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Relation between Nose Scale and Sleep Disorder Breathing Among Spice Factory Workers in Semarang

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Abstract

Sleep-disorder breathing covers a broad spectrum of breathing-related sleep disorders. Nasal obstruction has been identified as a modifiable risk of sleep-disordered breathing and is a common complaint in sleep-disordered breathing patients. The nose scale is a simple standard instrument that can estimate those at risk of developing sleep-disordered breathing. To determine the relationship between nose scale and sleep disorder breathing in spice factory workers in Semarang. Analytical observational research with a cross-sectional design involving 530 spice factory workers in Semarang. The independent variable of the study was the risk of developing sleep-disordered breathing based on the body mass index value. The dependent variable of the research is the Nose Scale score. Analysis was by the Independent T-test, Mann- Whitney, Chi-square, and Fisher exact tests. Results are significant if p<0.05. Complaints of a blocked nose (6% vs 3%), blocked nose (8% vs 7%), difficulty breathing (4% vs 3%), difficulty sleeping due to nasal problems (5% vs 3%), and difficulty breathing air (6 % vs 4%) was more common in the high risk sleep disorder breathing group (BMI >25kg/m2) than in the low risk sleep disorder breathing group (BMI <25kg/m2), respectively. The high risk sleep disorder breathing group (BMI >25kg/m2) also reported a higher mean Nose Scale score than the low risk sleep disorder breathing group (BMI <25kg/m2), namely 5.81 vs 3.95. Individuals with high risk sleep disorder breathing (BMI >25kg/m2) have higher complaints of nasal problems than individuals with low risk sleep disorder breathing (BMI <25kg/m2).

Introduction

Sleep disorder breathing encompasses a broad spectrum of respiratory-related sleep disorders, including obstructive sleep apnea (OSA), central sleep apnea, sleep-related hypoventilation, and sleep-related hypoxemia. The prevalence of sleep-disorder breathing is estimated between 6.5%-9% in women and between 17%-31% in men. Several predisposing factors for the condition include obesity, genetic or congenital, nasal/pharyngeal abnormalities, and structural abnormalities of the upper airway (Foldvary-Schaefer *et al.*, 2017).

The diagnosis of sleep disorder breathing is established by conducting anamnesis regarding sleep patterns, physical examination, radiological examination, and special supporting examinations. Nasal obstruction has been identified as a modifiable risk of sleep-disordered breathing and is a common complaint in patients with sleep-disordered breathing. Several studies have reported that there is a subpopulation of patients with nasal obstruction who also experience undiagnosed sleep-disordered breathing (Sawa *et al.*, 2020).

A simple standard instrument, the nose scale, assesses knowledge of snoring status, and clinical examination, can predict individuals at risk for sleep-disordered breathing and refer patients for further management. This literature

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review discusses the relationship between NOSE scores and sleep-disordered breathing. This study aims to determine the relationship between nose scale and breathing sleep disorder in spice factory workers in Semarang.

Method

This analytical observational study used a cross-sectional design involving 530 spice factory workers in Semarang. The inclusion criteria were 1) age 18-55 years and 2) willingness to be a research subject. The exclusion criteria of the study were 1) a history of head trauma and 2) suffering from nasal polyps or tumors. The independent variable was the risk of sleep-disordered breathing based on body mass index values. The dependent variable of the study was the Nose Scale score. The analysis used the Independent T-test, Mann-Whitney, Chi-square, and Fisher exact tests. Significant results if $p < 0.05$.

Results

Evaluation of nose scale and sleep disorder breathing scores was conducted on 600 spice factory workers around Semarang City. As many as 550 workers met the inclusion criteria, and 20 subjects had exclusion criteria. So, a total of 530 workers became subjects in this study. Subjects were grouped regarding the risk of sleep disorder breathing by referring to BMI scores. As many as 258 workers were included in the low-risk sleep disorder breathing group (BMI <25kg/m^2), and 272 workers were in the high-risk sleep disorder breathing group (BMI>25kg/m^2).

