Understanding the Dynamic Relationship between Genetics, Environment, and Early Childhood Development on Risk of Obesity

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Genomics

We have 22-24,000 active genes, but Pinor Noir has 29,000- more doesn’t mean more functional complexity

Our genetic code is a record of our past (with more than 120,000 silent genes) and a record of our present, what determines our future?

If 99.9% of all human beings have the same genetics, why are there so many differences?
Genetics x Environment

In all living species, environment affects gene expression. The social life of genes can be short or long acting.

- The case of the Killer bees versus European bees
Rapid Infant Growth

Rapid weight gain in the first 3 months of life is associated with obesity and its complications (insulin resistance, increased abdominal circumference, dyslipidemia) in early adulthood (ages 18-24).

Genetics x Environment x Development

Epigenetics: changes in gene expression via post-translational and post-transcriptional modifications.

- You can’t change your genes, but you can change the way your genes behave
- We are permeable, fluid beings rather than stable unitary isolates
- This leads to the recursive, reconstruction of the self

The Social Life of Genes, David Dobbs, Pacific Standard (based on Steve Cole’s work)
Workshop Objectives

1. Identify epigenetic-mediated relationships between exposure to risk factors during sensitive periods of development (gestation through age 3) and subsequent obesity-related health outcomes.

2. Explore the science around periods of plasticity and potential reversibility of obesity risk in the context of early childhood development.

3. Examine the translation of epigenetic science to guide early childhood obesity prevention and intervention to reduce obesity risk.
Understanding how the science fits together

Session 1: The Role of Epigenetics in Pediatric Obesity- A Conceptual Overview
Session 2: Etiology and Causal Inference
Session 3: Opportunities for Intervention and Prevention
Session 4: Real World Application
Session 5: Data Gaps and Future Directions
EARLY ORIGINS OF OBESITY
The Role of Epigenetics and Opportunities for Intervention

MULTI-LEVEL ENVIRONMENT
- ENVIRONMENT
  - Cultural & Economic
- COMMUNITY
- FAMILY
- CHILD
- METABOLISM & PHYSIOLOGY

TOXIC STRESS
Health & Economic Disparities

MATERNAL & PATERNAL STRESS

MATERNAL NUTRITION, PHYSICAL ACTIVITY, TOXIC STRESS

APPETITE & SATIETY
Rapid Infant Growth

EPIGENETICS (Human and Microbiotic)

GENETICS (Human and Microbiotic)

PRE-CONCEPTION
GESTATION
INFANCY
EARLY CHILDHOOD
ADOLESCENCE
ADULTHOOD

PLASTICITY & REVERSIBILITY

The model presented is intended to highlight the workshop objectives, rather than to be fully comprehensive.
All levels of the internal and external environment interact with each other in a dynamic manner.