Prenatal exposure to BPA affects fat tissues in sheep

June 17 2013

New research suggests that fetal exposure to the common environmental chemical bisphenol A, or BPA, causes increased inflammation in fat tissues after birth, which can lead to obesity and metabolic syndrome. Results of the animal study were presented Monday at The Endocrine Society's 95th Annual Meeting in San Francisco.

Found in plastic water bottles, older baby bottles and many other consumer products, BPA is a known hormone disrupter with estrogen-like properties. Prior research has linked BPA in both animals and humans to obesity and the metabolic syndrome, which is a cluster of metabolic risk factors that increase the chance of later developing diabetes, heart disease and stroke.

"This research is the first study to show that prenatal exposure to BPA increases postnatal fat tissue inflammation, a condition that underlies the onset of metabolic diseases such as obesity, diabetes and cardiovascular disease," said the study's lead author, Almudena Veiga-Lopez, DVM, PhD, a research investigator at the University of Michigan, Ann Arbor.

She said the study, which examines the effects of BPA on sheep, improves the understanding of how prenatal BPA exposure regulates the inflammatory response in offspring in the tissues that are relevant to development of metabolic disease. The study was conducted in the laboratory of Vasantha Padmanabhan, MS, PhD, Professor at the University of Michigan, Ann Arbor, with funding from the National Institutes of Health's National Institutes of Environmental Health

Sciences. Veiga-Lopez said sheep have similar body fat to that in humans, including visceral (deep belly) fat and subcutaneous fat, which is directly below the skin.

The researchers fed two groups of pregnant sheep corn oil, either with nothing added to it or with added BPA at a dose needed to achieve BPA levels similar to those seen in human cord blood in the umbilical cord blood of the sheep offspring. Of the female offspring from the sheep, half from each group were overfed at approximately 6 weeks of age. All female offspring then were divided into four groups of nine to 12 animals each: (1) non-BPA-exposed controls that received a normal diet, (2) BPA-exposed offspring that received a normal diet, (3) overfed, obese controls and (4) overfed, obese BPA-exposed offspring.

At 15 months of age, sheep underwent a glucose tolerance test, to measure their insulin and blood sugar levels. Seven months later, the researchers collected samples of the animals' visceral and subcutaneous fat tissues to evaluate levels of two biological markers of inflammation. These biomarkers were CD68, a marker for inflammatory cells, and adiponectin, a molecule with a known role in the development of metabolic syndrome. When the adiponectin level decreases or CD68 expression increases, inflammation is worse, according to Veiga-Lopez.

Adiponectin was decreased and CD68 expression was raised in the visceral fat of both obese groups, and CD68 expression also was raised in the subcutaneous fat in normal weight, BPA-exposed female offspring, Veiga-Lopez reported. She said these results suggest that "prenatal BPA exposure and postnatal diet may interact to modulate inflammatory mechanisms in fat deposits."

Both obese groups had hyperinsulinemia, or high insulin levels, a precursor to insulin resistance, which is a prediabetic state, Veiga-Lopez reported. However, she said prenatal exposure to BPA did not lead to

insulin resistance in sheep, as was true in a previous mouse study. She speculated that the hyperinsulinemia in obese offspring stems from changes that occurred in the two inflammatory markers in the visceral fat deposit.

Provided by The Endocrine Society