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Editorial

The Genome-Food Interface in Fetal Period Is Determinant for Adulthood Susceptibility to Breast and Other Cancers

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Recent studies revealed that some risk factors in adult chronic diseases like cancer, obesity and related disorders are associated with the maternal nutritional condition (Crowe and Allison, 2015; Waterland and Jirtle, 2004; Wolff et al., 1998). In mammalians, fetal development is in close relation with maternal nutrition (nutrigenomics), and the genome of the preimplantation mammalian embryo undergoes vast methylation, demethylation and remethylation epichanges that are extremely susceptible to metabolic and environmental factors (Feng et al., 2010). Lipotropes are of methyl group-containing essential nutrients (folate, vitamin B_{12} and methionine) affecting extensively one-carbon metabolism. Prolong impacts of high-dose maternal dietary intake of lipotropes (five folds higher than in the control diet) on the development and progression of mammary tumors in rat offspring during adulthood is well known. Tumor incidence was significantly decreased among the offspring born from mothers fed high-dose lipotropes (Cho et al., 2012). In humans, consumption of soy genistein or some other bioactive food components during infancy reduces later breast cancer risk, although the benefit may be considerably limited if consumed during adulthood. So, food ingredients may be more effective in decreasing of cancer risk in some periods of life span than others. Several dietary exposures can modify fetal and postnatal hormonal environment, including changing concentrations of estrogens, leptin, etc. The changes in hormonal concentration then may induce insistent epigenetic changes by affecting gene promoter CpG island regions or by bringing histone modifications that influencing chromatin transcription (Hilakivi-Clarke, 2007). The xenoestrogen bisphenol A (BPA) exposure caused higher amounts of pro-activation histone H3K4 trimethylation at the transcription initiation stage of the alpha-lactalbumin gene at post natal day 4, concurrently increasing expression of this gene. These results show that fetal BPA exposure triggers changes in the postnatal and adult mammary gland epigenome and changes gene expression modality (Chango and Pogribny, 2015). These events may provide deriving force for development of neoplastic lesions that evident during adulthood (Dhimolea et al., 2014). Therefore, the great concerns developed in recent years about uncontrolled and rapid incidence of cancers worldwide including Iran, may, at least partly, stem from the fetal period in response to fetal environmental nutrients and hormones quantity and quality. So, it would be a vise strategy to shift our cancer preventive measures, instead of postnatal period to fetal life with simple intervention in nutritional aspects of pregnant women.

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