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Short Communication
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Pregnancy and Carbohydrate Metabolism

Maria Trapali*

Department of Biomedical Sciences, University of West Attica, Greece

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***Corresponding author:** Maria Trapali, Chemist, Ph.D Biochemistry, Department of Biomedical Sciences, University of West Attica, Greece

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Short Communication

Adjustments of nutrient metabolism in the mother are evident as early as the first weeks after conception and continue to develop throughout pregnancy. Many of these adjustments are made to ensure nutrient availability ingredients for the fetus during periods of increased nutrient requirements. Fetal nutrient requirements vary according to genetically determined fetal tissue growth and development sequences. The amount and types of essential nutrients depend on the type and amount of nutrients required for the functioning of specific metabolic pathways and the development of embryonic structures. Nutrients should be available at the time they express those genes that control fetal growth and development [1]. A normal pregnancy is characterized by increased insulin resistance, mild fasting hypoglycaemia, and prolonged hyperglycaemia after the meal [2]. The supply of glucose to the placenta dictates growth rate fetus. Glucose is the preferred substrate for the fetus [1,3], which depletes maternal reserves and further reduces the mother's ability to synthesize glucose, using amino acids [4].

Maternal insulin resistance usually develops early in pregnancy (a phenomenon that leads to fat production from the body), limiting the consumption of glucose by the mother and allowing the movement of more part of the fetus [5]. Changes that increase maternal insulin resistance (sometimes referred as the diabetic effect of pregnancy) give normal pregnant women during the 3rd trimester of pregnancy a mild carbohydrate intolerance [1,3]. Placental lactogen, estrogen and progesterone play a role in insulin resistance [2]. Insulin does not have the ability to cross the placenta to reach the fetus, so in early pregnancy the pancreas gradually reduces its production. As the pregnancy progresses, the placenta increases in size and produces increasing amounts of hormones (human chorionic somatropin, estrogen and progesterone). At the same time, the production of cortisol is increasing from the adrenal glands. These four hormones reduce capacity of mother to use insulin. This fact ensures abundant glucose for the needs of the placental unit.

So, the mother has gradually increasing need for insulin, until the end of pregnancy. The normal β cells of the islets of Langerhans in the pancreas can respond to this need for insulin [4], showing a 10-20% increase in their mass, resulting in both cell hypertrophy and hyperplasia. However, it is reflected by higher synthesis and secretion of insulin per cell, although the mechanisms are not well known [5]. But if the pancreas is unable to cope with these increased demands, gestational diabetes develops [6]. Insulin resistance in the mother's liver increases production glycogen (gluconeogenesis) by 30%. At the same time, insulin resistance in the muscles, the largest store of glucose in the body, restricts glucose consumption by the mother. Insulin resistance in adipose tissue dramatically increases lipolysis, providing fatty acids as an alternative fuel for maternal consumption and glycerol as a substrate for gluconeogenesis in liver. Glycerol is the preferred substrate for gluconeogenesis during pregnancy, thus preserving the amino acids for fetal development. In short, pregnancy reprograms maternal metabolism (with reduced conversion of glucose to glycogen and fat, reduced utilization of glucose from the body and increased glucose production by liver) to maximize glucose shift in the fetus). Fetal consumption is quite extensive with the result that the mother's fasting glucose is reduced by 10% in advanced pregnancy, despite insulin resistance [5]. However, postprandial blood glucose concentrations increase and remain high for a longer period of time than before pregnancy [1].

After fasting more than 12 hours, the maternal metabolism rapidly turns to the utilization of glycogenic or glycogenetic amino acids (amino acids such as alanine and glutamic acid that can be converted to glucose), fat oxidation and increased ketone production (metabolic byproducts of fatty acid catabolism for energy production, β-hydroxybutyric acid, acetone are the main ketones or ketone bodies). Decreased glucose levels and plasma insulin and elevated triglyceride-free levels fatty acids and ketones are observed hours earlier than in non-pregnant women fasting. The rapid transition to fasting metabolism allows pregnant women to use stored fat as a prime source energy, keeping glucose and amino acids available for use by the fetus [1]. Although these metabolic adjustments contribute to ensuring a continuous supply of glucose to the fetus, fasting increases over time the dependence of the fetus on ketone bodies, as a source of energy. Prolonged use of fetal ketone bodies, as seen in women with inadequate diabetes management or in those who lose weight during pregnancy (either for a certain period of time or for the entire duration), is associated with a disorder of the physical and mental development of the newborn [1,7].

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