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Prevalence of Comorbididites in Obstructive Sleep Apnea Population Waiting for Bariatric Surgery

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Abstract

Background: According to the American Diabetes Association, 9.3% of the US population had diabetes in the year 2012, which corresponds to 29.1 million people. On the other hand, the overall prevalence of hypertension amongst individuals in the US, that were 18 years or older was 29.1% between 2011 and 2012. The intention of this study was to focus on comparing the prevalence of co-morbidities amongst the bariatric surgery patients who also have OSA.

Methods: In this retrospective study, we collected data from 749 obese outpatients, which had been scheduled for bariatric surgery. We then selected the data of the 258 patients that had been recommended to undergo the overnight sleep study. The patients were interviewed by a Board Certified Sleep Physician. The sleep studies were conducted at a 9-bed sleep facility. During the initial consultation, we focused on collecting data related to various comorbidities including hypertension, diabetes, hypothyroidism, asthma, and COPD. The OSA diagnosis and severity was based on Apnea Hypopnea Index (AHI), which is defined as the total events of apnea and hypopnea per hour of sleep. An AHI, which is less than or equal to 5 AHI/hour is considered normal, 6 to 15 AHI/hour as mild OSA, 16 to 30 AHI/hour is moderate and greater than 30 AHI/hour is considered severe OSA.

Results: Our research showed that the bariatric surgery patients that had been diagnosed with OSA on the basis of abnormal polysomnography results, were more prevalent to have Hypertension, diabetes mellitus, and hypothyroidism. Whereas, asthma was more common in the patients that did not have OSA (56%). HTN and DM were more prevalent in the category of patients that had moderate to Severe OSA. Whereas, asthma and hypothyroidism were less likely in the patients that had a higher AHI, as compared to those patients that had an AHI less than 15.

Conclusions: We concluded that the comorbidities like HTN, DM, and hypothyroidism were more prevalent in the diagnosed OSA patients (by polysomnography) that were undergoing bariatric surgery. While, asthma was more prevalent in the non-OSA population. Among the OSA population, HTN and DM were more prevalent in the patients that had moderate to severe OSA. Whereas, asthma and hypothyroidism were more common in the patients that had been diagnosed with mild OSA.

Keywords: OSA, Obesity, Bariatric surgery, Hypertension, Diabetes Mellitus, AHI

Introduction

According to the American Diabetes Association, 9.3% of the US population had diabetes in 2012, which corresponds to 29.1 million people. Among these, only 21 million were diagnosed whereas the remaining 8.1 million were left undiagnosed [1]. On the other hand, overall prevalence of hypertension in 18 years or older in US was 29.1% between 2011 and 2012.This prevalence was nearly equal among women (28.5%) and men (29.7%) [2]. Approximately 82% of the adults having hypertension in 2009-2010 were aware of their condition, and nearly 76% were on antihypertensive drugs [3]. In the reviewed data, the definition of hypertension is described as either having blood pressure ≥140/90, 130/85, 130/80 mmHg or the use of antihypertensive drugs [4,5]. The prevalence of hypertension tends to increase by decreasing the cut off points of diagnostic blood pressures [4,5].

Certain environmental factors like lack of exercise and physical activity due to sedentary life style and dependence on transportation may also promote obesity [6]. This goes hand in hand with the easy availability of inexpensive, palatable unhealthy

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foods; attractive marketing gimmicks and whole sale distribution [6]. Because of this increasing obesity, it is becoming a challenge for the health care providers to control the weight of people suffering from DM2 [7,8]. Due to a global increase in the prevalence of DM2, it is expected to hit 552 million in about 15 years from now [9].

OSA is particularly prevalent in middle aged obese man but now its existence is increasingly noted among women and even in lean subjects [10]. Despite the strong association of OSA with obesity and cardiovascular problems, it is recognized and managed as a 'localized respiratory track disorder' rather than a 'systemic problem [11]. In obese individuals, the increased burden and work load affects the respiratory system [12,13]. Other than pulmonary embolism, OSA, asthma, pneumonia and reactive airway disease, a number of respiratory disorders are related to obesity [14].

