Sleep Apnea During Pregnancy Is Not Good for Mother or Baby

Boston (March 30, 2015)—Sleep apnea, a disorder in which breathing repeatedly stops during sleep, is a potentially serious condition because it deprives the body of oxygen. It becomes an even more serious condition in pregnant women—who can be more prone to it—because the oxygen deprivation may affect the baby. Researchers at the University of Western Ontario in Canada observed that female rats that were regularly deprived of air during their pregnancy had pups that could not handle glucose as well, making their pups more at risk for metabolic disease as adults.

The team, led by John Ciriello, exposed pregnant rats to chronic intermittent hypoxia—bouts of no oxygen—during their pregnancy and observed that the offspring had higher levels of proteins that encourage the liver to release, and not store, glucose. According to the researchers, the data suggest that reoccurring oxygen deprivation, as in sleep apnea, during pregnancy causes long-term changes in the offspring’s liver’s ability to maintain blood glucose level.

The team has been able to follow the effects into adulthood. At 12 weeks old, “the fasted offspring of mothers exposed to chronic intermittent hypoxia were hyperglycemic and hyperinsulinemic,” says Ciriello. Further tests showed that “adult offspring of mothers exposed to chronic intermittent hypoxia exhibit poor glucose tolerance,” he continues. “Our findings indicate that these adult offspring have a decreased sensitivity to insulin but have not developed a complete resistance to its signaling effects at this age.”

The group’s recent findings are consistent with a separate study that reported that “rodents overexpressing 11β-HSD1 (11β-hydroxysteroid dehydrogenase type I)—seen in our studies—develop obesity, have elevated circulating corticosteroid levels—also seen in our studies—and develop type 2 diabetes,” Ciriello says.

“This further supports our suggestion that the adult offspring of mothers exposed to chronic intermittent hypoxia during gestation are at a higher risk for developing some aspects of the metabolic syndrome, including type 2 diabetes,” says Ciriello.

Full Abstract
Chronic intermittent hypoxia (CIH) is the underlying pathophysiological condition seen in individuals with sleep apnea. Exposure of pregnant females to CIH results in offspring that are hyperglycemic, hyperinsulinemic and have impaired early-stage glucose tolerance. However, the underlying mechanisms for these changes remain elusive. This study was done to determine changes in glucoregulatory function of the liver in offspring as a result of CIH exposure during gestational development. Female Sprague-Dawley rats were mated and exposed daily to CIH from gestational day one to day 20. Offspring were sacrificed at postnatal day one or six weeks of age to determine immediate and long-term changes in liver function. By six weeks of age, male CIH offspring had higher plasma corticosterone concomitant with higher hepatic protein expression of glucocorticoid receptor (GR), and this was associated with lower hepatic Liver X Receptor (LXR) expression, the nuclear receptor involved in suppressing glucose-6-phosphatase (G6Pase) and 11β-hydroxysteroid dehydrogenase type I (11β-HSD1) expression. Analysis of both GR and LXR-direct target genes in gluconeogenesis showed that
hepatic G6Pase and 11b-HSD1 were higher in the CIH offspring, while no changes in phosphoenolpyruvate carboxykinase protein levels were observed. These data suggest that gestational exposure to CIH results in long-term alteration of glucose homeostasis via increased glucocorticoid signaling and augmented expression of gluconeogenic enzymes due in part to LXR signaling in the liver.

NOTE TO JOURNALISTS: To schedule an interview with a member of the research team, please contact Stacy Brooks at sbrooks@the-aps.org or (240) 432-9697.

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