Research results point to dangers of endocrine-disrupting chemicals

Several researchers shared their findings in studies reviewing the effects of various endocrine-disrupting chemicals, including bisphenol A (BPA) and triclocarban during a morning session yesterday. Studies indicated a role for BPA in increased risk of prostate cancer, increased incidence of cryptorchidism and increased inflammation in fat tissues after birth, which can lead to obesity and metabolic syndrome.

Investigators at the University of Illinois at Chicago have determined that exposure to low doses of BPA are linked to an increased risk of prostate cancer in human stem cells.

The study used human prostate stem cells from organ donors to grow prostate tissue in mice. To simulate human BPA exposure, the investigators fed BPA at levels found in humans to the study mice for the first two weeks of the prostate tissue formation.

Early BPA exposure increased risk of both prostate cancer and prostate epithelial neoplasia (PIN) to 33 to 45 percent of tissue exposed to BPA compared with 12 percent in the non- BPA exposed tissue, according to lead study author Gail S. Prins, PhD, Professor of Physiology and Urology at UI-Chicago.

“These results suggest that stem cells are direct BPA targets which may explain the long-lasting effects of this chemical throughout the body,” she said. “They provide the first direct in vivo evidence that developmental exposure to environmentally relevant levels of BPA increases human prostate cancer risk.”

BPA and Cryptorchidism

A study performed in France links fetal exposure to BPA to defects of the testicular hormone insulin-like peptide 3 in newborn boys with undescended testicles.

Patrick Fenichel, MD, PhD, Professor and head of reproductive endocrinology at the University Hospital of Nice in France, said that while their study did not provide definitive evidence of a solely environmental cause of cryptorchidism, it does suggest for the first time a link that could contribute to one co-factor. Investigators studied 180 boys born after 34 weeks gestation, 52 of which were born with one or two undescended testicles and 128 of which did not have the birth defect.

They measured both newborn groups’ levels of BPA, insulin-like peptide 3 (INSL3) and testosterone. The testosterone levels didn’t differ between the groups, but infants with cryptorchidism had significantly lower levels of INSL3. In all 180 infants, the BPA levels inversely correlated with the level of INSL3; the higher the BPA level, the lower the hormone level.

“You have an indirect negative relation between the two and you can suggest that bisphenol A, which has an estrogen effect, may repress the expression of INSL3, and induce with or without other factors cryptorchidism,” said Dr. Fenichel.

BPA and Inflammation of Fat Tissue

An animal study suggests that fetal exposure to BPA causes increased inflammation in fat tissues after birth, which can lead to obesity and metabolic syndrome.

Almudena Veiga-Lopez, DVM, PhD, a research investigator at the University of Michigan, Ann Arbor, said their study looked at two groups of pregnant sheep, one fed sheep corn oil with no additives and one with BPA added in a dose to achieve an umbilical cord level in the sheep’s offspring similar to those seen in human cord blood. Sheep were selected for the study because body fat is similar to that in humans.

The female offspring were then divided into four groups: non-BPA exposed fed a normal diet; BPA exposed fed a normal diet; overfed obese control; overfed obese, BPA exposed. Investigators then measured the sheep offsprings’ insulin and blood sugar levels at 15 months and evaluated two biomarkers (CD68 and adiponectin) from the offsprings’ visceral and subcutaneous fat tissues at 22 months.

The research showed adiponectin was decreased and CD68 expression was raised in the visceral fat of both obese groups, and CD68 expression also was raised in subcutaneous fat in the normal weight, BPA-exposed groups, suggesting that “prenatal BPA exposure and postnatal diet may interact to modulate inflammatory mechanisms in fat deposits.”

Maternal Exposure to Triclocarban

Research also was presented that showed that a mother’s exposure to triclocarban while nursing shortens the life of her female offspring in rats. Pregnant rats were fed rat chow supplemented with no triclocarban or 0.2 or 0.5 percent triclocarban. Doses found in the maternal rats’ blood corresponded to that in humans after a 15-minute full body shower using bar soap with 0.6 percent triclocarban.

Rebekah Kennedy, a graduate student in the Department of Public Health at the University of Tennessee, Knoxville, said investigators cross matched the litters so that each mother nursed two of her own pups and two pups from each of the other two groups. While body weight did not differ at birth among the groups, by day 3, pups nursed by the control rats were heavier than the triclocarban exposed group.

Additionally, none of the rats nursed by rats that received the highest triclocarban dose survived past day 6, and only 13 percent of those nursed by rats receiving the lower dose survived by weaning.

“Our data suggests that the critical exposure window affecting rat pup survival is related to lactation, as all pups raised by control rats survived regardless of triclocarban exposure status during gestation,” she said.

Restrict feeding by time or amount consumed

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