Are Symptoms of Obstructive Sleep Apnea During Pregnancy Associated With Autism Spectrum Disorder in Children: A Case-Control Study

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ABSTRACT

Background: Obstructive sleep apnea complicates 10% to 32% or greater of pregnancies, however, reports on long-term effects on the children of pregnancies affected by obstructive sleep apnea are limited.

Objective: We sought to test the hypothesis that the children of pregnant people with symptoms of obstructive sleep apnea during pregnancy have an increased incidence of autism spectrum disorder.

Methods: This was a case-control study comparing the pregnancies of people whose children were later diagnosed with autism spectrum disorder without a known associated genetic condition to those whose children were diagnosed with autism spectrum disorder with a known associated genetic condition.

Results: Of the 51 total parents who were eligible and consented to participate, 4 had a child with autism associated with a known genetic condition, and 47 had a child with autism with no known genetic condition. The prevalence of any snoring (50.0% and 36.2%, respectively) and daytime tiredness (75.0% and 89.4%, respectively) were similar between both groups.

Conclusions: In this study, the prevalence of any snoring and falling asleep while driving during pregnancy was higher in the sampled population than typically reported in pregnant people. While the sample size for this study was small, our preliminary results suggest that parents of children with autism have a high prevalence of sleep-related concerns during their pregnancies, which indicates the need for further investigation – especially for obstructive sleep apnea. Future studies exploring the neurodevelopmental outcomes of children of a cohort of pregnant people with known presence or absence of obstructive sleep apnea during pregnancy is warranted.

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BACKGROUND

Obstructive sleep apnea (OSA) complicates 10% to 32% or greater of pregnancies, with a pooled worldwide prevalence of at least 15% (95% CI, 12%-18%) in a recent meta-analysis.[1–3] Some physiologic changes of pregnancy, namely increased oxygen consumption and decreased functional residual capacity, can lead to low maternal oxygen reserves and result in rapid oxygen desaturation if apnea or hypopnea events occur.[4] OSA is characterized by recurrent partial or complete airway collapse during sleep, which results in varying degrees of oxygen desaturation and microarousals and associated catecholamine release.[5] Recurrent desaturations cause increased release of reactive oxygen species, which act together to increase inflammation, dysregulate endothelial function, and increase blood pressure.[5] However, despite these known pathophysiologic pathways, OSA that both predates and which is incident during pregnancy remains underdiagnosed.[2,5] Two pregnancy-specific screening tools have been generated that boast high sensitivity and specificity for predicting OSA in pregnancy.[2,6] Both incorporate a combination of body mass index (BMI), maternal age, and snoring into their model, with less emphasis on sleepiness or other symptoms that are generally more common and less specific for sleep apnea during pregnancy.[1,2,6]

When OSA is diagnosed, systematic reviews and meta-analyses have demonstrated an increased risk of adverse pregnancy outcomes, namely hypertensive disorders of pregnancy,[4,5,7] gestational

diabetes, fetal growth restriction, and preterm birth.[5,7,8] However, reports on long-term effects on the children of pregnancies affected by OSA are more limited.

Emerging animal models suggest that gestational intermittent hypoxia (aiming to simulate recurrent desaturations typical of OSA) is associated with neuroinflammation in offspring.[9] Neuroinflammation and microglia or astrocyte dysfunction are associated with neurodevelopmental disorders, including autism spectrum disorder (ASD).[10,11]

In humans, one small prospective study and one population-based study have queried the association between OSA and aberrant behavioral development in offspring. The population-based study found that maternal OSA during pregnancy was associated with developmental vulnerability in male (but not female) children, defined as scoring below the 10th percentile in one or more assessment domains, including social competence, language and cognitive skills, communicative skills, and general knowledge.[12] A small prospective study showed that participants with confirmed OSA during pregnancy who lacked any other known pregnancy complications were statistically more likely (2.5-fold increased risk) to have children with low scores on social assessments at 1 year of age compared to participants with normal gestational sleep evaluations.[13] The mechanism for these neurobehavioral changes in the children resulting from these pregnancies may be related to OSA-induced sleep microfragmentation and macrofragmentation, which could manifest as daytime tiredness or fatigue during pregnancy. It also may be that cumulative hypoxia due to recurrent desaturations with OSA during pregnancy leads to neuroinflammation as the animal studies suggest. In general, ASD is conceptualized to be multifactorial in nature with both genetic and environmental contributing factors, and the role of OSA during pregnancy is one possible environmental factor worth exploring.