Subjects in the low-risk sleep disorder breathing group (BMI <25kg/m^2) had an average age of 35.91 years with a standard deviation of 12.12 years, a median value of 30 years with the youngest age of 18 years, and the oldest age of 55 years. Meanwhile, the high-risk sleep disorder breathing group (BMI>25kg/m^2) had an average of 40.17 years with a standard deviation of 11.10 years, a median value of 42 years, with the youngest age of 19 years and the oldest age of 55 years. There was a significant difference in the distribution of age between the study groups ($p<0.001$), where older ages were found in the high-risk sleep disorder breathing group (BMI>25kg/m^2). Subjects in the low-risk sleep disorder breathing group (BMI <25kg/m^2), were 234 (91%) male and 24 (9%) female. In the high-risk sleep disorder breathing group (BMI >25kg/m^2), there were 255 male subjects (94%) and 17 female subjects (6%). There was no significant difference in gender distribution between study groups ($p=0.189$).

Regarding the history of hypertension, in the low-risk sleep disorder breathing group (BMI <25kg/m^2), there were 12 subjects (5%) who had a history of hypertension. In the high-

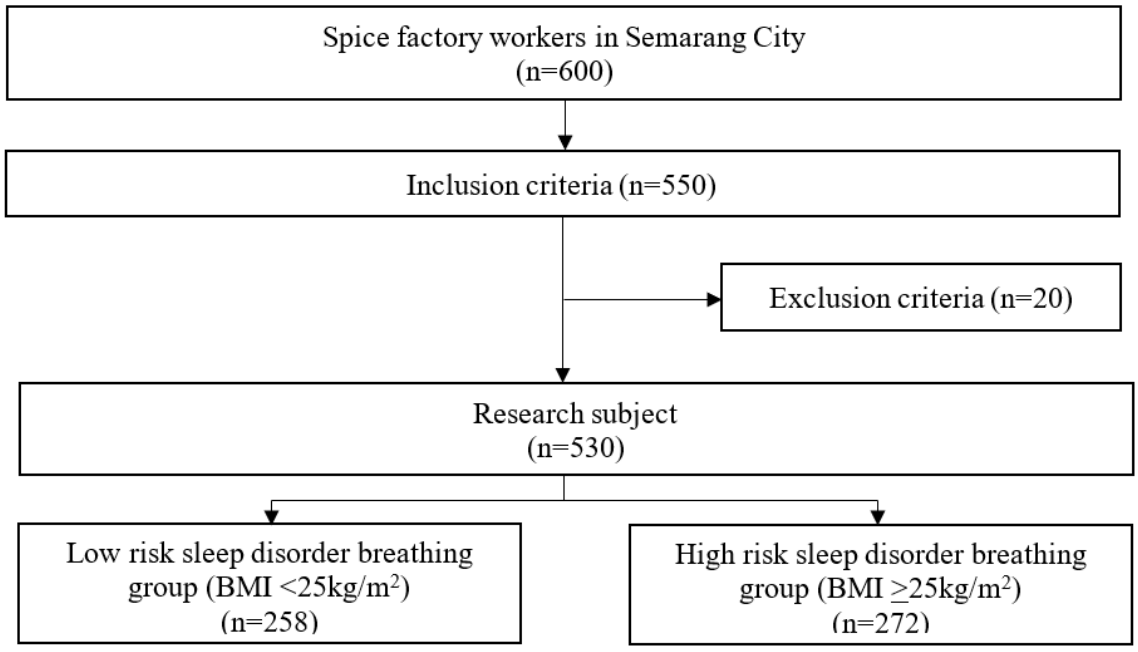

Table 1. Demographics of Research Subjects

Variables	*Low risk sleep disorder breathing* (BMI <25kg/m²)		*High risk sleep disorder breathing* (BMI >25kg/m²)		*p*
	n (%)	Mean ± SD; Median (min-max)	n (%)	Mean ± SD; Median (min-max)	
Age	-	35.91 ± 12.12; 30 (18-55)	-	40.17 ± 11.10; 42 (19-55)	**<0.001**¶
Gender Male Female	 234 (91) 24 (9)	-	 255 (94) 17 (6)	-	0.189£
Hypertension history Yes No	 12 (5) 246 (95)	-	 39 (14) 233 (86)	-	**<0.001**£
History of diabetes mellitus Yes No	 7 (3) 251 (97)	-	 16 (6) 256 (94)	-	0.073£

¶Mann Whitney U; £Chi-square; significance p<0.05

Table 2. Nose Scale Score and Risk of Sleep Disorder Breathing Based on Body Mass Index

