# In This Issue

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A brief summary of the articles appearing in this issue of *Biological Psychiatry*.

### Special Issue: Prenatal Programming of Neuropsychiatric Disorders Across the Lifespan

Stress promotes inflammation, and maternal stress during pregnancy has been associated with greater risk for poor neuropsychiatric outcomes in offspring. However, the role of inflammation as a mediator between maternal psychosocial stress and offspring neuropsychiatric outcomes has not been extensively studied in humans. In this review, **Hantsoo *et al.*** (pages 97–106) summarize the impacts of specific types of stress on maternal immune function and propose inflammation as a mediator in the relationship between psychosocial stress and offspring neuropsychiatric outcomes.

Inflammation during pregnancy has been linked to risk for neuropsychiatric disorders in offspring. Here, **Gumusoglu and Stevens** (pages 107–121) review the strengths and limitations of the preclinical models that have been developed to study the psychiatric risk contributed by maternal immune activation. Animal models have shown that maternal immune activation is associated with effects on a diverse range of offspring outcomes, including development of anxiety- and depression-like behaviors, changes in sensorimotor and social behaviors, and alterations to microglia, brain growth, neural cell populations, and synaptic structure and function. They emphasize that the interaction of maternal physiology and offspring brain developmental processes is a critical factor for understanding translatable mechanisms of neuropsychiatric risk.

Maternal nutrition is critically important for fetal development. This review by **DeCapo *et al.*** (pages 122–134) presents evidence from animal models examining how maternal nutrition influences offspring behaviors relevant to neuropsychiatric disorders, with a specific focus on the translational aspects of these models. It concludes with a discussion of future directions, including the need for additional studies examining how the sex of offspring affects the contribution of maternal nutrition to offspring risk of neuropsychiatric disorders.

Maternal diet and stress during pregnancy are each known to influence fetal development and to potentially affect long-term child health. However, diet and stress also influence one another, and their combined effects in pregnancy may worsen outcomes for offspring health and development. Here, **Lindsay *et al.*** (pages 135–149) review evidence from both animal and human studies that have investigated the interaction between prenatal diet and stress and the resulting consequences for offspring brain development. A broad pattern of results suggests that higher fat diets and higher intake of targeted nutrients may mitigate the effects of prenatal stress on offspring brain development.

The human body is host to trillions of microbes, collectively termed the microbiota, and emerging evidence suggests that they are intimately connected to the brain. This interconnected pathway, the gut-brain axis, begins development shortly after birth and is responsible for many physiological processes. This review by **Codagnone *et al.*** (pages 150–163) outlines the role of microbiota in the prenatal and postnatal development of the brain, with implications for health and disease.

There is a growing appreciation for the importance of the paternal preconception environment on offspring neurodevelopmental disease risk. Animal studies suggest that the germline transmission of information about the paternal experiences likely contributes to these changes in disease risk or resilience. This review by **Morgan *et al.*** (pages 164–171) examines the molecular components mediating these intergenerational effects, focusing on the candidate factors acting as 1) a vector to carry any signal from the paternal compartment to the maternal reproductive tract and future embryo; 2) a molecular signal, encoded by a paternal experience, to carry this "memory" and enact downstream responses; and 3) a target cell or tissue to receive the signal and convert it into an effect on embryonic development. They also explore the potential role of paternal extracellular vesicles.

Cortisol plays a key role in the body's response to stress and may be important for intergenerational transmission of stress and risk for psychiatric disorders. In this longitudinal study, **Graham *et al.*** (pages 172–181) report that higher maternal cortisol during pregnancy was associated with greater neonatal amygdala connectivity in girls. Further, elevated maternal cortisol was associated with higher internalizing symptoms at 24 months, but only in girls, an effect that was mediated by stronger neonatal amygdala connectivity. These data suggest that maternal cortisol during pregnancy may impact offspring brain and behavior in a sex-specific manner.

 

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