The purpose of this project was to evaluate whether the children of pregnant people at risk of OSA during pregnancy have an increased incidence of ASD. A case-control design was chosen, with cases representing children with ASD resulting from no known or identified genetic etiology and controls representing children with ASD associated with known genetic conditions to minimize recall bias. Our hypothesis was that more of the birthing parents of children with ASD without a known genetic etiology would be at risk of OSA during pregnancy than the birthing parents of children with ASD with a known genetic etiology.

METHODS

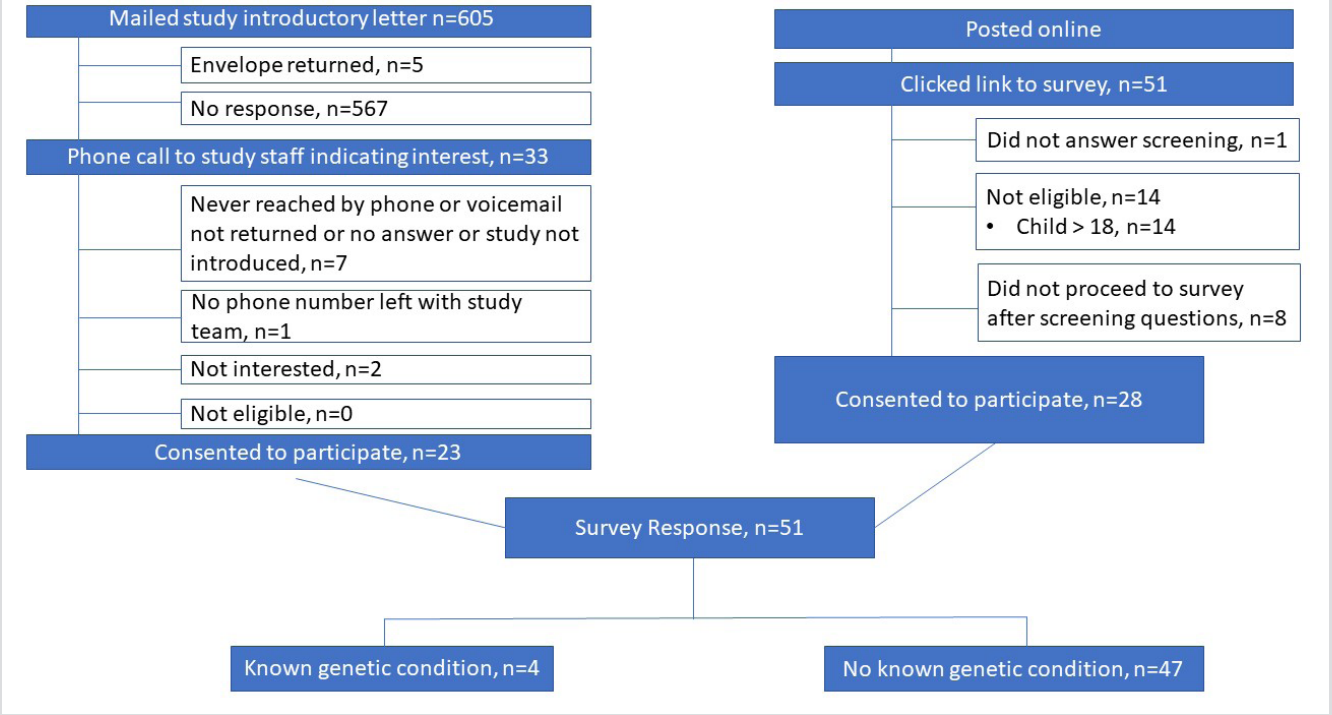
This study was reviewed by the Institutional Review Board (IRB) at the University of Wisconsin School of Medicine and Public Health (IRB 2021-1042). This survey study was initially designed to be administered via telephone to the parent who had been pregnant with a child who was later diagnosed with ASD. This survey was later adapted to an online format to allow wider participation.

