The effect of maternal Inflammation on foetal programming of metabolic disease - DTU Orbit (25/12/2018)

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Maternal obesity during pregnancy increases the child’s risk of developing obesity and obesity-related diseases later in life. Key components in foetal programming of metabolic risk remain to be identified; however, chronic low-grade inflammation associated with obesity might be responsible for metabolic imprinting in the offspring. We have therefore surveyed the literature to evaluate the role of maternal obesity-induced inflammation in foetal programming of obesity and related diseases. The literature on this topic is limited, so this review also includes animal models where maternal inflammation is mimicked by single injections with lipopolysaccharide (LPS). An LPS challenge results in an immunological response that resembles the obesity-induced immune profile, although LPS injections provoke a stronger response than the subclinical obesity-associated response. Maternal LPS or cytokine exposures result in increased adiposity and impaired metabolic homeostasis in the offspring, similar to the phenotype observed after exposure to maternal obesity. The cytokine levels might be specifically important for the metabolic imprinting, as cytokines are both transferable from maternal to foetal circulation and have the capability to modulate placental nutrient transfer. However, the immune response associated with obesity is moderate and therefore potentially weakened by the pregnancy-driven immune modulation, dominated by anti-inflammatory Treg and Th2 cells. We know from other low-grade inflammatory diseases, such as rheumatoid arthritis, that pregnancy can improve disease state. If pregnancy is also capable of suppressing the obesity-associated inflammation, the immunological markers might be less likely to affect metabolic programming in the developing foetus than otherwise implied.

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