



ISSN: 0975-833X

RESEARCH ARTICLE

TO ANALYSE THE IMPACT OF FAMILY HISTORY OF DIABETES ON SUBJECTIVE SLEEP QUALITY IN I YEAR MEDICAL STUDENTS

*Dr. Sunitha Priya, A. K.

Assistant Professor, Department of Physiology, Jubilee Mission Medical College and Research Institute, Thrissur, Kerala-680005

ARTICLE INFO

Article History:

Received 05th September, 2017
Received in revised form
12th October, 2017
Accepted 10th November, 2017
Published online 27th December, 2017

Key words:

Diabetes Mellitus;
Students,
Medical,
Sleep Quality.

ABSTRACT

Background: India being the “diabetes capital of the world” demands a pressing need to keep this diabetic epidemic in control. Poor sleep quality and decreased sleep duration have been linked to being one of the causal factors of diabetes. Thus sleep is not only a process of restoration of bodily function, but plays a major role in metabolic, specifically in glucose homeostasis. Though the link between poor sleep quality and diabetes is well established and proven to be bi-directional, the effect of sleep quality on the offspring of diabetics is not documented. So the objective of our study is to look at the effect of family history of diabetes on subjective sleep quality in I year Medical students.

Methods: After obtaining the informed consent, students filled out questionnaire with details of family history of diabetes, based on which they were classified into FH-, FH+1 and FH+2 groups which represents 0, 1, and 2 diabetics in the family respectively. Sleep quality was assessed by Pittsburgh sleep Quality index (PSQI) Questionnaire which is a subjective assessment of self-reported sleep quality.

Results: The global PSQI score was significantly different across the groups with FH+2 having highest (6.86) score. Pearson chi square test showed significant association between poor sleep quality and strong history of diabetes.

Conclusions: Strong family history of diabetes is associated with a poor sleep quality. An inheritable, overactive central sympathetic activity could be the possible link between sleep quality and family history of diabetes.

Copyright©2017, Dr. Sunitha Priya. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation: Dr. Sunitha Priya A.K. 2017. “To analyse the impact of family history of diabetes, on subjective sleep quality in I year Medical students.”, *International Journal of Current Research*, 9, (12), 62449-62452.

INTRODUCTION

The number of diabetics in the world has increased from 108 million in 1980 to 422 million in 2014, an increase in the prevalence of global diabetes from 4.7% to 8.5% (Kaveeshwar, 2014). India being the “diabetes capital of the world” will be host to around 79 million diabetics by 2030. “Every fifth diabetic in the world is an Indian” which demands a pressing need to keep this diabetic epidemic in control (Kumar *et al.*, 2015). The multi factorial causes of diabetes are obesity, genetic factors, environmental factors, socioeconomic, behavioral, and demographic factors and the interaction between genetics and these factors (Touma *et al.*, 2011). Recently poor sleep quality and decreased sleep duration have been linked to being one of the causal factors of diabetes (Kita *et al.*, 2012; Lee *et al.*, 2016; Ayas *et al.*, 2003; Lou *et al.*, 2012). Sleep was thought only as a crucial process of restoration of bodily function but recent studies have proven its importance in metabolic, specifically in glucose homeostasis.

*Corresponding author: Dr. Sunitha Priya A.K.

Assistant Professor, Department Of Physiology, Jubilee Mission Medical College and Research Institute, Thrissur, Kerala-680005.

Human sleep is composed of REM (Random eye movements) and NREM sleep (Non-random eye movement). The sleep cycle begins with a short period of NREM sleep followed by REM sleep and this alternates for 4-5 times during a normal night's sleep (Kumar, 2008). NREM is further subdivided into 4 stages as I, II, III, and IV of which the last two stages represent the deeper stages of sleep which are found during the first one-third of sleep. Hence, Stages III and IV are thought to be the most restorative and are also called as slow-wave sleep (SWS) (Tasali *et al.*, 2008). During SWS, less glucose is utilized by the brain, the sympathetic nervous system is less active, and the vagal tone is increased compared to wake and REM sleep (Zoccoli *et al.*, 2002; Somers *et al.*, 1993). SWS is also associated with a repressed pituitary-adrenal activity. Considering these changes, it is not surprising that a chronic poor sleep quality could affect overall glucose homeostasis. There is an alarming increase in Sleep loss and sleep disturbances in our society. This self-imposed sleep deprivation is due to longer working hours, more shift-work, and less time for sleep.

