Published in final edited form as:
Am J Obstet Gynecol. 2010 August ; 203(2): 142.e1–142.e5. doi:10.1016/j.ajog.2010.03.041.

Self-reported short sleep duration and frequent snoring in pregnancy: Impact on glucose metabolism

Francesca L. FACCO, MD[1], **William A. GROBMAN, MD, MBA**[1], **Jamie KRAMER, MD**[2], **Kim H. HO, BS**[1], and **Phyllis C. ZEE, MD, PhD**[3]

[1] Northwestern University, Feinberg School of Medicine, Department of Obstetrics and Gynecology

[2] New York Presbyterian Hospital-Cornell Medical Center, Department of Obstetrics and Gynecology

[3] Northwestern University, Feinberg School of Medicine, Department of Neurology

Abstract

Objective—To evaluate the impact of short sleep duration (SSD) and frequent snoring (FS) on glucose metabolism during pregnancy.

Study Design—Prospective, cohort study of healthy nulliparas who participated in a sleep survey study. SSD was defined as < 7 hours of sleep/night and FS, as snoring ≥3 nights/week. Outcomes included one-hour oral glucose tolerance (OGT) results and the presence of gestational diabetes (GDM). Univariate and multivariate analyses were performed.

Results—189 women participated; 48% reported a SSD and 18.5% complained of FS. SSD and FS were associated with higher OGT values: SSD (116±31 vs. 105±23, p=.008), FS (118±34 vs. 108±25, p= 0.04). Both SSD (10.2% vs. 1.1%, p=.008) and FS (14.3% vs. 3.3%, p=.009) were associated with a higher incidence of GDM. Even after controlling for potential confounders, SSD and FS remained associated with GDM.

Conclusions—SSD and FS are associated with glucose intolerance in pregnancy.

Keywords

gestational diabetes; glucose metabolism; sleep disorders in pregnancy

Introduction

Numerous studies have demonstrated associations between abnormal sleep patterns and a wide spectrum of medical conditions.[1, 2] In particular, poor sleep has been linked to insulin resistance, glucose intolerance, and type 2 diabetes. [3] The two sleep disorders that have been

Corresponding Author: Francesca Facco, MD, Northwestern University, Prentice Women's Hospital, 250 East Superior Street, Suite 05-2175, Chicago, IL 60611 312-472-4671, f-facco@md.northwestern.edu.

Prior Presentations

Facco FL, Grobman WA, Lu B, Kramer J, Ho K, Zee P. Frequent Snoring During Pregnancy is Associated with an Increased Risk of Gestational Diabetes. Annual Meeting of the Associated Professional Sleep Societies, Seattle, Washington, June. 6–11, 2009

Facco FL, Grobman WA, Lu B, Kramer J, Ho K, Zee P. Short Sleep Duration During Pregnancy: Impact on Glucose Metabolism. Society of Maternal-Fetal Medicine Annual Meeting, Chicago, IL, Feb. 3–6, 2010

most consistently associated with abnormal glucose metabolism are short sleep duration and sleep disordered breathing. [4–8]

Short sleep duration (SSD) has a variety of definitions, though most commonly it is defined as sleeping on average less than 7 hours per night. According to recent data from the 2004–2007 National Health Interview Survey, over 28% of the U.S adult population sleeps less than 7 hours/night. [9] Sleep disordered breathing (SDB) is a chronic condition characterized by repeated episodes of hypopnea and apnea during sleep. It is estimated that approximately 7% of adults in the general population have SDB of moderate to severe severity.[10] Frequent snoring is a common self-reported measure of SDB.[11–13] In non-pregnant adults, laboratory studies have shown that SSD and SDB are associated with impairments in glucose metabolism, [14–17] and epidemiologic studies have demonstrated a link between these two sleep disorders and the risk of developing diabetes. [5, 8, 18–21].

While studies have shown that SSD and frequent snoring are common complaints among pregnant women, [22–25] there are limited data regarding the relationship between these sleep disturbances and glucose metabolism during pregnancy. Abnormal glucose metabolism during pregnancy is associated with adverse maternal and neonatal outcomes.[26, 27] If SSD and SDB during pregnancy contribute to maternal glucose intolerance, screening for and treating these sleep disorders during pregnancy may lessen maternal glucose intolerance and perhaps improve pregnancy outcomes. The objective of this study was to evaluate the impact of self-reported SSD and SDB symptoms on glucose metabolism during pregnancy.

