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European Congress on Obesity

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New evidence links hormone-mimic chemicals to obesity

Geneva, Switzerland: Exposure during development either in the womb or during infancy to chemicals used to make products such as baby bottles, the lining of food tins and some plastic food wraps and containers, may contribute to the development of obesity, according to new research presented at the European Congress on Obesity.

While eating too much and exercising too little are still considered the major cause of obesity, scientists have recently started investigating whether chemicals known as endocrine disruptors, which mimic or alter the effects of hormones in the body, could also play a role in making people fat.

In a special session at the European Congress on Obesity supported by the National Institute of Environmental Health Sciences (NIEHS) in the USA and the Swiss National Science Foundation, experts reported new evidence that mice exposed to endocrine disrupting chemicals during pregnancy – at levels either comparable to or approaching those that humans are exposed to - produced offspring that became fat as adults and had altered gene and metabolic functions involved in regulating weight.

“The findings from these studies suggest that susceptibility to obesity is developed in the womb or early in life and that exposure to a variety of common household chemicals can, probably along with foetal nutrition, play a role in increasing that susceptibility,” said Jerry Heindel of the US National Institute of Environmental Health Sciences, an expert in the field, who was unconnected with the studies highlighted at the conference.

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“This information has the potential to change how people view and treat obesity. If these findings are proven to be true in humans, then the focus must change from losing weight as adults to prevention of weight gain during development, through reducing the exposure to such substances.”

One of the chemicals under scrutiny is Bisphenol A, or BPA, an ingredient in polycarbonate plastics. Past research has found evidence that it leaches from plastic food containers and bottles, from plastic wrap and from the resin that lines food cans. It has been found in a large percentage of people examined in developed countries. Besides urine and blood, it has been noted in amniotic fluid, placenta, umbilical cord blood and breast milk. Laboratory experiments have found that BPA can increase the production of fat cells.

In one study, Professor Beverly Rubin, a neuroendocrinologist at Tufts University in the United States, found that female mice whose mothers were exposed to BPA from early pregnancy through day 16 of lactation showed increased weight in adulthood. Food intake and activity levels were no different between the mice who became fat and those that did not. The study also found disturbance in insulin sensitivity and glucose balance and in the weight regulating hormone leptin.

“This study indicates that developmental exposure to this chemical prior to and just after birth can exert a long lasting influence on body weight regulation,” Professor Rubin said.

In another experiment outlined at the conference, Suzanne Fenton, a research biologist at the US Environmental Protection Agency, found that when the chemical perfluorooctanoic acid (PFOA) - a greaseproofing agent used in scores of products from microwave popcorn bags to pizza box liners and other food containers - was given to pregnant mice, their offspring were unusually small at birth then became overweight as adults. In contrast, the mice whose mothers were not exposed to the chemical had a normal growth pattern, as did mice that were exposed as adults only. PFOA is detected in the blood of people around the globe, but is detected at up to 100 times higher concentrations in people living in industrially polluted areas.

“Our mouse study involved the lowest doses we have investigated to date and it shows the weight effect does occur at fairly low doses. In fact, similar to what has been seen with other compounds, we only see this effect when the dose is low, which indicates that perhaps different doses trigger health problems in the body by various mechanisms or that the high doses cause more serious problems, and potentially mask the abnormal weight gain,” Fenton said. “What we need to do next is find the mechanism of action for this chemical to determine whether there is a corresponding health risk for the human population.”

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A third study presented at the conference, outlined by Dr Bruce Blumberg, a developmental biologist at the University of California at Irvine, found that when scientists treated pregnant mice with the chemical tributyltin at a dose comparable to that found in humans, a genetic programme was switched on in the offspring, that programmes them to become fat later in life. Tributyltin is used in boat paint, in plastic food wrap and as a fungicide on crops and has been found to leach into food.

“Developmental exposure is probably more serious than adult exposure because the data with other such exposures suggest that the pro-obesity reprogramming is irreversible, which means you will spend your life fighting weight gain,” said Blumberg. “Whether the effect on animals is the same on humans will depend on the levels in the human population. The data we do have suggest that, at least for this chemical, it is in the range that we see effects, but more research on that is needed to determine the magnitude of the risk.”

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Session: Role of endocrine disruptors from the environment in the aetiology of obesity and diabetes

Time: Wednesday 1000 CET – 1700 CET

Location: Room F

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