Variables	*Low Risk Sleep Disorder Breathing* (BMI <25kg/m²)		*High Risk Sleep Disorder Breathing* (BMI >25kg/m²)		*p*
	n (%)	Mean ± SD; Median (min-max)	n (%)	Mean ± SD; Median (min-max)	
Weight	-	60.30 ± 7.06; 60.35 (37-81)	-	76.67 ± 10.30; 74.75 (55-125)	
Height	-	165.40 ± 6.74; 165 (144-184)	-	164.43 ± 6.35; 164.5 (143-188)	0.087‡
Body mass index	-	22.03 ± 2.13; 22.45 (15.5-24)	-	28.32 ± 3.23; 27.3 (25-44.8)	
Complaints of stuffy nose Yes No	 8 (3) 250 (97)	-	 17 (6) 255 (94)	-	0.087£
Complaints of blocked nose Yes No	 17 (7) 241 (93)	-	 21 (8) 251 (92)	-	0.614£
Complaints of difficulty breathing Yes No	 8 (3) 250 (97)	-	 11 (4) 261 (96)	-	0.559£

Variables	*Low Risk Sleep Disorder Breathing* (BMI <25kg/m²)		*High Risk Sleep Disorder Breathing* (BMI >25kg/m²)		*p*
	n (%)	Mean ± SD; Median (min-max)	n (%)	Mean ± SD; Median (min-max)	
Difficulty sleeping due to nasal problems		-		-	0.322£
Yes	8 (3)		13 (5)		
No	250 (97)		259 (95)		
Complaints of difficulty breathing air		-		-	0.214£
Yes	10 (4)		17 (6)		
No	248 (96)		255 (94)		
NOSE SCALE Score		3.95 ± 15.01; 0 (0-100)		5.81 ± 19.59; 0 (0-100)	0.486ᶠ
No blockage	233 (90)		242 (89)		
Mild obstruction	14 (5)		10 (4)		
Moderate blockage	4 (2)		8 (3)		
Heavy blockage	2 (1)		2 (1)		
Very heavy blockage	5 (2)		10 (4)		

¶Mann Whitney U;‡Independent T-test; £Chi-square;ᶠFischer exact; significance p<0.05

risk sleep disorder breathing group (BMI>25kg/m²), there were 39 subjects (14%) who had a history of hypertension. There was a significant difference in the distribution of hypertension history between study groups (p<0.001). Where the incidence of hypertension was more common in the high-risk sleep disorder breathing group (BMI>25kg/m²). Regarding the history of diabetes mellitus, in the low-risk sleep disorder breathing group (BMI <25kg/m²), there were 7 subjects (3%) who had a history of diabetes mellitus. In the high-risk sleep disorder breathing group (BMI>25kg/m²), there were 16 subjects (6%) who had a history of diabetes mellitus. There was no significant difference in the distribution of diabetes mellitus history between study groups (p=0.073).

Regarding body weight, the low-risk sleep disorder breathing group (BMI <25kg/m²) had an average of 60.3 kg with a standard deviation of 7.06 kg, a median value of 60.35 kg with the smallest body weight of 37 kg, and the largest body weight of 81 kg. The high-risk sleep disorder breathing group (BMI>25kg/m²) had an average of 76.67 kg with a standard deviation of 10.3 kg, a median value of 74.75 kg with the lowest body weight of 55 kg, and the highest body weight of 125 kg. There was a significant difference in the distribution of body weight between study groups (p<0.001), where greater body weight was found in the high-risk sleep disorder breathing group (BMI>25kg/m²).

Regarding height, the low-risk sleep disorder breathing group (BMI <25kg/m²) had an average of 165.4 cm with a standard deviation of 6.74 cm, a median value of 165 cm with the lowest height of 144 cm, and the highest height of 184 cm. The high-risk sleep disorder breathing group (BMI>25kg/m²) had an average of 164.43 cm with a standard deviation of 6.35 cm, a median value of 164.5 cm with the lowest height of 143 cm, and the highest height of 188 cm. There was no significant difference in the height distribution between the study groups (p=0.087).