Material and Methods

This study was a planned secondary analysis of data from a prospective, observational study designed to evaluate the prevalence of and trends in sleep disturbances across pregnancy.[25] The study was approved by the Institutional Review Board of Northwestern University. Patients were recruited in the outpatient setting from among women who received care at Northwestern Memorial Hospital affiliated practices. These practices serve women who have both government-based and private health insurance. Women were approached for participation if they were nulliparous and had a singleton gestation. Women with the following medical conditions were excluded: chronic hypertension, heart disease, chronic lung disease, pre-gestational diabetes, chronic renal disease, and autoimmune disease (excluding treated hypothyroidism). Women who were eligible and agreed to participate provided informed consent.

The population was derived as a convenience sample (i.e., non-probability sampling, the patients are selected, in part or in whole, at the convenience of the researcher). Study participants were asked to complete a sleep questionnaire in early pregnancy (6–20 weeks) and then again in the third trimester (28–40 weeks). This questionnaire included demographic information such as maternal age, race and ethnicity, and pre-pregnancy weight. Subjects were followed prospectively and pregnancy outcomes were abstracted from the medical record by study personnel. Obstetric health care providers as well as study personnel who abstracted the medical record were not aware of the results of the sleep survey.

A full description of the sleep questionnaire used for this study has been reported elsewhere.[25] The questionnaire included items that addressed sleep duration and SDB symptoms. Participants were asked “During the past month, how many hours of *actual* sleep did you get at night?” The questionnaire specified that this number may be different than the number of hours spent in bed. SSD was defined as sleeping less than 7 hours per night. Participants were also asked if they snored (self-report). If they reported snoring they were asked to

choose one of the following snoring frequencies: nearly every day, 3–4 nights per week, 1–2 nights per week, 1–2 nights per month or never/nearly never. Frequent snoring, used to represent SDB, was defined as snoring ≥3 nights per week. Outcomes in women who complained of SSD or frequent snoring while pregnant (early and/or late pregnancy) were compared to outcomes in women without these sleep complaints.

Outcomes examined included mean 1-hour oral glucose tolerance (OGT) values, 1-hour OGT values ≥130, and gestational diabetes (GDM). Results of 1-hour OGT screening were obtained from the prenatal record. At our institution this testing is performed between 24–28 weeks using a 50 gram glucose load which is administered without regard to the time of the last meal, in accordance with The American College of Obstetricians and Gynecologists (ACOG) guidelines.[28] Women who screened positive on the 1-hour OGT went on to do a 100 gram, 3-hour OGT test. The prenatal record and delivery record were searched and the presence of GDM was based on documentation in the medical record. At the time of this study physicians at our institution utilized the diagnostic thresholds established by the National Diabetes Data Group (NDDG) to diagnose GDM.[29]

Outcomes and demographic characteristics in women with and without SSD and frequent snoring were compared using the t-test for continuous data, and the χ^2 and Fischer exact tests for categorical data. Multivariable logistic regression models were used to control for potential confounders. Covariates included in the multivariable regression models included maternal age, race or ethnicity, and pre-pregnancy BMI. All tests were two-tailed and a P value of less than 0.05 was considered statistically significant. Statistical analyses were performed using SPSS 17.0 statistical software (SPSS Inc., Chicago, IL).

Results

Of the 224 eligible women who were approached, 202 (90%) agreed to participate and completed the baseline survey. One hundred and eighty-nine of these women participated in the third-trimester survey as well. The mean gestational age was 13.8 ± 3.8 and 30.0 ± 2.2 weeks at the first and second survey, respectively. Demographic characteristics of the study population, as a whole, and stratified by sleep complaints, are provided in Table 1. Results of 1-hr OGT screening were available for 182 women. For six women the prenatal record only stated that the screening test was “normal”, and for one subject there was no documentation of a 1-hr OGT. Complete prenatal records that allowed for the ascertainment of GDM were available for 188 women.