The decreased sleep duration and sleep disturbance contribute to poor sleep quality which is linked to many behavioral, cognitive and mood disturbances (Kumar, 2008; US Centers for Disease Control and Prevention, 2009; Panda *et al.*, 2012). Recently human sleep deprivation studies have thrown light on its effect on Insulin sensitivity. A one-week sleep restriction in healthy normoglycemic men was associated with a 20% decrease in Insulin sensitivity (Buxton, 2010). A prospective study on Japanese workers with 4 years follow-up also has proven a poor sleep quality to be a major risk factor for the development of diabetes independent of other confounding factors (Kita *et al.*, 2012). Though the link between poor sleep quality and diabetes is well established and proven to be bi-directional (Cunha, 2008), the effect of sleep quality on the offspring of diabetics is not well documented. So the objective of our study is to look at the effect of family history of diabetes on subjective sleep quality in 1 year Medical students.

MATERIALS AND METHODS

This is a cross-sectional, questionnaire based study conducted in April 2017. The study population was 1 year MBBS students in a Medical college in Kerala, India. Students willing to participate in the study formed the inclusion criteria. After obtaining the Institutional ethical approval and informed written consent, 100 students filled out their personal information with details of family history of diabetes and Pittsburgh sleep quality questionnaire. Out of 100, two questionnaires were incomplete and had to be excluded from the analysis. Positive history of diabetes in parents and grandparents were taken into account and the number of diabetics in the family was calculated as 0, 1, and 2 which were represented as FH-, FH+1 and FH+2 respectively.

Assessment of sleep Quality: Sleep quality can be assessed subjectively or objectively. Subjective assessment is based on sleep Questionnaires while the objective assessment is by polysomnography. The gold standard for assessing sleep quality is the laboratory polysomnogram (PSG) which involves noninvasive sensors like skin electrode, EEG, ECG, and EOG electrodes and sensors to measure arterial oxygen saturation (Carley, 2016). This objective measurement is not accessible for everyone and hence a subjective assessment of self-reported sleep quality was developed. Though numerous instruments have been standardized, the Pittsburgh sleep Quality index is extensively used and validated (Buysse *et al.*, 1989). Studies have shown Subjective sleep quality to be a measure of slow wave sleep and sleep continuity. Sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI), a self-administered questionnaire for evaluating the quality of sleep in the last one month. It has seven components namely sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleep medication, and daytime dysfunction. Each component in the PSQI is scored from 0 to 3, whereby 0 means the positive extreme and 3 means the negative extreme on the Likert scale; the PSQI global score is the sum of these subcategories. The global score range from 0 to 21 and a score of 5 or more is defined as Poor sleep quality and a score less than five as good sleep quality.

Statistical Analysis

The computer-based analysis program SPSS version 20.0 was used for all statistical analyses.

The data was normally distributed and hence were represented as Mean \pm SE. The students were classified into 3 groups as FH-, FH+1 and FH+2 based on the number of diabetics in the family. One Way ANOVA was done to analyse the statistical difference in the sleep quality across group. Sleep quality was divided into two categories as poor (PSQI score $<$ 5) and good (PSQI score $>$ 5) and Chi-square was done to look at the association between the categorical variables sleep quality and number of diabetics in the family.

RESULTS

The mean age of our study population was 19 years. There were 26 students without any family history of diabetes. Of the remaining 78 students, 51 students had one diabetic and 27 students had 2 diabetics in the family. Since complete sampling method was used, we could not have equal number of students in each group.

Table 1 illustrates the subject characteristics namely height, weight, waist circumference, hip circumference and waist-hip ratio of 3 groups. The anthropometric measures were not different between the groups and hence one way ANOVA showed no statistical difference across group.