Regarding body mass index, the low-risk sleep disorder breathing group (BMI <25kg/m²) had an average of 22.03 kg/m² with a standard deviation of 2.13 kg/m², a median value of 22.45 kg/m² with the lowest body mass index of 15.5 kg/m² and the highest body mass index of 24 kg/m². The high-risk sleep disorder breathing group (BMI>25kg/m²) had an average of 28.32 kg/m² with a standard

deviation of 3.23 kg/m^2, a median value of 27.3 kg/m^2 with the lowest body mass index of 25 kg/m^2 and the highest body mass index of 44.8 kg/m^2. There was a significant difference in the distribution of body mass index between study groups (p<0.001), where a higher body mass index was found in the high-risk sleep disorder breathing group (BMI>25kg/m^2).

Regarding stuffy noses, in the low-risk sleep disorder breathing group (BMI <25kg/m^2), there were 8 subjects (3%) complaining of stuffy noses. In the high-risk sleep disorder breathing group (BMI >25kg/m^2), 17 subjects (6%) complained of stuffy nose. There was no significant difference in the distribution of stuffy noses between study groups (p=0.087). Regarding nasal congestion, in the low-risk sleep disorder breathing group (BMI <25kg/m^2), there were 17 subjects (7%) complaining of nasal congestion. In the high-risk sleep disorder breathing group (BMI >25kg/m^2), 21 subjects (8%) complained of nasal congestion. There was no significant difference in the distribution of nasal congestion complaints between study groups (p=0.614).

Regarding difficulty breathing in the low-risk sleep disorder breathing group (BMI <25kg/m^2), 8 subjects (3%) complained of difficulty breathing. In the high-risk sleep disorder breathing group (BMI>25kg/m^2), 11 subjects (4%) complained of difficulty breathing. There was no significant difference in the distribution of complaints of difficulty breathing between research groups (p=0.557). Regarding difficulty sleeping, in the low-risk sleep disorder breathing group (BMI <25kg/m^2), 8 subjects (3%) complained of difficulty sleeping. In the high-risk sleep disorder breathing group (BMI >25kg/m^2), 13 subjects (5%) complained of difficulty sleeping. There was no significant difference in the distribution of complaints of difficulty sleeping due to nasal problems between study groups (p=0.322). Regarding difficulty in breathing, in the low-risk sleep disorder breathing group (BMI <25kg/m2), 10 subjects (4%) complained of difficulty in breathing. In the high-risk sleep disorder breathing group (BMI >25kg/m2), 17 subjects (6%) complained of difficulty in breathing. There was no significant difference in the distribution of complaints of difficulty in breathing between research groups (p=0.214).

Regarding the NOSE SCALE score, the low-risk sleep disorder breathing group (BMI <25kg/m^2) obtained an average score of 3.95 with a standard deviation of 15.01, a median value of 0 with the lowest score of 0, and the highest score of 100. There were 233 subjects (90%) who did not have nasal obstruction, 14 subjects (5%) had mild nasal obstruction, 4 subjects (2%) had moderate nasal obstruction, 2 subjects (1%) had severe nasal obstruction and 5 subjects (2%) had very severe nasal obstruction. In the high-risk sleep disorder breathing group (BMI >25kg/m^2), the average score was 5.81 with a standard deviation of 19.59, a median value of 0 with the lowest score of 0 and the highest score of 100. There were 242 subjects (89%) who had no nasal obstruction, 10 subjects (4%) had mild nasal obstruction, 8 subjects (3%) had moderate nasal obstruction, 2 subjects (1%) had severe nasal obstruction and 10 subjects (4%) had very severe nasal obstruction. There was no significant difference in the NOSE SCALE score between study groups (p=0.486).

Significantly older age was found in the high-risk sleep disorder breathing group (BMI >25kg/m^2) with an average of 40 years compared to the low-risk sleep disorder breathing group (BMI <25kg/m^2) with an average of 35 years. A history of hypertension was also significantly more common in the high-risk sleep disorder breathing group (BMI >25kg/m^2). Khabazkhoob M *et al*., who assessed the prevalence of overweight and obesity in the middle-aged population, found that individuals aged > 54 years tended to have a BMI > 25kg/m2 compared to individuals aged 40-44 years with a significant difference (p <0.001). 3 As age increases, hormonal changes, decreased physical activity, and particular cultural beliefs may contribute to the incidence of overweight and obesity. Punjabi N *et al*., who evaluated the prevalence of hypertension in patients with sleep-disordered breathing (SDB), found that the prevalence of hypertension and SDB (AHI3a ≥ 5 events/hour) was high, with estimates of 53.8% and 82.8%, respectively. In men without HIV, SDB was associated with the risk of hypertension (OR: 2.93; 95% CI: 1.46 to 5.86) (Feng *et al*., 2023; Punjabi *et al*., 2023).