At the time of the initial survey 26% of participants reported SSD (48/183). This increased to 40% (73/183) in the third trimester (p=.001).[25] In all, 48% of women (88/183) reported a SSD in early and/or late pregnancy. The prevalence of frequent snoring rose from 11% (21/189) in early pregnancy to 16% (31/189) in late pregnancy (p=.03). [25] In total, 18.5% (35/189) of women complained of frequent snoring while pregnant. Markers of glucose intolerance, stratified by presence of abnormal sleep patterns, are shown in Table 2. Both SSD and frequent snoring during pregnancy were associated with higher 1-hour OGT values. Women who reported SSD during pregnancy also were more likely to have 1-hour OGT values of ≥130 (OR 2.6, 95% CI 1.3, 5.7). The prevalence of 1-hour OGT ≥130 was greater in women with frequent snoring (32% vs. 20%); however, this difference was not statistically significant. This evidence of glucose intolerance was not limited to higher values on the 1-hour OGT, as women with SSD and frequent snoring were also noted to have a greater frequency of overt GDM: OR 10.6 (95% CI 1.3, 85.5) and OR 4.9 (95% CI 1.3, 18.1), respectively.

Multivariable logistic regression models were used to adjust for potential confounding factors. Even after controlling for age, race/ethnicity, and pre-pregnancy BMI, and adjusting for frequent snoring, SSD continued to be significantly associated with 1-hour GTT values ≥130 (adjusted OR 2.4, 95% CI 1.1,5.3) and the development of GDM (adjusted OR 11.7, 95% CI 1.2, 114.5). Likewise, after adjusting for demographic factors and SSD, frequent snoring remained associated with an increased risk of GDM (adjusted OR 6.7, 95% CI 1.4, 33.8). We performed an analysis to assess for an interaction between SSD and frequent snoring and no significant interaction was observed.

Discussion

This study examined glucose metabolism and risk of GDM in healthy nulliparous women reporting SSD and frequent snoring during pregnancy. Our findings suggest that women with these sleep disturbances are at increased risk of impaired glucose tolerance and GDM. Studies of non-pregnant individuals have linked SSD and SDB to fasting hyperglycemia, impaired glucose tolerance and type 2 diabetes.[14–21] Yet, evidence for the association between sleep and pregnancy abnormalities has been lacking. There is biologic plausibility for this association, as research has found that SSD and SDB are associated with elevated levels of pro-inflammatory cytokines and oxidative stress markers. It is thought that the enhanced inflammatory and oxidative stress response caused by these sleep disorders promotes insulin resistance which ultimately leads to impaired glucose tolerance and diabetes. [30, 31]

Women with GDM are at increased risk for pregnancy complications including preeclampsia and cesarean delivery, and their neonates are at risk for macrosomia, birth injury, hypoglycemia and hyperbilirubinemia. The Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) trial demonstrated that even women who have a more modest degree of glucose intolerance, less than that required for the diagnosis of GDM, are at greater risk of adverse pregnancy outcomes. [27] Known risk factors for GDM or impaired glucose tolerance during pregnancy include advanced maternal age, obesity, multiple gestations, and a family history of diabetes. This study suggests that poor sleep during pregnancy may also increase a women's risk of hyperglycemia during pregnancy, and that this risk is independent of maternal characteristics such as BMI.

The potential association of sleep abnormalities with glucose intolerance has clinical relevance, as abnormal sleep patterns are potentially modifiable risk factors. It is possible, for example, that screening for and treating sleep disorders during pregnancy could improve glucose metabolism, decrease the incidence of GDM, and improve pregnancy outcomes. Studies of non-pregnant individuals have shown just such a benefit with regards to treatment of SDB with nasal continuous positive airway pressure (nCPAP), and improvements in glucose metabolism. [32–34] Yet, evidence is lacking that diabetes itself, or adverse health outcome related to diabetes, can be prevented by interventions related to sleep. In non-pregnant individuals this type of study would be relatively difficult to accomplish since subjects would require such long term follow-up. Conversely, pregnancy may be a perfect setting to evaluate the possibility that treatments to improve abnormal sleep patterns can improve outcomes, given the abbreviated time course required for the manifestation of GDM and its consequences.