In the last ten years prior to the new millennium, studies showed that 4% of men and 2% of women between the ages of 45-65 were suffering from OSA [15]. However, more recent studies show that 10% of men and 3% of women between the ages of 30-49 have OSA [16]. Currently, with the rapid rise in the prevalence of obesity in our society, it is being considered as an epidemic. OSA has been shown to be even more prevalent in obese individuals, with studies showing figures as high as 60 to 70% amongst the obese population [17].

OSA has been characterized as the periodic hypopneas and apneas during sleep, due to the obstruction in the upper respiratory tract [18]. The consistent deoxygenation of hemoglobin results in increased respiratory efforts [12,13], interrupted sleep and frequent night time awakenings [19]. This drastically affects daily living, as it results in excessive daytime tiredness and sleepiness [20]. OSA patients have an increased prevalence of DM2 [21], hypertension [22] and cardiovascular risks, like stroke [23]. Most of these OSA patients go untreated even though the prevalence of OSA is very high [20].

For the management of OSA, the most recently recommended treatment options that are offered to patients include traditional weight loss methods, customized oral apparatus and positive airway pressure machines [24,25]. Weight loss has shown to dramatically decrease the symptoms of OSA [26]. The data also declares that weight loss impressively decreases the severity and risk of other obesity related comorbidities [27,28]. Patients that don't succeed in losing weight through the traditional methods of weight loss management have shown to benefit from Bariatric Surgery [29,30]. The universal guidelines for Bariatric Surgery consists of the following constituents: being 18-65 years of age and having a BMI equal to or more than 40 kg/m^2 or a BMI equal to or more than 35 kg/m^2 and the presence of any obesity related comorbidities (resistant hypertension, established heart disease, severe degenerative osteoarthritis or respiratory failure) [31,32]. Patients that suffer from OSA should be informed about an increased risk of intra-operative complications if they wish to undergo Bariatric Surgery.

The OSA patients that are undergoing bariatric surgery are at an increased risk of general anesthesia related complications like hypertension, hypoxemia, arrhythmias and hypercapnia. Due to the altered lung physiology and dysmorphic chest anatomy, there are increased chances of complications [33,34]. Bariatric surgeons have decided to make OSA evaluation a compulsory pre-operative screening, regardless of symptoms [35] due to the significant number of undiagnosed OSA patients that plan on going for the procedure [36-40].

Polysomnography has been declared the standard screening method for diagnosing OSA. The patient requires an overnight stay at a sleep laboratory to meet the demands of a standardized sleep study. The physical examination and the patient history are insignificant in diagnosing obstructive sleep apnea [41,42].

The intention of this study was to focus on multi-ethnic bariatric surgery patients of underserved areas of the New York City and compare the prevalence, severity and co-morbidities of OSA in obese versus non-obese patients.

Method

In this retrospective study, we collected data from 749 obese outpatients, scheduled for bariatric surgery. They were referred by four different surgeons of the hospital, situated in New York City, between January 1st 2015 to December 31st 2015, regardless of having OSA symptoms or not. Among those, we selected 258 patients, who went for the overnight sleep study.

The sleep studies were conducted by a Board Certified Sleep Physician at 9 bed sleep clinic. The data regarding co-morbidities that was collected during the initial consultation included hypertension, diabetes mellitus, asthma and hypothyroidism. The study was broadly categorized into OSA and non-OSA and then further sub-stratified into mild and moderate to severe OSA on the basis of apnea/hypopnea index (AHI).

The overnight polysomnography was conducted by using a digital system (Alice Phillips Respironic Sleepware G3 version 7.3.4). The polysomnographic data was scored manually and was interpreted by an experienced board certified sleep physician according to the current guidelines [43,44]. The most recent criteria was used for the purpose of scoring sleep stages [44]. The guidelines suggest that the occlusion of airflow in the upper respiratory tract for more than ten seconds is an "apnea event", while a "hypopnea event" is defined as a nasal pressure decrement of 30% or more, together with an episode lasting more than ten seconds with 3% or more oxyhemoglobin desaturation. The OSA diagnosis and severity was based on Apnea Hypopnea Index (AHI), which is defined as the total events of apnea and hypopnea per hour of sleep. Less than equal to 5 AHI/hour is considered normal, 6 to 15 AHI/hour as mild OSA, 16 to 30 AHI/hour moderate and greater than 30 AHI/hour is considered severe OSA.

