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Editorial

A Note on Prenatal Nutrition

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During the fetal period there is a very special demand of nutrients that would participate in the processes of organogenesis, particularly in the conformation of the Central Nervous System (CNS). The encephalon will contain all the cerebral structures that are of a great diversity, morphologically and functionally. Since the prenatal stage the different nutrients: proteins, carbohydrates, lipids, vitamins, minerals, oxygen, and water, have a variety of crucial functions at the cell, tissue and organic levels, for molecular and cell construction of tissues and fetal organs.

Some brain components, like auditory and visual cerebral cortices, the hippocampus, the corpus striatum and their neural circuits, develop rapidly in particular in the human last trimester of gestation. Making them particularly vulnerable to environmental aggressions, among them deficiencies in nutrients´ accessibility.

Altered accessibility or lack of nutriments during gestation, is frequent, sometimes not so evident, and may provoke intrauterine undernutrition with growth restriction, general deficiencies or lack of specific nutrients in the case of malnutrition in-utero. It is obvious that mother´s well nutritional state is critical, as it is the availability of nutrients through a healthy placenta. A defective placenta may cause the known clinical picture: Placental Insufficiency. A faulty placenta, may carry the fetus to a deficient growth, altering neuronal development and brain function (Intrauterine Growth Restriction (IURG). It has been observed in experimental animals and human babies that an altered availability of some nutrients like some amino acids that have the biochemical role to be precursor molecules in the synthesis of some important neurotransmitters, like L-Tryptophan (L-Trp) precursor of the brain serotonin (5HT) biosynthetic path.

Across a long lasting series of experimental work, we were able to postulate that altering L-Trp availability, early in gestation, the formation and functional development of the sensory cortex was significantly modified, showing an altered function, particularly the auditory cortex, whose responses to specific stimuli are significantly disturbed in experimental animals a human baby that suffered IUGR. And very probably the visual and somatosensory cortices are too subjected to anomalous development when the synthetic brain serotonin path is experimentally altered. IUGR causes a malfunction of the main enzymes of this path with the acceleration of the activity of the rate-limiting enzyme, increasing brain serotonin synthesis. During IUGR, we observed that the precursor amino acid, L-Trp, starts an anomalous behavior since before it passes from plasma to the brain, through the brain blood barrier (BBB). Its competition with other plasma aromatic amino acids to pass to the brain through the BBB, change and more L-Trp is transported to the brain so activating the biosynthetic 5-HT path with more serotonin available during early stages of embryogenesis which very alters its important role in brain morphogenesis; and its binding to plasma albumin is also altered in neonates with IUGR, being significantly less, so allowing an increase in the free fraction of the amino acid (L-Trp) in plasma under IUGR or intrauterine malnutrition, secondary to a faulty placental functioning or to mother´s undernutrition. These data may be clinically relevant since the published biochemical and physiological alterations, have been observed in human neonates and nursing babies up to three months of age, the anomalous metabolic and functional disturbances are still present. Likewise, the neurobiochemical and neurophysiological anomalous metabolic features described, neonatally and postnatally, persist in experimental rats up to adulthood. Suggesting that an epigenetic phenomenon might be involved.

The main line of thought that was followed in this long lasting project was the well-known information published about the important role of the brain serotonin system, in brain morphogenesis and development, since very early stages of gestation. In this respect we have been able to describe in the fetal brain of experimental animals, the metabolic behavior of the serotonergic system

and amplify already published knowledge. Underlining and enlarging the data about the relevant role of the serotonergic system in the fetal brain including the existence, at the molecular level, of a 5-HT receptor´s signalization, specific uptake and a releasing system, Na+ and Ca+ dependent in growth cones´ neuronal fractions from the E17rat fetal brain. Systems that do appear very early in brain morphogenesis, both in the experimental rats and it seems that also in humans, due to its crucial role in cerebral cortical formation.

So, we can make a well based conclusion, supported by our data, that allow us to strongly state that the effect of early modification, during fetal life, of specific nutrients availability may provoke deleterious effects in brain cortical conformation and functional integration, secondary to a malfunction of the prenatal brain serotoninergic system.

Our results strongly suggest that this physio-metabolic brain developmental disturbance in brain function, that recalls the so called: "Inborn errors of metabolism", may be reflected later in life in a deficient adaptability of children to their social or school environmental conditions.

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