Initially, people whose children were enrolled in a research registry at the Waisman Center at the University of Wisconsin-Madison with a diagnosis of ASD were mailed an introductory letter and printed informed consent information. Those who were interested were instructed to call the telephone number for study participation. If the study staff were not directly reached, a voicemail message requested that those interested in participating in the study record their name, contact phone number, and preferred time for a return call. A researcher (JN) then called the parent at the parent's preferred time and conducted the survey via telephone. The survey consisted of screening questions to confirm eligibility and questions about snoring, tiredness, and suspicion for sleep apnea during pregnancy. We also asked demographic questions about age, BMI, and the presence of comorbidities. The symptom of snoring was queried because it is a predictor of OSA and is included in most screening questionnaires for OSA used during pregnancy[6,14] and in the general population.[15–18] Snoring also has been used as a pseudo-surrogate for OSA in some studies.[19–22] Similarly, BMI, or obesity, is a predictor of OSA and is included in screening questionnaires.[6,15–18] Tiredness also is included in some, but not all, questionnaires used to screen for OSA.[15–17] While we could not retrospectively test those surveyed for the presence of OSA during their pregnancy since their pregnancies were all completed, these clinical measures and symptoms were queried because they are well-established risk factors for OSA.

When the response rate was low, the IRB protocol was amended to allow us to reach out to all parents of children who were patients of the Autism and Developmental Disabilities Clinic at the Waisman Center at the University of Wisconsin-Madison with a diagnosis of ASD. These parents were invited to participate via a mailed introductory letter and printed informed consent document. A list of children under 18 was generated by the clinic, and the letter was mailed to the listed parent of these children. Parents were specifically invited to participate as this project sought to interview the individual who was pregnant with the child who was later diagnosed with ASD. Those interested in participating were invited to call the study staff at a listed number. Their calls were returned by a researcher (JN) in same manner as noted above.

Due to low recruitment, the study protocol was modified to allow recruitment of survey responses from online communities for parents of children with ASD. Accordingly, the survey was modified slightly to allow it to be completed online via a survey format rather than as a study form, and a link to the survey was posted on social media sites inviting the parents of children with ASD to participate. Specifically, an invitation to participate and a survey link were posted on the UW Obstetrics and Gynecology's Twitter and Facebook page, Waisman Center Facebook page, Physician Mom Group Facebook page, Madison Mom Facebook page, Reddit, and a University of Wisconsin research email listserv.

Figure. Recruitment and Participation



Study fliers with a quick-response (QR) code link to the survey also were posted at the Waisman Center's clinic and the University of Wisconsin's pediatric neurology clinic. The online survey allowed participants to complete the entire survey online. Both the telephone survey and online survey were hosted on the University of Wisconsin Institute for Clinical and Translational Research's REDCap server.[23,24] REDCap (Research Electronic Data Capture) is a secure, web-based software platform designed to support data capture for research studies that also hosts and allows electronic distribution of online surveys.[23,24]

The population surveyed were biological parents who had been pregnant with a child who was later diagnosed with ASD. Cases were selected as those whose children did not have an identified genetic condition associated with ASD. Controls were those whose children had an identified genetic condition associated with ASD. This population was selected as controls because their lived experiences since birth would overall be as similar as possible to the cases with the intention of minimizing recall bias. Specific genetic etiologies considered to represent genetic conditions associated with ASD or similar neurobehavioral changes included the following: MeCP2 genetic variant/Rett syndrome; LIS1, GRIN2B, and COL4A mutations; fragile X syndrome; and other microarray findings consistent with known genetic conditions associated with ASD based upon review by a trained pediatric neurologist and developmental pediatrician (KS and KK). The presence or absence of a genetic condition was assessed by the clinical team rather than as part of participation in this study, and genetic testing was not offered. Other inclusion criteria included parental age of 18 or greater at the time of participation in the survey. Exclusion criteria included neonatal history of intraventricular hemorrhage, hypoxic ischemic encephalopathy, neonatal sepsis, known perinatal stroke, or death of the child.