Data was organized by using Microsoft excel and mean, percentages and relative risk were calculated where appropriate.

Results

Table 1 data suggests that out of 258 people, 124 (48%) had OSA. Hypertension, diabetes mellitus, and hypothyroidism were more prevalent in bariatric surgery candidates diagnosed as OSA on polysomnography. Whereas, asthma was more common in normal patients (56%). The probability of having diabetes mellitus was 1.93 times more in OSA patients under going bariatric surgery as compared to non OSA bariatric surgery candidates. Similarly, OSA diagnosed population waiting for bariatric surgery was 1.57 times more likely to have hypertension

n=258		NORMAL	OSA	Relative Risk
Participants	(n)	134	124	
	%	51.9379845	48.0620155	
Female	(n)	119	84	
	%	46.124031	32.5581395	
Male	(n)	15	40	
	%	5.81395349	15.503876	
Asthma	(n)	45	35	0.84
	%	17.4418605	13.5658915	
DM	(n)	19	34	1.93
	%	7.36434109	13.1782946	
HTN	(n)	31	45	1.57
	%	12.0155039	17.4418605	
Hypothyroid	(n)	4	9	2.41
	%	1.5503876	3.48837209	

Table 1: Prevalence of comorbidities among OSA and normal population.

n=258		Control	Moderate	Severe
Participants	(n)	193	29	36
	%	74.8062016	11.2403101	13.9534884
Female	(n)	167	19	17
	%	64.7286822	7.36434109	6.58914729
Male	(n)	26	10	19
	%	10.0775194	3.87596899	7.36434109
Asthma	(n)	64	7	9
	%	24.8062016	2.71317829	3.48837209
DM	(n)	32	9	12
	%	12.4031008	3.48837209	4.65116279
HTN	(n)	49	11	16
	%	18.9922481	4.26356589	6.20155039
Hypothyroid	(n)	11	2	0
	%	4.26356589	0.7751938	0

Table 2: Prevalence of comorbidities among mild, moderate and severe OSA population.

as compared to non OSA population. On the other hand, Asthma was 0.84 times less likely in bariatric surgery candidates having OSA. Data further suggests that pre-operative bariatric surgery patients having AHI more than 5 were 2.41 times more probable of having hypothyroidism as compared to those having AHI less than 5.

Table 2 compares mild from moderate to severe OSA population. HTN and DM were more prevalent in moderate to Severe OSA candidates, whereas, despite of having high AHI, asthma and hypothyroidism were less likely in the group as compared to those having AHI less than 15.

Discussion

When we look at the statistics it becomes evident that both, hypertension and diabetes mellitus have some sort of direct or indirect co-relation with obesity [45]. Patients who suffer from DM2 or obesity face the same sort of challenges as they struggle to lose their body weight.

In the present era, in US (United States) alone, a disturbing 35% of the population above 20 years of age are found to be obese [46]. The most prevalent form of sleep disorder breathing (SDB) associated with hypertension, metabolic syndrome and DM2, which results in the increased risk of cardiovascular disease is Obstructive Sleep Apnea (OSA) [47]. In patients suffering from OSA the prevalence of metabolic syndrome has been shown to be on the higher side. The main risk factor for OSA is obesity [48]. Now OSA is being considerably counted as the most common cause of secondary arterial hypertension [48].

According to Min H J et al., 87.7% of their selected population had OSA with resistant HTN, as both disease have similar risk factors [49]. Whereas, in our study only 36% of the population had OSA with HTN. Henderson LA et al., postulated that central nervous system changes especially in brain stem circuitry and sympathetic activity contributes to the prevalence of HTN in OSA patients. They further mentioned that positive pressure ventilation can reverse these alterations in central nervous system [50]. Pengo MF and his team also discovered a considerable decrement in blood pressure and heart rate after using positive airway pressure for two weeks in OSA patients associated with HTN [51].

