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Editorial

Diabetes, Metabolic Syndrome, Early Malnutrition: Different Faces of the Same Process

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After dealing for some years with experimental and Clinical Diabetes Mellitus (DM), Metabolic Syndrome (MS) and Intrauterine Growth Restriction, (all technical details have been properly published and can be found in the corresponding literature) strongly called our attention the similarities of origin of all these three metabolic disturbances, briefly: all of them share the antecedent of early age abnormal metabolic traits, like an early abnormal handling of carbohydrates and lipids, with clinical signs of obesity and cholesterol mishandling. Other common trait is Insulin resistance, these metabolic changes may start in the prenatal period including nutritional stress particularly in the case of IUGR, predisposing to late adult illness. They also share the consequences of these early changes, namely cardiovascular alterations, at older ages. Following this line of thought we observed that they share pathophysiological metabolic abnormalities in adulthood and course also with an abnormal feeding behaviour and a high BMI.

But, they show other very interesting pathophysiological common feature, they also share significant alterations of brain function in an important neurotransmission system the serotonin brain metabolism and function as early as the prenatal period, affecting the important participation of the serotonin system in the corticogenesis processes, particularly in the formation of the sensory cortex, affecting its function in a long lasting way. In the case of DM and IUGR, opportune treatment does not reverse the damage on the brain serotonin neurotransmission system and the individual seem to carry the neurochemical and functional abnormalities for life. Plasma neutral amino acids (AAN) mishandling, change their relation with the free fraction of the main serotonin synthesis precursor, namely Free fraction of L-Tryptophan (FFL-Trp), provoking a less transport of this precursor to the brain, in the case of MS and DM, with a less concentration of the brain neurotransmitter during development and adulthood. In the case of IUGR the opposite occurs with the AAN decreasing in plasma, provoking an increase of the synthesis of brain serotonin, because of an increase of the FFL-TRP passing to the brain and activating the T5H, s long lastingly in these individuals, both experimental animals and human babies. So, for us, our data strongly suggest that all these three processes are different faces of the same developmental metabolic problem.