Table 1. Subject characteristics

Variable	FH- (n=26)	FH+1 (n=51)	FH+2 (n=21)
Weight (Kg)	60.21 \pm 2.03	58.21 \pm 1.67	55.78 \pm 2.07
Height (Cm)	166.52 \pm 1.75	165.07 \pm 1.27	160.68 \pm 1.85
Waist Circumference(Cm)	73.35 \pm 1.43	71.82 \pm 1.23	70.73 \pm 1.72
Hip Circumference(Cm)	94.5 \pm 1.21	93.77 \pm 0.95	93.75 \pm 1.52
W/H Ratio	0.77 \pm 0.01	0.76 \pm 0.01	0.75 \pm 0.01

Data represented as Mean \pm SEM. One way ANOVA showed no significant difference between the groups. FH- No Family H/O Diabetes. FH+1 one diabetic in the family. FH+2 two diabetics in the family

Table 2. Subjective sleep quality score

Variable	FH- (n=26)	FH+1 (n=51)	FH+2 (n=21)
Sleep quality (C1)	1.07 \pm 0.13	0.98 \pm 0.10	1.48 \pm 0.16*
Sleep latency (C2)	0.63 \pm 0.16	0.35 \pm 0.07	0.71 \pm 0.19
Sleep duration (C3)	1.48 \pm 0.11	1.55 \pm 0.11	1.76 \pm 0.17
Habitual sleep efficiency (C4)	0.07 \pm 0.050	0.04 \pm 0.03	0.19 \pm 0.09
Sleep disturbance (C5)	0.85 \pm 0.07	1.02 \pm 0.06	1.09 \pm 0.12
Use of sleep medication (C6)	0	0	0
Daytime dysfunction (C7)	1.11 \pm 0.14	1.31 \pm 0.12	1.61 \pm 0.17
GPSQI	5.26 \pm 0.36	5.25 \pm 0.28	6.86 \pm 0.5**

Data represented as Mean \pm SEM. One way ANOVA showed significant difference in C1 and GPSQI across the groups. *p $<$ 0.05, **p $<$ 0.01. FH- No Family H/O Diabetes. FH+1 one diabetic in the family. FH+2 two diabetics in the family

Table 2 represents the Pittsburgh sleep quality score and its component score. The global score is significantly different across the groups with FH+2 having highest (6.86) score. Perceived sleep quality is also higher in FH+2 (1.48). Though other components of PSQI were still higher in the FH+2 group they were not statistically significant.

FH- and FH+1 had similar scores in all categories. Table 3 represents Pearson chi-square test between family history of diabetes (FH-, FH+1 and FH2) and sleep quality categorized as poor and good. A significant association was found between poor sleep quality and strong family history of diabetes (p=0.004).

Table 3. Cross tabulation between sleep quality and number of diabetics in the family

Variable	Sleepgroup		Total	p value
	<5 (Good sleep quality)	>5 (Poor sleep quality)		
Number of Diabetics in the family	0	13	13	$\chi^2(2)=11.2, p = 0.004$
	1	35	16	
	2	6	15	
Total		54	44	98

Chi-square test showed significant association between sleep quality and the number of diabetics in the family.

DISCUSSION

Our study illustrates that students with a strong family history of diabetes have poor sleep quality compared to other groups. Also, students with one diabetic in the family had similar sleep quality as those without family history of diabetes, while students with more than one diabetic in the family seem to have the worst sleep quality. A family history of diabetes is a high-risk factor for the eventual development of diabetes in an individual (Ramachandran, 2001; Mohan, 2003) and studies have shown insulin resistance exhibited by normoglycemic, young off springs of diabetic parents (Arslanian, 2005). Also, these off springs have increased central sympathetic nerve activity proportional to the increase in insulin resistance. Similarly, Anderson *et al* showed that hyperinsulinemia secondary to insulin resistance causes sympathetic activation in humans (Anderson, 1991). In addition, chronic Sympathetic nervous system (SNS) over activity itself can contribute to a further decline in insulin sensitivity. Hence, the two possible mechanisms are: offspring of type 2 diabetic subjects develop insulin resistance through sympathetic activation or hyperinsulinemia can lead to hyperactive central sympathetic drive. In a 10 year follow-up study in normoglycemic Japanese population, it was found that sympathetic hyperactivity preceded hyperinsulinaemia. Thus, this insulin-mediated increase in SNS is mediated via the arcuate nucleus of the hypothalamus²⁴. The highly permeable capillaries in the arcuate nucleus (AN) allows insulin to activate receptors without a specific transport mechanism (Ciofi, 2011; Dampney, 2011).

