions are primarily due to posterior movement of the soft palate and tongue and thickening of lateral walls. These data provide important insights into OSA pathogenesis.

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