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What is and what is not-Type 2 diabetes mellitus

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Albeit diagnosed and defined as a "primary sugar disease of the adult"; i.e, also as namely a disease of the carbohydrate metabolism by mostly authors; so called type 2 diabetes mellitus should be better defined as a "No Man´s Land" state of disease in adults, at most diagnosed by a fasting glycaemia equal or higher than 126 mg/dL. And why this reality? Because in a global epidemic, which is badly out of control, among other reasons. there is not much time to loose. So let´s get into some facts! Despite all controversies surrounding the etiology, pathogenesis, and therapeutic roles for hyperglycaemia in type 2 diabetes mellitus, newer anti-hyperglycaemic drugs are still getting onto the market at a high speed, due to the overconfidence in HbA1c as a surrogate outcome for microvascular complications; albeit. All large recent randomised clinical trials and meta-analysis have shown that trying to achieve glycaemic levels close to the normal range did not reduce the most clinically important microvascular or macrovascular hard endpoints as end-stage renal disease, vision loss, stroke, cardiovascular and total mortality, with the added harm of substantial increase in the number of hypoglycaemic episodes, and even death rates. If glucose or HbA1c were good surrogate disease markers why the increased mortality in the ACCORD trial and the recent rosiglitazone saga, among other anti-hyperglycaemic drugs? The above, among other core issues, will be covered in our talk.

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Insulin/adenosine signaling axis in gestational diabetes

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Gestational diabetes mellitus (GDM) is a disease that occurs during pregnancy and is associated with maternal and fetal hyperglycaemia. Women with GDM are treated via diet to control their glycaemia; however, a proportion of these patients does not achieve the recommended values of glycaemia and are subjected to insulin therapy until delivery. Even if a diet-treated GDM pregnancy leads to normal maternal and newborn's glucose levels, fetoplacental vascular dysfunction remains evident. Thus, control of glycaemia via diet does not prevent GDM-associated fetoplacental vascular and metabolic alterations and hence, we presented evidence regarding insulin therapy in the context of its potential consequences for fetoplacental vascular function in GDM. The results suggest that in this insulin therapy, for it to produce normoglycaemia in the mother and newborn may require additional therapeutic measures to restore the normal metabolic condition of the vascular network in GDM. It was also observed that A1, A2A adenosine receptors and insulin receptors A and B has a potential functional link in the cell signaling, which is associated with the activation of these receptors. This possibility could be helpful for the planning of strategies, including adenosine receptor-improved insulin therapy, for the treatment of GDM patients, thereby promoting the well being of the growing fetus, newborn and mother.

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