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The obesogen tributyltin.

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Abstract

The obesogen hypothesis postulates the role of environmental chemical pollutants that disrupt homeostatic controls and adaptive mechanisms to promote adipose-dependent weight gain leading to obesity and metabolic syndrome complications. One of the most direct molecular mechanisms for coupling environmental chemical exposures to perturbed physiology invokes pollutants mimicking endogenous endocrine hormones or bioactive dietary signaling metabolites that serve as nuclear receptor ligands. The organotin pollutant tributyltin can exert toxicity through multiple mechanisms but most recently has been shown to bind, activate, and mediate RXR-PPAR γ transcriptional regulation central to lipid metabolism and adipocyte biology. Data in support of long-term obesogenic effects on whole body adipose tissue are also reported. Organotins represent an important model test system for evaluating the impact and epidemiological significance of chemical insults as contributing factors for obesity and human metabolic health.

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- Adipogenesis /drug effects *
- Adipose Tissue /drug effects /metabolism /pathology
- Animals
- Endocrine Disruptors /toxicity *
- Environmental Exposure /adverse effects *
- Environmental Pollutants /toxicity *
- Epigenesis, Genetic /drug effects
- Humans
- Lipid Metabolism /drug effects
- Models, Biological *
- Obesity /chemically induced * /metabolism /pathology
- Tributyltin Compounds /toxicity *

