CHE Collaborative for Health & Environment

Webinar Highlights

Obesogens: A Unifying Theory for the Global Rise in Obesity

Despite varied treatment, mitigation, and prevention efforts, the prevalence and severity of obesity continue to rise. As researchers look for the reasons to explain the global rise in obesity, one thing has become clear — chemicals in our food, packaging, personal care items, and other products are playing a key role.

Evidence is mounting that certain chemicals called obesogens can cause the human (and animal) body to produce more fat than it normally would. In this EDC Strategies Partnership webinar, **Dr. Jerry Heindel** presented a combined model of obesity causation that considers the role of obesogens, linking four general models to create a unifying paradigm. Study coauthors **Dr. Robert Lustig** and **Dr. Barbara Corkey** joined for the Q&A portion of the discussion.

Featured Speaker: Jerry Heindel, PhD, Director of the Environmental Health Sciences Program, Healthy Environment and Endocrine Disruptor Strategies, speaking March 19, 2024.

This fact sheet has been created by CHE based on information presented in an EDC Strategies Partnership webinar. Selected quotes in bold are from the webinar speaker(s). For the full set of resources provided by the webinar presenters, see the webinar page, where you'll also find associated slides and resources.

The Problem

The cause of obesity is unknown, but it is multifactorial. This lack of understanding makes it harder to address the obesity crisis as a public health issue.

Currently, there are four different models that researchers use in studying obesity. The study presented by Dr. Heindel examined the models.

- The first model is the "calories in - calories out" idea: you gain weight if you eat more calories than you expend. This model also says that something in the

nutritional environment has changed over the last 50 or so years, likely related to availability and consumption of ultra-processed food. This has led to increased food consumption and increased weight.

- The second model states that sugar is the culprit – specifically, high glycemic index carbohydrates that stimulate insulin. The increased insulin stimulates fat storage and increases how much we eat.
- The third model — the obesogen model — states that obesogens alter metabolism. Most obesogens disrupt hormonal signaling pathways in various tissues that control energy intake, nutrient handling, and body weight.
- The fourth model states that tissues have metabolism sensors that detect when there is sufficient fuel. These sensors produce small amounts of reactive oxygen species (ROS) that tell the pancreas to release more insulin and fat tissue to make more fat.

The authors proposed an integrated model that puts exposure to obesogens as a key cause of obesity. Obesogens are a unifying and key part of all of these obesity models.

"Each model, we believe, makes essential contributions to the pathogenesis of obesity and we shouldn't be arguing one over the other but try to look at how they work together."

This integrated model can explain developmental programming and effects across the lifespan and generations, an altered metabolic set point, alterations in mitochondrial efficiency, and signals across metabolic tissues that convey a modified nutritional state. Through effects on gene expression and ROS, obesogens alter the function of metabolic tissues such that people are more sensitive to diet-induced weight gain and less sensitive to weight loss.

Recommendations

With an integrated model, we can take a new approach to the obesity pandemic that focuses on prevention:

- We know many obesogens by name. They include chemicals that come from fossil fuels and plastics (e.g., BPA, phthalates, and PFAS), flame retardants and some food additives, and emulsifiers and colorants found in ultra-processed food.
- We know where these exposures come from.
- For many obesogens, we know how they act to increase weight gain.

Decreasing human exposure to obesogens, particularly early in life, will prevent obesity.

Personal changes can help, like filtering drinking water and using organic household products, cleaners, and pesticides. Individuals can also try to limit exposures to plastics, canned food, and ultra-processed food. However, it is unrealistic to expect consumers or clinicians to prevent obesity alone. Instead, policymakers and regulators need to develop public health policies that will regulate and remove these harmful chemicals from products.

To Find Out More

- Watch the March 19, 2024 webinar: Obesogens: A unifying theory for the global rise in obesity
- Read the presentation slides: The Obesogen Model as a Unifying Theory for the Global Rise in Obesity
- Read the blog post, crossposted with *Environmental Health News*: Untangling the causes of obesity
- Read the study: Obesogens: A unifying theory for the global rise in obesity

About the Speaker

Jerrold (Jerry) J. Heindel has a Ph.D. in Biochemistry from the University of Michigan and worked in reproductive biology and toxicology at the University of Texas Medical School at Houston and the University of Mississippi. He moved to the National Institute of Environmental Health Sciences (NIEHS) and headed their Reproductive and Developmental Toxicology group. He then moved to the Division of Extramural Research and Training at NIEHS. He was responsible for developing and administering the NIEHS grants program in endocrine disruption, the developmental basis of diseases and obesity and diabetes for over 25 years. He is now retired from NIEHS and is currently the director of the Environmental Health Sciences Program, Healthy Environment and Endocrine Disruptor Strategies, www.heeds.org.