
IN A DIFFERENT BUT RELATED FIELD DEALING WITH THE EARLY DETERMINATION OF NEUROENDOCRINE FUNCTION NEEDED LATER IN LIFE, NAMELY THE SPECIFICATION OF SEXUALLY DIMORPHIC PATTERNS IN THE MAMMALIAN FOREBRAIN (4), RECENT ADVANCES HAVE BEEN MADE: SPECIFICALLY THE SURGE OF SEX STEROID SECRETION IN EARLY NEONATAL LIFE HAS BEEN LINKED TO NEUROANATOMICAL AND FUNCTIONAL CHANGES IN THE FOREBRAIN (5–7).


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Leptin treatment of neonatal ob-mice on days 4–12 of life reversed the reduction in nerve fibres underlining the specific role of leptin in neuron formation to a level seen in wild-type littermates (9). Most importantly, treatment of leptin-deficient adult mice with leptin for 20 days did not result in the formation of anorexigenic and orexigenic nerve fibres. This confirms that there is a critical period for the formation of leptin-dependent neurons in mice.

Interestingly, in the same issue of Science in which the article by Bouret and co-workers (11) appeared, Pinto and colleagues (12) present data on the role of leptin on synapse formation in the arcuate nucleus of the hypothalamus. In adult ob-mice leptin leads to the formation of new synapses even if the animals had been leptin deficient before. This suggests that leptin still has effects on hypothalamic neuronal plasticity in the adult animal, despite the reduction in anorexigenic and orexigenic nerve fibres.

In summary, the work by Bouret and co-workers (11) suggests a potential mechanism by which changes in leptin concentration during fetal and neonatal life may alter hypothalamic structures that influence food intake and energy metabolism until adult life. This might be the first step in elucidating mechanisms being important to explain the concept of fetal programming of adult disease.

References
1 Barker DJP. In utero programming of chronic disease. Clinical Science 1998 95 115–128.