OSA and resistant hypertension share some very similar risk factors, such as obesity and metabolic syndrome, and indeed, a high prevalence of OSA has been reported in patients with resistant hypertension, estimated to range from 64% to 83%. OSA is now recognized as the most common cause of secondary hypertension. An association between OSA and hypertension has been demonstrated in wide cross-sectional studies as well as longitudinal ones in the general population. Although some of the association is explained by established risk factors, such as obesity, there is substantial data to suggest that the role of OSA in resistant hypertension is independent of other confounding factors. In the Wisconsin Sleep Cohort study, a significant dose-response relationship was observed between baseline sleep-disordered breathing and hypertension 4 years later, independent of confounding factors. Subjects with an AHI of 0.1 to 4.9 events per hour at baseline had a 1.4-fold increased odds of developing hypertension, whereas subjects with an AHI of 5.0 to 14.9 events per hour showed an almost 3-fold increased odds of hypertension compared with subjects with 0 events per hour. Similarly, another large prospective cohort study with a median follow-up period of 12.2 years showed that the presence of OSA was associated with an increased risk of incident hypertension. These data provide vital evidence that OSA is an independent risk factor for hypertension and that the severity of hypertension increases with increasing severity of OSA (Wang, 2014).

Several mechanisms have been proposed to play a significant role in the development of resistant hypertension in OSA. OSA causes chronic intermittent hypoxia, which induces an imbalance of parasympathetic and sympathetic nerves, resulting in sympathetic activation, activation of the renin-angiotensin-aldosterone system, inflammation, and increased oxidative stress, increased endothelin release with vasoconstriction and endothelial dysfunction (Parati *et al.,* 2013). Patients with OSA commonly have a narrow pharynx due to neck fat deposits, micrognathia, tonsillar hypertrophy, or pharyngeal edema, which reduces the pharyngeal lumen. At the onset of sleep, the pharyngeal dilator muscles relax, causing partial or complete collapse of the pharynx, leading to obstructive hypopneas and apneas. Repeated cessation of airflow during sleep leads to repeated cycles of hypoxemia and hypercapnia, which stimulate the cardiac parasympathetic and sympathetic nervous systems. The final response depends on the balance between the parasympathetic and sympathetic nervous systems and airflow. Sympathetic activation is one of the key mechanisms involved in the genesis of resistant hypertension in OSA. Recurrent apnea episodes increase sympathetic activity during sleep and wakefulness through activation of the chemo-reflex, which plays a key role in regulating ventilation and circulatory responses to changes in arterial oxygen saturation and carbon dioxide. An activated renin-angiotensin-aldosterone system is another vital pathomechanism linking OSA to resistant hypertension. Previous studies have shown that OSA subjects have increased plasma angiotensin II and aldosterone levels compared with controls. CPAP therapy significantly reduced blood pressure, as well as plasma renin and angiotensin II levels. The decrease in blood pressure correlated with lower plasma renin and angiotensin II levels. Treatment with angiotensin II receptor blockers abolished the increase in blood pressure induced by intermittent hypoxia. These data suggest that OSA mediates hypertension at least in part through increased angiotensin II production (Wang, 2014).

Intermittent hypoxia and post-apneic reperfusion lead to the formation of free oxygen radicals, increase oxidative stress, and activate the inflammatory cascade. Reactive oxygen species and inflammation reduce nitric oxide levels and impair endothelium-dependent vasodilation, which may contribute to increased blood pressure independently of sympathetic activation. Studies have shown impaired endothelium-dependent vasodilation and decreased nitric oxide levels in OSA subjects, which improved after CPAP therapy.[6] Complaints of nasal obstruction (6% vs 3%), nasal congestion (8% vs 7%), difficulty breathing (4% vs 3%), difficulty sleeping due to nasal problems (5% vs 3%), and difficulty breathing air (6% vs 4%) were more common in the high-risk sleep disorder breathing group

(BMI >25kg/m^2) compared to the low-risk sleep disorder breathing group (BMI <25kg/m^2), respectively. The high-risk sleep disorder breathing group (BMI >25kg/m^2) also reported a higher mean NOSE SCALE score than the low-risk sleep disorder breathing group (BMI <25kg/m^2), which was 5.81 vs 3.95. It indicates that individuals with high-risk sleep disorder breathing (BMI >25kg/m^2) have higher nasal problems than individuals with low-risk sleep disorder breathing (BMI <25kg/m^2).

