

Inflammation may link obesity and adverse pregnancy outcomes

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A number of different immunological mechanisms ensure the successful establishment and maintenance of pregnancy. Imbalance in these mechanisms is associated with adverse pregnancy outcomes. In a review published in *Advances in Neuroimmune Biology*, researchers from the Institute of Life Science, College of Medicine at Swansea University in the UK examine the impact of maternal obesity on the inflammatory responses in tissues of both the mother and the child.

"While great progress has been made in elucidating the immunological mechanisms that ensure [reproductive success](#), we now need to understand the impact of a very modern epidemic on immune response at the materno-fetal interface, as well on the mother and the child," said lead investigator Catherine A. Thornton, PhD. "Inflammation may have a key role in many of the detrimental effects of obesity in non-pregnant individuals, and emerging data suggest that inflammation also links obesity and adverse [pregnancy outcomes](#)."

Evidence of altered inflammatory status with obesity in the circulation of both the mother and child in pregnancy is emerging. For example, obese pregnant women have elevated levels of interleukin-6 (IL-6). IL-6 is also increased in the cord plasma of offspring of [obese mothers](#), and is associated with increased fetal adiposity and, in a [rat model](#), to hypertension and increased hypothalamic-pituitary-adrenal axis activity in adulthood. Altered inflammatory status of the placenta in association with maternal obesity may have a critical role in the short term programming of health and disease in the offspring, the researchers

commented. Maternal obesity is associated with an inflammatory response by the placenta including elevated pro-inflammatory cytokine gene expression.

The negative impact of maternal obesity on the [immune function](#) of mother and child includes an increased risk for preeclampsia, likely mediated via inflammation and [triglycerides](#). Increased maternal [body mass index](#) is associated with an increased risk of neonatal early onset group B streptococcal disease, and an increased risk of respiratory tract infections. The [inflammatory response](#) and immune function of the newborn might relate to later health outcomes. Hyper-responsiveness of inflammatory function at birth is linked to the development of allergic disease in infancy.

Maternal metabolic status during pregnancy and weaning is particularly relevant to healthy development of hypothalamic neurones that regulate weight and feeding in offspring, the researchers report. One study demonstrated that a high-fat diet during pregnancy can induce the expression of hypothalamic peptides involved in the regulation of food intake and body composition in weanling rats. More recently, female offspring of fathers fed a chronic high fat diet had impaired glucose tolerance and insulin secretion. "These findings lead to the suggestion that such programmed expression has a role to play in adult physiology, including increased food intake, preference for a fat-rich diet, weight gain, and metabolic dysfunction," says Dr. Thornton.

"Diseases once found only in adults are increasing in the paediatric population. The focus has been on diseases with a clear metabolic component and it remains relatively unknown what risk [maternal obesity](#) during pregnancy imposes for the development of autoimmune diseases, allergy and asthma, and neurodevelopment and cognitive behavior," stated Dr. Thornton. "Animal models indicate that the provision of a normal diet to the offspring once weaned does not overcome the effects

of maternal overnutrition, so simple dietary changes may prove ineffective. Targeted maternal immunomodulation might be needed to curtail this potential pandemic."

More information: The article is "Inflammation, Obesity, and Neuromodulation in Pregnancy and Fetal Development," by C.A. Thornton, R.H. Jones, A. Doekhie, A.H. Bryant, A.L. Beynon, and J.S. Davies. *Advances in Neuroimmune Biology* 1 (2011) 193-203. [DOI 10.3233/NIB-2011-015](https://doi.org/10.3233/NIB-2011-015)

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