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Reactivating Nuclear Receptor NR2F2 in Adipose Stem Cells to Reverse Omega-6 Fatty Acid-Induced Developmental Programming

Background

Childhood obesity remains a public health crisis with long-term metabolic disease risks. Obesity is a complex gene-nutrient-lifestyle interaction disease defined by excess white adipose tissue (WAT) and is rooted in development. Early-life exposures shape adipose tissue development and systemic metabolism through developmental programming. Dietary fatty acid (FA) composition, especially a high omega-6 (n6) to omega-3 (n3) ratio, has emerged as a driver of accelerated infant adiposity and obesity risk, but the mechanisms by which n6-FA program adipocyte fate and metabolism remain unclear.

Methods

C57BL/6J dams were randomized to either n6-rich or balanced n6/n3 control diets at the time of mating and underwent normal gestation and parturition. On postnatal day 12 (PND12), indirect calorimetry quantified whole-body metabolism using ^{13}C-nutrient tracing. Adipocyte Stem-like Cells (ASCs) were isolated by flow cytometry for in vitro differentiation, global gene expression, proteomics, and cellular oxidation assays. NR2F2 was transiently re-activated in vitro in ASCs with its ligand (gain-of-function), and NR2F2 was ablated in ASCs ex vivo using homozygous floxed Nr2f2 pups (loss-of-function).

Results

12-day-old pups exposed to developmental n6-FA accumulated hypertrophic, white adipocytes instead of beige adipocytes. Molecularly, Nuclear Receptor 2 group F member 2 (NR2F2), a ligand-activated transcription factor that regulates metabolic genes, was suppressed in n6-FA ASCs. In vitro, NR2F2-low ASCs differentiated into adipocytes with decreased metabolic regulators peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC1α), peroxisome proliferator-activated receptor gamma (PPARγ), PR domain containing 16 (PRDM16), and uncoupling protein 1 (UCP1). Cellularly, resultant adipocytes oxidized lipid and glucose at lower rates and had increased lipogenic enzymes.

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Conclusion

Our findings indicate that excess developmental n6-FA disrupts NR2F2-mediated ASC fate determination, leading to formation of nutrient-storing, lipogenic adipocytes.

Keywords: Developmental programming, adipose metabolism, fatty acids

Biography

My research focuses on understanding how early-life biological signals durably shape lifelong metabolic health. Specifically, my laboratory investigates how developmental lipid signals are received by cells and tissues during the perinatal period, and how these signals establish lasting metabolic programs that influence obesity susceptibility or resilience later in life. I am particularly interested in how lipid-mediated developmental cues regulate adipose tissue growth and function, and whether these signals become stored as forms of “metabolic memory” persisting beyond the developmental exposure. This problem has clinical importance because developmental programming studies increasingly demonstrate that early-life nutritional exposures shape long-term metabolic disease risk.

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