Table 1. Characteristics of the Pregnant Parent at the Time of the Index Pregnancy

	ASD Not Related to Known Genetic Cause N = 47	ASD Related to Known Genetic Cause N = 4	*P* value[a]
Age, mean (SD)	30.6 (6.3)	29.0 (7.0)	0.621
BMI, median (IQR)			
Prepregnancy	26.7 (22.1 – 33.6)	28.8 (26.4 – 34.4)	0.323
During pregnancy	30.8 (27.0 – 38.1)	36.4 (33.2 – 41.5)	0.153
Chronic hypertension, n (%)	1 (2.2)[b]	0 (0)	1.00
Diabetes, n (%)	1 (2.1)	1 (25.0)	0.221

Abbreviation: ASD, autism spectrum disorder; BMI, body mass index.

[a] *t* test for age, Wilcoxon rank sum test for BMI, Fisher exact test for chronic hypertension and diabetes.

[b] One person did not answer this question.

For statistical analysis, Fisher exact test, *t* test, and Wilcoxon rank-sum were performed where appropriate. Given the paucity of data and unknown effect size, an a priori sample size was not performed. All statistical analyses were performed using Stata

(16.1, StataCorp LLC, College Station, Texas).

RESULTS

From November 11, 2021 through November 29, 2021, 60 letters of introduction were mailed to registry participants introducing this study. From March 22, 2022 through April 7, 2022, 545 letters of introduction were sent to parents of clinic patients. Of 605 letters of introduction mailed to potential participants, 5 envelopes were returned due to changes of address, and we received 33 telephone calls indicating interest. One person did not leave a telephone number on the voicemail, thus could not be reached. Seven interested people were never reached by telephone. Following a verbal description of the study, 2 people were not interested in participating and 23 consented to participate. On May 24, 2022, the online survey was approved for use, and social media posts were approved on June 14, 2022. The survey was then made available via online links and posted on social media, shared via email correspondence, and available by scanning a QR code on recruitment posters. Fifty-one potential participants clicked on the survey link. Following a written introduction, 1 person did not respond to the screening questions, 14 people were not eligible due to their child being over 18 years old, 8 did not proceed to the survey itself after reviewing the screening questions, and 28 consented to participate. No respondents were excluded due to not having been pregnant with their child themselves or due to the presence of hypoxic ischemic encephalopathy or other exclusion criteria.

Of the 51 completed survey responses, 4 were from the parent of a child whose diagnosis was associated with a known genetic condition and 47 were from the parent of a child whose diagnosis was not known to be associated with a known genetic condition (Figure).

As shown in Table 1, demographic characteristics of the parents who had been pregnant with children with ASD with or without associated genetic conditions were similar. Symptoms of OSA, such as snoring, daytime tiredness, and falling asleep while driving, also were similar (Table 2). Snoring was reported by 36.2% of parents whose child with autism did not have a known genetic cause and 50.0% of parents whose child with autism did have a known genetic cause ($P = 1.00$). None of the parents whose child with autism was associated with a known genetic condition fell asleep while driving, compared to 6.4% of those whose child with autism was not associated with a known genetic condition ($P = 1.00$). One person whose child with autism did not have a known genetic condition had a confirmed diagnosis of OSA during their pregnancy; in her case, her diagnosis was made prior to pregnancy. The number of participants and the number of participants whose children have ASD with an associated genetic condition were low, precluding more detailed statistical analysis.

Table 2. Sleep Characteristics at the Time of the Index Pregnancy

	ASD Not Related to Known Genetic Cause N = 47	ASD Related to Known Genetic Cause N = 4	*P* value[a]
Any snoring during pregnancy, n (%)[b]	17 (36.2)	2 (50.0)	1.00
Loud snoring	7 (41.2)	1 (50.0)	1.00
Snoring 3 or more times per week	9 (52.9)	1 (50.0)	1.00
Cessation of breathing during sleep, n (%)[c]	1 (2.8)	0 (0)	1.00
Daytime tiredness, n (%)[d]	42 (89.4)	3 (75.0)	0.404
Fell asleep while driving, n (%)[e]	3 (6.4)	0 (0.0)	1.00
Ever diagnosed with sleep apnea, n (%)[e]	9 (19.1)	1 (25.0)	1.00
Diagnosed with sleep apnea before pregnancy[f]	1 (16.7)	0 (0)	1.00
Diagnosed with sleep apnea during pregnancy	0 (0)	0 (0)	N/A
Parent-suspected sleep apnea, n (%)[g]	4 (8.5)	0 (0)	0.152

Abbreviation: ASD, autism spectrum disorder.

