Appetite and satiety demonstrate a remarkable heterogeneity among humans, with a spectrum contributing to both anorexia and hyperphagia.

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Developmental Programming of Appetite/Satiety
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Key insights
Obesity in childhood and in later life may be the result of developmentally programmed hyperphagia. Recent data strongly suggest that maternal and/or fetal under- or overnutrition predisposes the offspring to become hyperphagic. Infants born small for gestational age and those born to obese mothers are at risk.

Current knowledge
Appetite is regulated by a complex circuit of hypothalamic nuclei involved in the generation of appetite versus satiety signals. The arcuate nucleus (ARC) is the predominant appetite regulatory site in the brain and receives signals from different areas of the brain and other tissues. The ARC contains at least two populations of neurons with opposing actions on food intake. Specific alterations in the fetal metabolic/energy environment can alter the equilibrium between orexigenic and anorexigenic neuronal systems, thus affecting appetite and satiety.

Practical implications
Imbalances in maternal nutrition (under- or overnutrition, low-protein or high-fat diets) result in a cascade of genetic, biochemical and cellular effects that can ultimately bias the appetite regulatory networks of the offspring towards hyperphagia. Specific epigenomic studies are necessary in order to pinpoint the genomic sites that act as targets for early nutritional effects.

Maternal nutrition affects appetite in the offspring. Perturbations in maternal nutrition can alter nutrient sensors, neuroendocrine levels and signaling, neurogenesis and neuropeptide levels. These pathways interact and ultimately influence appetite.

A greater understanding of the development of appetite regulatory pathways opens the door for novel interventions for the prevention and treatment of obesity.

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