Magliulo G *et al*., who evaluated the olfactory function in patients with OSA found that olfactory dysfunction occurred in 22 (36.6%) patients in the study group, hyposmia occurred in 19 patients (86.4%) and anosmia in 3 patients (13.6%). The average TDI score in the study group was 30. A strong correlation was found between olfactory dysfunction and the severity of OSA as measured using the AHI. Patients with OSA appear to have a high incidence of olfactory dysfunction. The degree of olfactory dysfunction seemed to be related to the severity of the disease. However, other factors, such as nasal obstruction and reduced mucociliary clearance, play a role in the etiology of this condition (Magliulo *et al.,*2018). Another study conducted by Shin DH *et al*., who assessed the effect of sleep disorder breathing on olfactory function based on the Apnea-Hypopnea Index (AHI) scores obtained similar results that AHI was related to the odor threshold score (odor), and the average SaO2 was related to the odor discrimination score (odor). Hypoxia and low nasal airflow caused by OSAS also affect olfactory function. In addition, low mean oxygen is a major risk factor in determining olfactory function (Shin *et al*., 2017). Kaya KS *et al*., found an increase in the olfactory threshold in OSA patients who received PAP treatment thought to be able to improve olfactory dysfunction (Kaya *et al*., 2020). Siegel JK *et al*., who evaluated sleep disorder breathing against the incidence of odor identification disorders in the elderly, found that 29% of elderly adults in the US reported symptoms of sleep disorder breathing (apnea or snoring at night). Based on this number, only 32% were diagnosed with sleep apnea. Older adults with SDB (those who reported symptoms or had been diagnosed with sleep apnea) were significantly more likely to have impaired odor identification (odds ratio 2.13, 95% confidence interval 1.19–3.83, p = 0.012) in analyses that accounted for age, sex, race/ethnicity, education, cognition, comorbidities (including depression), and body mass index (Siegel *et al*., 2021).

Assessment of activated brain areas in OSA patients during the olfactory test was conducted by Invitto S *et al*., who found that OSA patients showed faster N1 latency and greater amplitude. The same trend was found in the Late Positive Component (LPC), where OSA showed decreased latency and increased amplitude during stimulation using rose flowers, in the right inferior frontal cortex and faster latency in the left centroparietal cortex. The OERP results showed a decrease in the endogenous component. These results may be a consequence of the exogenous perception difficulties highlighted in the N1 component. Increased arousal could also be associated with the respiratory activity during the olfactory task (Invitto *et al*., 2019).

In OSA patients, olfactory dysfunction is associated with various mechanisms. Several studies have reported cognitive deficits in OSA patients, including memory, attention, and sensory and olfactory functions. Decreased (poor) neurocognitive performance found to be associated with olfactory dysfunction through impaired olfactory discrimination and decreased threshold. On the other hand, decreased nasal airflow, intermittent hypoxia, chronic irritation, and upper airway mucosal damage may be additional causes associated with the mechanisms responsible for olfactory dysfunction in OSA (Kaya *et al*., 2020).

The mechanisms that affect olfactory function in OSA may be related to many factors, including oxygen saturation, disease duration, and degree of upper airway inflammation. Several studies have reported that airway inflammation in OSA patients experiencing intermittent nocturnal hypoxia increases pro-inflammatory markers such as interleukin 8 and TNF-α. Positive airway pressure (PAP) administration may contribute to olfactory function by reducing mucosal inflammation in OSA. The mechanisms that affect olfactory function in OSA may be related to many factors,

including oxygen saturation, disease duration, and degree of upper airway inflammation. Several studies have reported that airway inflammation in OSA patients experiencing intermittent nocturnal hypoxia increases pro-inflammatory markers such as interleukin 8 and TNF-α. Positive airway pressure (PAP) administration may contribute to olfactory function by reducing mucosal inflammation in OSA (Wang *et al*., 2015).

