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Editorial

Hunger May Subtly Disturb the Brain Function

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Through the History of Mankind famine have always been present. Malthus (Thomas R. Malthus 1766-1834) predicted that the food production will be surpassed by the population growth in the world. In the long run, in several aspects, he was wright. But he could not predict one substantial variable: The Des-humanization of a significant part of Humanity. Neither the "human ego-centrism", and the unequal distribution of food around the world. The squandering and the inflated cost of food, inaccessible for the poor people., with the unacceptable result of children and adult´s undernutrition that may damage the body functions, brain and mental development, not always carrying to death but in many cases subtly curtailing social, cultural and productive development and performance and therefore human well-being.

The concepts described here are supported by extensive experimental work, widely published, in experimental animal models of intrauterine growth restriction (IUGR), secondary to undernutrition. The interesting relation between the brain serotonin synthesis and the availability of precursor nutrients, in this case the amino acid L-Tryptophan (L-Trp) and some vitamins like pyridoxine. We pointed our research on the effects of a deficiency in the availability of these precursor nutrients, for the whole metabolic path of brain serotonin synthesis since the prenatal, peri and postnatal life. We evaluated the translation of this approach to human babies born to mothers with the diagnosis of Placental Insufficiency (PI) or Gestational Malnutrition, whose offspring suffered IUGR. The results summarized here come from several clinical trials under collaboration with expert neonatologists, pediatricians, endocrinologists and obstetricians from de National Medical Center of México, with the approval of the local Biomedical Research Ethical Committee., and the Committee for the Use and Production of Experimental Animals. All neuro-biochemical, physiologic and morphologic studies were first done in Wistar laboratory rats whose IUGR offspring resulted from gestational undernourished mothers or from a PI model secondary to ligation of one branch of the uterine artery to the gestating mother-rat. With the experimental information obtained in many trials of gestational undernutrition in these animal models, we translated this experience to IUGR human babies born to mothers with PI or gestationally undernourished. With the use of non- invasive methods, parameters related to the brain serotonin system were measured in IUGR human newborns and nursing babies in micro-blood samples obtained from the umbilical cord first, and after by micro-puncture up the 3rd month of postnatal life. Briefly, the results showed: An increase in the plasma concentration of the free fraction of the precursor amino acid L-Trp (FFT) which is the fraction of the plasma L-Trp that passes to the brain for activation of the serotonin synthetic pathway, secondary to a decrease in its binding -rate to plasma albumin. Indeed in the rat´s IUGR offspring a similar result was previously observed; but besides and very interesting, the whole synthetic-path of brain 5-HT, was accelerated, including the whole L-Trp-5-Hydroxylase (T5H) activity, the limiting step in the biosynthesis of brain serotonin. All these changes were present in the IUGR rat brain up to adulthood, in spite of nutritional recovery. In the same line of thought we observed that both T5H isoforms 1 and 2, are expressed in the IUGR brainstem with increased activity. In the human IUGR infants the FFT was also elevated, as mentioned, and its binding to plasma albumin diminished suggesting that the serotonin synthesis in their brain is also disturbed in the same sense as in the IUGR nursing rats what was confirmed by a non-invasive electro-physiologic method to assess the auditory cortical response which is directly related to intensive 5-HT afferent terminals

innervation. The wave´s N1-P2 segment amplitude of the intensity dependent cortical auditory response to specific stimuli was dramatically diminished in IUGR infants, as compared to normal age matched controls. It was concluded that in IUGR human infants there is also an alteration of the brain serotonin synthetic-path and function, since the prenatal stage that disturbs the normal physiologic role of this amine in the sensory cortex developing functional and morphological processes, failing to develop a normal sensory cortex malfunctioning up to three months of postnatal age in human nursing infants and up to adulthood in experimental animals., where an alteration of the growing thalamo-cortical fibers toward S1, was also observed. The Electro physiologic evoked auditory cortex (A1) abnormal responses mentioned, suggested that very possibly the A1 function and morphology in human infants was also damaged by IUGR secondary to the serotonergic changes caused by IUGR undernutrition. The pathophysiological mechanism involved could have been the alteration in the availability of the precursor nutrient (L-Trp) and other nutrients involved in the normal biosynthetic serotonin-pathway in their brains as we have demonstrated in the IUGR rat brain, consisting in an acceleration of the serotonin synthesis secondary to a significant increase of the T5H activity with a significant change in the kinetics of this important enzyme system. Changes that last up to 3 months of postnatal age suggesting their long lasting effects on the sensory cortex function. Nutritional recovery in these IUGR infants did not lead these changes to normality therefore. It was concluded that IUGR secondary to undernutrition may provoke abnormal brain developmental long lasting changes in these infants that could, in time, lead to an abnormal cognitive maturation secondary to an abnormal sensory brain function limiting their mental, learning capacity and intellectual general output, being a fertile social discriminatory target.

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