British Journal of Nutrition (2008), **99**, 1391–1392
doi: 10.1017/S0007114507871704

Letter to the Editor

Perinatal nutrition and obesity

(First published online 21 December 2007)

Stimuli or insults during the perinatal period can have lifetime consequences and this long-term effect is called 'programming'. Early nutrition is an important environmental signal that can induce lifetime effects on metabolism, growth and neurodevelopment and on major disease processes such as hypertension, diabetes, and obesity[1–3]. For instance, exclusive breast-feeding is an early environmental stimulus that is known to influence the development of insulin resistance, obesity, hypertension and type 2 diabetes mellitus in later life[4–6]. In this context, the results of the study published recently in the *British Journal of Nutrition* by Bayol *et al.* [7] are interesting.

Appetite is controlled by appetite-stimulating neuropeptide Y (NPY) and agouti-related peptide (AgRP), and the appetite-inhibitory molecules pro-opiomelanocortin (POMC) and cocaine and amphetamine-regulated transcript (CART) which regulate energy balance[8]. Hypothalamic appetite regulatory centres develop during the perinatal period[1]. Hence, factors that influence brain growth and development will have substantial impact on the development of appetite regulatory centres that, in turn, determine food intake in later life. For instance, postnatal over-nutrition in rats leads to increased early weight gain and fat deposition, hyperphagia, obesity, hyperleptinaemia, hyperglycaemia, hyperinsulinaemia and insulin resistance and the over-fed rats show decreased mean areas of neuronal nuclei and cytoplasm within the paraventricular (PVN), ventromedial (VMN), and arcuate (ARC) nuclei of the hypothalamus and a significant increase in the number of NPY-containing neurons within the ARC and decreased immunostaining for both POMC and α-melanocyte-stimulating hormone[9,10]. Furthermore, neuropeptides NPY, AgRP, POMC and CART showed significant changes in their concentrations in the various regions of the hypothalamic nuclei in sheep in response to intrafetal infusion of glucose between 130 and 140 days of gestation[11]. These results indicate that neuropeptides which regulate appetite centres and their responses to stimuli such as glucose, insulin and other stimuli are 'programmed' in the fetal and perinatal stages of development. This could explain why a maternal junk-food diet in pregnancy and lactation promoted an exacerbated taste for similar food and greater propensity for obesity in rat offspring[7]. Maternal junk-food intake programmed the offspring hypothalamus to crave for junk food.

The brain is rich in PUFA especially arachidonic acid (AA) and DHA which constitute as much as 30 to 50 % of the total fatty acids in the brain, where they are predominantly associated with membrane phospholipids. These PUFA activate syntaxin 3, a plasma membrane protein that has an important role in the growth of neurites[12]. Junk food is known to be energy-dense and rich in saturated and *trans* fatty acids that could interfere with the metabolism of essential fatty acids[13] and so could potentially lead to PUFA deficiency in the mother and offspring during the critical period of brain growth, development and maturation leading to inappropriate synaptic connections of hypothalamic neurons. This may lead to the hypothalamic 'body weight–appetite–satiety set point' set such that it promotes an exacerbated taste for similar food and greater propensity for obesity in rat offspring. If this proposal is true, it implies that provision of PUFA during the critical perinatal period of growth would prevent the development of obesity and metabolic syndrome X.

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British Journal of Nutrition