Clinical studies have shown that PAP treatment contributes to respiratory and cognitive performance in OSA (Xu *et al.,* 2017). PAP administration can improve olfactory dysfunction in OSA patients through various interactions (Boerner *et al.,* 2017). The cholinergic neurotransmitter system is involved in many cognitive functions and is sensitive to cerebral hypoxemia. PAP, by normalizing neurotransmitter synthesis can improve cognitive dysfunction in OSA patients. Increased oxygen saturation positively affect neurocognitive functions, including olfaction (Salihoğlu *et al*., 2014). Inflammation of the nasal mucosa with altered nasal mucociliary clearance is a common cause of olfactory neuroepithelial dysfunction. Typically, poor mucociliary clearance prevents the interaction between odorant molecules contained in inspired air and the olfactory epithelium. Most patients with OSAS are known to have decreased mucosal clearance even in the absence of overt sinonasal inflammatory disease. A recent clinical study of nasal mucociliary clearance in patients with obstructive sleep apnea syndrome showed that the nasal mucociliary system showed significant impairment in patients with severe OSAS (Deniz *et al*., 2014; Cai *et al*., 2020).

The underlying mechanism of the correlation between OSAS and olfactory function is thought to be that intermittent hypoxia and sleep fragmentation can cause neuronal loss in the hippocampus and prefrontal cortex, areas closely related to memory and executive function (Salihoğlu *et al.,* 2014; Peng *et al*., 2018). The impact of cognitive decline on smell can lead to decreased discrimination and identification functions. It is known that OSA is characterized by cessation of breathing (apnea) or reduced airflow (hypopnea) during sleep. Increased AHI is associated with decreased nasal airflow. Changes in nasal airflow affect olfactory function, especially the odor threshold. Therefore, the negative correlation between AHI and odor threshold is thought to indicate changes in nasal airflow in OSA patients (Fu *et al*., 2015).

Conclussions

Individuals with high-risk sleep disorder breathing (BMI >25kg/m^2) have higher nasal problems (complaints of nasal obstruction, nasal congestion, difficulty breathing, difficulty sleeping due to nasal problems, and difficulty breathing air) compared to individuals with low-risk sleep disorder breathing (BMI <25kg/m^2).

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References

Boerner, B., Tini, G.M., Fachinger, P., Graber, S.M., & Irani, S., 2017. Significant Improvement of Olfactory Performance in Sleep Apnea Patients After Three Months of Nasal CPAP Therapy – Observational Study and Randomized Trial. *PLoS One*, 12(2), pp.e0171087.

Cai, Y., Goldberg, A.N., & Chang, J.L., 2020. The Nose and Nasal Breathing in Sleep Apnea. *Otolaryngologic Clinics of North America*, *53*(3), pp.385-395.

Deniz, M., Gultekin, E., Ciftci, Z., Alp, R., Ozdemir, D.N., Isik, A., & Demirel, O.B., 2014. Nasal Mucociliary Clearance in Obstructive Sleep Apnea Syndrome Patients. *Am J Rhinol Allergy*, 28(5), pp.e178–80.

Feng, X., Zhu, J., Hua, Z., Shi, Q., Zhou, J., & Luo, P., 2023. The Prevalence and Determinant of Overweight and Obesity Among Residents Aged 40–69 Years in High-Risk Regions for Upper Gastrointestinal Cancer in Southeast China. *Sci Rep*, 13(1), pp.8172.

Foldvary-Schaefer, N.R., & Waters, T.E., 2017. Sleep-Disordered Breathing. *Continuum Lifelong Learning in* Neurology, 23(4), pp.1093–116.

Fu, D., Pinto, J.M., Wang, L., Chen, G., Zhan, X., &

Wei, Y., 2015. The Effect of Nasal Structure on Olfactory Function in Patients with OSA. *European Archives of Oto-Rhino-*Laryngology, 272(2), pp.357–62.

Invitto, S., Calcagnì, A., Piraino, G., Ciccarese, V., Balconi, M., De Tommaso, M., & Toraldo, D.M., 2019. Obstructive Sleep Apnea Syndrome and Olfactory Perception: An OERP Study. *Respir Physiol Neurobiol*, 259, pp.37–44.