The main strengths of this study are its prospective design as well as the limited potential for ascertainment bias. The category of a patient's exposure was not known by either the individuals who cared for the patient or by the researcher who abstracted data from the clinical chart. Moreover, because sleep is not routinely evaluated in pregnancy, and there is not a well known association between sleep patterns and adverse pregnancy outcomes, there

is little reason to believe that ascertainment bias could have been introduced during regular clinical care. The principle limitation is that we assessed subjective sleep duration and symptoms of SDB. Self-reports of SSD and SDB have a reasonable although not exact correlation with objective measures (i.e., actigraphy or polysomnography) of sleep.[35, 36] Yet, if bias were to be introduced from this lack of correlation, it should be against the finding of an association. There is no reason to believe that women who are destined to develop GDM are more likely to incorrectly report the presence of abnormal sleep long before the GDM develops. Also, subjects were unaware what criteria would be used to define abnormal sleep patterns. Another limitation is that due to our sample size, stratified analyses to determine whether the associations we reported are present or different in certain populations (e.g., younger vs. older women) were not able to be adequately explored given the lack of power for informative subgroup analyses. On the whole, our results provide an estimate of the association between SSD and SDB during pregnancy and GDM that ideally would be confirmed by larger studies using objective measures of sleep (i.e., actigraphy or polysomnography). Additionally, this was an observational study and while we report a relationship between SSD and frequent snoring and impaired glucose tolerance during pregnancy, our study cannot infer causation. Furthermore, our study population was modestly diverse and recruited from a single institution, limiting the generalizability of our findings. Lastly, while our findings were statistically significant our confidence intervals were wide and further studies with greater power are required to provide more precise estimates of the association.

In summary, we found that self reported SSD and SDB are common during pregnancy and are associated with impaired glucose tolerance and GDM in healthy nulliparous women. Further studies, and in particular prospective investigations with objective sleep measures, are needed to confirm this association in a larger cohort of pregnant women. If our findings are confirmed, the next step would be to design studies to evaluate if screening for and treatment of sleep disorders during pregnancy can improve pregnancy outcomes.

Acknowledgments

Support

Supported in part by research grant 1K12HD050121 from the National Institutes of Health, National Institute of Child Health and Human Development

Refrences

1. Zee PC, Turek FW. Sleep and health: Everywhere and in both directions. Arch Intern Med. Sep 18; 2006 166(16):1686–1688. [PubMed: 16983044]
2. Foley D, Ancoli Israel S, Britz P, Walsh J. Sleep disturbances and chronic disease in older adults: results of the 2003 National Sleep Foundation Sleep in America Survey. J Psychosom Res. May; 2004 56(5):497–502. [PubMed: 15172205]
3. Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. Sleep Med Rev. Jun; 2007 11(3):163–178. [PubMed: 17442599]
4. Al Lawati NM, Patel SR, Ayas NT. Epidemiology, risk factors, and consequences of obstructive sleep apnea and short sleep duration. Prog Cardiovasc Dis. Jan–Feb; 2009 51(4):285–293. [PubMed: 19110130]
5. Ayas NT, White DP, Al Delaimy WK, et al. A prospective study of self-reported sleep duration and incident diabetes in women. Diabetes Care. Feb; 2003 26(2):380–384. [PubMed: 12547866]
6. Beihl DA, Liese AD, Haffner SM. Sleep duration as a risk factor for incident type 2 diabetes in a multiethnic cohort. Ann Epidemiol. May; 2009 19(5):351–357. [PubMed: 19362278]

