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# Influence of Intrauterine Malnutrition on Brain Development: Alteration of Myelination

*J.M. Bourre, O. Morand, C. Chanez, O. Dumont and M.A. Flexor*

Laboratoire de neurochimie, Inserm U–134, Hôpital de la Salpétrière and Centre de recherches biologiques néonatales, Inserm U–29, Hôpital de Port-Royal, Paris

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**Abstract.** As compared to other organs, in intrauterine growth retardation brain is less affected; however, forebrain weight and total lipids are reduced. Myelin quantity is reduced by 27, 17, 9 and 6% at 15, 18, 30 and 60 days after birth, respectively. Thus, intrauterine undernutrition followed by normal diet after birth affects myelination, a postnatal event. Prenatal growth retardation moderately but irreversibly impairs brain maturation, whereas the composition of myelin during maturation is close to normal (when density profile, lipid amount and the fatty acid pattern are considered).

In humans, malnutrition in early life exposes to many immediate dangers, and in the long term, early malnutrition seems to be associated with physical stunting and mental retardation. Owing to the difficulties in assessing the long-term physical and functional effects of early nutritional deprivation in man, animal models have generally been used. From experiments, we know that the malnutrition during the brain growth spurt (1–3 weeks of age in the rat, from 3 months before birth to about 18 months of age in humans) tends to produce permanent deficits in brain weight and cell number (1), and that the degree of physical deficit depends on the timing, severity and duration of malnutrition (1, 2).

Intrauterine malnutrition was developed in rats by producing animals with intrauterine growth retardation (IUGR). With this method, brain is not drastically affected in contrast to other organs (5); however, synaptosomes present some biochemical alterations (6) and the outgrowth of IUGR rat cerebellum cultivated *in vitro* was retarded (7). As early postnatal starvation causes lasting brain hypomyelination (8), this work was undertaken to determine the effect on myelination of intrauterine malnutrition followed by normal postnatal nutrition.