In our selected population, 27.4% people having DM were also diagnosed with OSA. Among those, 78% had AHI greater than 15, indicating that DM is more prevalent in patients with moderate to severe OSA. Similarly, Lam DC et al., found 54% prevalence of OSA in diabetic patients, which is much higher than ours, but only 33% of those had moderate to severe OSA, which is lower as compared to our data [52]. The metabolic syndrome in diabetic patients with OSA improved after one year of CPAP treatment [53]. Ronksley PE et al.,evaluated that DM is more prevalent in severe OSA patients [54]. Our data also suggests that DM is more prevalent (62%) in moderate to severe OSA.

Bahammam SA et al., showed 0.4% prevalence of clinical hypothyroidism in OSA patients [55], whereas in our study the prevalence was 0.07%. According to Kapur VK et al., hypothyroidism is not associated with OSA and the treatment of hypothyroidism has no effect on OSA [56].

According to Jonassen TM et al., asthma is associated with OSA [57], but our selected population showed less prevance of asthma in OSA than non OSA population with 0.84 times relative risk reduction. Teodorescu M et al., support severe OSA to be more prevalent in asthmatic patients [58]. Whereas, among the OSA population, our data suggests that asthma is more prevalent in mild (32%) than moderate to severe OSA patients (24%).

Currently, our data is limited to a single lab which can cause selection bias. We were unable to collect data regarding Epworth scoring scale and Stop Bang criteria related to severity based OSA groups. In future studies, we will focus on gathering the data on the basis of ethnicities.

We concluded that HTN, DM and hypothyroidism were more prevalent in obese population under going bariatric surgery, which were diagnosed as OSA on polysomnography. Asthma was more prevalent in non OSA population. Among the OSA population, HTN and DM were more prevalent in moderate to severe OSA, whereas, asthma and hypothyroidism were more common in mild OSA patients.

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Conflict of Interest

All authors certify that they have no affiliations.