Likewise, a transport mediated uptake of peripheral insulin across the blood-brain barrier is also existent in the hypothalamus. Patients with type 2 diabetes tend to sleep poorly¹⁶ and studies have demonstrated that curtailment of sleep duration and poor sleep quality was associated with increased SNS activity (Somers, 1995; Spiegel, 1999). However, selective SWS deprivation caused SNS hyperactivity with an associated increase in plasma catecholamines and a decrease in insulin sensitivity (Spiegel 1999). Studies on young male twins showed that amount of SWS is a stable individual trait that is highly heritable (Linkowski, 1999). Though sleep quality can also be markedly affected by non-genetic factors, significant heritability for sleep duration and sleep quality have been documented (Watson, 2010). Also, sympathetic activity per se is also genetically determined. An individual with a strong family history of diabetes has hyperinsulinemia and increased SNS compared to non-diabetic offspring. Thus it could be the heritable nature of abnormal SWS and/or SNS hyperactivity which could contribute to the future development of diabetes in individuals with family history of diabetes. To the best of our belief, we did not find any scientific article linking family history of diabetes and sleep quality and hence we are the first to report this finding.

Limitations of the study: Though, PSQI questionnaire is a validated tool for assessing sleep quality, the gold standard is the polysomnography which could not be used in this study. Also, our findings cannot be extrapolated to the general population because of the narrow range of the age group of the study sample.

Conclusion

The present study concludes that students with a strong family history of diabetes have poor sleep quality which may lead on to diabetes in later part of their life. An increased central sympathetic drive could be the possible link. This finding can be confirmed only with prospective studies in the future.

Acknowledgement

We wholeheartedly thank our students for participating in this study.

Conflict of interest: the authors have no conflict of interest

REFERENCES

- Anderson, E. A., Hoffman, R. P., Balon, T. W., Sinkey, C. A. and Mark. A. L. 1991. Hyperinsulinemia produces both sympathetic neural activation and vasodilation in normal humans. *J Clin Invest.*, Jun; 87:2246-52
- Arslanian SA, Bacha F. Saad R, Gungor N. 2005. Family history of type 2 diabetes is associated with decreased insulin sensitivity and an impaired balance between insulin sensitivity and insulin secretion in white youth. *Diabetes Care.* Jan; 28:115-9
- Ayas NT1, White DP, Al-Delaimy WK, Manson JE, Stampfer MJ, Speizer FE, Patel S, Hu FB. 2003. A prospective study of self reported sleep duration and incident diabetes in women. *Diabetes Care.* Feb; 26:380-4.
- Buxton OM1, Pavlova M, Reid EW, Wang W, Simonson DC, Adler GK. 2010. Sleep restriction for 1 week reduces insulin sensitivity in healthy men. *Diabetes.* Sep; 59:2126-33.
- Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. 1989. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res.* 28:193- 213.
- Carley DW1, Farabi SS. 2016. Physiology of Sleep. *Diabetes Spectr.* Feb; 29:5-9
- Cassaglia, P. A., Hermes, S. M., Aicher, S. A. and Brooks. V. L. 2011. Insulin acts in the arcuate nucleus to increase lumbar sympathetic nerve activity and baroreflex function in rats. *J Physiol.*, Apr 1; 589:1643-62.
- Ciofi. P. 2011. The arcuate nucleus as a circumventricular organ in the mouse. *NeurosciLett.* Jan 7; 487:187-90.
- Cunha MC1, Zanetti ML, Hass VJ. 2008. Sleep quality in type 2 diabetics. *Rev Lat Am Enfermagem.* Sep-Oct; 16:850-5.

