How does a maternal high-fat diet during pregnancy impact the brain development of a fetus?

- Maternal diet and obesity has been a key factor in developing fetal brains, having persisting effects in postnatal development.
- Increased proliferation of neural progenitors in neuroepithelium of the hippocampus and cortex and decreased cell death in the Ammon's horn and fimbria areas of the hippocampus indicate decreased early neuronal differentiation.
- Altered rates of cell proliferation in the hypothalamus and hippocampus of developing Sprague Dawley rats suggests developmental delay.
- Castrin, a marker for recently generated neurons, has been observed at decreased levels in fetal rats, suggesting reduced early neuronal maturation.

Is a high-fat diet sustainable?

- Western world has increased access and abundance to foods high in fats and carbohydrates, resulting in higher rates of obesity.
- Important to reduce obesity preconception and eat a diet with moderate amounts of fats and carbohydrates during pregnancy to avoid the “potentially hazardous neurotoxic environment to the developing nervous system” (Page, Jones and Anday, 2014).
- Hypothesized that energy-dense diets, high in sugars and fats are the lowest costing diets. Studies in Europe have shown that healthier diets (with increased fruits and vegetables) cost more.

How does a high-fat diet influence cognition and juvenile brain development?

- In juvenile (3 weeks old) mice, a high fat diet decreases hippocampal neurogenesis, making the juvenile period vulnerable to the adverse effects high fat diets can have on hippocampal function due to HFD promoting higher levels of pro-inflammatory cytokines.
- Adverse effects of high fat diets on hippocampal function: impaired long-term spatial memory and spatial reversal learning (measured using Morris Water Maze) (Boitard, C, 2014).
- Animals that are able to learn a spatial memory task faster have more brain-derived neurotrophic factor (BDNF) mRNA and protein in the hippocampus.
- BDNF, a mediator of neuronal vitality and function, is key in neuronal events underlying learning and memory, and is reduced in mice with HF diets compared to LF.
- Animals with HF diets had decreased: (1.) synapsin I protein, important for neurotransmitter release; (2) cyclic AMP-response element-binding protein (CREB); CREB is required for various forms of memory and is under regulatory control of BDNF; (3) growth-associated protein 43 (GAP43), important for neurite outgrowth, neurotransmitter release, learning and memory.
- Conclusion HFS diets reduce the levels of BDNF lowering the levels of BDNF controlled factors that regulate, axonal growth and neurotransmitter release resulting in deficiency in learning and memory ability.