Ethical approval

Not required

Informed consent

Not required

References

1. Statistics about Diabetes: American Diabetes Association website.
2. T Nwankwo T, Yoon SS, Burt V, Gu Q. Hypertension Among Adults in the United States: National Health and Nutrition Examination Survey, 2011-2012. *NCHS Data Brief*. 2013;(133):1-8.
3. Yoon SS, Burt V, Louis T, Carroll MD. Hypertension among adults in the United States, 2009-2010 *NCHS Data Brief*. 2012;(107):1-8.
4. Viswanathan V, Snehalatha C, Kumutha R, Nair BM, Ramachandran A. Impact of Joint National Committee VII recommendations on diabetic microvascular complications. *J Assoc Physicians India*. 2004;52:873-876.
5. Chen XY, Thomas GN, Chen YK, Chan JC, Wong KS. Atherosclerotic vascular disease rather than metabolic syndrome predicts ischemic stroke in diabetic patients. *Cerebrovasc Dis*. 2010;30(4):374-379.
6. Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. *Lancet*. 2011;378(9793):804-814.
7. Health and Environment Alliance (HEAL) Figures on Obesity and Diabetes in the European Union Brussels: HEAL; nd.
8. Cecchini M, Sassi F, Lauer JA, Lee YY, Guajardo-Barron V, Chisholm D. Tackling of unhealthy diets, physical inactivity, and obesity: health effects and cost-effectiveness. *Lancet.* 2010;376(9754):1775-1784.
9. IDF The global burden [web page on the Internet] IDF Diabetes Atlas 5th edBrussels: IDF; nd
10. Ogden CL, Carroll, Kit BK, Flegal KM. Prevalence of Childhood and Adult Obesity in the United States, 2011-2012. *JAMA*. 2014;311(8):806-814.
11. Vgontzas AN. Does obesity play a major role in the pathogenesis of sleep apnoea and its associated manifestations via inflammation, visceral adiposity, and insulin resistance? *Arch Physiol Biochem*. 2008;114(4):211-223.
12. Steier J, Jolley CJ, Seymour J, Roughton M, Polkey MI, Moxham J. Neural respiratory drive in obesity. *Thorax*. 2009;64:719-725.
13. Steier J, Jolley CJ, Seymour J, et al. Increased load on the respiratory muscles in obstructive sleep apnea. *Respir Physiol Neurobiol.* 2010;171(1):54-60.
14. Zammit C, Liddicoat H, Moonsie I, et al. Obesity and respiratory diseases. *Int J Gen Med*. 2010;3:335-343.
15. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med.* 1993;328(17):1230-1235.
16. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol*. 2013;177(9):1006-1014.
17. Aguiar IC, Freitas WR, Santos IR, et al. Obstructive sleep apnea and pulmonary function in patients with severe obesity before and after bariatric surgery: a randomized clinical trial. *Multidiscip Respir Med.* 2014;9(1):43.
18. Remmers JE, deGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol Respir Environ Exerc Physiol.* 1978;44(6):931-948.
19. Sharma SK, Agrawal S, Damodaran D, et al. CPAP for the metabolic syndrome in patients with obstructive sleep apnea. *N Engl J Med*. 2011;365:2277-2286.
20. Steier J, Martin A, Harris J, Jarrold I, Pugh D, Williams A. Predicted relative prevalence estimates for obstructive sleep apnoea and the associated healthcare provision across the UK. *Thorax* 2014;69(4):390-402.
21. West SD, Nicoll DJ, Stradling JR. Prevalence of obstructive sleep apnoea in men with type 2 diabetes. *Thorax* 2006;61(11):945-950.
22. Sjostrom C, Lindberg E, Elmasry A, Hagg A, Svardsudd K, Janson C. Prevalence of sleep apnoea and snoring in hypertensive men: a population based study. *Thorax*. 2002;57(7):602-617.
23. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med.* 2005;353(19):2034-2041.
24. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. *Lancet*. 1981;317(8225):862-875.
25. National Institute for Health and Clinical Excellence. Continuous positive airway pressure for the treatment of obstructive sleep apnoea/hypopnoea syndrome, 2008:26.
26. Haines KL, Nelson LG, Gonzalez R, et al. Objective evidence that bariatric surgery improves obesity-related obstructive sleep apnea. *Surgery.* 2007;141(3):354-368.
27. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA*. 2004;292(14):1724-1737.
28. Mostaedi R, Lackey DE, Adams SH, Dada SA, Hoda ZA, Ali MR. Prevalence of undiagnosed and inadequately treated type 2 diabetes mellitus, hypertension, and dyslipidemia in morbidly obese patients who present for bariatric surgery. *Obes Surg*. 2014;24(6):927-935.
29. Alqahtani AR, Antonisamy B, Alamri H, Elahmedi M, Zimmerman VA. Laparoscopic sleeve gastrectomy in 108 obese children and adolescents aged 5 to 21 years. *Ann Surg*. 2012;256(2):266-273.
30. Yermilov I, McGory ML, Shekelle PW, Ko CY, Maggard MA. Appropriateness criteria for bariatric surgery: beyond the NIH guidelines. *Obesity (Silver Spring)*. 2009;17(8):1521-1527.
31. Gasa M, Salord N, Fortuna AM, et al. Obstructive sleep apnoea and metabolic impairment in severe obesity. *Eur Respir J*. 2011;38(5):1089-1097.
32. Ravesloot MJ, van Maanen JP, Hilgevoord AA, van Wagensveld BA, de Vries N. Obstructive sleep apnea is underrecognized and underdiagnosed in patients undergoing bariatric surgery. *Eur Arch Otorhinolaryngol.* 2012;269(7):1865-1871.
33. Chung SA, Yuan H, Chung F. A systemic review of obstructive sleep apnea and its implications for anesthesiologists. *Anesth Analg*. 2008;107(5):1543-1563.
34. Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. *Am J Cardiol*. 1983;52(5):490-514.
35. O'Keeffe T, Patterson EJ. Evidence supporting routine polysomnography before bariatric surgery. *Obes Surg*. 2004;14(1):23-26.
36. Rasmussen JJ, Fuller WD, Ali MR. Sleep apnea syndrome is significantly underdiagnosed in bariatric surgical patients. *Surg Obes Relat Dis.* 2012;8(5):569-573.
37. Carneiro G, Florio RT, Zanella MT, et al. Is mandatory screening for obstructive sleep apnea with polysomnography in all severely obese patients indicated? *Sleep Breath*. 2012;16(1):163-168.
38. Nepomnayshy D, Hesham W, Erickson B, MacDonald J, Iorio R, Brams D. Sleep apnea: is routine preoperative screening necessary? *Obes Surg*. 2013;23(3):287-291.
39. Yeh PS, Lee YC, Lee WJ, et al. Clinical predictors of obstructive sleep apnea in Asian bariatric patients. *Obes Surg*. 2010;20(1):30-35.
40. Farinholt GN, Carr AD, Chang EJ, Ali MR. A call to arms: obese men with more severe comorbid disease and underutilization of bariatric operations. *Surg Endosc*. 2013;27(12):4556-4563.
41. Hoffstein V, Szalai JP. Predictive value of clinical features in diagnosing obstructive sleep apnea. *Sleep*. 1993;16(2):118-122.
42. Viner S, Szalai JP, Hoffstein V. Are history and physical examination a good screening test for sleep apnea? *Ann Intern Med*. 1991;115(5):356-369.
43. Iber C, Ancoli-Israel S, Chesson AL, et al. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester, IL: American Academy of Sleep Medicine; 2007.