7. Knutson KL, Ryden AM, Mander BA, Van Cauter E. Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus. Arch Intern Med. Sep 18; 2006 166(16):1768–1774. [PubMed: 16983057]
8. Punjabi NM, Shahar E, Redline S, Gottlieb DJ, Givelber R, Resnick HE. Sleep disordered breathing, glucose intolerance, and insulin resistance: the Sleep Heart Health Study. Am J Epidemiol. Sep 15; 2004 160(6):521–530. [PubMed: 15353412]
9. Krueger PM, Friedman EM. Sleep duration in the United States: a cross sectional population based study. Am J Epidemiol. May 1; 2009 169(9):1052–1063. [PubMed: 19299406]
10. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. Am J Respir Crit Care Med. May 1; 2002 165(9):1217–1239. [PubMed: 11991871]
11. Brietzke SE, Katz ES, Roberson DW. Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. Otolaryngol Head Neck Surg. Dec; 2004 131(6):827–832. [PubMed: 15577775]
12. Kump K, Whalen C, Tishler PV, et al. Assessment of the validity and utility of a sleep symptom questionnaire. Am J Respir Crit Care Med. Sep; 1994 150(3):735–741. [PubMed: 8087345]
13. Tami TA, Duncan HJ, Pfleger M. Identification of obstructive sleep apnea in patientswho snore. Laryngoscope. Apr; 1998 108(4 Pt 1):508–513. [PubMed: 9546261]
14. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet. Oct 23; 1999 354(9188):1435–1439. [PubMed: 10543671]
15. Stamatakis K, Punjabi NM. Effects of Sleep Fragmentation on Glucose Metabolism in Normal Subjects. Chest. Jun 19.2009
16. Stamatakis K, Sanders MH, Caffo B, et al. Fasting glycemia in sleep disordered breathing: lowering the threshold on oxyhemoglobin desaturation. Sleep. Jul 1; 2008 31(7):1018–1024. [PubMed: 18652097]
17. Trento M, Broglio F, Riganti F, et al. Sleep abnormalities in type 2 diabetes may be associated with glycemic control. Acta Diabetol. Dec; 2008 45(4):225–229. [PubMed: 18685806]
18. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. Ann N Y Acad Sci. 2008; 1129:287–304. [PubMed: 18591489]
19. Marshall NS, Wong KK, Phillips CL, Liu PY, Knuiman MW, Grunstein RR. Is sleep apnea an independent risk factor for prevalent and incident diabetes in the Busselton Health Study? J Clin Sleep Med. Feb 15; 2009 5(1):15–20. [PubMed: 19317376]
20. Gottlieb DJ, Punjabi NM, Newman AB, et al. Association of sleep time with diabetes mellitus and impaired glucose tolerance. Arch Intern Med. Apr 25; 2005 165(8):863–867. [PubMed: 15851636]
21. Reichmuth KJ, Austin D, Skatrud JB, Young T. Association of sleep apnea and type II diabetes: a population based study. Am J Respir Crit Care Med. Dec 15; 2005 172(12):1590–1595. [PubMed: 16192452]
22. Hedman C, Pohjasvaara T, Tolonen U, Suhonen-Malm AS, Myllyla VV. Effects of pregnancy on mothers' sleep. Sleep Med. Jan; 2002 3(1):37–42. [PubMed: 14592252]
23. Mindell JA, Jacobson BJ. Sleep disturbances during pregnancy. J Obstet Gynecol Neonatal Nurs. Nov–Dec; 2000 29(6):590–597.
24. Pien GW, Schwab RJ. Sleep disorders during pregnancy. Sleep. Nov 1; 2004 27(7):1405–1417. [PubMed: 15586794]
25. Facco FL, Kramer J, Ho KH, Zee PC, Grobman WA. Sleep disturbances in pregnancy. Obstet Gynecol. Jan; 115(1):77–83. [PubMed: 20027038]
26. Landon MB, Spong CY, Thom E, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. N Engl J Med. Oct 1; 2009 361(14):1339–1348. [PubMed: 19797280]
27. Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med. May 8; 2008 358(19):1991–2002. [PubMed: 18463375]
28. ACOG Practice Bulletin. Clinical management guidelines for obstetrician gynecologists. Number 30, September 2001 (replaces Technical Bulletin Number 200, December 1994). Gestational diabetes. Obstet Gynecol. Sep; 2001 98(3):525–538. [PubMed: 11547793]
29. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes. Dec; 1979 28(12):1039–1057. [PubMed: 510803]

30. Jelic S, Le Jemtel TH. Inflammation, oxidative stress, and the vascular endothelium in obstructive sleep apnea. Trends Cardiovasc Med. Oct; 2008 18(7):253–260. [PubMed: 19232954]

31. Mullington JM, Haack M, Toth M, Serrador JM, Meier Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. Prog Cardiovasc Dis. Jan–Feb; 2009 51(4):294–302. [PubMed: 19110131]

