# 23

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**Developmental factors in adult obesity**

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Chemicals used in a wide range of household products have the capacity to disrupt the chemical messengers by which cells communicate and are thus referred to as "endocrine disrupting chemicals". Environmental estrogens bind to nuclear estrogen receptors that regulate tissue differentiation during fetal life, which involves "programming" of gene activity. The typical view of estrogen is that it is associated with a reduction in food intake and body weight in adults. However, fetal exposure to bisphenol A, the estrogenic chemical used to make polycarbonate plastic food and beverage containers as well as the resin lining of metal food and beverage cans, causes an increase in postnatal growth in male and female mice. This occurs at doses of bisphenol A within the range of exposure of human fetuses to this ubiquitous environmental contaminant. In addition to environmental chemicals, placental blood flow is a critical factor in fetal nutrition and growth and is related to postnatal obesity and associated metabolic diseases. Restriction of placental blood flow results in reduced fetal growth, and depending on the postnatal environment, growth restricted babies may exhibit a high velocity of growth and become obese (as predicted by the "thrifty phenotype" hypothesis). These findings suggest a complex interaction between prenatal and postnatal factors in adult obesity. However, the primary focus on obesity in the medical community is on food consumption and energy expenditure, with relatively little attention being paid to the contribution of developmental factors. We have developed a mouse model in which reduced placental blood flow and placental nutrient transport result in intrauterine growth restriction (IUGR), which is followed by a dramatic increase in postnatal growth; the IUGR animals are being compared to siblings with a high rate of fetal growth and a lower rate of postnatal growth. This model is being used to determine whether during critical periods in development, estrogenic chemicals such as bisphenol A interact with fetal nutrition to impact postnatal growth and adult obesity in selected sub-populations of mice. This research is funded by the National Institute of Environmental Health Sciences.