[a]Fisher exact test used for all *P* values.

[b]Nine people in the nongenetic group did not recall; percent with loud snoring and frequent snoring only reported for those who snored at all. Three in the nongenetic group did not know whether they snored loudly. Five in the nongenetic group and 1 in the genetic group did not know whether they snored frequently.

[c]Two people in the nongenetic group did not recall and 11 did not answer. Two in the genetic group did not answer.

[d]Two people in the nongenetic group did not recall or did not know.

[e]One person in the nongenetic group did not recall.

[f]Three in the nongenetic group did not respond.

[g]Twelve people in the nongenetic group did not recall, 3 people in the genetic group did not recall.

DISCUSSION

The number of participants in this survey study was low, which precluded meaningful statistical analysis of this case-control study as intended. In this small sample, we found that the prevalence of the exposure of snoring and tiredness were similar between the birthing parent of children with ASD with a known genetic etiology and those without a known genetic etiology.

One unexpected finding was that the prevalence of any snoring during pregnancy in our population was overall higher than expected at 36.2% to 50%. Snoring is estimated to affect 1.2% to 35% of pregnancies.[19,25–27] While our overall numbers are small, the prevalence of snoring is higher than in reports in the general population and also higher than in pregnant people later diagnosed with OSA.[19,25-27] While snoring is not equivalent to a diagnosis of OSA, it is a clinical finding indicative of increased upper airway resistance and included in screening tools used for pregnant[2,6] and nonpregnant people.[15,16,18] The high prevalence of snoring is an important finding that merits future validation in larger populations and, ideally, corroboration with objective testing for OSA during pregnancy.

In this sample, we also found that 6.4% of the parents whose child with autism was not associated with a known genetic condition fell asleep while driving during their pregnancy. This is a dangerous occurrence, and the prevalence in this small sample is higher than reported in the general population. While the general prevalence of falling asleep while driving during pregnancy is not known, 4% of people in the US in general report having fallen asleep while driving in the prior 30 days,[28] and 4.26% of one pregnant population reported falling asleep while driving.[29] However, the sample size in our study was small; thus, this finding may represent random variation.

Our study has many limitations. First, analysis was limited by our sample size, which led to this analysis being underpowered. We attempted to increase recruitment by reaching out to a registry in addition to our local patients. We also transitioned the survey to an online format, posted links on social media, shared via an email listserv of individuals who may be interested in participating in research, and posted signs locally at the pediatric neurology clinic. A second limitation was that only one of the participants had a confirmed diagnosis of OSA during their pregnancy, which limits our ability to discern any impact of diagnosed OSA. Accordingly, and because we expected this based upon the low prevalence of screening and diagnostic testing for OSA during pregnancy, we used surrogates for OSA, such as snoring and tiredness, which can be indicators of OSA but do not constitute diagnostic criteria alone. Third, our retrospective design is limited by recall and susceptible to recall bias. Participants in this survey knew that the survey was evaluating prenatal sleep-related exposures that may relate to autism in the offspring. This may have affected who chose to participate at all and the responses of those who did participate.

This study raises an interesting finding regarding the higher-than-expected prevalence of snoring. While it is likely related to participation bias, it builds the case for further investigation into potential relationships between OSA during pregnancy and ASD in the children of these pregnancies. In particular, it would be helpful to query cohorts of pregnant people with or without known diagnoses of OSA during pregnancy for neurobehavioral outcomes in their offspring. As above, confirmation of OSA diagnosis through objective testing could also bring considerable value, by allowing phenotyping and endotyping of gestational pathophysiology with possible contributions to offspring ASD and point to downstream mechanistic pathways of interest for translational diagnostic and therapeutic studies. A coordinated effort to screen, test, and treat pregnant people for OSA, particularly if risk factors such as obesity and hypertension exist, may aid these efforts.

CONCLUSIONS

While the sample size for this study was small, our preliminary results suggest that parents of children with autism have a high prevalence of sleep-related concerns during their pregnancies, which indicates the need for further investigation – especially for OSA. Future studies exploring the neurodevelopmental outcomes of children of a cohort of pregnant people with known presence or absence of OSA during pregnancy is warranted.

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