- Dampney. R. A. L. 2011. Arcuate nucleus—a gateway for insulin's action on sympathetic activity. *J Physiol.* May 1; 589:2109-10.
- Kaveeshwar SA, Cornwall J. 2014. The current state of diabetes mellitus in India. *AMJ*; 7:45-48
- Kita T1, Yoshioka E, Satoh H, Saijo Y, Kawaharada M, Okada E, Kishi R. 2012. Short sleep duration and poor sleep quality increase the risk of diabetes in Japanese workers with no family history of diabetes. *Diabetes Care.* Feb;35 :313-8
- Kita T1, Yoshioka E, Satoh H, Saijo Y, Kawaharada M, Okada E, Kishi R. 2012. Short sleep duration and poor sleep quality increase the risk of diabetes in Japanese workers with no family history of diabetes. *Diabetes Care.* Feb; 35:313-8
- Kumar A, Goel MK, Jain RB, Khanna P, Chaudhary V. 2013. India towards diabetes control: Key issues. *AMJ* 6:524-531
- Kumar VM. 2008. Sleep and sleep disorders. *Indian J Chest Dis Allied Sci.*, Jan-Mar;50 :129-35.
- Lee JA1, Sunwoo S1, Kim YS1, Yu BY2, Park HK3, Jeon TH4, Yoo BW5. 2016. The Effect of Sleep Quality on the Development of Type 2 Diabetes in Primary Care Patients. *J Korean Med Sci.* Feb;31 :240-6
- Linkowski, P. 1999. EEG sleep patterns in twins. *J Sleep Res.* Jun; 8:11-3
- Lou P1, Chen P, Zhang L, Zhang P, Yu J, Zhang N, Wu H, Zhao J. 2012. Relation of sleep quality and sleep duration to type 2 diabetes: a population-based cross-sectional survey. *BMJ Open.* Aug 7; 2: 1-5.
- Mohan V, Shanthirani CS, Deepa R. 2003. Glucose intolerance (diabetes and IGT) in a selected South Indian population with special reference to family history, obesity and lifestyle factors—the Chennai urban population study (CUPS14). *J Assoc Physicians India.* Aug;51:771-7
- Panda S1, Taly AB, Sinha S, Gururaj G, Girish N, Nagaraja D. 2012. Sleep-related disorders among a healthy population in South India. *Neurol India.* Jan-Feb; 60:68-74
- Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK *et al.* 2001. High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes survey. *Diabetologia.* 44:1094-1101
- Rewer M, Hamman RF. 1995. Risk factors for non-insulin-dependent diabetes. In *Diabetes in America*. 2nd ed. National Diabetes data group, Ed. Bethesda, MD, National Institute of Health, p.179-220.
- Somers VK, Dyken ME, Mark AL, Abboud FM. 1993. Sympathetic-nerve activity during sleep in normal subjects. *N Engl J Med.*, 328:303–307.
- Somers, V. K., Dyken, M. E., Clary, M. P. and Abboud, F. M. 1995. Sympathetic neural mechanisms in obstructive sleep apnea. *J Clin Invest.*, Oct; 96:1897-904
- Spiegel, K1, Leproult, R., Van Cauter, E. 1999. Impact of sleep debt on metabolic and endocrine function. *Lancet.* Oct 23; 354:1435-9.
- Tasali E1, Leproult R, Ehrmann DA, Van Cauter E.2008. Slow-wave sleep and the risk of type 2 diabetes in humans. *Proc Natl Acad Sci U S A.* Jan 22; 105:1044-9
- Touma C, Pannain S. 2011. Does lack of sleep cause diabetes? *Cleve Clin J Med.* Aug;78 :549-58
- US Centers for Disease Control and Prevention (CDC). Perceived insufficient rest or sleep among adults—United States, 2008. *MMWR Morb Mortal Wkly Rep* 2009; 58:1175–1179.
- Watson, N.F.1, Buchwald, D., Vitiello, M.V., Noonan, C., Goldberg, J. 2010. A twin study of sleep duration and body mass index. *J Clin Sleep Med.* ,Feb 15; 6:11-7.
- Zoccoli G, Walker AM, Lenzi P, Franzini C. 2002. The cerebral circulation during sleep: regulation mechanisms and functional implications. *Sleep Med Rev.* 6:443–455.
