Exposure to BPA substitute, BPS, multiplies breast cancer cells

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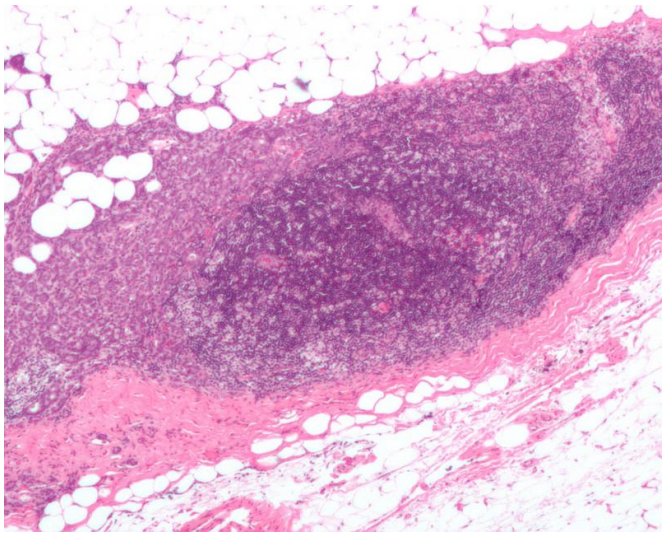

Micrograph showing a lymph node invaded by ductal breast carcinoma, with extension of the tumour beyond the lymph node. Credit: Nephron/Wikipedia

Bisphenol S (BPS), a substitute for the chemical bisphenol A (BPA) in the plastic industry, shows the potential for increasing the aggressiveness of breast cancer through its behavior as an endocrine-disrupting chemical, a new study finds. The results, which tested BPS in human breast cancer cells, will be presented Saturday at ENDO 2017, the Endocrine Society's 99th annual meeting in Orlando, Fla.

BPS is found in polycarbonate hard plastics, currency bills and thermal paper receipts as well as many products touted to be free of BPA, a known endocrine-disrupting chemical suspected of having multiple possible health risks.

"Despite hopes for a safer alternative to BPA, studies have shown BPS to exhibit similar estrogen-mimicking behavior to BPA," said the study's principal investigator, Sumi Dinda, Ph.D., associate professor at Oakland University School of Health Sciences, Rochester, Mich.

Their study confirmed that BPS acts like estrogen in breast cancer cells, Dinda said, adding, "So far, BPS seems to be a potent endocrine disruptor."

He and his colleagues studied the effects of BPS on estrogen receptor-alpha and the BRCA1 gene. Most breast cancers are estrogen receptor positive, and, according to the National Cancer Institute, 55 to 65 percent of women who inherit a harmful mutation in the BRCA1 gene will develop breast cancer.

Using two commercially available breast cancer cell lines obtained from women with estrogen-receptor-positive breast cancer, the research team exposed the cancer cells to varying strengths of BPS or to an inactive substance as a control.

The investigators also treated the breast cancer cells with estradiol (estrogen) and found that BPS acted like estrogen in multiplying breast cancer cells, Dinda said. Compared with the control, BPS heightened the protein expression in estrogen receptor and BRCA1 after 24 hours, as did estrogen. After a six-day treatment with BPS, the breast cancer cells in both cell lines reportedly increased in number by 12 percent at the lowest dose (4 micromolars) and by 60 percent at 8 micromolars.

The research team then blocked the BPS-induced proliferation of breast cancer cells by treating the cells with anti-estrogen drugs, which are used to block estrogen's action onto estrogen binding proteins (estrogen receptors) in breast cancer cells.

Dinda said their findings suggest that BPS may cause breast cancer to become more aggressive. Although further study of BPS in breast cancer cells is needed for confirmation, he suggested that "if a woman has a mutated BRAC1 gene and uses

products containing BPS, her risk for developing breast cancer may increase further."

Co-author Katie Aleck, a research assistant at Oakland University, will present the study results at the meeting.

Provided by The Endocrine Society