Endocrine disruptors cause fatty liver

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Exposure to low doses of hormone-disrupting chemicals early in life can alter gene expression in the liver as well as liver function, increasing the susceptibility to obesity and other metabolic diseases in adulthood, a new study finds. Results of the animal study will be presented Friday at the Endocrine Society's 97th annual meeting in San Diego.

Brief exposure in infancy to several industrial chemicals that are common in the human environment, particularly bisphenol A (BPA), caused fatty liver disease in adulthood, the researchers found in rats.

"Even a short exposure to these endocrine disruptors at the wrong time of development has a lifelong effect on the individual," said the study's senior investigator, Cheryl Lyn Walker, PhD, director of the Texas A&M University Health Science Center Institute of Biosciences and Technology, Houston.

Because the changes occur at the molecular level, they are not evident until later in life, she said.

Scientists have suspected that exposure early in life to BPA and certain other chemicals that disrupt the action of hormones, called endocrine disruptors, may promote obesity in adulthood, but the exact cause is unknown. In research funded by the National Institute of Environmental Health Sciences, Walker and her colleagues focused on the liver, which plays a central role in fat metabolism and obesity.

The researchers gave groups of newborn rats low doses of one of four

different endocrine disruptors during a critical period of liver development: the three days immediately after birth. They then examined liver tissue from these chemically exposed animals either immediately after exposure or 70 days later, when the rats were adults. They compared liver samples to those obtained from nonexposed control rats.

BPA and another endocrine disruptor, tributyltin—an additive in paint and textiles—caused liver damage consistent with human nonalcoholic hepatic steatosis, or fatty liver, the investigators reported.

Analysis of liver gene expression patterns of the exposed rats found that the endocrine disruptors induced developmental reprogramming of the animals' epigenomes. In both rodents and humans, the epigenome programs the genome, our complete set of DNA.

Walker said they suspect that "these chemical exposures interfere with the epigenetic 'programmers' to do their job. It's like a glitch on your computer that causes a software program to get installed incorrectly."

This reprogramming of the liver could potentially drive obesity, she explained. Unlike genetic defects, however, epigenetic programming can be reversed.

"It may be possible to reset the malprogramming, or 'malware', to reduce the risk of obesity and associated diseases," Walker said.

With additional research and knowledge, she said, it may also be possible one day to use these epigenetic changes as markers, which can tell that a child is at risk of obesity and other diseases.

Provided by The Endocrine Society