Kaya, K.S., Akpınar, M., Turk, B., Seyhun, N., Cankaya, M., & Coskun, B.U., 2020. Olfactory Function in Patients with Obstructive Sleep Apnea Using Positive Airway Pressure. *Ear Nose Throat J.*, 99(4), pp.239–44.

Khabazkhoob, M., Emamian, M.H., Hashemi, H., Shariati, M., & Fotouhi, A., 2017. Prevalence of Overweight and Obesity in the Middle-age Population: A Priority for the Health System. *Iran J Public Health*, 46(6), pp.827–34.

Magliulo, G., De Vincentiis, M., Iannella, G., Ciofalo, A., Pasquariello, B., Manno, A., Angeletti, D., & Polimeni, A., 2018. Olfactory Evaluation in Obstructive Sleep Apnoea Patients. Acta *Otorhinolaryngologica Italica*, 38(4), pp.338–45.

Parati, G., Lombardi, C., Hedner, J., Bonsignore, M.R., Grote, L., Tkacova, R., Lévy, P., Riha, R., Bassetti, C., Narkiewicz, K., Mancia, G., & McNicholas, W.T., 2013. Recommendations for the Management of Patients with Obstructive Sleep Apnoea and Hypertension. *European Respiratory Journal*, 41(3), pp.523–38.

Peng, M., Chen, R., Cheng, J., Li, J., Liu, W., & Hong, C., 2018. Application Value of the NoSAS Score for Screening Sleep-Disordered Breathing. *Journal of Thoracic Disease*, *10*(8), 4774.

Punjabi, N.M., Brown, T.T., Abreu, A.R., Aurora, R.N., Patel, S.R., Stosor, V., Cho, J.H., D'Souza, G., Wallace, D., Margolick, J.B., 2023. Sleep-Disordered Breathing and Prevalent Hypertension in Men with and Without HIV. *JAIDS Journal of Acquired Immune Deficiency Syndromes*, 94(4), pp.349–54.

Salihoğlu, M., Kendirli, M.T., Altundağ, A., Tekeli, H., Sağlam, M., Çayönü, M., Şenol, M.G., & Özdağ, F., 2014. The Effect of Obstructive Sleep Apnea on Olfactory Functions. *Laryngoscope*, 124(9), pp.2190–4.

Sawa, A., Suzuki, H., Niwa, H., Oguchi, S., Yagi, T., Iwata, Y., Makiyama, Y., Chow, C.M., & Komiyama, O., 2020. Assessment of Screening for Nasal Obstruction among Sleep Dentistry Outpatients with Obstructive Sleep Apnea. *Dent J (Basel)*, 8(4), pp.1–10.

Shin, D.H., Ahn, S.H., Yang, Y., Choi, S., Cho, J.H., Hong, S.C., & Kim, J.K., 2017. The Effect of Sleep Disordered Breathing on Olfactory Functions: Analysis by Apnea-Hypopnea Index. *Clin Exp Otorhinolaryngol*, 10(1), pp.71–6.

Siegel, J.K., Yuan, X., Wroblewski, K.E., McClintock, M.K., & Pinto, J.M., 2021. Sleep-Disordered Breathing Is Associated with Impaired Odor Identification in Older U.S. Adults. *The Journals of Gerontology*, 76(3), pp.528–33.

Wang, A.Y.M., 2014. Sleep-Disordered Breathing and Resistant Hypertension. *Semin Nephrol.*, 34(5), pp.520–31.

Wang, Y., Hu, K., Liu, K., Li, Z., Yang, J., Dong, Y., Nie, M., Chen, J., Ruan, Y., & Kang, J., 2015. Obstructive Sleep Apnea Exacerbates Airway Inflammation in Patients with Chronic Obstructive Pulmonary Disease. *Sleep Med.*, 16(9), pp.1123–30.

Xu, H., Wang, H., Guan, J., Yi, H., Qian, Y., Zou, J., Xia, Y., Fu, Y., Li, X., Jiao, X., Huang, H., Dong, P., Yu, Z., Yang, J., Xiang, M., Li, J., Chen, Y., Wang, P., Sun, Y., Li, Y., Zheng, X., Jia, W., & Yin, S., 2017. Effects of Continuous Positive Airway Pressure on Neurocognitive Architecture and Function in Patients with Obstructive Sleep Apnoea: Study Protocol for a Multicentre Randomised Controlled Trial. *BMJ Open*, 7(5), pp.e014932.