Conferencias y Simposios

Simposio: el embarazo de Iris

Coordinador: Dr. Jorge Alvariñas

Función de la célula beta del feto, epigenética y otras variables

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El páncreas fetal comienza su desarrollo durante el primer trimestre del embarazo. La historia clínica de Iris nos muestra que es una paciente que, si bien bajó de peso en forma preconcepcional, en el primer trimestre de embarazo presenta obesidad y en el segundo trimestre de embarazo desarrolla diabetes gestacional (DG). En el primer trimestre, su obesidad conducirá a un entorno prooxidante y proinflamatorio intrauterino que podrá inducir fallas en el proceso de diferenciación de la célula beta pancreática en la etapa embrionaria y afectar la regulación fina de la interrelación de las células del islote pancreático durante su desarrollo. Éstas son posibles causas del elevado riesgo de la disfunción metabólica que tendrá la progenie de Iris a lo largo de su vida.

Cuando en su segundo trimestre de embarazo la paciente Iris desarrolla DG, a los efectos adversos del entorno prooxidante y proinflamatorio intrauterino que se exacerban a medida que progresa el embarazo, se suma el efecto adverso de la hiperglucemia. Este exceso de glucosa circulante será transportada a través de la placenta, estimulando en forma temprana a un páncreas que si bien presenta inmadurez, es funcional. Esto generará un exceso de insulina fetal, promotora del sobrecrecimiento y adiposidad fetal, y una desregulación de la interrelación de órganos con función metabólica. Estos cambios conducirán a la programación del metabolismo fetal, que será evidente a lo largo de la vida de la descendencia de Iris.

Los mecanismos implicados en el impacto del entorno prooxidante y proinflamatorio durante el desarrollo de la célula beta y la conformación del islote son múltiples, siendo relevantes los cambios en la regulación epigenética y en la regulación de los múltiples factores de transcripción que conducen al desarrollo del páncreas endocrino. Se discutirá el impacto de agentes nutrigenómicos capaces de regular el desarrollo del páncreas endocrino y prevenir la programación fetal.

Palabras clave: entorno prooxidante; entorno proinflamatorio intrauterino; epigenética.

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Symposium: Iris's pregnancy

Coordinator: Dr. Jorge Alvariñas

Function of the fetal beta cell, epigenetic and other variables

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The fetal pancreas develops during the first trimester of pregnancy. The clinical history of the patient Iris shows that she was obese at the preconceptional stage, losses weight, but was still obese during the first trimester of pregnancy. Maternal obesity leads to a prooxidant and proinflammatory intrauterine environment. This may affect both the embryo beta cell differentiation process and the fine regulation needed for a proper interrelationship of the developing pancreatic islets. These are possible causes of the increased risks of metabolic dysfunction that the offspring of Iris will present during their lifetime.

The patient Iris developed gestational diabetes in the second trimester of pregnancy. Thus, in addition to the adverse effects induced by the prooxidant and proinflammatory intrauterine environment, which are increased as long as the pregnancy develops, the adverse effect of hyperglycemia arises. An excess of maternal circulating glucose will be transported to the fetal circulation through the placenta, leading to an overstimulation of the fetal pancreas, which is immature but secrete insulin. This will generate an excess of fetal insulin, and promote fetal growth, fetal adiposity and a dysregulation of the metabolic organs, which are still immature, leading to alterations that will impact the offspring in the form of metabolic programming that will be evident later in life.

The mechanisms involved in the impact of the prooxidant and proinflammatory intrauterine environment during beta cell development and pancreatic islet formation and intracellular regulations are multiple. Among them, it is relevant the epigenetic regulation and the changes in functionality of multiple transcription factors involved in the development and maturation of the endocrine pancreas. The role of nutrigenomic agents leading to the regulation of development of the endocrine pancreas to prevent fetal programming will be discussed.

Key words: proinflammatory intrauterine environment; prooxidant intrauterine environment; epigenetic.

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