32. Babu AR, Herdegen J, Fogelfeld L, Shott S, Mazzone T. Type 2 diabetes, glycemic control, and continuous positive airway pressure in obstructive sleep apnea. Arch Intern Med. Feb 28; 2005 165(4):447–452. [PubMed: 15738376]

33. Dawson A, Abel SL, Loving RT, et al. CPAP therapy of obstructive sleep apnea in type 2 diabetics improves glycemic control during sleep. J Clin Sleep Med. Dec 15; 2008 4(6):538–542. [PubMed: 19110882]

34. Lam JC, Lam B, Yao TJ, et al. A randomized controlled trial of nCPAP on insulin sensitivity in obstructive sleep apnoea. Eur Respir J. Jul 16.2009

35. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med. Oct 5; 1999 131(7):485–491. [PubMed: 10507956]

36. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self reported and measured sleep duration: how similar are they? Epidemiology. Nov; 2008 19(6):838–845. [PubMed: 18854708]

Table 1

Demographics of participants

	All participants N=189	SSD N=88	No SSD N=95	P value	Frequent Snoring N=35	No frequent snoring N=154	P value
Age (years)							
Mean ± SD	29.7 ±5.5	30.6±4.9	29.3±5.7	0.1	30.3±6.5	29.6±5.3	0.5
18–24 n (%)	32 (17)	8 (9)	20 (21)	0.08	6 (17)	26 (17)	0.8
25–34 n(%)	126 (67)	65 (74)	60 (63)		22 (63)	104 (67)	
≥35 n (%)	31 (16)	15 (17)	15 (16)		7 (20)	24 (16)	
Ethno-racial status							
White n (%)	117 (62)	47 (53)	68 (72)	0.02	20 (57)	97 (63)	0.01
Black n (%)	28 (15)	13 (15)	11 (11)		11 (31)	17 (11)	
Hispanic n (%)	21 (11)	16 (18)	5 (5)		2 (6)	19 (12)	
Other n (%)	23 (12)	12 (14)	11 (11)		2 (6)	21 (14)	
Pre-pregnancy BMI							
Mean ± SD	24.1 ±5.4	24.8±5.7	23.4±5.0	0.08	25.3±5.1	23.9±5.4	0.2
<18.4 n (%)	10 (5)	3 (3)	7 (7)	0.04	1 (3)	9 (6)	0.3
18.5–24.9 n (%)	119 (63)	49 (56)	67 (71)		19 (54)	100 (65)	
25–29.9 n (%)	34 (18)	21 (24)	11 (12)		7 (20)	27 (17)	
≥30 n (%)	26 (14)	15 (17)	10 (10)		8 (23)	18 (12)	
Insurance							
Public aid n (%)	36 (19)	16 (18)	16 (17)	0.8	8 (23)	28 (18)	0.5
Private n (%)	153 (81)	72 (82)	79 (83)		27 (77)	126 (82)	

Table 2

1-hour OGT and GDM results

	SSD	No SSD	P value	Crude OR(95% CI)	Adjusted OR*(95% CI)	Frequent snoring	No frequent snoring	P value	Crude OR (95% CI)	Adjusted OR** (95% CI)
1-hour OGT mean ±SD	115.7 ± 31.2	104.6 ± 23	0.008	N/A	N/A	118.2 ± 34	107.5 ± 25.4	0.04	N/A	N/A
1-hour OGT ≥130 n/N (%)	27/84 (32.1%)	14/92	0.008	2.6 (1.3,5.5)	2.4 (1.1, 5.3)	11/34 (32.4%)	30/148 (20.3%)	0.1	1.9 (0.8,4.3)	1.9 (0.7,4.7)
GDM n/N (%)	9/88 (10.2%)	1/94 (1.1%)	0.008	10.6 (1.3, 85.5)	11.7 (1.2,114.5)	5/35 (14.3%)	5/135 (3.3%)	0.009	4.9 (1.3,18.1)	6.9 (1.4,33.9)

* Adjusted for age, ethno-racial status, BMI, and frequent snoring

** Adjusted for age, ethno-racial status, BMI, and SSD