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GRK 2576 Guest Lecture

Title: Epigenetic programming by adverse (nutrition in) pregnancy

Speaker: Prof. Dr. Torsten Plösch, PhD

University of Oldenburg (D) and University Medical Center Groningen (NL)

Date: 30. November 2021
Time: 14:00 h CET
Location: virtual - Cisco Webex

(https://hhu.webex.com/hhu-en/j.php?MTID=mbc765847f4ea7b369f9ab6330b032aff)
Meeting number (access code): 2733 260 9024
Meeting password: n6TSJJPs3D3

Biosketch

Prof. Dr. Torsten Plösch spent most of his scientific career at the University Medical Center Groningen, the Netherlands. There, he is Adjunct Professor for Experimental Perinatology from 2019 onwards. From 2020 on he shares his work between the UMCG and adjacent Oldenburg university in Germany, where he is the head of the research lab Pediatrics. He has authored a total of 98 peer-reviewed publications so far.

Prof. Dr. Plösch studies how the fetal and neonatal environment determines the health of the offspring at adult age (nutritional or developmental programming; BARKER hypothesis, DOHaD hypothesis, Developmental Origins of Health and Disease). This includes studies on the acute effects of early nutrition and environmental stress, but also on adult metabolic regulation. His work includes mouse models but also studies on human cohorts, where he explores new, easily available sources of biomaterials for epigenetic research.

Selected publikation

Klerk DH, Plösch T, Verkaik-Schakel RN, Hulscher JBF, Kooi EMW, Bos AF. DNA Methylation of *TLR4, VEGFA*, and *DEFA5* Is Associated With Necrotizing Enterocolitis in Preterm Infants. **Front Pediatr.** 2021 Mar 4;9:630817. doi: 10.3389/fped.2021.630817. PMID: 33748044; PMCID: PMC7969816.

Dijkstra DJ, Verkaik-Schakel RN, Eskandar S, Limonciel A, Stojanovska V, Scherjon SA, Plösch T. Mid-gestation low-dose LPS administration results in female-specific excessive weight gain upon a western style diet in mouse offspring. **Sci Rep.** 2020 Nov 12;10(1):19618. doi: 10.1038/s41598-020-76501-8. PMID: 33184349; PMCID: PMC7665071.

Stojanovska V, Sharma N, Dijkstra DJ, Scherjon SA, Jäger A, Schorle H, Plösch T. Placental insufficiency contributes to fatty acid metabolism alterations in aged female mouse offspring. **Am J Physiol Regul Integr Comp Physiol.** 2018 Dec 1;315(6):R1107-R1114. doi: 10.1152/ajpregu.00420.2017. Epub 2018 Sep 12. PMID: 30207754.

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Talk teaser

Guest lecture with Prof. Dr. Torsten Plösch

Epigenetic programming by adverse (nutrition in) pregnancy

The early environment has a strong influence on adult health. The key idea is that nutrients, metabolites and other signals induce epigenetic changes in the embryo, fetus or newborn organism which persist into adulthood. Often, these epigenetic changes act on genes with important regulatory functions for metabolism, for example nuclear receptors. These changes can present beneficial adaptations to the environment but also lead to mismatches, which hence influence the susceptibility to develop chronic disease. Therefore, in my research the offspring is often confronted with a second challenge, for example a high-fat diet. During the last couple of years, my research has focused on the molecular and epigenetic regulation of metabolic processes in rodent models and humans. DNA methylation is currently the most important epigenetic feature studied.

Two aspects are key to my research:

- First, the acute influence of external factors (diet, stress, microbiota) on metabolic regulation, namely mediated by nuclear receptor signaling. This includes the questions how external signals interact with those receptors, how the receptors influence physiology on a cellular and organismal level, and how we can utilize this for better diagnosis or treatment of diseases like the metabolic syndrome.
- Second, I am interested in how the long-term regulation of this interactions is achieved by means of epigenetic modifications. As this is the key focus of my research, I use several animal models and human cohort studies. In these models, first the embryo/fetus is confronted with an adverse environment. The read-out for these models is the change in physiology of the offspring (often after a second hit) and DNA methylation differences in different organs.