44. Silber MH, Ancoli-Israel S, Bonnet MH, et al. The visual scoring of sleep in adults. *J Clin Sleep Med*. 2007;3(2):121-131.

45. Colosia AD, Palencia R, Khan S. Prevalence of hypertension and obesity in patients with type 2 diabetes mellitus in observational studies: a systematic literature review. *Diabetes Metab Syndr Obes*. 2013;(6):327-338.

46. Seetho IW, Wilding JP. Sleep-disordered breathing, type 2 diabetes and the metabolic syndrome. *Chron Respir Dis.* 2014;11(4):257-275.

47. Schulz R, Eisele HJ, Reichenberger F, Seeger W. Obstructive sleep apnoea and metabolic syndrome. *Pneumologie*. 2008;62(2):88-91.

48. Vgontzas AN, Bixler EO, Chrousos GP. Sleep apnea is a manifestation of the metabolic syndrome. *Sleep Med Rev*. 2005;9(3):211-224.

49. Hyun Jin Min, Yang-Je Cho, Chang-Hoon Kim. Clinical Features of Obstructive Sleep Apnea That Determine Its High Prevalence in Resistant Hypertension. *Yonsei Med J*. 2015;56(5):1258-1265.

50. Henderson LA, Macefield VG. Obstructive Sleep Apnoea and Hypertension: the Role of the Central Nervous System. *Curr Hypertens Rep*. 2016;18(7):59.

51. Pengo MF, Ratneswaran C, Berry M, et al. Effect of Continuous Positive Airway Pressure on Blood Pressure Variability in Patients With Obstructive Sleep Apnea. *J Clin Hypertens (Greenwich)*. 2016;(11):1180-1184.

52. Lam DC, Lui MM, Lam JC, Ong LH, Lam KS, Ip MS. Prevalence and recognition of obstructive sleep apnea in Chinese patients with type 2 diabetes mellitus. *Chest*. 2010;138(5):1101-1117.

53. Oktay B, Akbal E, Firat H, Ardiç S, Kizilgun M. CPAP treatment in the coexistence of obstructive sleep apnea syndrome and metabolic syndrome, results of one year follow up. *Acta Clin Belg*. 2009;64(4):329-334.

54. Ronksley PE, Hemmelgarn BR, Heitman SJ, Hanly PJ, Faris PD, Quan H, Tsai WH. Obstructive sleep apnoea is associated with diabetes in sleepy subjects. *Thorax*. 2009;64(10):834-849.

55. Bahammam SA, Sharif MM, Jammah AA, Bahammam AS. Prevalence of thyroid disease in patients with obstructive sleep apnea. *Respir Med*. 2011;105(11):1755-1760.

56. Kapur VK, Koepsell TD, deMaine J, Hert R, Sandblom RE, Psaty BM. Association of hypothyroidism and obstructive sleep apnea. *Am J Respir Crit Care Med*. 1998;158(1):1379-1383.

57. Jonassen TM, Eagan TM Bjorvatn B, Lehmann S. Associations between obstructive lung disease and symptoms of obstructive sleep apnoea in a general population. *Clin Respir J*. 2016.

58. Mihaela Teodorescu, Jodi H. Barnet, Erika W. Hagen, Mari Palta, Terry B. Young, Paul E. Peppard. Association between asthma and risk of developing obstructive sleep apnea. *JAMA*. 2015;13;313(2):156-164.